Environmental Chemistry for a Sustainable World 20

Pallavi Saxena
Anju Srivastava *Editors*

Air Pollution and Environmental Health

Springer

Environmental Chemistry for a Sustainable World

Volume 20

More information about this series at http://www.springer.com/series/11480

Pallavi Saxena • Anju Srivastava
Editors

Air Pollution and Environmental Health

Springer

Editors

Pallavi Saxena
Department of Environmental
Sciences, Hindu College
University of Delhi
Delhi, India

Anju Srivastava
Department of Chemistry, Hindu College
University of Delhi
Delhi, India

ISSN 2213-7114 ISSN 2213-7122 (electronic)
Environmental Chemistry for a Sustainable World
ISBN 978-981-15-3480-5 ISBN 978-981-15-3481-2 (eBook)
https://doi.org/10.1007/978-981-15-3481-2

© Springer Nature Singapore Pte Ltd. 2020
This work is subject to copyright. All rights are reserved by the Publisher, whether the whole or part of the material is concerned, specifically the rights of translation, reprinting, reuse of illustrations, recitation, broadcasting, reproduction on microfilms or in any other physical way, and transmission or information storage and retrieval, electronic adaptation, computer software, or by similar or dissimilar methodology now known or hereafter developed.
The use of general descriptive names, registered names, trademarks, service marks, etc. in this publication does not imply, even in the absence of a specific statement, that such names are exempt from the relevant protective laws and regulations and therefore free for general use.
The publisher, the authors, and the editors are safe to assume that the advice and information in this book are believed to be true and accurate at the date of publication. Neither the publisher nor the authors or the editors give a warranty, expressed or implied, with respect to the material contained herein or for any errors or omissions that may have been made. The publisher remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

This Springer imprint is published by the registered company Springer Nature Singapore Pte Ltd.
The registered company address is: 152 Beach Road, #21-01/04 Gateway East, Singapore 189721, Singapore

Contents

About the Editors

Pallavi Saxena is an Assistant Professor of Environmental Science at Hindu College, University of Delhi, India. She was repeatedly selected for the DST Fast Track Young Scientist Award (2014–2017) at the School of Environmental Sciences, Jawaharlal Nehru University, New Delhi, India, together with numerous other awards. She holds a PhD in Environmental Science from the University of Delhi and completed her Post Doc at the Space and Atmospheric Sciences Division, Physical Research Laboratory (PRL), Ahmedabad, India. She has published 21 research papers in prominent journals.

Anju Srivastava has been the Principal of Hindu College, University of Delhi since December 2018, after having served as the officiating Principal since 2014. She has also been an Associate Professor of Organic Chemistry at the Department of Chemistry, Hindu College, for over 20 years. Holding a Master's degree and PhD from IIT Delhi, she was selected as a Fellow of the ILLL (Institute of Lifelong Learning)—an organization at the University of Delhi engaged in the development of e-content for undergraduates of the university. She was presented with the "Distinguished Teacher" Award by Former President of India, Dr. A. P. J. Abdul Kalam, for her contributions to teaching and mentoring. She is an active member of the Royal Society of Chemistry, London and the Green Chemistry Task Force set up by the Department of Science and Technology, Govt. of India. She has also published several articles on Green Chemistry and Environmental Sciences in prominent journals.

Chapter 1
Introduction

Pallavi Saxena and Anju Srivastava

Abstract Air pollution is regarded as a global burden disease in the recent years, even after the implementation of various significant control policies and measures. Air pollution is also one of the important elements of health risk assessment studies. Overpopulation, drastic increase in urban settlements, and set up of various industries lead to the problem of increment of primary and secondary air pollutants which are the major cause for deterioration of plant and human health. These hazardous agents emitted through anthropogenic and biogenic sources contribute to high concentrations in the atmosphere. Comprehensive studies on various air pollutants, their impacts on plant as well as human health and possible control measures for the same are very less reported. Such studies can help the policy makers to suggest some cost-effective measures for the control of these air pollutants. This chapter focuses on the nature, chemistry, and sources of air pollutants and their impact on plant as well as human health. It also summarizes the highlights of different chapters signifying various aspects of air pollutants in consideration with plant as well as human health.

Keywords Air pollutants · Plant health · Human health · Hazardous · Environment

1.1 Introduction

On worldwide level, air pollution is one of the major environmental issues due to its harmful impact on flora and fauna and overall environmental health (Kampa and Castanas 2008). Higher concentrations of both primary as well as secondary

---

P. Saxena (✉)
Department of Environmental Sciences, Hindu College, University of Delhi, Delhi, India
e-mail: pallavienvironment@gmail.com; pallavisaxena@hinducollege.ac.in

A. Srivastava
Department of Chemistry, Hindu College, University of Delhi, Delhi, India

P. Saxena, A. Srivastava (eds.), *Air Pollution and Environmental Health*,
Environmental Chemistry for a Sustainable World 20,
https://doi.org/10.1007/978-981-15-3481-2_1

pollutants have been reported in metropolitan areas (Jim and Chen 2008; Nowak et al. 2006, 2014). The air quality is majorly deteriorated by man-made activities (Kampa and Castanas 2008; Speak et al. 2012). As per the recent findings by World Health Organization (WHO), approximately 4 million death count was recorded annually and respiratory disorders were the most dominant among all the diseases due to bad air quality, particularly in developing countries (WHO 2015, 2016). Various atmospheric processes, environmental health (flora and fauna), crop yield, material health, and weather phenomena are strongly affected by various primary and secondary air pollutants emitted from different sectoral sources like transport, industrial, commercial, residential, domestic, and vegetation zones (Saxena and Ghosh 2011). Atmospheric processes like climate change, secondary organic aerosol formation, cloud formation, and other significant related processes are generally affected by primary and secondary air pollutants. Air quality is degraded due to gaseous and particulate air pollutants like sulfur dioxide (SO_2), oxides of nitrogen (NO_x), volatile organic compounds (VOCs), carbon monoxide (CO), and particulate matter (PM) and ozone (O_3) (Sonwani and Saxena 2016). Among all the sources of air pollution, transport sector is the main reason for deteriorating the air quality. It has been reported that in developing countries most of the air pollution (approximately 70–80%) is caused by vehicular emissions, particularly from larger number of older vehicles with low vehicle maintenance, low fuel quality, and improper road infrastructure (Wang et al. 2010; Bigazzi and Figliozzi 2014). Since last three decades, air quality of developing countries such as India has been drastically declining due to rapid urbanization and industrialization. In fact, these factors are majorly responsible for the increment of air pollutants in many continental areas like Asia, Africa, and Latin America (Ashmore 2005; Saxena and Sonwani 2019a, b). In 1960, less than 22% of developing world's population was urban but by 1990 it increased to 34% (Grant 2012), and by 2020, global urban population is projected to increase by 50% (World Bank 2009; UN Report 2017).

Various types of air pollutants are emitted from single point (e.g., emission from factories or chimneys, tail-pipe emissions from vehicles) or multiple point sources (ozone emissions from vehicles, industries, domestic sectors, or from biomass burning). Dense vegetative zones act as "sinks" of various air pollutants and relatively higher concentrations of gaseous and particulate air pollutants are found in these areas than urban zones (Sonwani and Maurya 2018). The dynamics, emission, and transboundary movement of air pollutants are the key factors for control of these pollutants at local, national, and international level. Many of the atmospheric and biotic processes are affected by transboundary movement of air masses from adjoining or nearby regions and are one of the main reasons for climate change and health risk assessment studies (Saxena and Sonwani 2019a, b). The most important and well-known example of the effect of air pollutant emissions and transport on the constituents of global atmosphere is "Antarctic Ozone Hole." It was reported by various scientists that large amount of loss in the concentrations of stratospheric ozone over the southern pole in each spring was observed due to the formation of secondary products by chlorofluorocarbons (Molina and Rowland 1974; Crutzen and Arnold 1986; Solomon et al. 1994). Another famous example of the impact of air pollutants on atmosphere is The Arctic haze (the reddish brown springtime haze present in the polar region) which was caused by emission of gaseous and particulate air pollutants from industries situated in European region and also the transport of various air pollutants from other regions too which would further affect the air quality of rural areas (Barrie 1986).

The impacts of atmospheric pollutants on living beings like flora, fauna, and human are deleterious. Primary and secondary air pollutants, like sulfur dioxide and ozone, respectively, have a major impact on physiology and morphology of plant species and lead to huge agricultural yield loss (Heck et al. 1988; Saxena et al. 2020). With respect to impact of air pollutants on human health, a huge risk of heart-related diseases, pulmonary disorders, and diabetes in adults and children are also been reported (Sadanaga et al. 2003). In particular, tropospheric ozone (O_3) is considered as the most hazardous air pollutant that can affect the crop yield and result in decline in biochemical and physiological processes of plant species. Tropospheric ozone is also called phytotoxic agent (Saxena et al. 2019). Such impacts can result in producing imbalance in carbon dioxide uptake process and lead to significant change in the physiology of plant species (Saxena et al. 2017). Another significant phenomenon is acid rain which also produces negative change in enzymatic and other biochemical processes of various crop species. During the process of acid rain, two main acid products are formed, such as sulfuric acid and nitric acid, which are the main cause for producing membrane permeability loss, lack of stomatal conductance, and decrease in enzymatic activities (Pant and Harrison 2013). The main cause of acid rain is high industrial emissions from nearby regions, either local or regional, and is responsible for lowering down the pH of rain up to 4–5.8. Acid rain is highly responsible for causing impact on living and nonliving materials. Due to acid rain, a number of fertile lands are also converted into wastelands due to large deposition of acids that can affect the physicochemical properties of soil (Liu and Diamond 2005; Davis 2008). Another important atmospheric phenomenon that is causing harmful impact on environment is climate change. Climate change is caused by elevated increase in the number of selected air pollutants that are popularly called greenhouse gases (Karanasiou et al. 2014). These greenhouse gases, mainly CO_2, CH_4 N_2O, and O_3, create negative alteration in Earth's radiation budget due to absorption or emittance of longwave radiation that is responsible for increasing the temperature of earth. Among all the greenhouse gases, few of them are responsible for either absorbing or reflecting the incoming shortwave radiation that results in either heating or cooling of earth's surface, respectively (IPCC 2013; Saxena and Sonwani 2019a, b).

The famous metropols located in various continents of the world like Asia, Africa, and Latin America have reported a number of air pollution-related problems (Ashmore 2005). In a country like India, the air quality has worsen day by day as per the past two decades. The most polluted city in India is Delhi, which is also getting affected by severe air problem due to emission of different atmospheric pollutants even after the implementation of various control policies and measures (Saxena et al. 2012; Sonwani and Kulshrestha 2019). Delhi is on rank 2 in terms of bad air quality among all the other air polluted cities of the world (WHO 2014–15). As per the reports, approximately 1.7 million people are killed every year due to bad air quality.

India is also the world's highest death rate count country due to severe respiratory disorders and asthmatic diseases.

Bad outdoor air quality has considerable impact on the human health. Contact with the air pollution occurs primarily by inhalation and ingestion. Contamination of ambient air and subsequent fallout of pollutants further contribute to contamination of water and food, posing additional hazard toward ingestion of pollutants (Saxena and Naik 2018). Air pollution can cause some minor and acute risks such as shortness of breath, nausea, dizziness and dermatological disorders and asthma, bronchitis and cancer respectively. This type of pollution is also responsible for frequent abortions, birth defects, and deformities in infants (Ritz et al. 2002). The immunity of the individuals also gets affected due to exposure to various air pollutants and results in chronic diseases (Calderón-Garcidueñas et al. 2008). In case of air pollution-related diseases, both short-term and long-term exposure due to air pollution is harmful to human health which resulted in various diseases and even mortality in many of the cases.

Some of the air pollution episodes such as photochemical smog over Meuse valley, Belgium (Nemery et al. 2017), London smog (Bell et al. 2004) and leakage of hazardous methyl isocyanate (MIC) during the Bhopal gas tragedy (Sriramachari 2004) have already been reported at well-known level all over the world. A number of air pollution-related medical cases has been registered daily in the past two decades particularly in India and is directly proportional to the amount of pollutants emitted in the atmosphere (Brunekreef and Holgate 2002; Fischer et al. 2004; Liu et al. 2013). The degree of toxicity of a particular air pollutant is governed by the key factors: (1) composition (quality) of the air pollution, (2) concentration (quantity) of pollutants in the air, and (3) duration of exposure to the air pollutants.

1.2 Summary of Chapters

On the basis of discussion on a number of issues, it has been noticed that the problem of air pollution especially in Asian cities is primarily because of increasing urbanization and industrialization. This book, "Perspectives of Air Pollution Toward Environmental Health" covers the important topics related to air pollution sources, their impacts and control strategies. Chapter 2 summarizes a brief overview of the state-of-the-art techniques to measure air pollution. This chapter also explains about various types of air pollutants, their impacts and emission sources. Several studies have been carried out for air pollution measurement using different measurement methods worldwide. It also describes specific techniques employed for different types of air pollutants and how advanced and right measurement technique will help the policy makers to improve the air quality for the protection of this sensitive environment without further waiting for legislative laws. Chapter 3 deals with the philosophical and practical discussion and its implications vis-à-vis mathematical air quality modelling. An evaluation of pertinent mathematical modelling techniques viz. Box, Gaussian, Eulerian, Lagrangian, and Particle modelling approaches are provided. Chapter 4 highlights the effect of different air pollutants on plant health and activation of antioxidant system as a defense mechanism. It also explains the negative role of various air pollutants on morphology and physiology of different plant species. Chapter 5 describes how C3 and C4 crop plant responses to elevated CO_2 and higher temperature. These responses are experimentally verified and tested in vitro as well as in vivo with the help of various physiological and biochemical parameters, which ultimately affected the crop yield and product quality. Finally, it also discusses about mitigation and adaptation strategies to improve agricultural crop production which minimizes the production risk. Chapter 6 discusses the effect of various air pollutants on the stress physiological parameters. These studies are crucial because one of the major responses to plant pollutants is the inhibition of photosynthesis. This inhibition of photosynthesis not only alters the growth pattern and longevity but also changes plant phenology. Chapter 7 aims at identifying different sources of air pollution, types of pollutants and mainly their impact on overall human health and functioning of different organs and systems in human body independently and in combined manner. It also describes that controlled exposure studies of toxic air pollutants solely cannot explain the effects of air pollutants on human health along with that animal toxicology, occupational and epidemiology studies together can conclude the effect of a single pollutant and complex pollutants on human health. Chapter 8 reviews about different particulate matter (PM) exposure-based epidemiological studies with more focus on high ambient Total Suspended Particulate (TSP) levels. It has also been found that overall absolute risk for mortality due to PM exposure is higher for cardiovascular compared to pulmonary disorders in case of both acute and chronic exposures. Chapter 9 gives an idea about the deterioration of air quality due to emission of various pollutants, their formation and management methods along with the concerned health issues. This will serve as an imperative document for the scientific community and policy makers to develop effective mitigation policies with respect to air quality improvement particularly for health risk assessment studies. This book ends up with Chap. 10 focusing on the developments and implementations in air quality management and the possible sustainable measures for the same. The chapter highlights the importance of laws and policies and their role in reducing air pollution. It critically analyzes various regulatory measures and policies in most polluted countries of the world. This is also a significant chapter as it highlights the developmental agenda for the future which reflects the links between socioeconomic and environmental sustainability and protects and reinforces the environmental pillar.

1.3 Conclusion

The comprehensive information based on several studies concludes that air pollutant concentrations are still increasing in developing countries even after adoption of several new policies and control technologies. The source apportionment studies of many air pollutants shows that particulate matter are still facing the challenge to minimize their levels so that they can come under permissible limit. Very less attention has been given to biogenic sources as compared to anthropogenic ones, though in case of volatile organic compounds, ozone and secondary aerosol production, they contribute largely and are disturbing the air quality budget. Impact studies related to air pollution and human health are also indicating toward a serious problem which needs to be addressed because the death toll rate due to respiratory illness is increasing day by day. At this stage of sensitive situation, there is an urgent call for stringent control policies or methods to curb air pollution to save the environment. Thus, the different chapters in this book explains about sources of air pollution, i.e., both anthropogenic and biogenic, impact studies on human health, environment and economy and control methods and policies to curb air pollution are given in this book.

References

Ashmore MR (2005) Assessing the future global impacts of ozone on vegetation. Plant Cell Environ 28:949–964

Barrie L (1986) Arctic air pollution. Cambridge University Press, Cambridge, 328 pp

Bell ML, Davis DL, Fletcher T (2004) A retrospective assessment of mortality from the London smog episode of 1952: the role of influenza and pollution. Environ Health Perspect 112:6–8

Bigazzi AY, Figliozzi MA (2014) Review of urban bicyclists intake and uptake of traffic-related air pollution. Transp Rev 34:221–245

Brunekreef B, Holgate ST (2002) Air pollution and health. Lancet 360:1233–1242

Calderón-Garcidueñas L, Solt AC, Henríquez-Roldán C, Torres-Jardón R, Nuse B, Herritt L, Villarreal-Calderón R, Osnaya N, Stone I, García R, Brooks DM, González-Maciel A, Reynoso-Robles R, Delgado-Chávez R, Reed W (2008) Long-term air pollution exposure is associated with neuroinflammation, an altered innate immune response, disruption of the blood-brain barrier, ultrafine particulate deposition, and accumulation of amyloid β-42 and α-synuclein in children and young adult. Toxicol Pathol 36:289–310

Crutzen PJ, Arnold F (1986) Nitric acid cloud formation in the cold Antarctic atmosphere: a major cause for springtime ‘ozone hole’. Nature 324:651–655

Davis LW (2008) The effect of driving restrictions on air quality in Mexico City. J Polit Econ 116:38–81

Fischer PH, Brunekreef B, Lebret E (2004) Air pollution related deaths during the 2003 heat wave in the Netherlands. Atmos Environ 38:1083–1085

Grant U (2012) Urbanization and employment opportunities of youth in developing countries. UNESCO Report, pp 1–37

Heck WW, Taylor OC, Tingey DT (1988) Assessment of crop loss from air pollutants. Elsevier Applied Science, London

IPCC (2013) Climate change 2013. In: The physical science basis. IPCC, Geneva, pp 119–1523

Jim CY, Chen WY (2008) Assessing the ecosystem service of air pollutant removal by urban trees in Guangzhou (China). J Environ Manag 88(4):665–676

Kampa M, Castanas E (2008) Human health effects of air pollution. Environ Pollut 151(2):362–367

Karanasiou A, Viana M, Querol X, Moreno T, de Leeuw F (2014) Assessment of personal exposure to particulate air pollution during commuting in European cities—recommendations and policy implications. Sci Total Environ 490:785–797

Liu JG, Diamond J (2005) China’s environment in a globalizing world. Nature 435:1179–1186

Liu H-Y, Bartonova A, Schindler M, Sharma M, Behera SN, Katiyar K, Dikshit O (2013) Respiratory disease in relation to outdoor air pollution in Kanpur, India. Arch Environ Occup Health 68:204–217

Molina JS, Rowland FS (1974) Stratospheric sink for chlouoromethanes: chlorine atom-catalized destruction of ozone. Nature 249:810–812

Nemery B, Hoet PHM, Nemmar A (2017) The Meuse Valley fog of 1930: an air pollution disaster. Lancet 357:704–708

Nowak DJ, Crane DE, Stevens JC (2006) Air pollution removal by urban trees and shrubs in the United States. Urban Urban Green 4(3–4):115–123

Nowak DJ, Hirabayashi S, Bodine A, Greenfield E (2014) Tree and forest effects on air quality and human health in the United States. Environ Pollut 193:119–129

Pant P, Harrison RM (2013) Estimation of the contribution of road traffic emissions to particulate matter concentrations from field measurements: a review. Atmos Environ 77:78–97

Ritz B, Yu F, Fruin S, Chapa G, Shaw GM, Harris JA (2002) Ambient air pollution and risk of birth defects in Southern California. Am J Epidemiol 155:17–25

Sadanaga Y, Matsumoto J, Kajii Y (2003) Photochemical reactions in the urban air: recent understandings of radical chemistry. J Photochem Photobiol C Photochem Rev 4:85–104

Saxena P, Ghosh C (2011) Variation in the concentration of ground level ozone at selected sites in Delhi. Int J Environ Sci 1(7):1899–1911

Saxena P, Naik V (eds) (2018) Air pollution: sources, impacts and controls. CABI, Wallingford

Saxena P, Sonwani S (2019a) Primary criteria air pollutants: environmental health effects. In: Criteria air pollutants and their impact on environmental health. Springer, Singapore, pp 49–82

Saxena P, Sonwani S (2019b) Secondary criteria air pollutants: environmental health effects. In: Criteria air pollutants and their impact on environmental health. Springer, Singapore, pp 83–126

Saxena P, Bhardwaj R, Ghosh C (2012) Status of air pollutants after implementation of CNG in Delhi. Curr World Environ 7(1):109–115

Saxena P, Sharma Y, Chugh M (2017) Assessment of ozone phytotoxic potential of dense vegetation cover of Delhi by AOT40. In: AGU Fall Meeting Abstracts, December

Saxena P, Srivastava A, Tyagi M, Kaur S (2019) Impact of tropospheric ozone on plant metabolism – a review. Pollut Res 38(1):175–180

Saxena P, Srivastava A, Verma S, Singh L, Sonwani S (2020) Analysis of atmospheric pollutants during fireworks festival 'Diwali' at a residential site Delhi in India. In: Measurement, analysis and remediation of environmental pollutants. Springer, Singapore, pp 91–105

Solomon S, Burkholder JB, Ravishankara AR, Garcia RR (1994) Ozone depletion and global warming potentials of CH3I. J Geophys Res 99:20929–20935

Sonwani S, Kulshrestha UC (2019) PM 10 carbonaceous aerosols and their real-time wet scavenging during monsoon and non-monsoon seasons at Delhi, India. J Atmos Chem 76:171–200

Sonwani S, Maurya V (2018) Impact of air pollution on the environment and economy. In: Air pollution: sources, impacts and controls. CABI, Oxford. ISBN 9781786393890

Sonwani S, Saxena P (2016) Identifying the sources of primary air pollutants and their impact on environmental health: a review. IJETR 6(2):111–130

Speak AF, Rothwell JJ, Lindley SJ, Smith CL (2012) Urban particulate pollution reduction by four species of green roof vegetation in a UK city. Atmos Environ 61:283–293

Sriramachari S (2004) The Bhopal gas tragedy: an environmental disaster. Curr Sci 86:905–920

United Nations, Department of Economic and Social Affairs, Population Division (2017)

Wang S, Zhao M, Xing J, Wu Y, Zhou Y, Lei Y, He K, Fu L, Hao J (2010) Quantifying the air pollutants emission reduction during the 2008 Olympic Games in Beijing. Environ Sci Technol 44:2490–2496

WHO (2014–15) Air quality guidelines for Europe, 2nd edn. WHO Regional Publications, Copenhagen. European Series, p 91

WHO (2015) World Health Statistics Report. World Health Organization, Geneva. http://apps.who.int/iris/bitstream/10665/170250/1/9789240694439_eng.pdf

WHO (2016) World Health Statistics Report. World Health Organization, Geneva. http://apps.who.int/iris/bitstream/10665/206498/1/9789241565264_eng.pdf

World Bank (2009) The world bank annual report 2009. Year in review. http://siteresources.worldbank.org/EXTAR2009/Resources/6223977-1252950831873/AR09_Complete.pdf

Chapter 2
Monitoring and Assessment of Air Pollution

Shani Tiwari, Bing Chen, and A. K. Singh

Abstract During the last few decades, the rapid increase of industrialization and urbanization are the two major factors responsible for high levels of various air pollutants all over the globe. The enhancement of air pollutants into the atmosphere from different natural and anthropogenic emission sources have many observable hostile effects on biota, air quality (like impacts on health, air quality, agriculture, economy, etc.) and future climate change too. Thus, the accurate measurement of air pollution on a global scale is highly needed which will be helpful for the policymakers to improve the air quality management and understanding of future climate change. In the last few decades, the continuous development in technology resulting in development of a highly sophisticated and accurate instrument which is capable of measuring air pollutants and trace gases present in polluted air and widely used on global as well as regional scale. Currently, ample numbers of monitoring and controlling programs are in progress to overcome the complexity of air pollution. The present chapter summarizes a brief overview of the state of different techniques to measure air pollution.

Keywords Air Pollution · Climate Change · Remote Sensing

2.1 Introduction

Currently, air pollution is one of the major concerns among the global scientific communities due to their detrimental effects on air quality, environment, climate, and human health. In recent years, a rapid economic development demonstrated a

---

S. Tiwari (✉) · B. Chen
Environmental Research Institute, Shandong University, Qingdao, Shandong, China
e-mail: pshanitiwari@gmail.com

A. K. Singh
Atmospheric Research Lab., Department of Physics, Banaras Hindu University, Varanasi, India

P. Saxena, A. Srivastava (eds.), *Air Pollution and Environmental Health*,
Environmental Chemistry for a Sustainable World 20,
https://doi.org/10.1007/978-981-15-3481-2_2

strong positive correlation with air pollution worldwide (especially in Asia) and exceeded the WHO's annual particulate exposure guideline ($PM_{2.5} \sim 10\ \mu g/m^3$) resulting in nearly 90% population being exposed to polluted air (WHO 2018; Saxena and Sonwani 2019a, b, c). Air pollution is the greatest environmental health risk today and cause of millions of premature deaths (Lelieveld et al. 2015) which results it to become the fourth leading contributor of premature death and with the global burden of the annual cost of 225 billion USD (World Bank 2016; Sonwani and Kulshreshtha 2016). More than half a million premature deaths are reported over India due to air pollution (Ghude et al. 2016; Chowdhury and Dey 2016). Air pollution is widely divided into two major groups viz. (a) outdoor air pollution which includes ozone, SO_2, CH_4, particulate matters of all sizes and (b) indoor air pollution mainly includes gaseous pollutants (e.g., NO_2, CO) and ultrafine particles emitted from combustion sources. These air pollutants are present in the atmosphere from both primary (directly from natural and anthropogenic sources) as well as secondary emission sources (gas to particle conversion). These pollutants have harmful effects on human health for short time scale (e.g., eye irritation, respiratory irritation, ear infection) as well as on long time scale (e.g., lung cancer, heart disease) (Deng et al. 2017; Kampa and Castanas 2008). Atmospheric aerosols (also known as particulate matter) are one of the major air pollutants present in the atmosphere and have significant impact on the earth's radiation budget through the scattering/absorption of solar and longwave radiation, the modification of cloud optical properties as well as through various complex atmospheric processes (Tiwari et al. 2015, 2016, 2018; Seinfeld et al. 2016; Fan et al. 2016; Vinoj et al. 2014; IPCC 2013; Ramanathan et al. 2001). They also have the ability to disturb the global circulation pattern, hydrological cycle, and carbon cycle (Lau et al. 2006; Mahowald 2011). It is well known that trace gaseous pollutants not only have significant impacts on the earth's radiation budget (IPCC 2013) but they also play a vital role in the formation of secondary organic aerosol (SOA) and volatile organic compounds (VOCs) which are one of the most crucial atmospheric pollutants (Sonwani et al. 2016).

Over the last few decades, several instruments have been developed to measure air pollution. Every instruments and measurement techniques have their own merits, demerits and limitations. For example, the ground-based measurements of air pollution could not be possible on a global scale due to the different constraints like instrumental cost, geographical location, etc. However, the measurement of air pollution from space is a good technique and relatively cheaper than in situ measurement barring certain constraints like temporal and spatial resolution, coverage of limited number of air pollutants, etc. Thus, satellite observations are one part which provides complete global coverage and will serve only as a complement to ground observations. Due to the recent developments in technologies, remote sensing is one of the most effective and economical tools for air pollution measurements. It has the capability to measure the vertical distribution of air pollutants which play a substantial role in the calculation of the radiation budget. Therefore, for better utilization of the air quality measurement, the scientific community should have combined the spaceborne measurements with the information from ground-based sensors and models, which will improve the air quality forecast, characterization of

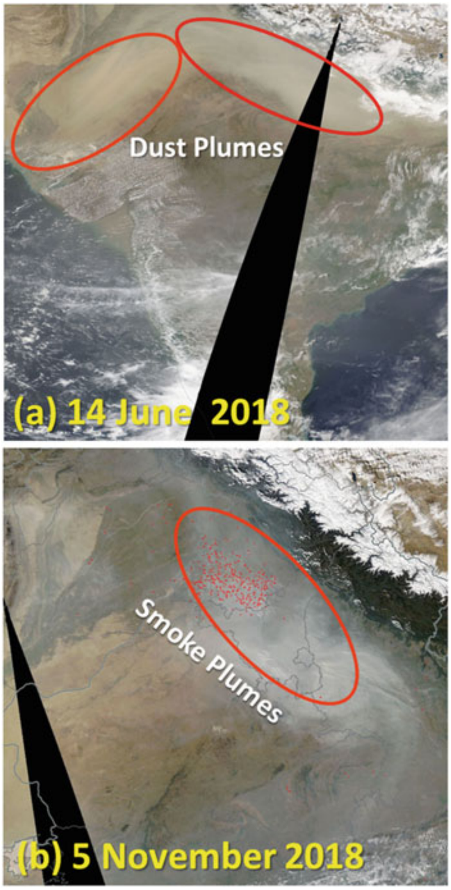


Fig. 2.1 MODIS True color images showing the intense dust plumes over the entire Indo-Gangetic Basin on June 14, 2018 (**a**) and smoke plumes on November 5, 2018. The red dots in (**b**) are MODIS active fire hot spots

surface-level air pollution, and emission inventories. The spaceborne measurements also have the capability to capture the natural hazardous events like a dust storm, haze, fog, etc., which enhance the concentration of air pollutants into the atmosphere. Figure 2.1a represents the MODIS true color images of dust plumes during the dust storm event on June 14, 2018 which spread over entire Indo-Gangetic Basin (IGB) and moving toward northeast causing dust deposition on the Himalayan foothills. Earlier researchers have also reported the dust plumes over the different region of the world from the spaceborne measurements (Singh et al. 2016; Alam et al. 2014; Gautam et al. 2013; Hsu et al. 2013). On the other hand, Fig. 2.1b shows MODIS true color image of smoke plumes on November 5, 2018 over the IGB which is mainly due to the biomass burning from the Northern India (mainly Haryana and Punjab) which is further confirmed from the MODIS fire hot spot (red dots) showing maximum density over the region. Recently, Sarkar et al. (2018) also reported the dense haze over the northern IGB using the synergetic measurement of the ground and spaceborne radiometers.

Therefore, the accurate measurement of air pollutants is highly needed which will be helpful for the policymakers at regional as well as global scale to improve the air quality for the protection of this sensitive environment without further waiting for legislative laws. The synergetic use of ground-based and spaceborne measurement provides a unique platform for in-depth study of air pollution, their transportation, and adverse impacts. In the upcoming section, brief descriptions of different air pollutant measurements techniques from the ground as well as space are discussed.

2.2 Impacts of Air Pollution

In the twenty-first century, air pollution is a major threat to human health and causes a number of potentially deadly diseases and illness, e.g., lung disease and other respiratory problems. Many studies have shown that air pollution is directly responsible for the development of asthma, Chronic Obstructive Pulmonary Disorder (COPD), cardiovascular and heart disease. Annually, nearly 7 million premature deaths are reported mainly due to air pollution (WHO 2018; Saxena and Sonwani 2020). Earlier studies also reported that the higher exposure of particulate matter can cause preterm birth thus raising the new born babies' risk of health complications (Malley et al. 2017; Nieuwenhuijsen et al. 2014). Air pollution is also hazardous for the plants and vegetation causing a reduction in crop yields production (Burney and Ramanathan 2014). They cause many hazardous environmental phenomena like acid rain, smog, haze, etc. which lead to affect human life in different ways. Many atmospheric pollutants (like CO_2, black carbon) have the ability to trap the atmospheric heat and affect the earth's radiating budget (Saxena and Naik 2018). Particulate matter (PM) can also change the cloud properties resulting in changes in cloud albedo as well as total rainfall (Seinfeld et al. 2016). The net positive radiative forcing is reported for different atmospheric pollutants present into the atmosphere associated with a large error (Fig. 2.2) suggesting that further in-depth investigation of the radiative impacts these air pollutants, is required (IPCC 2013). Figure 2.2 reflects net positive radiative forcing (2.83 Wm^{-2}) due to well-mixed traces gases among which CO_2 has the maximum value. The tropospheric ozone has positive radiative forcing while stratospheric ozone has vice versa. Apart from this, the radiative forcing due to the atmospheric aerosol show both positive and negative radiative forcing with a large error indicating toward a major contributor of net global radiative forcing. A number of studies also reported both positive and negative radiative forcing for different types of aerosol over the different regions of the world from different measurements (Boiyo et al. 2019; Zhu et al. 2019; Bibi et al. 2017; Tiwari et al. 2015).

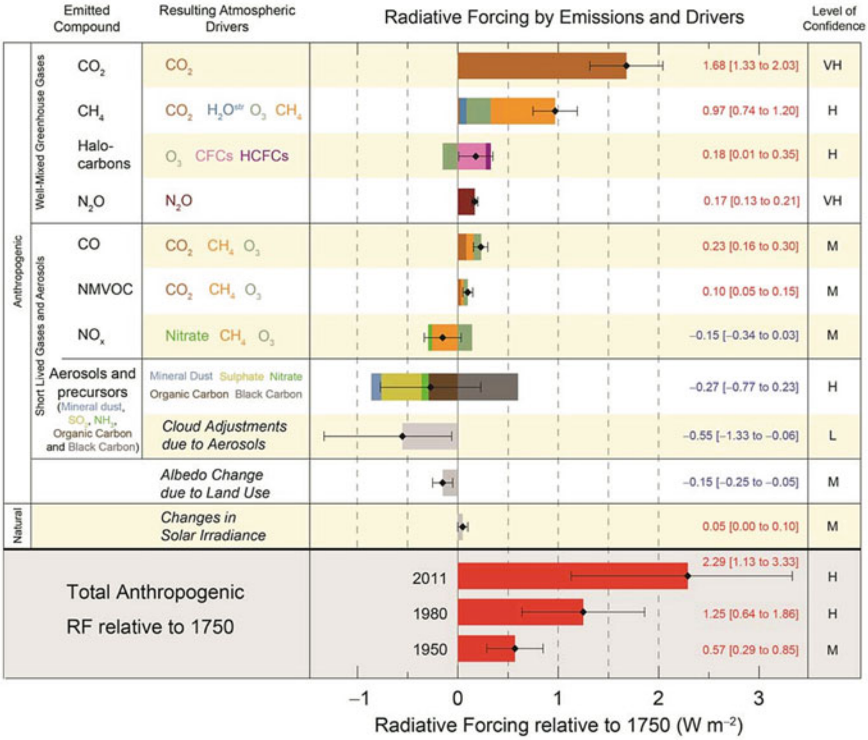


Fig. 2.2 Global radiative forcing of various air pollutants for the period 1750–2011 (adopted from the IPCC 2013)

2.3 Measurement of Air Pollution

The accurate measurements of air pollutants are needed for mitigation and future air quality-related policies. Several techniques, i.e., spectroscopic, chromatography, remote sensing techniques along with sampling and size distribution measurement are quite popular and widely used for the measurement of air pollutants. A brief description of the state-of-art view of various different air pollution measurement techniques are described as below.

2.3.1 Spectroscopic Techniques

The spectroscopic techniques are one of the most popular methods to measure air pollutants present in the atmosphere. These techniques are based on the interaction of electromagnetic radiation with atoms and molecules of interest and also used in the spaceborne measurement of air pollutants. Numbers of instruments are developed based on different spectroscopic techniques viz. emission spectroscopic, infrared absorption spectroscopic, ultraviolet-visible spectroscopic, mass spectroscopic, etc. In emission spectroscopic techniques, the air pollutants are excited to a higher electronic state and the intensity of emitted radiation is measured. The mass spectroscopic technique is used in the development of aerosol mass spectrometers which can analyze the size and chemical composition of individual particles within a few seconds. A number of aerosol mass spectrometers are developed like time-of-flight mass spectrometers (TOFMS), Quadrupole mass spectrometers (QMS), Magnetic sector mass spectrometer (MSSS), and Ion trap mass spectrometers (ITMS) depending on their ion separating method. An example of the measurement of gaseous air pollutants (i.e., SO_2, NO_2, CO, and O_3) using spectroscopic techniques, over China, during 2014–2016 is shown in Fig. 2.3. This figure reflects the interannual variability of pollutants which shows the decreasing trends (excluding O_3) from 2014 to 2016 with the mean concentrations of SO_2, CO, and NO_2 as 33.77 ± 18.47 mg m^{-3} to 21.86 ± 11.79 mg m^{-3}, from 1.19 ± 0.40 mg m^{-3} to 1.01 ± 0.32 mg m^{-3}, and from 36.17 ± 11.01 mg m^{-3} to 32.00 ± 9.51 mg m^{-3}, respectively. The significant decrease in SO_2 particularly in the North China and North China Plain (NCP) is mainly attributed due to the up-gradation of flue gas desulfurization (FGD) systems in thermal power units and adoption of electrostatic precipitators (ESP) for the particulate matter (Li et al. 2017b; Hua et al. 2016). Although the number of vehicles has linearly increased during 2014–2016, yet the reduction in NO_2 emission is mainly because of the up-gradation of fuel quality standards and implementation of effective emission control policies to power plants (Tian et al. 2013; Li et al. 2016). The decrease in CO is mainly due to a decrease in coal consumption and straw recycling instead of open biomass burning (Lindner et al. 2013). On the other hand, the concentration of O_3 showed an opposite trend (i.e., increasing) with concentration 87.65 ± 16.74 mg m^{-3} in 2014 to 98.57 ± 14.86 mg m^{-3} in 2016. Remarkable enhancement in O_3 over Yang River Delta is mainly due to the oxidation of aromatic hydrocarbons emitted from the industrial emission sources (Zhang et al. 2015).

2.3.2 Sampling Methods

The measurement of aerosol mass and number concentration is very important for the air quality-related policies as well as for the study of long-term climate change (Saxena and Sonwani 2020). Most of the countries have their own national ambient air quality standards based on the concentration of particulate matter and other pollutants. Thus, particulate matter concentrations are routinely measured worldwide. The most common method to measure the PM concentration is filtration method in which the filters are weighed under the control temperature and relative humidity before and after sampling. Then, PM concentration is calculated from the increase in filter mass and the volume of the air sample. This method has an added advantage that particles collected on the filter can be also analyzed chemically.

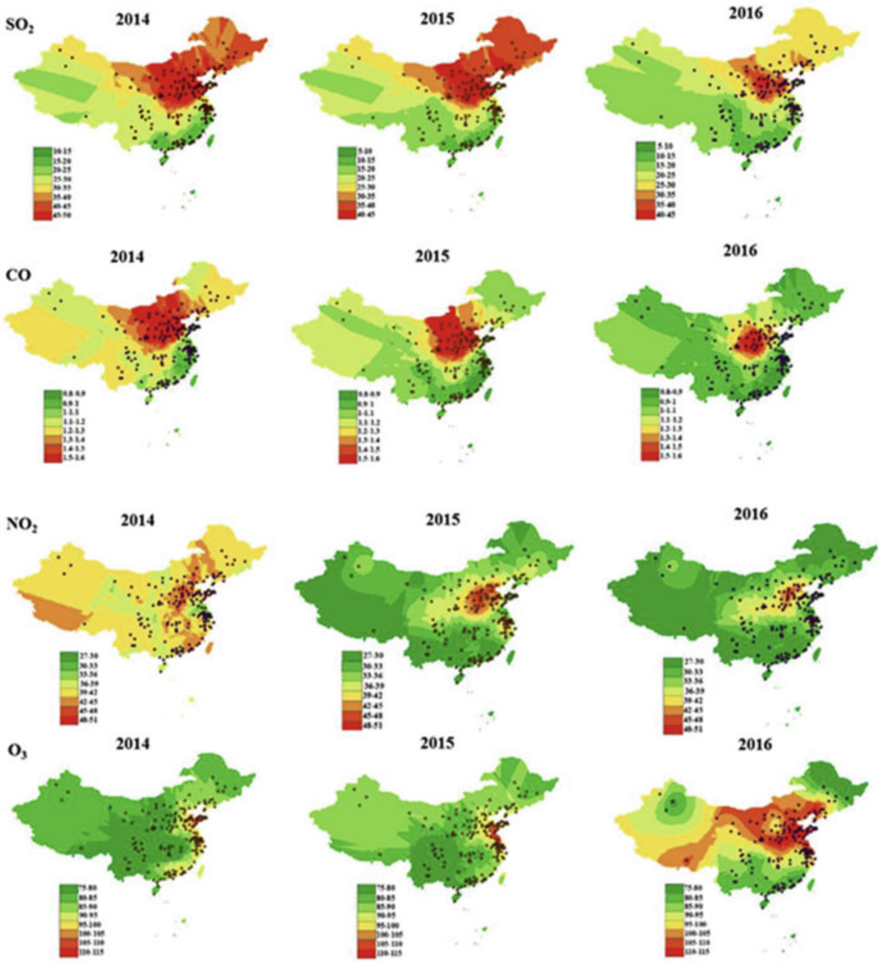


Fig. 2.3 Spatial variability of gaseous pollutants (i.e., SO_2, NO_2, CO, and O_3) during 2014–2016 over the mainland of China (adopted by Li et al. 2017b)

Recent development of instrumentation has resulted in some automatic sampling instruments (like Beta attenuation monitoring: BAM1020) which provide the PM_{10} and $PM_{2.5}$ concentration ($\mu g\ m^{-3}$) at high time resolution in the actual condition. It measures the attenuation of beta radiation through a particle loaded filter and the degree of attenuation is closely proportional to the mass of the sample. In addition to this, Tapered Element Oscillating Microbalance (TEOM) is another fully automatic sampling instrument, which is based on the microbalance method and provides PM concentration at very high time resolution. In TEOM, a filter is mounted at the end of a hollow tapered quartz tube which is free to oscillate. Particles are collected on the filter and cause the changes in the tube's frequency of the oscillation which is directly related to the mass of the sample. An example of variation in daily and monthly mean concentration of particulate matter (both PM_{10} and $PM_{2.5}$) and their ratio at Bhubaneswar during 2012–2014 is shown in Fig. 2.4a–c, respectively. The concentration in particulate matter showed a large daily and inter-/intra-seasonal variation with the annual mean value of 30.6 ± 22.1 $\mu g\ m^{-3}$ ($PM_{2.5}$) and 88.3 ± 30.6 $\mu g\ m^{-3}$ (PM_{10}). Significant variation in concentration of $PM_{2.5}$ and PM_{10} is mainly due to various aerosol types, their emission sources and long-range transportation. A pronounced peak in particulate matter loading is observed in winter followed by the post-monsoon season associated with a higher value of $PM_{2.5}/PM_{10}$ signifying toward the higher concentration of fine mode aerosol particles. Relatively higher concentration of fine mode aerosol is also reported over IGB during the post-monsoon and winter season (Sarkar et al. 2018; Tiwari et al. 2016, 2018; Badarinath et al. 2009).

2.3.3 Chromatography Techniques

Chromatography is an analytical technique to separate and identify the components of the complex mixture of air pollutants by differential movement through a two-phase system, i.e., stationary and mobile phases. The mobile phase can be liquid or gas and carries the mixture of substance in the sample through the adsorptive material (e.g., Kaolin, alumina, silica) also known as the stationary phase. This is a highly sophisticated and accurate technique used to distinguish the different constituents of air pollutants present in the environment. Depending on the type of stationary phase, nature of adsorptive forces, and mobile phase, different types of chromatographic techniques are used in different instruments. For example, in gas chromatography (GC), an inert gas is used as mobile phase and volatile organic compounds (VOCs) present in the atmosphere are separated in column and measured by the detector. An ion exchange resin is used as a stationary phase in ion chromatography (IC) which separates ions and polar molecules based on their affinity to the ion exchange. In adsorption chromatography, the stationary phase is an adsorbent material while in high-pressure liquid chromatography (HPLC), the mobile phase is liquid which is forced under high pressure through a column packed with the sorbent. Figure 2.5 shows an example of the measurement of VOCs and O_3 variability at Rocky Mountain National Park (ROMO) from July to October 2014 during the Front Range Air Pollution and Photochemistry Experiment (FRAPPÉ) campaign. Figure 2.5 suggest that during most of the times, all the observed VOCs had generally similar temporal variation suggesting that the regional meteorology plays a crucial role in their concentration changes. However, in some cases, some differences can also be seen for which sources are mainly due to the specific emission (Benedict et al. 2019).

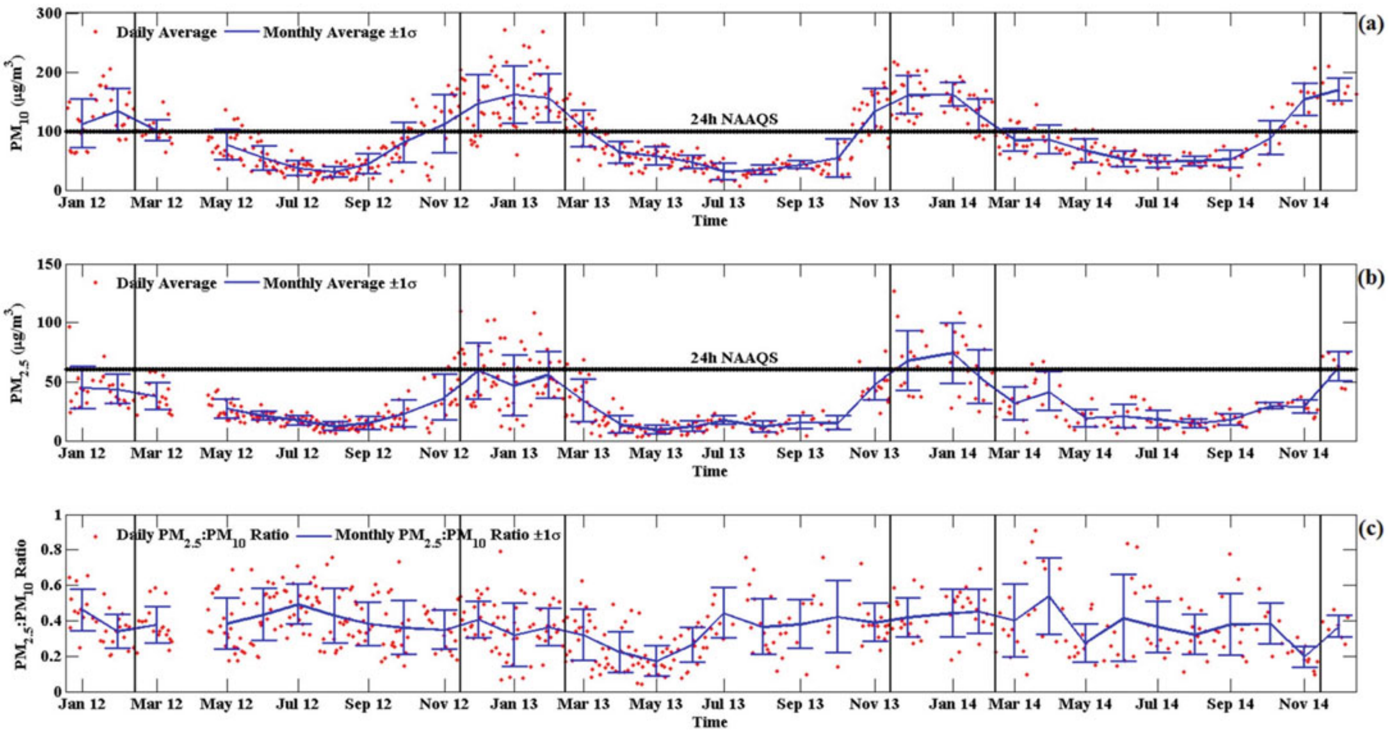


Fig. 2.4 Daily and monthly mean variation of (**a**) PM$_{10}$ (μg m^{-3}) (**b**) PM$_{2.5}$ (μg m^{-3}) and (**c**) PM$_{2.5}$/PM$_{10}$ at Bhubaneswar during 2012–2014. The vertical lines represent the ±1σ of the monthly mean (adopted Mahapatra et al. 2018)

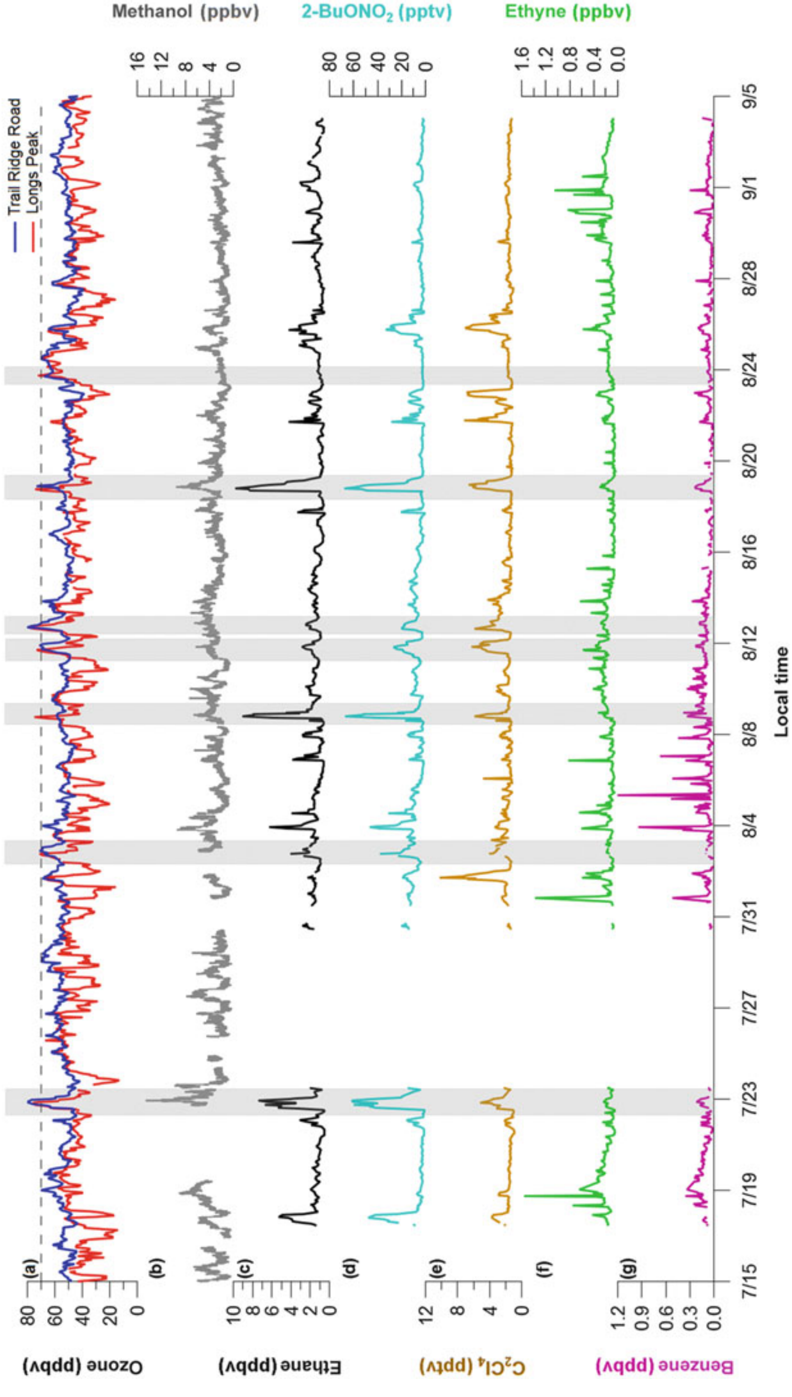


Fig. 2.5 Day-to-day variation in a subset of VOCs along with ozone measured at the Rocky Mountain National Park ROMO-LP site from July 17, 2014 to September 4, 2014 (adopted by Benedict et al. 2019)

2.3.4 Size Measurement Techniques

The size of the particulate matter has crucial importance to the human health, air quality as well global climate (Tiwari et al. 2018; Deng et al. 2017; Seinfeld et al. 2016; IPCC 2013). The size measurement of particulate matter has been carried out using different instruments like Differential Mobility Analyzer (DMA), Differential Mobility Particle Sizer (DMPS), Scanning Mobility Particle Sizer (SMPS), Optical Particle Counters (OPCs), Aerodynamic Particle Sizers (APS) etc. based on variety of approaches and can measure the aerosol particle in wide size range. In DMA, particles are classified based on their electrical mobility which is a function of particle charge, particle geometric size, and have ability to provide particle size in a large range 3–1000 nm. In OPCs (GRIMM Aerosol Spectrometer), the particle size is calculated based on light scattering technique. A fraction of scattered light is collected and converted into voltage pulses which provide the particle size using a calibration curve. It can measure the particle size in the range of 0.2 μm to up to several tens of microns in diameters. Apart from this, APS can provide the real-time measurement of particle size and their number concentration simultaneously at very high resolution. In APS, aerodynamic particle size is measured by the particle velocity which is the function of time of flight taken by the particle to travel the distance. The measurement of the smallest size of the particle depends on the design of the instrument and reported as minimum as 0.2–0.5 μm in diameter. Figure 2.6 shows the normalized average number and surface area size distributions of submicron aerosol measured by the SMPS on board the aircraft during the Dynamics–Aerosol–Chemistry–Cloud Interactions in West Africa (DACCIWA) campaign in June–July 2016. Figure 2.6 clearly reflects two different size modes suggesting a relatively higher value of accumulation mode particles indicating toward more aged aerosol. The smaller Aitken mode aerosol particles are highly variable which could be easily seen with a large spread in data suggesting that region contains fresher

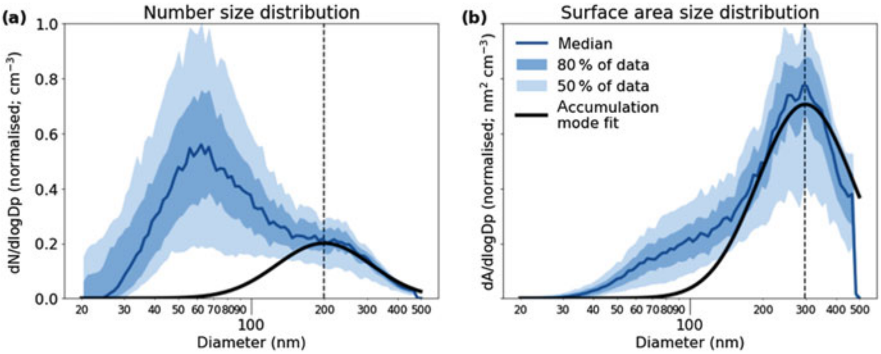


Fig. 2.6 The aerosol number size distribution (**a**) and volume size distribution (**b**) of submicron aerosol during the DACCIWA campaign. The rapid drop-off at the high end of the surface area distribution is an artifact of the SMPS size limit (adopted by Haslett et al. 2019)

emission source of finer aerosol (Haslett et al. 2019). However, accumulation mode aerosol particles are relatively stable during the whole campaign.

2.3.5 Remote Sensing: An Important Tool for Air Pollution Measurement

The remote sensing is a technique in which the information about the earth is taken without taking the physical sample of the earth's surface. In this technique, a sensor is used to measure the reflected energy from the earth. It can be used for the study of land-use change, air pollution tracking, and their transportation as well as in defense system also. Based on the emission sources and working principle, remote sensing is mainly divided into two major parts, i.e., (a) active and (b) passive remote sensing. In active remote sensing system, the sensor itself emits the radiation toward the object and measured the reflected signal (e.g., LIDAR: Light Detection and Ranging for optical domain and RADAR: Radio wave Detection and Ranging for radio wave domain, CALIPSO: Cloud-Aerosol Lidar and Infrared Pathfinder Satellite Observation); however, in passive remote sensing, system measures the energy/radiation from the earth's surface and atmosphere (AERONET: Aerosol Robotic Network, MODIS: Moderate Resolution Imaging Spectroradiometer, AIRS: Atmospheric Infrared Sounder, etc.). Passive remote sensing is the most popular and widely used in air pollution measurement from space due to their higher quality of satellite imaginary. The information collected from the different platform is fed to the centralized data collection center. The spatial and temporal resolution of the measurement is mainly depending on the orbit of the satellite and the viewing/scanning properties of the payloads (instruments). During each orbit, satellites cover a definite track on the earth, often called swath width having a range in between less than 100–3000 km which depend on the viewing/scanning properties of the payloads. Satellites having higher swath width (>2000 km) covers the whole earth in a day. Most of the satellites are nadir viewing (looking down toward the earth) providing the columnar information of air pollutants (up to 60–100 km, depending on the profile); however, those satellites having limb view (view atmosphere from sideways) can provide air pollutants profile for stratosphere only because of the large spatial variability of the troposphere. Further, a brief description of the measurement of air pollutants using different remote sensing techniques is discussed below.

2.3.5.1 Measurement of Atmospheric Aerosol

In the last two decades, atmospheric aerosol is recognized as one of the most important air pollutants due to their adverse impacts on the earth's system in different ways. Sun photometer/Sky radiometer is one of the most popular ground-based measurements for aerosol. A number of studies have been carried out for a better understanding of various aerosol properties under the various research program viz. AERONET (Aerosol Robotic Network) from NASA, CARSNET (China AeRoSol NETwork), SKYNET (SKY radiometer NETwork), ARFINET (Aerosol Radiative Forcing NETwork) worldwide (Tiwari et al. 2013, 2018; Moorthy et al. 2013; Xia et al. 2016). Apart from this, LIDAR is also another popular instrument for the ground-based measurement of aerosol. On the other hand, satellite remote sensing is mostly useful for the study of aerosol characteristics on a global scale. Numerous spaceborne sensors (e.g., MODIS, MISR, OMI, AVHRR, CALIPSO, etc.) are launched onboard different satellites which have different spatial and temporal resolution and can provide the different aerosol parameters (e.g., aerosol optical depth: AOD, Angstrom Exponent: AE; Single Scattering Albedo: SSA, etc.). The advanced retrieval of aerosol from spaceborne is well calibrated with ground-based instruments (AERONET) at a different region of the world which enhance the data quality assurance (Rupakheti et al. 2019; Tiwari and Singh 2013). The correlation between satellite retrieves and ground-based AOD shows a large variation on seasonal as well as a regional scale that may be because of the difference in surface reflectance, retrieval algorithm, etc. For example, the quality of MODIS data for AOD over the ocean is better than land (Prijith et al. 2013). The uncertainty associated in the MODIS retrieved AOD (τ) with AERONET is well documented as $\Delta\tau = \pm(0.05 + 0.15\tau_{\text{MODIS}})$ over land and $\Delta\tau = \pm(0.03 + 0.05\tau_{\text{MODIS}})$ over ocean, respectively (Remer et al. 2008; Levy et al. 2010). CALIPSO has ability to provide the vertical distribution of aerosol which is very crucial in the identification of different aerosol types and their effects on radiative forcing (Tiwari et al. 2016; Mishra and Shibata 2012). Based on sunphotometer measurements, different types of aerosol are also identified using various classification techniques (Rupakheti et al. 2019; Tiwari et al. 2015, 2018; Kumar et al. 2015). The aerosol climatology obtained from the AERONET measurement over Kanpur for the period of 2000–2010 is shown in Fig. 2.7a (Kaskaoutis et al. 2012) which represents a strong day-to-day and inter-/intra-seasonal variability with increasing aerosol trend over Kanpur. The slope of linear regression along with the percentage difference and confidence level are given inside the figure (black for $\text{AOD}_{500\text{ nm}}$, green for Angstrom Exponent for the pair of wavelengths 440–870 nm, i.e., $\text{AE}_{440\text{–}870\text{ nm}}$, blue for $\text{AE}_{380\text{–}500\text{ nm}}$ and red for $\text{AE}_{675\text{–}870\text{ nm}}$. Figure 2.7a reflects an increasing trend in AOD loading (7.69%) over Kanpur during the study period which is statistically quite significant ($p < 0.5$). The identification of different aerosol types based on AERONET fine mode fraction (FMF) and single scattering albedo (SSA) measured data, at four different locations viz. Karachi, Lahore, Jaipur, and Kanpur are shown in Fig. 2.7b (Tiwari et al. 2015). Four different types of aerosol are classified viz. polluted dust (PD), polluted continental (PC), black carbon enriched (BCE), and organic carbon enriched (OCE). Relatively lower FMF value associated with higher SSA value is observed for PD demonstrating the higher concentration of larger size particles. However, for OCE higher value of SSA along with higher FMF value imparting toward the scattering nature of aerosol particles having a larger fraction of fine particles. These observed aerosols have a different annual contribution in total aerosol loading which also showed a significant seasonal variation (Tiwari et al. 2015). On the other

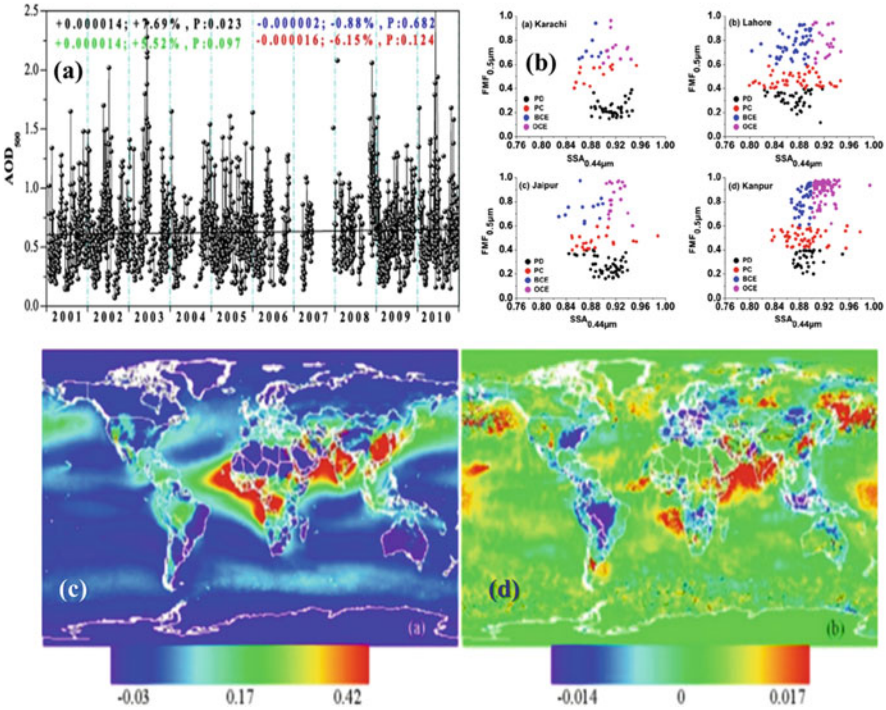


Fig. 2.7 Variability of aerosol climatology over Kanpur during 2001–2010 using AERONET data (**a**), scatter plot of FMF vs. SSA for the aerosol classification over Karachi, Lahore Jaipur, and Kanpur (**b**) spatial variation of mean AOD during 2002–2012 obtained from MODIS (**c**) and spatial variation of rate of change of AOD during 2002–2012 (**d**) [adopted from Kaskaoutis et al. 2012 (**a**), Tiwari et al. 2015 (**b**), Mao et al. 2014 (**c**) and (**d**)]

hand, MODIS observation suggests the relatively higher value of AOD over China, India, and Africa region than rest of the world during 2002–2012 (Fig. 2.7c) having the positive rate of change of AOD (Fig. 2.7d). Dey and Di Girolamo (2010) also found an increasing trend over the Indian region using MISR data set.

2.3.5.2 Measurement of Ozone

Ozone (O_3) is a reactive oxidant gas and one of the most crucial air pollutants into the atmosphere having ability to affect human health, vegetation at surface level (Liu and Peng 2018; Yue and Unger 2018), and the earth radiation budget at a columnar level. Most of the earth's atmospheric ozone is found into the stratosphere (~90%) which forms a thick layer (about 15–30 km), also known as "Ozone layer" (Schultz et al. 2017). The ozone hole (the hole in the ozone layer) over Antarctica because of air pollution became an international issue among scientific communities, but a recent study reported that it is recovering slowly (Hossaini et al. 2017). This layer

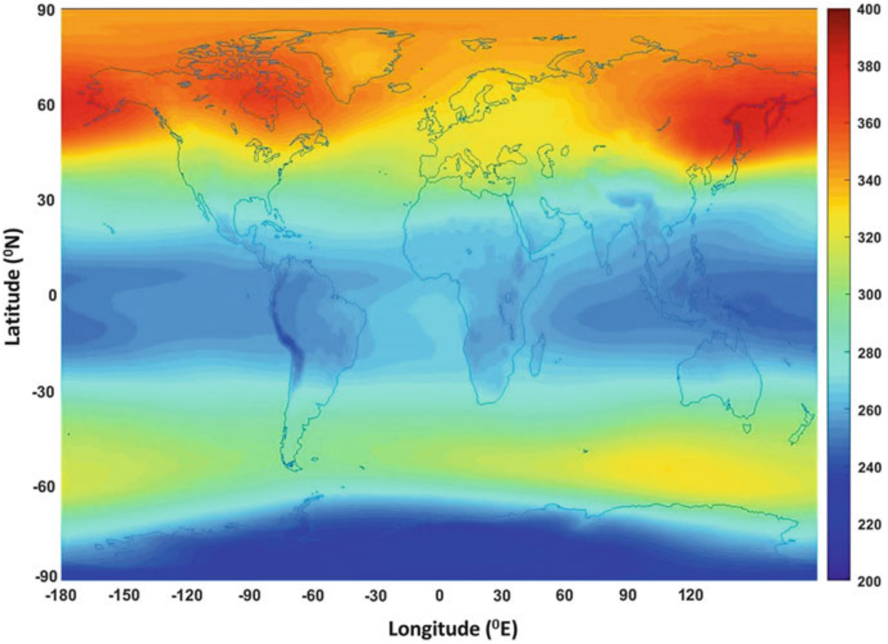


Fig. 2.8 Global monthly mean climatology of total columnar ozone (in Dobson Unit) subcontinent for 2005–2015 obtained from OMI

absorbs solar UV radiation (mainly in the wavelength range from 290 to 320 nm) which can damage the vegetation and could be one of the reasons for skin cancer in human being (Eastham and Barrett 2016). On the other hand, remaining 10% of the ozone is found into the lower troposphere which is mainly from the by-products of industrial productions and through chemical reactions including hydrocarbon, nitrogen oxides, etc., resulting in relatively higher concentration over urban areas (Maji et al. 2019; Saxena and Sonwani 2019a, b, c).

The tropospheric ozone is more toxic and has the ability to affect vegetation and air quality causing many breathing problems in human beings. The tropospheric ozone causes heating of the atmosphere with positive radiative forcing while stratospheric ozone has the opposite nature (IPCC 2013). The Ozone Monitoring Instrument (OMI), Atmospheric Infrared Sounder (AIRS), and Total Ozone Mapping Spectrometer (TOMS) are very popular spaceborne sensors onboard different satellites which are capable to measure the total columnar ozone with different spatial resolution. OMI is nadir viewing with wide swath having 20 hyperspectral imaging spectrometers is capable to provide global coverage of top of the atmosphere upwelling radiances in the ultraviolet and visible (270–500 nm) regions of the solar spectrum with high resolution (i.e., 13 × 24 km). Further details of the OMI sensor are discussed elsewhere (Levelt et al. 2006; Tiwari et al. 2016). The climatology of total column ozone (in Dobson Unit: DU) is shown in Fig. 2.8. Figure 2.8

suggest a higher value of Ozone over the Arctic region while relatively lower ozone value is observed over the equator.

2.3.5.3 Measurement of Tropospheric Nitrogen Dioxide

Nitrogen dioxide (NO_2) is highly reactive and notorious air pollutant present into the atmosphere. They are mainly emitted from the anthropogenic sources like vehicle engines biomass fuel and crop residue burning, soil emissions and power plants at a surface level while in the troposphere the main source of NO_2 is atmospheric lighting (Cheng et al. 2012; Finney et al. 2016). Tropospheric NO_2 is mainly formed by oxidation process of nitrogen monoxide (NO) which is further converted into NO through photolysis process in the daylight (Werner et al. 2013). It also produces methane and aldehydes, ozone through the photochemical reactions in the presence of volatile organic compounds (VOCs) in sunlight, hence also known as "indirect greenhouse gas" (Finney et al. 2016, U.S. CAR 2010). At higher concentration, it has adverse effects on respiratory systems of human beings, visibility and can lead to the formation of acid rain, smog, etc. (Lamsal et al. 2015; Kampa and Castanas 2008). A number of spaceborne measurements for columnar tropospheric NO_2 has been conducted since 1995 (Burrows et al. 1999) and well corroborated with NO_x emission (Streets et al. 2013) and monitoring of air pollution over a large spatio-temporal scale (Lamsal et al. 2015; Duncan et al. 2016). Significant numbers of satellites have been launched to monitor tropospheric NO_2 such as SCanning Imaging Absorption spectroMeter for Atmospheric CHartographY (SCIAMACHY), Global Ozone Monitoring Experiment-2 (GOME-2), and Ozone Monitoring Instrument (OMI) which have different spatial as well as temporal resolution. Currently, OMI is the state-of-the-art instrument for tropospheric NO_2 from space which board on NASA Aura satellite, because of their relatively higher spatial resolution and continuous measurement over a decade and widely used in emission trend studies (Boersma et al. 2011; Krotkov et al. 2016; Liu et al. 2017; Lamsal et al. 2015; Duncan et al. 2016). In addition, the emission of NOx can also be determined using the inverse modeling in which satellite data in combination with model output data is used to improve the source strengths. The spatial variability of tropospheric NO_2 over the United State for 2005 and 2013 is shown in Fig. 2.9. Figure 2.9 clearly reflect a substantial decrease in NO_2 over the United States from 2005 (about to 30–40%) which is mainly due to effective environmental policies and technologies changes (Lamsal et al. 2015). The reduction in NO_2 also causes the reduction of ozone by 14% over the United States (EPA 2012). An increasing trend in tropospheric NO_2 (nearly 52%) is also reported over 48 different Chinese cities during 2005–2011 while after that it decreased interestingly by 21% during 2011–2015 which is because of different emission control policies (Liu et al. 2017). The surface concentration of NO_2 in Beijing is showed a decreasing trend during 2008–2015 (Cheng et al. 2018). On the other hand, an increasing trend (10% a year) of NO_2 is found over the Canadian oil sands due to the intensive surface mining (McLinden

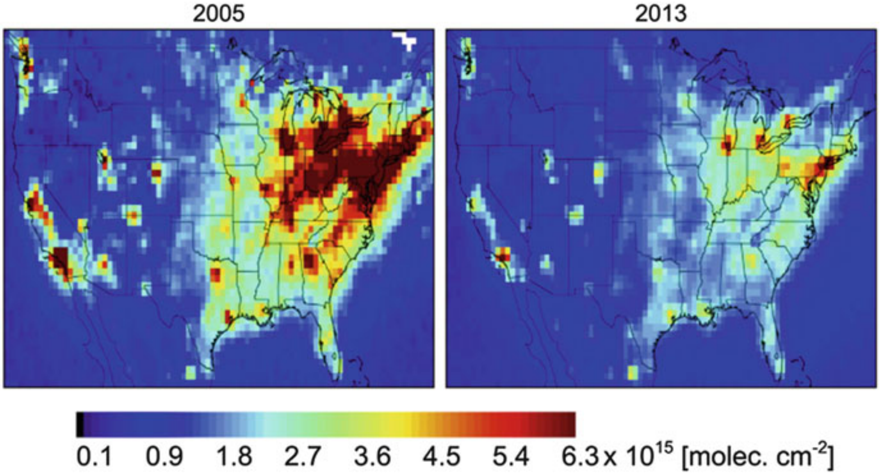


Fig. 2.9 Annual average OMI NO_2 over the United State during 2005–2013 (adopted by Lamsal et al. 2015)

et al. 2016) suggesting that the main cause for the change in the tropospheric NO_2 concentrations is the change in the source strengths.

2.3.5.4 Measurement of Carbon Monoxide

Carbon monoxide (CO) is reactive toxic gas and largely distributed into the atmosphere. The major sources of CO are from anthropogenic sources like biomass burning, fossil fuel combustion, oxidation of methane, volatile organic carbons (VOCs) resulting in the nearly two-third contribution of total emission (Deeter et al. 2017; Seinfeld and Pandis 2010). The maximum concentration of CO is found within the boundary layer and can transport over a large distance. They have a significant impact on global climate change with positive indirect radiative forcing 0.2 Wm^{-2} (Myhre et al. 2013). CO also served as an ozone precursor and used in different studies of pollution sources, their transportation as well as air quality forecasting (Inness et al. 2015). MOPITT is thermal nadir-viewing gas-correlation radiometer and the most popular spaceborne measurement for the tropospheric CO. It provides the information about the CO emission separately for lower troposphere (700–500 hPa) and upper troposphere (300–200 hPa) with a horizontal resolution of 22 km (Deeter et al. 2003; Drummond 1992). Further details about measurement and retrieval techniques from MOPITT have been described elsewhere (Edwards et al. 1999; Deeter et al. 2003). Figure 2.10 shows the spatial variability of CO and its transportation over western coastal India and the Arabian Sea at 850 hPa measured by MOPITT for the period of (a) November 1–10, 2007; (b) November 11–20, 2007; and (c) November 21–30, 2007. A relatively higher concentration of CO loading was observed over the Arabian Sea due to long-range

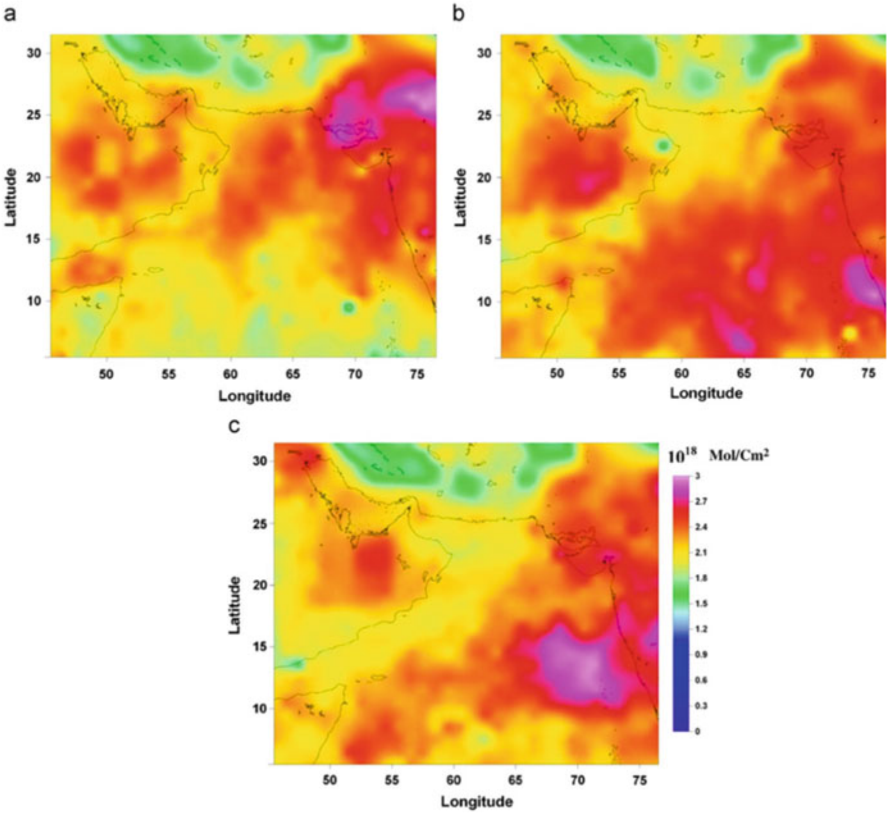


Fig. 2.10 Spatial distribution of MOPITT CO Columnar concentration at 800 hPa during (**a**) November 1–10, 2007, (**b**) November 11–20, 2007 and (**c**) November 21–30, 2007 (adopted by Badarinath et al. 2009)

transport of anthropogenic aerosols and trace gases which are mainly emitted from crop residues burning in the IGB and extensive fireworks during Diwali festival (Badarinath et al. 2009). MOPITT derived CO shows a decreasing trend i.e. ~−1% on a global scale while relatively higher decreasing trend (~−1.6%) over East Asia during 2005–2016 (Worden et al. 2013). The spatial variability of tropospheric CO trends obtained from MOPITT over East Asia is shown in Fig. 2.11. Relatively lower decreasing trend is found over western China than Eastern China due to upwind of the heavily industrialized region. While on the other hand, higher decrease in CO (~−1% year^{-1}) is also observed over the coastal region of East Asia and Eastern Pacific which suggest the reduction in export of CO through westerlies winds (Zheng et al. 2018).

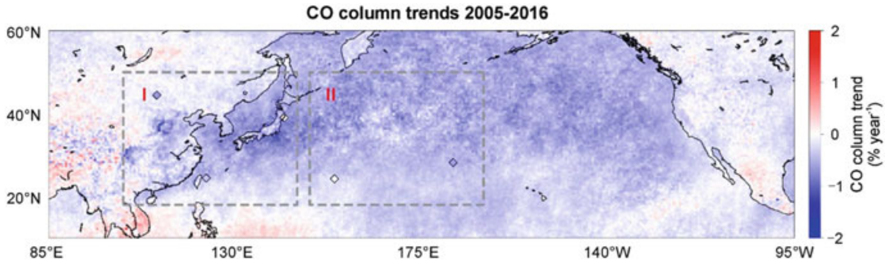


Fig. 2.11 Tropospheric CO column trends derived from MOPITT V7 data over East Asia during 2005–2016 (adopted by Zheng et al. 2018)

2.3.5.5 Measurement of Sulfur Dioxide

Sulfur dioxide (SO_2), one of the trace gases, mainly emitted into the atmosphere from both natural (e.g., volcanic eruption, oxidation of dimethyl sulfate) as well as anthropogenic sources (combustion of fossil fuel). The coal power plants are one of the largest emission sources of SO_2 and make a significant contribution global sulfur budget which causes the degradation in visibility and air quality (Valverde et al. 2016; Vet et al. 2014). Most of the SO_2 are found within the boundary layer because of their near-surface emission sources resulting in hazardous for human health (Chiang et al. 2016). Studies suggest that they could contribute to the acid rain and damage the respiratory system by affecting lung function as we well as the growth of plants (Saygın et al. 2017). The atmospheric lifetime of SO_2 is very short and it can produce sulfate aerosols through an oxidation process which is a major contributor to $PM_{2.5}$ chemical composition and have a significant climate impact (IPCC 2013). Among several instruments, OMI is widely used for the measurement due to its higher resolution. Southeast Asian countries are the highest coal consumption countries of the world and China/India is on top among them (Li et al. 2017a, b). Studies reported a decreasing trend of coal consumption during last two decade due to several strict and effective environmental policies (e.g., Sulfuric acid industrial pollution prevention and control technology policy) implemented from the central and state government resulting in the decreasing trend of associated air pollutants in China (Li et al. 2017a, b). While on the other hand, an increasing trend of coal consumption and fossil fuel by India has triggered severe air pollution every year which causes millions of premature deaths (Chowdhury and Dey 2016). The change in SO_2 loading measured from OMI from 2005 to 2016 over India and China is shown in Fig. 2.12. Figure clearly reflects an enhancement in SO_2 from 2005 to 2016 over Indian region while vice versa for China. In 2005, SO_2 has a higher value over entire Northern China Plain with hot spots having value >2 DU over Hebei and Henan, mainly because of numerous power plants and cement industries (Fig. 2.12a). A deduction in SO_2 (<1 Du) can easily be seen in 2016 over China because of different policies and legislative environmental laws. On the other hand, in 2005 the isolated hot spots of SO_2 in India are near the coal power plants in Odisha, Jharkhand, Chhattisgarh (Fig. 2.12a) with value 0.5 DU which extends over

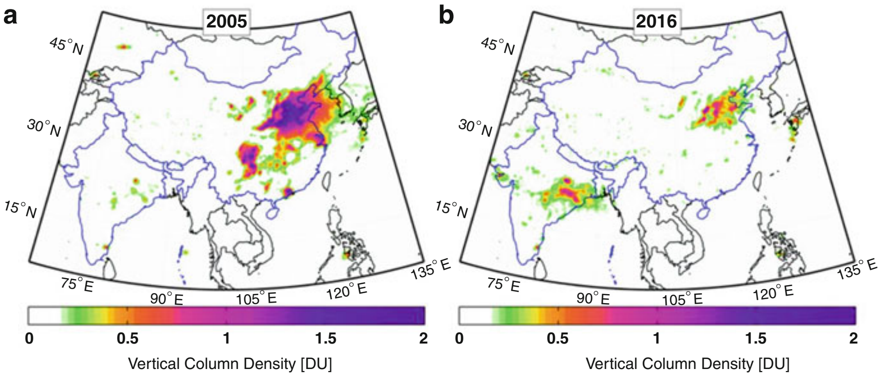


Fig. 2.12 (a) Spatial variability of average SO_2 vertical column amounts over India and China for 2005 (**b**) showing a significant increase and decrease of SO_2 over India and China (adopted by Li et al. 2017a)

another region due to emission from the new power plants. The western part of India (mainly near Jamnagar) had few new hot spots of SO_2 and can also easily see where the expansion of a large oil refinery and construction of the largest power plant in India took place during 2008–2012.

2.3.5.6 Measurement of Methane

Methane is the second most important anthropogenic greenhouse gas after CO_2 which has positive radiative forcing 0.97 Wm^{-2} (Myhre et al. 2013; IPCC 2013). Methane is mainly emitted from the industrial emission of oil and gas industry, coal mining, crop residue burning (mainly paddy residue burning) and wetlands (Jain et al. 2014). The major sink of methane is oxidation by the hydroxyl radical imposing atmospheric larger lifetime (about 10 years) (IPCC 2013). It can also impact the oxidative capacity of the atmosphere and stratospheric water vapor. The global burden of atmospheric methane has been increasing over the past decade (Turner et al. 2019) and about 8% of total global CH_4 emission occurs from South Asia (Chandra et al. 2017). The spatial variation of methane over the Indian subcontinent obtained from the Atmospheric Infrared Sounder (AIRS) measurement is shown in Fig. 2.13. AIRS is a complex high spectral resolution scanning radiometer board on NASA's Aqua satellite. It is nadir viewing having 2387 channels covering wavelength 3.74–15.4 μm and CH_4 are retrieved at near channel 7.6 μm. A brief description of different technical aspects retrieval methodology is discussed elsewhere (Xiong et al. 2010). Figure 2.13 shows the AIRS daytime spatial variation of 10 years (2005–2015) mean of CH_4 for the October month over Indian region. A higher concentration of CH_4 is observed over the IGB than the rest part of India, with a positive gradient from western to eastern IGB, which may be mainly due to the crop residue burning which injects a huge amount fine particulate and trace gases

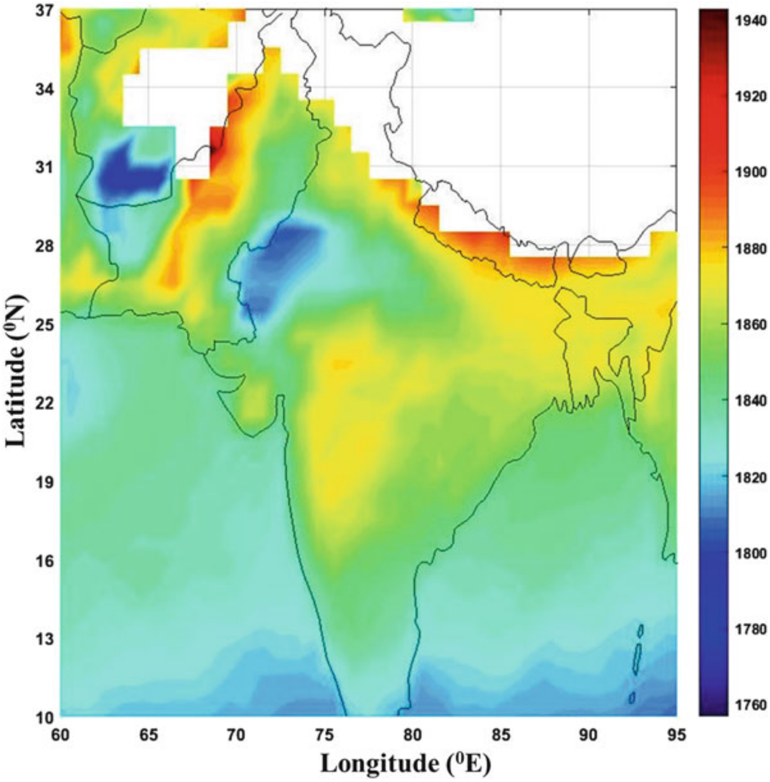


Fig. 2.13 Spatial variation of mean CH_4 (in ppbv) for October month from 2005 to 2015 at 850 hPa over the Indian subcontinent. White patches represent the unavailability of data

into the atmosphere. Earlier studies also found a higher concentration of methane over the north part of the Indian subcontinent (Chandra et al. 2017).

2.4 Conclusions

Air pollution is the most critical issue among the scientific community of the world. It has detrimental effects on human health, climate as well as agricultural production. Many instruments are developed based on different methods which have the ability to measure the air pollutants accurately at very high time resolution. Remote sensing techniques are also a cost-effective tool for the measurement of air pollution from both grounds as well as space. They have the ability to measure air pollution at high resolution and transportation. The main objective of the present chapter is to provide a brief description of the state-of-art view of instruments widely used in air pollution measurements. In this chapter, various types of air pollutants, their impacts and emission sources are discussed. Several studies have been carried out for air pollution measurement using different measurement methods worldwide. Although, a number of satellites are launched for the air pollution measurement from space yet there are significant uncertainties are still remaining which are mainly due to the different constraints in retrieval algorithm and spaceborne sensor. Thus, the improvement in satellite retrieval algorithm and spaceborne sensors are highly needed which will provide quality-assured data and enhance the database of air pollutants on a global scale which is helpful for air quality forecasting and future climate change using various climate model. These quality-assured data sets of air pollutants will be also helpful for the policymakers to improve the emission control policies and environmental legislative laws.

Acknowledgments We are thankful to the MODIS, OMI, AIRS, and AERONET NASA scientific research team for providing air pollution data. Author Shani Tiwari is also thankful to the Shandong University, China for the financial support under the International Postdoctoral Exchange Program.

References

Alam K, Trautmann T, Blaschke T, Subhan F (2014) Changes in aerosol optical properties due to dust storms in the Middle East and Southwest Asia. Remote Sens Environ 143:216–227

Badarinath KVS, Kharol SK, Sharma AR, Prasad VK (2009) Analysis of aerosol and carbon monoxide characteristics over Arabian Sea during crop residue burning period in the Indo-Gangetic Plains using multi-satellite remote sensing datasets. J Atmos Sol Terr Phys 71 (2009):1267–1276

Benedict KB, Zhou Y, Sive BC, Prenni AJ, Gebhart KA, Fischer EV, Evanoski-Cole A, Sullivan AP, Callahan S, Schichtel BA, Mao H, Zhou Y, Collett JL Jr (2019) Volatile organic compounds and ozone in Rocky Mountain National Park during FRAPPÉ. Atmos Chem Phys 19:499–521

Bibi S, Alam K, Chishtie F, Bibi H, Rahman S (2017) Observations of black carbon aerosols characteristics over an urban environment: radiative forcing and related implications. Sci Total Environ 603–604:319–329

Boersma KF, Eskes HJ, Dirksen RJ, van der RJ A, Veefkind JP et al (2011) An improved tropospheric NO2 column retrieval algorithm for the ozone monitoring instrument. Atmos Meas Tech 4:1905–1928

Boiyo R, Kumar KR, Zhao T, Guo J (2019) A 10 year record of aerosol optical properties and radiative forcing over three environmentally distinct AERONET sites in Kenya, East Africa. J Geophys Res. https://doi.org/10.1029/2018JD029461

Burney J, Ramanathan V (2014) Recent climate and air pollution impacts on Indian agriculture. PNAS 111(46):16319–16324

Burrows JP, Weber M, Buchwitz M, Rozanov V, Ladstätter-Weißenmayer A, Richter A, DeBeek R, Hoogen R, Klaus Bramstedt K, Eichmann KU, Eisinger M, Perner D (1999) The Global Ozone Monitoring Experiment (GOME): mission concept and first scientific results. J Am Meteorol Soc. https://doi.org/10.1175/1520-0469(1999)056<0151:TGOMEG>2.0.CO;2

Chandra N, Hayashida S, Saeki T, Patra PK (2017) What controls the seasonal cycle of columnar methane observed by GOSAT over different regions in India? Atmos Chem Phys 17:12633–12643

Cheng MM, Jiang H, Guo Z (2012) Evaluation of long-term tropospheric NO2 columns and the effect of different ecosystem in Yangtze River Delta. Procedia Environ Sci 13:1045–1056

Cheng N, Li Y, Sun F, Chen C, Wang B, Li Q, Wei P, Cheng B (2018) Ground – level NO2 in urban Beijing: trends, distribution, and effects of emission reduction measures. Aerosol Air Qual Res 18:343–356

Chiang TY, Yuan TH, Shie RH, Chen CF, Chan CC (2016) Increased incidence of allergic rhinitis, bronchitis and asthma, in children living near a petrochemical complex with SO2 pollution. Environ Int 96:1–7

Chowdhury S, Dey S (2016) Cause-specific premature death from ambient PM2.5 exposure in India: estimate adjusted for baseline mortality. Environ Int 91:283–290

Deeter MN et al (2003) Operational carbon monoxide retrieval algorithm and selected results for the MOPITT instrument J. Geophys Res Atmos 108:4399

Deeter MN, Edwards DP, Francis GL, Gille JC, Martinez-Alonso S, Worden HM, Sweeney C (2017) A climate-scale satellite record for carbon monoxide: the MOPITT Version 7 product Atmos. Meas Tech 10:2533–2555

Deng Q, Lu C, Jiang W, Zhao J, Deng L, Xiang Y (2017) Association of outdoor air pollution and indoor renovation with early childhood ear infection in China. Chemosphere 169:288–296

Dey S, Di Girolamo L (2010) A climatology of aerosol optical and microphysical properties over the Indian subcontinent from 9 years (2000–2008) of multiangle imaging spectroradiometer (MISR) data. J Geophys Res 115:D15204. https://doi.org/10.1029/2009JD013395

Drummond JR (1992) Measurements of pollution in the troposphere (MOPITT). In: Gille JC, Visconti G (eds) The Use of EOS for Studies of Atmospheric Physics. North-Holland, New York, pp 77–101

Duncan BN, Lamsal LN, Thompson AM, Yoshida Y, Lu Z, Streets DG, Pickering KE (2016) A space-based, high-resolution view of notable changes in urban NOx pollution around the world (2005–2014). J Geophys Res Atmos 121:976–996

Eastham SD, Barrett SRH (2016) Aviation-attributable ozone as a driver for changes in mortality related to air quality and skin cancer. Atmos Environ 144:17–23

Edwards DP, Halvorson C, Gille JC (1999) Radiative transfer modeling for the EOS Terra Satellite Measurement of Pollution in the Troposphere (MOPITT) instrument. J Geophys Res 104:16755

EPA (2012) Our Nation's Air: Status and Trends Through 2010. Tech. rep., Environmental Protection Agency, EPA-454/R-12001

Fan J, Wang Y, Rosenfeld D, Liu X (2016) Review of aerosol-cloud interactions: mechanisms, significance and challenges. J Atmos Sci. https://doi.org/10.1175/JAS-D-16-0037.1

Finney DL, Doherty RM, Wild O, Young PJ, Butler A (2016) Response of lightning NOx emissions and ozone production to climate change: insights from the atmospheric chemistry and climate model intercomparison project. Geophys Res Lett 43:5492–5500

Gautam R, Hsu NC, Lau WK-M, Yasunari TJ (2013) Satellite observations of desert dust-induced Himalayan snow darkening. Geophys Res Lett 40:988–993

Ghude SD, Chate DM, Jena C, Beig G, Kumar R, Barth MC, Pfister GG, Fadnavis S, Pithani P (2016) Premature mortality in India due to PM2.5 and ozone exposure. Geophys Res Lett 43:4650–4658

Haslett SL, Taylor JW, Deetz K, Vogel B, Babić K, Kalthoff N, Wieser A, Dione C, Lohou F, Brito J, Dupuy R, Schwarzenboeck A, Zieger P, Coe H (2019) The radiative impact of out-of-cloud aerosol hygroscopic growth during the summer monsoon in southern West Africa. Atmos Chem Phys 19:1505–1520

Hossaini R, Chipperfield MP, Montzka SA, Leeson AA, Dhomse SS, Pyle JJ (2017) The increasing threat to stratospheric ozone from dichloromethane. Nat Commun 8:15962. https://doi.org/10.1038/ncomms1596

Hsu SC, Tsai F, Lin FJ, Chen WN, Shiah FK, Huang JC et al (2013) A super Asian dust storm over the East and South China Seas: disproportionate dust deposition. J Geophys Res Atmos 118 (13):7169–7181

Hua SB, Tian HZ, Wang K, Zhu CY, Gao JJ, Ma YL et al (2016) Atmospheric emission inventory of hazardous air pollutants from China's cement plants: temporal trends, spatial variation characteristics and scenario projections. Atmos Environ 128:1–9

IPCC (2013) Fifth Assessment Report: Climate Change 2013. Cambridge University Press, New York, NY

Inness A, Blechschmidt A-M, Bouarar I, Chabrillat S, Cre-pulja M, Engelen RJ, Eskes H, Flemming J, Gaudel A, Hendrick F, Huijnen V, Jones L, Kapsomenakis J, Katragkou E, Keppens A, Langerock B, de Mazière M, Melas D, Parrington M, Peuch VH, Razinger M, Richter A, Schultz MG, Suttie M, Thouret V, Vrekoussis M, Wagner A, Zerefos C (2015) Data assimilation of satellite retrieved ozone, carbon monoxide and nitrogen dioxide with ECMWF's composition-IFS. Atmos Chem Phys 15:5275–5303

Jain N, Bhatia A, Pathak H (2014) Emission of air pollutants from crop residue burning in India. Aerosol Air Qual Res 14:422–430

Kampa M, Castanas E (2008) Human health effects of air pollution. Environ Pollut 151(2):362–367

Kaskaoutis DG, Singh RP, Gautam G, Sharma M, Kosmopoulos PG, Tripathi SN (2012) Variability and trends of aerosol properties over Kanpur, northern India using AERONET data (2001–10). Environ Res Lett 7(2012):024003. (9 pp)

Krotkov NA, McLinden CA, Li C, Lamsal LN, Celarier EA, Marchenko SV, Swartz WH, Bucsela EJ, Joiner J, Duncan BN, Boersma KF, Veefkind JP, Levelt PF, Fioletov VE, Dickerson RR, He H, Lu Z, Streets DG (2016) Aura OMI observations of regional SO2 and NO2 pollution changes from 2005 to 2015. Atmos Chem Phys 16:4605–4629

Kumar KR, Yin Y, Sivakumar V, Kang N, Yu X, Diao Y, Adesina AJ, Reddy RR (2015) Aerosol climatology and discrimination of aerosol types retrieved from MODIS, MISR and OMI over Durban (29.88°S, 31.02°E), South Africa. Atmos Environ 117:9–18

Lamsal LN, Duncan BN, Yoshida Y, Krotkov NA, Pickering KE, Streets DG, Lu Z (2015) U.S. NO2 trends (2005–2013): EPA air quality system (AQS) data versus improved observations from the ozone monitoring instrument (OMI). Atmos Environ 110:130–143

Lau KM, Kim MK, Kim KM (2006) Asian summer monsoon anomalies induced direct forcing: the role of the Tibetan plateau. Clim Dyn 26(7–8):855–864

Lelieveld J, Evans JS, Fnais M, Giannadaki D, Pozzer A (2015) The contribution of outdoor air pollution sources to premature mortality on a global scale. Nature 525:367–371

Levelt PF, van den Oord GHJ, Dobber MR, Malkki A, Huib V, de Johan V, Stammes P, Lundell JOV, Saari H (2006) The ozone monitoring instrument. IEEE Trans Geosci Remote Sens 44:1093–1101

Levy RC, Remer LA, Kleidman RG, Mattoo S, Ichoku C, Kahn R, Eck TF (2010) Global evaluation of the Collection 5 MODIS dark-target aerosol products over land. Atmos Chem Phys 10:10399–10420

Li Y, Meng J, Liu JF, Xu Y, Guan DB, Tao W, Huang Y, Tao S (2016) Interprovincial reliance for improving air quality in China: a case study on black carbon aerosol. Environ Sci Technol 50:4118–4126

Li C, McLinden C, Fioletov V, Krotkov N, Carn S, Joiner J, Streets D, He H, Ren X, Li Z, Dickerson RR (2017a) India is overtaking china as the world's largest emitter of anthropogenic sulfur dioxide. Sci Rep 7:14304. https://doi.org/10.1038/s41598-017-14639-8

Li R, Cui L, Li J, Zhao A, Fu H, Wu Y, Zhang L, Kong J, Chen J (2017b) Spatial and temporal variation of particulate matter and gaseous pollutants in China during 2014–2016. Atmos Environ 161:235–246

Lindner S, Liu Z, Guan D, Geng Y, Li X (2013) CO2 emissions from China's power sector at the provincial level: consumption versus production perspectives. Renew Sust Energy Rev 19:164–172

Liu JC, Peng RD (2018) Health effect of mixtures of ozone, nitrogen dioxide, and fine particulates in 85 US counties. Air Qual Atmos Health. https://doi.org/10.1007/s11869-017-0544-2

Liu F, Beirle S, Zhang Q, van der A RJ, Zheng B, Tong D, He K (2017) NOx emission trends over Chinese cities estimated from OMI observations during 2005 to 2015. Atmos Chem Phys 17:9261–9275

Mahapatra PS, Sinha PR, Boopathy R, Das T, Mohanty S, Sahu SC, Gurjar BR (2018) Seasonal progression of atmospheric particulate matter over an urban coastal region in peninsular India: role of local meteorology and long-range transport. Atmos Res 199:145–158

Mahowald N (2011) Aerosol indirect effect on biogeochemical cycles and climate. Science. https://doi.org/10.1126/science.1207374

Maji KJ, Ye WF, Arora M, Nagendra SMS (2019) Ozone pollution in Chinese cities: assessment of seasonal variation, health effects and economic burden. Environ Pollut 247:792–801

Malley CS, Kuylenstierna JCI, Vallack HW, Henze DK, Blencowe H, Ashmore MR (2017) Preterm birth associated with maternal fine particulate matter exposure: a global, regional and national assessment. Environ Int 101:173–182

Mao KB, Ma Y, Xia L, Chen WY, Shen XY, He TJ, Xu TR (2014) Global aerosol change in the last decade: an analysis based on MODIS data. Atmos Environ 94:680–686

McLinden CA, Fioletov V, Krotkov NA, Li C, Boersma KF, Adams C (2016) A decade of change in NO2 and SO2 over the Canadian oil sands as seen from space. Environ Sci Technol 50 (1):331–337

Mishra AK, Shibata T (2012) Synergistic analyses of optical and microphysical properties of agricultural crop residue burning aerosols over the Indo-Gangetic Basin (IGB). Atmos Environ 57:205–218

Moorthy KK, Babu SS, Manoj MR, Satheesh SK (2013) Buildup of aerosols over the Indian region. Geophys Res Lett 40:1011–1014. https://doi.org/10.1002/grl.50165

Myhre G, Shindell D, Bréon F-M, Collins W, Fuglestvedt J, Huang J, Koch D, Lamarque J-F, Lee D, Mendoza B, Nakajima T, Robock A, Stephens G, Takemura T, Zhang H (2013) Anthropogenic and natural radiative forcing. In: Stocker TF, Qin D, Plattner G-K, Tignor M, Allen SK, Boschung J, Nauels A, Xia Y, Bex V, Midgley PM (eds) Climate Change 2013. The Physical Science Basis. Contribution of Working Group I to the Fifth Assessment Report of the Intergovernmental Panel on Climate Change. Cambridge University Press, Cambridge, pp 659–740. https://doi.org/10.1017/CBO9781107415324.018

Nieuwenhuijsen MJ, Basagaña X, Dadvand P, Martinez D, Cirach M, Beelen R, Jacquemin B (2014) Air pollution and human fertility rates. Environ Int 17:09–14

Prijith SS, Rajeev K, Thampi BV, Nair SK, Mohan M (2013) Multi-year observations of the spatial and vertical distribution of aerosols and the genesis of abnormal variations in aerosol loading over the Arabian Sea during Asian summer monsoon season. J Atmos Sol Terr Phys 105–106:142–151

Ramanathan V, Crutzen PJ, Kiehl JT, Rosenfeld D (2001) Aerosols, climate, and the hydrological cycle. Science 294:2119–2124. https://doi.org/10.1126/science.1064034

Remer LA, Kleidman RG, Levy RC, Kaufman YJ, Tanre D, Mattoo S, Martins JV, Ichoku C, Koren I, Yu H, Holben BN (2008) Global aerosol climatology from the MODIS satellite sensors. J Geophys Res 113:D14S07. https://doi.org/10.1029/2007JD009661

Rupakheti D, Kang S, Rupakheti M, Cong Z, Panday AK, Holben BN (2019) Identification of absorbing aerosol types at a site in the northern edge of Indo-Gangetic Plain and a polluted valley in the foothills of the central Himalaya. Atmos Res 223:15–23

Sarkar S, Singh RP, Chauhan A (2018) Crop residue burning in Northern India: increasing threat to Greater India. J Geophys Res Atmos 123:6920–6934. https://doi.org/10.1029/2018JD028428

Saxena P, Naik V (eds) (2018) Air pollution: sources, impacts and controls. CABI

Saxena P, Sonwani S (2019a) Primary criteria air pollutants: environmental health effects, Criteria air pollutants and their impact on environmental health. Springer, Singapore, pp 49–82

Saxena P, Sonwani S (2019b) Policy regulations and future recommendations, Criteria air pollutants and their impact on environmental health. Springer, Singapore, pp 127–157

Saxena P, Sonwani S (2019c) Criteria air pollutants: chemistry, sources and sinks, Criteria air pollutants and their impact on environmental health. Springer, Singapore, pp 7–48

Saxena P, Sonwani S (2020) Criteria air pollutants and their impact on environmental health. Springer, Singapore

Saygın M, Gonca T, Öztürk Ö, Has M, Çalışkan S, Has ZG, Akkaya A (2017) To investigate the effects of air pollution (PM10 and SO2) on the respiratory diseases asthma and chronic obstructive pulmonary disease. Turk ToraksDergisi 18(2):33–39

Schultz MG, Schröder S, Lyapina O, Cooper OR, Galbally I, Petropavlovskikh I, et al. (2017) Tropospheric ozone assessment report: database and metrics data of global surface ozone observations. Elementa: Science of the Anthropocene 5

Seinfeld JH, Pandis SN (2010) Atmospheric chemistry and physics: from air pollution to climate change, 2nd edn. Wiley, New York

Seinfeld JH et al (2016) Improving our fundamental understanding of the role of aerosol−cloud interactions in the climate system. PNAS 113(21):5781–5790

Singh A, Tiwari S, Sharma D, Singh D, Tiwari S, Srivastava AK, Rastogi N, Singh AK (2016) Characterization and radiative impact of dust aerosols over northwestern part of India: a case study during a severe dust storm. Meteorog Atmos Phys 128(6):779–792

Sonwani S, Kulshreshtha U (2016) Particulate matter levels and it's associated health risks in East Delhi. Proceedings of Indian aerosol science and technology association conference on aerosol and climate change: insight and challenges. IASTA Bull 22(1–2). ISSN 09714510

Sonwani S, Saxena P, Kulshrestha U (2016) Role of global warming and plant signaling in BVOC emissions. In: plant responses to air pollution. Springer, Singapore, pp 45–57

Streets DG, Canty T, Carmichael GR, de Foy B, Dickerson RR, Duncan BN, Edwards DP, Haynes JA, Henze DK, Houyoux MR, Jacob DJ, Krotkov NA, Lamsal LN, Liu Y, Lu Z, Martin RV, Pfister GG, Pinder RW, Salawitch RJ, Wecht KJ (2013) Emissions estimation from satellite retrievals: a review of current capability. Atmos Environ 77:1011–1042

Tian HZ, Liu KY, Hao JM, Wang Y, Gao JJ, Qiu PP, Zhu CY (2013) Nitrogen oxides emissions from thermal power plants in China: current status and future predictions. Environ Sci Technol 47:11350–11357

Tiwari S, Singh AK (2013) Variability of aerosol parameters derived from ground and satellite measurements over Varanasi located in Indo Gangetic Basin. Aerosol Air Qual Res 13:627–638

Tiwari S, Srivastava AK, Singh AK (2013) Heterogeneity in pre-monsoon aerosol characteristics over the Indo-Gangetic Basin. Atmos Environ 77:738–747

Tiwari S, Srivastava AK, Singh AK, Singh S (2015) Identification of aerosol types over Indo-Gangetic Basin: implications to optical properties and associated radiative forcing. Environ Sci Pollut Res. https://doi.org/10.1007/s11356-015-4495-6

Tiwari S, Mishra AK, Singh AK (2016) Aerosol climatology over the Bay of Bengal and Arabian Sea inferred from space-borne radiometers and lidar observations. Aerosol Air Qual Res. https://doi.org/10.4209/aaqr.2015.06.0406

Tiwari S, Kaskaoutis D, Soni VK, Attri SD, Singh AK (2018) Aerosol columnar characteristics and their heterogeneous nature over Varanasi, in the central Ganges valley. Environ Sci Pollut Res 25(25):24726–24745

Turner AJ, Frankenberg C, Kort EA (2019) Interpreting contemporary trends in atmospheric methane. PNAS 116(8):2805–2813

U.S. CAR (U.S. Climate Action Report) (2010) U.S. Department of State. Global Publishing Services, Washington, DC

Valverde V, Pay MT, Baldasano JM (2016) A model-based analysis of SO2 and NO2 dynamics from coal-fired power plants under representative synoptic circulation types over the Iberian Peninsula. Sci Total Environ 541:701–713

Vet R, Artz RS, Carou S, Shaw M, Ro C-U, Aas W, Baker A, Bowersox VC, Dentener F, Galy-Lacaux C, Hou A, Pienaar JJ, Gillett R, Forti MC, Gromov S, Hara H, Khodzher T, Mahowald NM, Nickovic S, Rao PSP, Reid NW (2014) A global assessment of precipitation chemistry and deposition of sulfur, nitrogen, sea salt, base cations, organic acids, acidity and pH, and phosphorus. Atmos Environ 93:3–100

Vinoj V, Rasch PJ, Wang H, Yoon J-H, Ma P-L, Landu K, Singh B (2014) Short-term modulation of Indian summer monsoon rainfall by West Asian dust. Nat Geosci 7:308–313

Werner R, Valev D, Atanassov A, Guineva V, Kirillov A (2013) Analysis of variations and trends of the NO2 slant column abundance obtained by DOAS measurements at Stara Zagora and at NDACC European mid–latitude stations in comparison with subtropical stations. J Atmos Sol Terr Phys 99:134–142

Worden HM et al (2013) Decadal record of satellite carbon monoxide observations. Atmos Chem Phys 13:837–850

World Bank (2016). http://www.worldbank.org/en/news/press-release/2016/09/08/air-pollution-deaths-cost-global-economy-225-billion

World Health Organization (2018). www.who.int/airpollution/data

Xia X, Che H, Zhu J, Chen H, Cong Z, Deng X, Fan X, Fu Y, Goloub P, Jiang H, Liu Q, Mai B, Wang P, Wu Y, Zhang J, Zhang R, Zhang X (2016) Ground-based remote sensing of aerosol climatology in China: aerosol optical properties, direct radiative effect and its parameterization. Atmos Environ 124(B):243–251

Xiong X, Barnet C, Maddy E, Wei J, Liu X, Zhou L, Pagano ST (2010) Seven years observation of mid-upper tropospheric methane from atmospheric infrared sounder. Remote Sens 2:2509–2530. https://doi.org/10.3390/rs2112509

Yue X, Unger N (2018) Fire air pollution reduces global terrestrial productivity. Nat Commun 9(1). https://doi.org/10.1038/s41467-018-07921-4

Zhang Z, Wang X, Zhang Y, Lü S, Huang Z, Huang X, Wang Y (2015) Ambient air benzene at background sites in China's most developed coastal regions: exposure levels, source implications and health risks. Sci Total Environ 511:792–800

Zheng B, Chevallier F, Ciais P, Yin Y, Deeter MN, Worden HM, Wang Y, Zhang Q, He K (2018) Rapid decline in carbon monoxide emissions and export from East Asia between years 2005 and 2016

Zhu J, Penner JE, Yu F, Sillman S, Andreae MO, Coe H (2019) Decrease in radiative forcing by organic aerosol nucleation, climate, and land use change. Nat Commun 10:423. https://doi.org/10.1038/s41467-019-08407-7

Chapter 3
Air Pollution Modeling

Ravi Yadav, Pujal Trivedi, L. K. Sahu, G. Beig, and Nidhi Tripathi

Abstract Since over the mid of the last century, anthropogenic emissions of trace gases and aerosols are increasing because of the competitive rapid economic growth, industrial development, and emissions from biomass burning and fossil fuel combustion all over the world. These are the major causes for global environment changes including the global spread of air pollution, increase in the concentrations of tropospheric oxidants, global warming, and typical seasonal variability. To understand the major causes of air pollution-related issues, several platforms have been used including in situ measurements of trace gases and aerosol, emission inventories datasets, satellite observations, regional, local and global chemical transport models. This chapter discusses air pollution in terms of advancement of the modeling performance. In addition to this, the section includes the major scientific outputs of air pollution using measurements and modeling activities all over the world. The chapter concludes with a focus on air pollution research activities and policy questions in the next upcoming years that will be helpful for advance understanding of air pollution research activities as well as setting up the reference for the environmental policies all over the world.

Keywords Air quality · Atmosphere · Trace gases · Aerosols · Air pollution modeling

---

R. Yadav (✉) · G. Beig
Atmospheric Pollution and Transport Modeling Division, Indian Institute of Tropical Meteorology, Pune, India
e-mail: ravi.yadav@tropmet.res.in

P. Trivedi
Intel Corporation, Folsom, CA, USA
e-mail: pujal.jayesh.j.trivedi@intel.com

L. K. Sahu · N. Tripathi
Space and Atmospheric Science Division, Physical Research Laboratory, Ahmedabad, India

P. Saxena, A. Srivastava (eds.), *Air Pollution and Environmental Health*,
Environmental Chemistry for a Sustainable World 20,
https://doi.org/10.1007/978-981-15-3481-2_3

3.1 Introduction

> Fresh ambient air is considered to be an imperative necessity for human health. Still, air pollution continues to cause a significant threat to health worldwide (WHO 2005).

Present atmosphere is quite unlike from the natural atmosphere that existed before the Industrial Revolution in terms of chemical composition (Fig. 3.1). If the natural atmosphere is considered to be "clean," then this means that clean air cannot be found anywhere in today's atmosphere. Human activities have significantly impacted climate change and major environmental problems from the point the Industrial Revolution began around 1750 (WHO 2016; Maji et al. 2020; Saxena and Ghosh 2017).

World's population was about of 6.5 billion in 2005 and which is expected to drastically increase to 9.1 billion in 2050 (http://esa.un.org/unpd/wup/index.htm). The growth in the total population is paralleled by an expansion in the level of the world's residents, who are living in urban regions. Starting 2008, over a large portion of the total population (~50%) lives in urban regions which is greater than in 1950 (~30%) (See Fig. 3.2). By 2030, the proportion of the population living in urban areas is expected to be over 50% in all major geographical areas of the world (Fig. 3.3).

In recent years, urban population fractions are higher in developed than developing countries (Montgomery 2008). Dramatic increase in population and urbanization, especially in the developing world, has been accompanied by technological and economic growth and development, yielding changes in land use, energy use, and transportation. The resulting changes due to urbanization have dramatic impacts on anthropogenic and biogenic emissions and have notably altered local to global-scale atmospheric composition, increasing the importance of understanding the impacts of urbanization on atmospheric chemistry (Yadav et al. 2019a; Wang et al. 2017; Sahu et al. 2017; Sonwani and Kulshrestha 2018).

Typically, large portion of population is mainly busy with residential and industrial activities (for example: combustions of fossil fuels (vehicle exhaust, heat generation and industrial processes, gasoline, petrochemical industries, use of biofuels, liquefied petroleum gas for cooking purpose, and power plants, etc.)

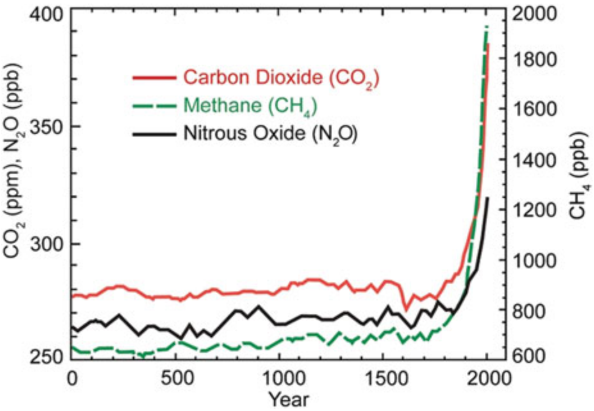


Fig. 3.1 Increase in the mixing ratios of atmospheric gases in the atmosphere over the last 2000 years (Source: IPCC Fourth Assessment Report—2007)

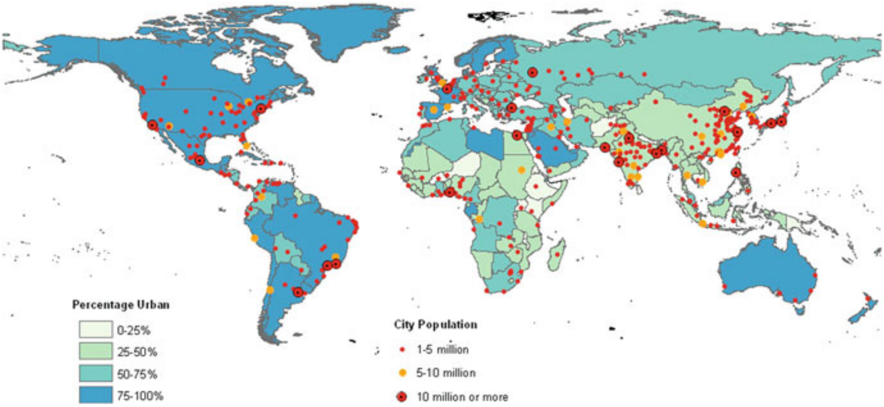


Fig. 3.2 Urban agglomeration in 2009 (urban proportion of the world population: 50.1%) (Source: UN Department of Economic and Social Affairs, 2010)

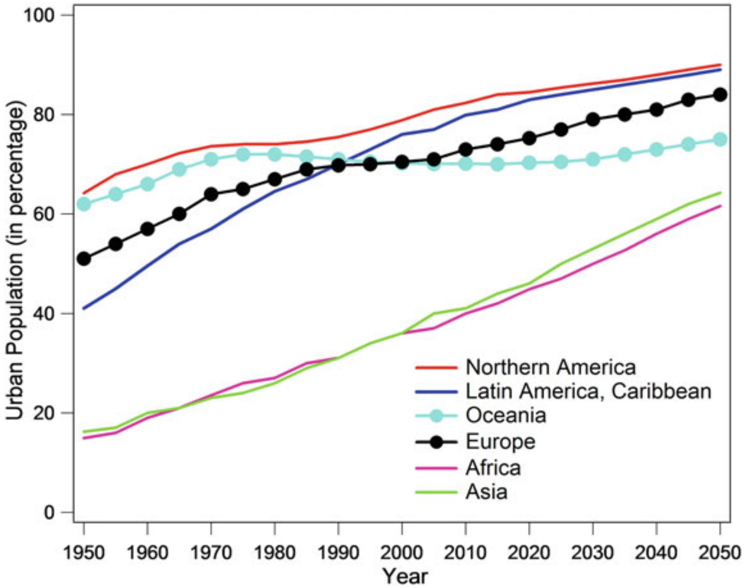


Fig. 3.3 Urban population by major geographical area (in percent of total population) (UN Department of Economic and Social Affairs, 2010)

(Simayi et al. 2020; Baudic et al. 2016; Sahu et al. 2011; Sahu and Lal 2006; Sonwani and Saxena 2016). These activities may enhance the emissions of long-lived greenhouse gases such as carbon dioxide (CO_2), nitrous oxide (N_2O), etc. and short-lived trace gases (for example: carbon monoxide (CO), methane (CH_4), volatile organic compounds (NMVOCs), nitrogen oxides (NOx) etc.) along with short-

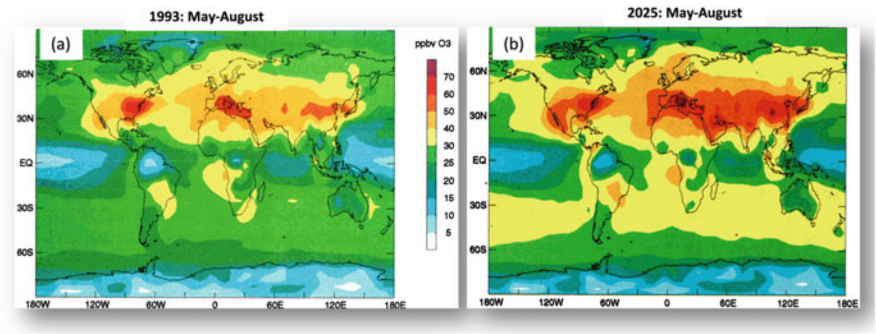


Fig. 3.4 (**a**) Global distribution of surface ozone concentrations based on the global three-dimensional chemistry-transport model (May–August 1993). (**b**) Modeling predication on geographical distribution of surface ozone concentrations in 2025 (mean from May to August). Figure adapted from Lelieveld and Dentener (2000)

lived aerosol particles such as black carbon, particulate matters, and dust (Beig et al. 2020; Mozaffar et al. 2020; Yadav et al. 2014a, b, c, 2017; Sahu and Saxena 2015; Jaffe et al. 2004; Karl et al. 2007).

It is well known that the photochemical reactions of short-lived trace gases in the presence of sunlight may lead to the production of surface ozone (O_3) and secondary organic aerosol (SOA) in the atmosphere (Seinfeld and Pandis 2006; Yadav et al. 2016; Sahu et al. 2016b; Saxena and Kulshrestha 2016; Fuzzi et al. 2015). In the troposphere, O_3 acts as a greenhouse gas which contributes to the global radiative forcing by +0.35 W m^{-2} because it absorbs radiation at 9.6 μm wavelength emitted from the Earth's surface (Akimoto 2003; Solomon et al. 2007). In addition to trapping heat, ground-level O_3 is a secondary pollutant that can cause respiratory health problems and damage crops and ecosystems (Cooper et al. 2010; Saxena et al. 2019).

In addition to this, the downdraft transport of O_3 from the stratosphere is another source of tropospheric O_3 which is mainly active in summer. Meanwhile, O_3 disappears due to the photochemical reactions with HOx radicals over the ocean, where NOx levels are usually low, and through decomposition by contact with the ground (Saxena et al. 2017). The amount of ozone due to photochemical reactions in the troposphere is much greater than the influx from the stratosphere, although the global mean net O_3 supplied by both sources is about the same, after counting the amount removed by decomposition in the troposphere. For example, the distributions of ozone mixing ratios at surface using 3D-chemical transport modeling (3D-CTM) are shown in Fig. 3.4. It shows that the mixing ratios of ozone were high in middle and high latitudes of the northern hemisphere, especially in regions where the emissions of air pollutants from human activities are large in volume.

Surface O_3 mixing ratios have increased 4–5 times the levels in preindustrial era. However, even if ozone concentrations were as high as 20 ppb at the time, recent mixing ratios of O_3 at remote and rural sites in the northern hemisphere have at least doubled since the preindustrial era. If human activities continue to cause emissions of pollutants that generate O_3, what will happen to ground level O_3 in the future?

Figure 3.4b is one example of modeling predication for surface O_3. It shows that the average mixing ratios of O_3 in North America, Europe, and East Asia, including Japan, from May to August will likely exceed 60 ppb. The atmosphere is a very complex reactive system in which various physical and chemical forms happen at the same time. To address the rapidly growing air pollution problem in World, it is very important to understand the in situ and satellite-based observations with chemical transport models and many efforts have been made in the past several years to understand the air quality research and the control of atmospheric composition. The combination of measurement and model sciences is a good approach for making real progress towards understanding the atmosphere. Here, we provide a brief overview of modeling which provides the vital framework for interactions.

3.1.1 Components of Air Pollution Modeling

Emission inventory, transport, physicochemical transformations, removal processes, and meteorology are the key components to develop air quality models. A flowchart of various components of a comprehensive atmospheric chemistry transport model (CTM) is shown in Fig. 3.5.

3.1.1.1 Inventory of Sources and Emissions

The air pollutants are emitted within the urban or regional region and source apportionment for the same are generally depicted by air pollution modeling and with the use of emission inventory. In urban regions, a wide range of emission sources such as fossil fuel, biofuel, power plants, and other industrial facilities are found which are taking into account while making emission inventory of that particular area (Yadav et al. 2016, 2019b; Sahu et al. 2016a; Beig et al. 2013).

Still, at receptor, the contributions of each source to the total concentration are summative, which is indirect type of modeling and is debatable when air pollutants from different sources may react with each other. As per the requirement, outflows from all few sources in every matrix region are frequently consolidated together into a larger zone resource, expecting that emanations are uniform in a particular area. Because vehicular emissions establish a noteworthy bit of sources in urban area, their successful stature of emissions is thought to be at the ground level; even though, some ongoing exploration may propose something different. Large stack emissions are typically considered as point sources whose powerful emission statures are determined from meteorological parameters and its given sources, utilizing proper plume-rise recipes. The dispersion is calculated from all of the main point sources and concentrations which are the result of a receptor point that are added to the contribution from area sources. The quantity of point sources considered along these lines can shift from tens to thousands, contingent upon the size and modern

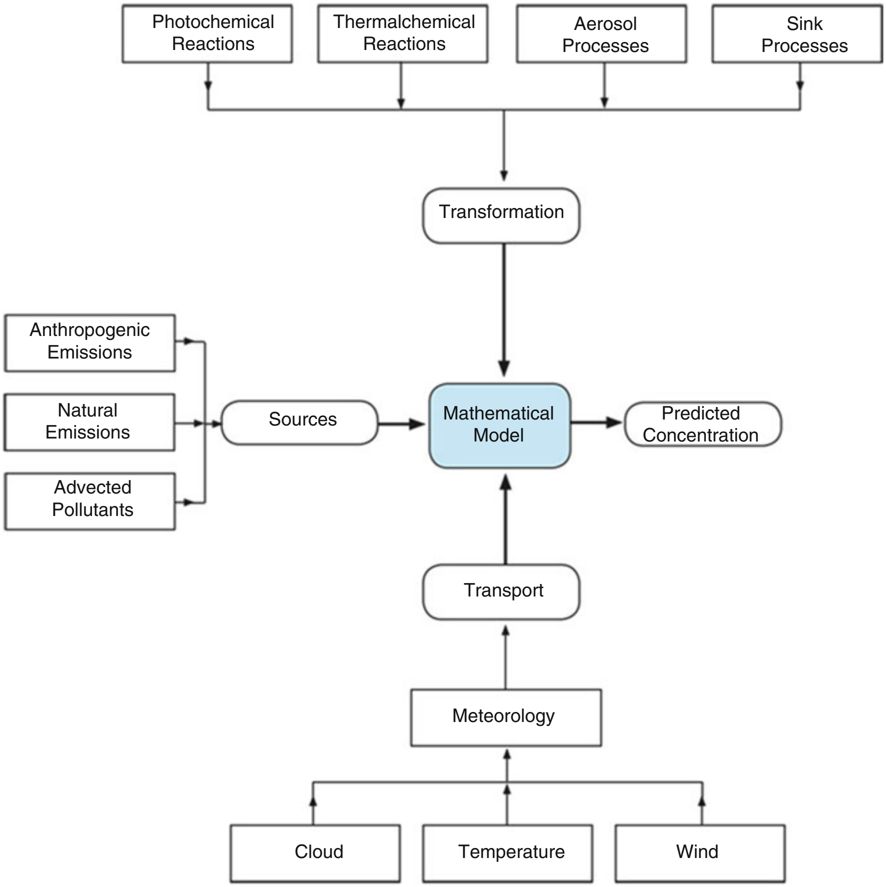


Fig. 3.5 Elements of a mathematical atmospheric chemical transport model (Source: Atmospheric Chemistry and Physics: From Air Pollution to Climate Change, Second Edition, by John H. Seinfeld and Spyros N. Pandis)

character of the urban region. The diurnal and seasonal variation of sources along with a perfect emissions inventory is also very prominent for air quality modeling.

3.1.1.2 Transport Circulation and Parameterization

In this section, two transport mechanisms are discussed which are important in conducting air pollution modeling namely, wind transport (the air pollutants are carried along with the time-averaged average wind called as advection process) and dispersion mechanism (transport due to the turbulence). Typically, atmospheric circulation (wind and turbulence) is frequently divided into four scales such as large, regional, local, and turbulence scales. For example, wind is fully responsible for the transport of the ambient compounds while turbulence resulted in mixing of constituents. Advection is the most effectively parameterized. The average time which determines the mean value of winds is identified with the spatial and temporal scales of the framework which is being modeled. Wind is the deterministic three-dimensional (3D) motion field which has time scales ranging from minutes to days and has spatial scales which range from kilometers to the planetary scale. Commonly, hourly or better midpoints of the winds are utilized. The turbulence describes the actually chaotic nature of fluid flow, which is manifested in the form of highly irregular, almost random, temporal, and spatial fluctuations in velocity, temperature, and scalar concentrations around their mean values, resulting from instability in a fluid at high Reynolds numbers. Reynolds number refers to the ratio of inertial forces to viscous forces. It is one of the most important dimensionless numbers in fluid dynamics. A low value refers to laminar flow and a high value refers to turbulent flow. It consists of Eddy motions having scales from a few millimeters up to perhaps a kilometer, and with time scales from about a second to 20 or 30 min. These parameters have special significance in determining the horizontal and vertical dispersion of pollutants. Turbulent fluxes are difficult to measure, and hence there are uncertainties in the various methods employed by models to describe them. K-theory is typically parameterized as being proportional to the gradient of mean mixing ratios. In this case the eddy diffusivity tensor, K, is determined as a function of atmospheric stability class and mixing height following some parameterizations. K-theory is valid only over short distances, and cannot simulate counter-gradient transport, which can be important in highly convective mixed layers.

In air quality models, statistical theories, gradient transport theories, and similarity theories are the most widely used descriptions of transport diffusion system. And the parameterization of horizontal and vertical diffusion is the significantly crucial component, usually combined with the empirical dispersion parameters obtained from tracer experiments over cities.

3.1.1.3 Chemical Transformations

One of the significant components of a photochemical model is atmospheric chemistry. In the atmosphere, there are a variety of pollutants which plays an important role in the chemical reaction. Moreover, these mechanisms are most important to know the dynamics of the ambient pollutants based on their reactivity. In the Earth's atmosphere, gas-phase chemistry and heterogeneous chemistry are most significant mainly for the formations of surface O_3, aerosol and acid deposition. In addition to this, aqueous-phase chemistry is significant for the formation of surface O_3 and sulfate in the presence of fog/clouds (Saxena and Kulshrestha 2015). Therefore, these types of chemical reactions are suitable for the formation of secondary air pollutants and are used in chemical-based models. The kinetic mechanisms of these chemical reactions are used in 3D numerical regional models.

3.1.1.4 Removal Process

The removal process including wet and dry depositions are extremely important in regional air quality models while in case of dispersion models, these are normally ignored. When the air pollutants are washed out by precipitation/fog droplets is known as wet deposition (natural processes), causing scavenging of air pollutants in the atmosphere (Saxena and Kulshrestha 2016). Clouds and precipitation are the prominent factors in surface chemistry in various ways. The following ways are: (a) aqueous-phase chemical reaction; (b) scavenging is the key sink for air pollutants; (c) the vertical motion of air pollutants due to the convection can often transport the pollutions from the planetary boundary layer-heights (PBL-H) to the free troposphere; and (d) the clouds itself change the solar radiation fields and photolysis rates. The prominent features of wet deposition are as follows: (a) the specific details of the cloud and precipitation fields in space and time; and (b) the relationship of trace gases and aerosols along with the clouds and precipitation (Sonwani and Kulshrestha 2019). Wet removal is regarded as first-order removal process. The removal rate (scavenging coefficient), that is established due to the empirical or microphysical reflections, and also found to be direct proportional to the precipitation rate. Later, with the help of cloud modules, the cloud and precipitation processes using meteorological parameters along with the profiles of trace gases and aerosols are also used in wet removal rate processes (Sonwani and Kulshrestha 2017). In dry deposition, the gases and aerosols fall on the surfaces (it may be surface of buildings, cars, and trees) from the atmosphere in the absence of the precipitation. Dry depositions of a gaseous species/particle are only managed by the level of turbulence, the properties of the deposition pollutants, and the nature of the surface itself. The level of turbulence in the atmosphere governs the rate at which pollutants are coming down to the surface. Solubility and the chemical reactivity may possibly affect gaseous pollutants uptake while in case of particles, size, density, and shape could determine whether capture by the surface occurs or not. The surface itself is a factor in dry deposition. A nonreactive surface could not allow the absorption or adsorption of certain gases; a flat surface may direct to particle bounce-off (Mills et al. 2018).

3.1.1.5 Meteorology

Meteorological conditions such as horizontal and vertical wind components, water vapor concentrations, temperature, atmospheric surface pressure, sunlight-based radiation, cloud division, rainfall, planetary boundary layer height (PBL-H), and turbulence play a prominent role in simulation change studies. In addition to this, it also includes numerous atmospheric processes which control or significantly impact the development of emission, atmospheric gases, and aerosols. These processes include horizontal and vertical transport, turbulent mixing, convection, lightning-induced generation of nitrogen oxides (NOx) as well as dry and wet deposition to the Earth’s surface. The chemical reactions and their reaction rate during the formation of secondary products are affected by the relative humidity, solar flux, temperature, and water vapor. Apart from that, simulation also affects due to the prominent uncertainties in the meteorological parameters. There are also some other essential meteorological factors which are also required for air quality models as horizontal and vertical wind components, temperature, water vapor mixing ratios, cloud portion and liquid water content, rainfall, sunlight-based actinic flux, sea level pressure, PBL-H, turbulence intensity, heat, moisture, and momentum. Wind and turbulence motions are separated normally in six scales namely, global, synoptic, mesoalpha, mesobeta, mesogamma, and turbulence in the more extensive meteorological network. Several air pollution models add meteorological fields from measurements which ignore the role of clouds and also overlook the impacts of aerosols. The advantage of utilizing interpolated meteorological fields is that, if adequate data are available, an added field is more exact than an analytical field. The drawbacks are: measured meteorological data are generally accessible in few areas and many of them are found closer to the surface. Therefore, data for elevated model layers are rare. Additionally, a model that uses a previous data base to anticipate meteorology is not analytical, since it can simulate in past decades.

3.2 Classification of Air Pollution Models

Physical and scientific models are atmospheric models. Physical models are used to simulate atmospheric processes with the help of various methods used for a small-scale illustration of the real framework, for example, a small scale model of an urban region in a wind tunnel. Issues related with appropriately copying the actual scales of atmospheric motions make physical models of this type in limited scope. From now, the focus on numerical models and the term model will allude carefully to scientific mathematical models. Most of the areas contain many monitoring networks which are conducted by administrative specialists and they reveal, every hour, the daily basis estimated values of different air pollutants present in a particular atmosphere. A lot of data is conceivably accessible in these colossal databases, and measurable examination of such information can give significant experiences.

3.2.1 Lagrangian and Eulerian Models

A wide range of atmospheric models have been proposed and utilized. A portion of simulate changes in the chemical composition of a given air parcel as it is advected in the atmosphere are depicted by Lagrangian models. However, Eulerian models express the concentrations in a range of fixed computational cells. A Lagrangian modeling demonstrates the structure that explains the movement of local wind where there is no mass exchange in the air parcel except for species emissions that are permitted to enter the package through its base. The air Parcel moves constantly, so

Table 3.1 Atmospheric chemical transport models defined according to spatial scale

Model	Typical domain scale	Typical resolution	Motion examples
Microscale	200 × 200 × 100 m	5 m	Molecular diffusion, molecular viscosity
Mesoscale (urban)	100 × 100 × 5 km	2 km	Eddies, small plumes, car exhaust, cumulus clouds
Regional	1000 × 1000 × 10 km	36 km	Gravity waves, thunderstorms, tornados, cloud clusters, local winds, urban air pollution
Synoptic (continental)	3000 × 3000 × 20 km	80 km	High and low pressure systems, weather fronts, tropical storms, hurricanes, Antarctic ozone hole
Global	65,000 × 65,000 × 200 km	4° × 5°	Global wind systems, rossby (planetary) waves, stratospheric ozone reduction, global warming

the model really simulates the concentrations at various areas with different time. On the contrary, an Eulerian modeling signifying the structure remains fixed in space. Species enter and leave every cell through its separators, and the model simulates the pollutant concentrations at all areas as a function of time.

3.2.2 *Spatial and Temporal Scale*

The simulations vary from a few hundred meters to thousands of kilometers (see Table 3.1) along with the meteorology on every scale and it basically depends on the domain area of atmospheric model. Typically, the computational domain is mainly composed of a variety of computational cells, each of having a uniform composition. The size of these cells decides the spatial resolution of the model. The distributions of the pollutant concentration at scales lesser than the model resolution cannot be resolved.

For example, the concentration distributions over the Los Angeles Basin cannot be described by a synoptic scale model that treats the entire area as one computational cell of uniform chemical composition. Urban air pollution studies are simulated over micro- to mesoscale domain, regional acid deposition is simulated over a meso- to synoptic scale domain and global climate change studies are simulated over a global scale domain. Meteorological events occur all over the scales. With respect to time, urban air pollution events are simulated over periods of hours to days, regional acid deposition events are simulated over periods of days to weeks and climate change events are simulated over periods of months to hundreds of years and beyond.

3.3 Dispersion Modeling

These models are the only method that quantifies the deterministic relationship between emissions and concentrations/depositions, including the consequences of past and future scenarios and the determination of the effectiveness of abatement strategies. Air pollution measurements give information about ambient concentrations and deposition at specific locations and times, without giving clear guidance on the identification of the causes of the air quality problem.

The air pollution models are the main method that evaluates the deterministic connection among emissions and concentrations/depositions, including the outcome of past and future situations and the assurance of the adequacy of reduction procedures. The measurements of air pollution provide the details of concentrations/depositions at explicit areas and times, without giving clear direction on the recognizable proof of the causes of the air quality issue. The concentrations of substances in the atmosphere are determined by: (1) transport, (2) diffusion, (3) chemical transformation, and (4) ground deposition. Transport phenomena, characterized by the mean velocity of the fluid, have been measured and studied for centuries. For example, the average wind has been studied by man for sailing purposes. The study of diffusion (turbulent motion) is more recent. Air pollutants in the encompassing air which get transported air wind streams from the longer distance and dispersed by turbulent streams and blend with the air. Dispersion is hard to understand because of the different scale of eddies as well as their complex interaction. Dispersion models will incorporate the physical and chemical procedures of transportation, change and dispersion of air pollutants in the ambient air to estimate the concentrations, and also provide the emission sources. In recent time, the models are intended to compute the concentrations of pollutants utilizing data of qualities of emission sources, emission rates, terrain discrepancy, meteorological variations, and background levels. Dispersion models utilize the scientific-based ideas of dispersion and diffusion with appropriate treatment of complex landscape and land use. The concentrations of air pollutants in the ambient air continually change which are affected by weather conditions.

Meteorological parameters such as large scale wind flows, atmospheric stability, vertical mixing, and local mesoscale winds play a significant role in the atmospheric dispersion. An appropriate structure and utilization of the dispersion models can be utilized for recognizable proof of patrons through foundation of source–receptor connections, assessment of air quality consistence with government standards, arranging new facilities, the models of emission at sources, forecast of high concentration episodes and providing data to hazard assessment agencies. Most importantly this type of modeling can also save the cost of continuous observations over the large regions for longer periods. All the model estimations are to be assessed to comprehend the exposure because of errors in emission strength, meteorological information, concentration estimations, and investigation before assessment of health impacts. There are two demonstrating approaches, following Lagrangian and Eulerian mechanics. In the Lagrangian approach, the way of an air parcel is followed and the progressions followed along the direction way. The study has been started to investigate the role of long-range transport on sulfur dioxide (SO_2) using Lagrangian modeling over large distances and longer periods (Fisher 1975). In Eulerian method, the three-dimensional atmosphere is divided into grid cells, both in horizontal and vertical directions, and the time changes of the properties are identified at each grid cell. Eulerian modeling was agreed to the studies of ozone (O_3) at urban areas (Reynolds et al. 1973); for SO_2 and for regional-scale sulfur. In addition to this, Eulerian modeling has been used only for specific episodes. Hybrid approaches were used by Friedlander and Seinfeld (1969), Eschenroeder and Martinez (1970), and Liu and Seinfeld (1974). It may be stated that prior to 1980, Lagrangian models were generally used for transport studies of sulfur and other particulate matter whereas Eulerian models were adopted for episodic events of secondary pollutants such as ozone. After 1980, the fundamental ideas were tweaked with advancement and utilization of 2D and 3D global troposphere models. AERMOD and CALPUFF models have been produced for simulation of nonreactive chemicals (e.g., SO_2). AERMOD is a steady-state Gaussian plume model which makes use of wind field derived from the surface, upper-air, and on location meteorological observations joined with landscape heights and land use. CALPUFF is a non-steady state Lagrangian puff dispersion model which simulates the transport in both the conditions, i.e., calm and stagnant, over complex territory and coastal regions with sea/land breezes. AERMOD is for short-range simulations but CALPUFF is suitable for both long-range and short-range simulations. There are various sorts of modeling approaches such as photochemical modeling, Plume Rise models, molecule models, Deposition Modules, Odor models, Statistical models, and positive factorization matrix (PMF) models.

3.3.1 Photochemical Modeling

The photochemical models are the large-scale air quality models that simulate the modifications of concentrations in the atmosphere with the help of mathematical equation which describes the chemical and physical processes in the atmosphere. These models are applicable for global, regional, and multiple spatial scales. A few examples of photochemical models are: Community Multi-scale Air Quality Model (CMAQ), Comprehensive Air quality Model with extensions (CAM_X), Weather Research and Forecasting model coupled with Chemistry WRF-Chem and so on. The CMAQ model has been developed for air quality having the capabilities to conduct the simulations for multiple air quality issues including surface O_3, fine particles, toxics, acid deposition, and visibility degradation [http://www.epa.gov/asmdnerl/CMAQ/index.html]. In addition to this, the model simulates the different chemical and physical processes that are believed to be significant for the understanding of ambient trace gases transformations and dispersions. CAMx model is applicable for the integrated assessment of gaseous and particulate pollutants [http://www.camx.com/]. The model has been intended to simulate the air quality over numerous geographic scales. Moreover, the model takes care of a wide range of inert and pollutants such as O_3, particulate matters ($PM_{2.5}$ and PM_{10}), and mercury and it also possesses plume-in-grid and source apportionment capabilities.

3.3.2 Plume Rise Models

For estimating the smoke concentrations, it is determined that the plumes circulate in the Gaussian pattern along with the center line of a steady wind trajectory. These models mainly assume the steady-state conditions during the life of the plume that means relatively constant emission rates, wind speed, and wind direction. They are to be used only to estimate the concentrations relatively near the source with short period with restrictions to the impact of the topography or land use. Some of air pollution models which include a computational module for plume rise, i.e., the initial behavior of a hot plume bring in vertically into a horizontal wind flow, e.g., PRIME in AERMOD and HYSPLIT.

3.4 WRF/Chem Model

The Weather Research and Forecasting–Chemistry model (WRF/Chem) is a latest regional air quality modeling system which has been developed by NOAA (National Oceanic and Atmospheric Administration) (Grell et al. 2005). The model makes use of the Eulerian mechanics, in which the meteorology and chemistry variables are forecasted at all the grid points in each time step. It is suitable for the prediction of air quality which requires regularly dealing with chemical species and meteorological parameters. In addition to this, it has two modules for meteorology and chemistry. The chemistry module constitutes the process of dry deposition, coupled with the soil/vegetation scheme; aqueous-phase chemistry together with some of the microphysics and aerosol schemes; biogenic emissions; anthropogenic emissions; gas-phase chemical reaction; photolysis schemes; and aerosol schemes with different choices. The air quality module of the model is fully consistent with the meteorological component; both components use the same transport scheme, the same horizontal and vertical grids, the same physics schemes and the same time step for transport and vertical mixing. The resolution of the model varies from a few kilometers to hundred kilometers. The modeling system is as great as coupled weather prediction/dispersion/air quality model to simulate release and transport of primary pollutants such as particulate matter and the prediction of secondary pollutants such as O_3. The model is able to run on parallel processing computer platforms to save computational time.

3.5 Overview of Air Pollution Modeling Studies

3.5.1 Role of Air Pollution on Human Health

Air pollution is an encompassing reason for death and sickness comprehensively. An expected ~4.2 million unexpected losses (premature death) comprehensively are related with ambient air pollution which causes the diseases like coronary illness, stroke, perpetual obstructive pneumonic sickness, lung malignant growth, and intense respiratory diseases in youngsters. The overall death and ailment rate from lung malignant growth, intense lower respiratory contamination, stroke, coronary illness, and endless obstructive aspiratory sickness are 29%, 17%, 24%, 25%, and 43%, respectively. Air pollutants which produced harmful health impacts include particulate matter (PM), ozone (O_3), nitrogen dioxide (NO_2), and sulfur dioxide (SO_2) (https://www.who.int/airpollution/ambient/health-impacts/en/). Ambient air pollution is ranked as fifth largest contributor in India as indicated by the Global Burden of Diseases (GBD) in WHO (2014). India is positioned second among other nations of the world influenced by $PM_{2.5}$ (Silva et al. 2013) and ozone (Lelieveld et al. 2015) pollution and responsible for causing acute and chronic respiratory diseases and related mortalities. Recently, one study has been done on premature mortality in India due to both $PM_{2.5}$ and O_3 (Ghude et al. 2016). In this study, O_3 and $PM_{2.5}$ simulation from WRF-Chem combined with the most recent populations to estimate exposure to air pollution on local and regional scales in India.

They used version 3.6.1 of the regional Weather Research and Forecasting model coupled with chemistry (WRF-Chem) to simulate hourly surface O_3 and $PM_{2.5}$ distributions for the year of 2011 at 36 km horizontal resolution in order to resolve urban and rural regions (Ghude et al. 2016) (Fig. 3.6). They also estimated that $PM_{2.5}$ exposure leads to about 570,000 untimely mortalities in 2011, and COPD mortalities because of ozone exposure, is around 12,000 individuals (Ghude et al. 2016).

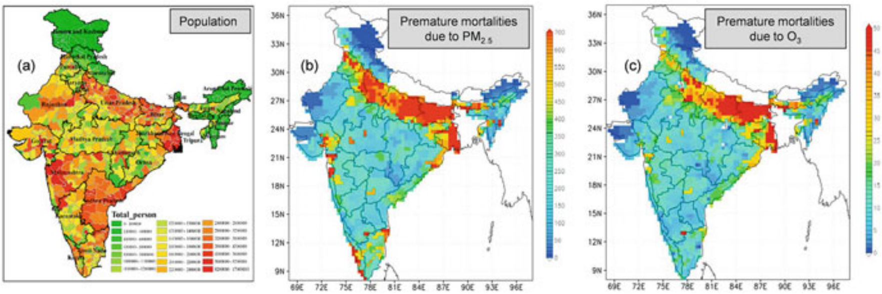


Fig. 3.6 Distribution of (**a**) district-wise India's population and total premature mortalities due to (**b**) $PM_{2.5}$ and (**c**) O_3 exposure in 2011 (Adapted from Ghude et al. 2016)

3.5.2 Dust Aerosols and Tropospheric Chemistry

Dust storms occur frequently in South Asia during the pre-monsoon season (Prasad and Singh 2007) and significantly affect the regional aerosol loadings as well as tropospheric chemistry. Kumar et al. (2014a) updated the dust emission scheme in WRF-Chem to capture dust storm-induced changes on regional aerosol loadings over northern India for a typical dust storm that occurred during April 17–22, 2010. WRF-Chem successfully simulated the temporal variations in Aerosol Robotic Network (AERONET) retrieved aerosol optical depth and Angstrom exponent at seven sites in South Asia. The model also captured the spatial distribution of satellite retrieved changes in AOD between low and high dust periods. The WRF-Chem simulations of dust aerosols conducted by Kumar et al. (2014b) included an updated photolysis rate scheme to account for effects of dust aerosols on photolysis frequencies and a heterogeneous chemistry module in the model to assess the effects of dust storms on surface ozone. The inclusion of these processes in WRF-Chem reduced the difference between observed and modeled O_3 from 16±9 to 2±8 ppbv and that in NOy from 2129±1425 to 372±1225 pptv compared to measurements at high altitude site Nainital in the central Himalayas (Fig. 3.7).

3.6 Summary and Conclusion

Fast growth in population over the past few decades has significantly elevated air pollution levels worldwide. Recent studies from India have shown that air pollution is adversely affecting human health. In this chapter, we briefly discussed about the modeling approach and their results, modeling long-term effects, as opposed to studying short-term episodes and present challenges. Apart from the additional input data requirements, running a model for a year takes 30 times as long (in computer time) as it does to run a 12-day episode. When this is compounded over the study of a number of scenarios, the cost quickly becomes prohibitive. Advances have been made in aggregation schemes, which assemble a long-term average from a suitable combination of representative short-term episodes, but further development is required.

Along with the model, an appropriate infrastructure is also essential. This will include all of the appropriate input data, a suitable computing platform, and skilled modelers to carry out the runs, and provide quality assurance and interpretation. These runs will involve scenarios, usually based on altered emissions, but possibly including other changes, such as different treatments of chemistry in the model, or altered meteorological conditions. Model results will be scrutinized, analyzed, and interpreted, before being presented to the policymakers. Chemical transport models have been very useful in advancing our understanding of air pollution but further efforts are required to reduce uncertainties in model simulations.

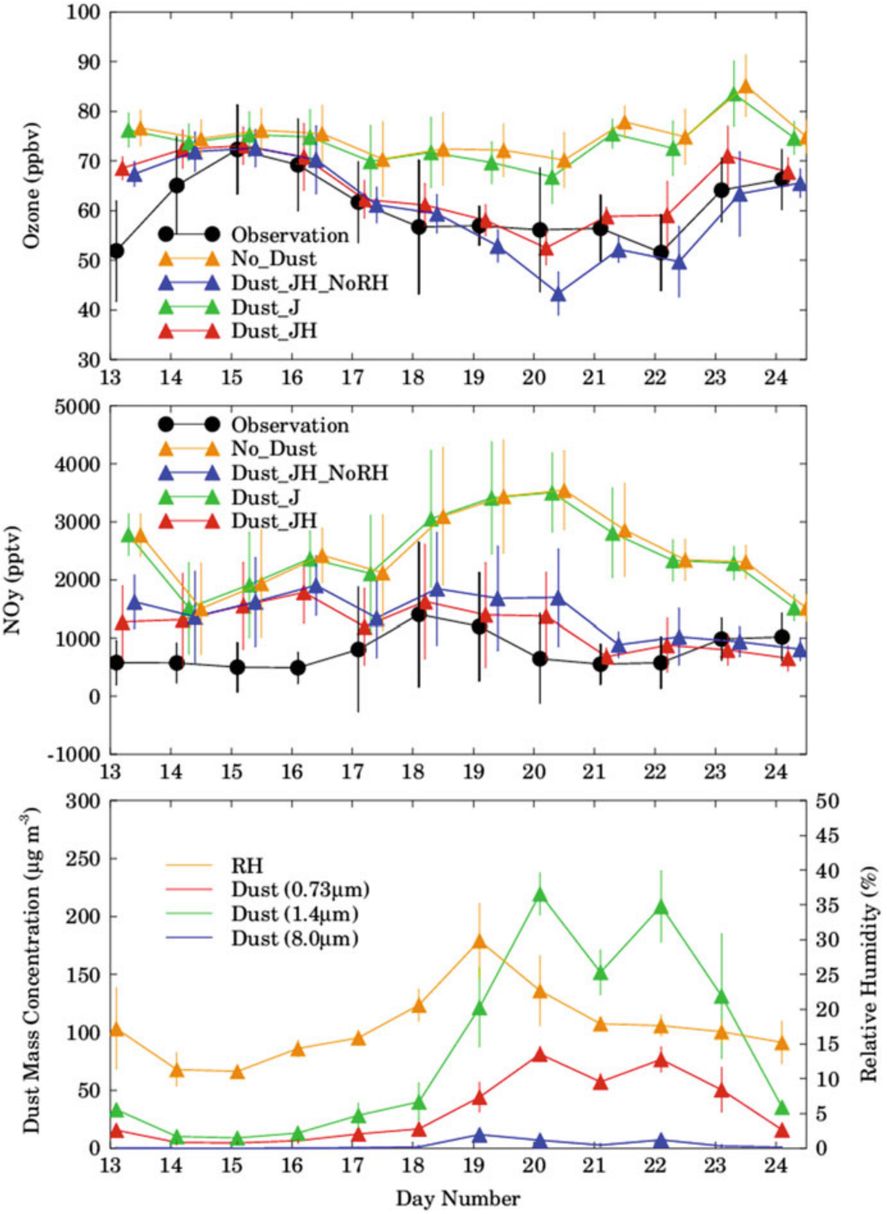


Fig. 3.7 Variations in observed and WRF-Chem simulated daily average O_3 (top panel) and NOy (middle panel) at Nainital during April 13–24, 2010. WRF-Chem simulated daily average mass concentration of dust particles of 0.73, 1.4, and 8.0 μm effective radii and relative humidity at Nainital are also shown. The vertical bars represent standard deviation in the average values. Dust_JH represents WRF-Chem simulation with both photolysis and heterogeneous chemistry with RH dependence of uptake coefficients included. Dust_JH_NoRH is same as Dust_JH but without RH dependence of uptake coefficients and DUST-J includes only photolysis effects of dust. Adapted from Kumar et al. (2014b)

References

Akimoto H (2003) Global air quality and pollution. Science 302(5651):1716–1719

Baudic A, Gros V, Sauvage S, Locoge N, Sanchez O, Sarda-Estève R, Kalogridis C, Petit J-E, Bonnaire N, Baisnée D, Favez O, Albinet A, Sciare J, Bonsang B (2016) Seasonal variability and source apportionment of volatile organic compounds (VOCs) in the Paris megacity (France). Atmos Chem Phys 16(18):11961–11989

Beig G, Chate DM, Ghude SD, Mahajan AS, Srinivas R, Ali K, Sahu SK, Parkhi N, Surendran D, Trimbake HR (2013) Quantifying the effect of air quality control measures during the 2010 Commonwealth Games at Delhi, India. Atmos Environ 80:455–463

Beig G, Sahu SK, Singh V, Tikle S, Sobhana SB, Prashant G, Ramakrishna K, Aditi R, Murthy BS (2020) Objective evaluation of stubble emission of North India and quantifying its impact on air quality of Delhi. Sci Total Environ 709:136126

Cooper OR, Parrish DD, Stohl A, Trainer M, Nédélec P, Thouret V, Cammas JP, Oltmans SJ, Johnson BJ, Tarasick D, Leblanc T, McDermid IS, Jaffe D, Gao R, Stith J, Ryerson T, Aikin K, Campos T, Weinheimer A, Avery MA (2010) Increasing springtime ozone mixing ratios in the free troposphere over western North America. Nature 463(7279):344–348

Eschenroeder AQ, Martinez JR (1970) Mathematical modeling of photochemical smog. Proceedings of the American Institute Aeronautics and Astronautics, Eight Aerospace Sciences Meeting, New York, 19–21 January

Fisher BEA (1975) The long-range transport of sulphur dioxide. Atmos Environ 9:1063

Forster P, Ramaswamy V, Artaxo P, Berntsen T, Betts R, Fahey DW, Haywood J, Lean J, Lowe DC, Myhre G, Nganga J, Prinn R, Raga G, Schulz M, Van Dorland R (2007) Changes in atmospheric constituents and in radiative forcing. In: Solomon S, Qin D, Manning M, Chen Z, Marquis M, Averyt KB, Tignor M, Miller HL (eds) Climate change 2007: the physical science basis. Contribution of Working Group I to the Fourth Assessment Report of the Intergovernmental Panel on Climate Change. Cambridge University Press, Cambridge

Friedlander SK, Seinfeld JH (1969) A dynamic model of photochemical smog. Environ Sci Technol 3:1175

Fuzzi S, Baltensperger U, Carslaw K, Decesari S, Denier van der Gon H, Facchini MC, Fowler D, Koren I, Langford B, Lohmann U, Nemitz E, Pandis S, Riipinen I, Rudich Y, Schaap M, Slowik J, Spracklen DV, Vignati E, Wild M, Williams M, Gilardoni S (2015) Particulate matter, air quality and climate: lessons learned and future needs. Atmos Chem Phys Discuss 15(1):521–744

Ghude SD et al (2016) Premature mortality in India due to PM2.5 and ozone exposure. Geophys Res Lett 43:4650–4658. https://doi.org/10.1002/2016GL068949

Grell GA, Peckham SE, Schmitz R, McKeen SA, Frost G, Skamarock WC, Eder B (2005) Fully coupled "online" chemistry within the WRF model. Atmos Environ 39:6957–6975

Jaffe D, Bertschi I, Jaegle L, Novelli P, Reid JS, Tanimoto H, Vingarzan R, Westphal DL (2004) Long-range transport of Siberian biomass burning emissions and impact on surface ozone in western North America. Geophys Res Lett 31(16):L16106

Karl T, Guenther A, Yokelson RJ, Greenberg J, Potosnak M, Blake DR, Artaxo P (2007) The tropical forest and fire emissions experiment: emission, chemistry, and transport of biogenic volatile organic compounds in the lower atmosphere over Amazonia. J Geophys Res 112: D18302. https://doi.org/10.1029/2007JD008539

Kumar R, Barth MC, Pfister GG, Naja M, Brasseur GP (2014a) WRF-Chem simulations of a typical pre-monsoon dust storm in northern India: influences on aerosol optical properties and radiation budget. Atmos Chem Phys 14:2431–2446. https://doi.org/10.5194/acp-14-2431-2014

Kumar R et al (2014b) Effects of dust aerosols on tropospheric chemistry during a typical pre-monsoon season dust storm in northern India. Atmos Chem Phys 14:6813–6834. https://doi.org/10.5194/acp-14-6813-2014

Lelieveld J, Dentener FJ (2000) What controls tropospheric ozone? J Geophys Res Atmos 105 (D3):3531–3551

Lelieveld J, Evans JS, Fnais M, Giannadak D, Pozzer A (2015) The contribution of outdoor air pollution sources to premature mortality on a global scale. Nature 525(7569):367–371. https://doi.org/10.1038/nature15371

Liu MK, Seinfeld JH (1974) On the validity of grid and trajectory models of urban air pollution. Atmos Environ 9:555–574

Maji S, Beig G, Yadav R (2020) Winter VOCs and OVOCs measured with PTR-MS at an urban site of India: role of emissions, meteorology and photochemical sources. Environ Pollut 258:113651

Mills G, Pleijel H, Malley CS, Sinha B, Cooper OR, Schultz MG et al (2018) Tropospheric ozone assessment report: present-day tropospheric ozone distribution and trends relevant to vegetation. Elem Sci Anth 6:47

Montgomery MR (2008) The urban transformation of the developing world. Science 319 (5864):761–764

Mozaffar A, Zhang Y-L, Fan M, Cao F, Lin Y-C (2020) Characteristics of summertime ambient VOCs and their contributions to O3 and SOA formation in a suburban area of Nanjing, China. Atmos Res 240:104923

Prasad AK, Singh RP (2007) Changes in aerosol parameters during major dust storm events (2001–2005) over the Indo-Gangetic Plains using AERONET and MODIS data. J Geophys Res 112:D09208. https://doi.org/10.1029/2006JD007778

Reynolds S, Roth P, Seinfeld J (1973) Mathematical modelling of photochemical air pollution. Atmos Environ 7

Sahu and Lal (2006) Distributions of C_2–C_5 NMHCs and related trace gases at a tropical urban site in India. Atmos Environ 40:880–891

Sahu SK, Beig G, Parkhi NS (2011) Emissions inventory of anthropogenic PM2.5 and PM10 in Delhi during Commonwealth Games 2010. Atmos Environ 45(34):6180–6190

Sahu LK, Saxena P (2015) High time and mass resolved PTR-TOF-MS measurements of VOCs at an urban site of India during winter: role of anthropogenic, biomass burning, biogenic and photochemical sources. Atmos Res 164:84–94

Sahu LK, Yadav R, Pal D (2016a) Source identification of VOCs at an urban site of western India: effect of marathon events and anthropogenic emissions weather conditions. J Geophys Res Atmos 121:2416–2433. https://doi.org/10.1002/2015JD024454

Sahu LK, Pal D, Yadav R, Munkhtur J (2016b) Aromatic VOCs at major road junctions of a Metropolis in India: measurements using TD-GC-FID and PTR-TOF-MS instruments. Aerosol Air Qual Res 16:2405–2420. https://doi.org/10.4209/aaqr.2015.11.0643

Sahu LK, Tripathi N, Yadav R (2017) Contribution of biogenic and photochemical sources to ambient VOCs during winter to summer transition at a semi-arid urban site in India. Environ Pollut 225:291–303

Saxena P, Ghosh C (2017) Role of biogenic VOCs in the production of tropospheric ozone in India. Int J Sci Eng Res 8(10):748–755

Saxena P, Kulshrestha UC (2015) Scavenging of urban air emissions by Fog at Delhi, India. In: AGU Fall Meeting Abstracts, December

Saxena P, Kulshrestha UC (2016) Contribution of carbonaceous species in SOA formation during fog and non-fog period. J Chem Biol Phys Sci 7(1):031–048

Saxena P, Sharma Y, Chugh M (2017) Assessment of ozone phytotoxic potential of dense vegetation cover of Delhi by AOT40. In: AGU Fall Meeting Abstracts, December

Saxena P, Srivastava A, Tyagi M, Kaur S (2019) Impact of tropospheric ozone on plant metabolism – a review. Pollut Res 38(1):175–180

Seinfeld JH, Pandis SN (2006) Atmospheric chemistry and physics: from air pollution to climate change. Wiley-Interscience, Hoboken, NJ. ISBN 13:978-0-471-72018-8

Silva A et al (2013) Global premature mortality due to anthropogenic outdoor air pollution and the contribution of past climate change. Environ Res Lett 8:034005. https://doi.org/10.1088/1748-9326/8/3/034005

Simayi M, Shi Y, Xi Z, Li J, Yu X, Liu H, Tan Q, Song D, Zeng L, Lu S, Xie S (2020) Understanding the sources and spatiotemporal characteristics of VOCs in the Chengdu Plain, China, through measurement and emission inventory. Sci Total Environ 714:136692

Solomon S et al (2007) Contribution of working group I to the fourth assessment report of the intergovernmental panel on climate change. Cambridge University Press, Cambridge

Sonwani S, Saxena P (2016) Identifying the sources of primary air pollutants and their impact on environmental health: a review. Int J Eng Tech Res 2:111–130

Sonwani S, Kulshrestha UC (2017) Wet scavenging of organic and elemental carbon during summer monsoon and winter monsoon seasons. In: AGU Fall Meeting Abstracts

Sonwani S, Kulshrestha U (2018) Morphology, elemental composition and source identification of airborne particles in Delhi, India. J Indian Geophys Union 22(6):607–620

Sonwani S, Kulshrestha UC (2019) PM 10 carbonaceous aerosols and their real-time wet scavenging during monsoon and non-monsoon seasons at Delhi, India. J Atmos Chem 76:171–200

United Nations, Department of Economic and Social Affairs (2010) Population division: World urbanization prospects, the 2009 revision: highlights. New York

Wang C, Huang X-F, Zhu Q, Cao L-M, Zhang B, He L-Y (2017) Differentiating local and regional sources of Chinese urban air pollution based on the effect of the Spring Festival. Atmos Chem Phys 17(14):9103–9114

WHO (2005) Air quality guidelines for particulate matter, ozone, nitrogen dioxide and sulfur dioxide. World Health Organization, Geneva

WHO (2016) Global urban ambient air pollution database. World Health Organization, Geneva

World Health Organization (WHO) (2014) Methods and data sources for country-level causes of death 2000–2012. World Health Organization, Geneva

Yadav R, Beig G, Jaaffrey SNA (2014a) The linkages of anthropogenic emissions and meteorology in the rapid increase of particulate matter at a foothill city in the Aravalli range of India. Atmos Environ 85:147–151

Yadav R, Sahu LK, Jaaffrey SNA, Beig G (2014b) Temporal variation of particulate matter (PM) and potential sources at an urban site of Udaipur in Western India. Aerosol Air Qual Res 14:1613–1629

Yadav R, Sahu LK, Jaaffrey SNA, Beig G (2014c) Distribution of ozone and related trace gases at a semi urban site of Udaipur in Western India. J Atmos Chem 71:125–144

Yadav R, Sahu LK, Beig G, Jaaffrey SNA (2016) Role of long range transport and local meteorology in seasonal variation of surface ozone and its precursors at an urban site in India. Atmos Res 176–177:96–107

Yadav R, Sahu LK, Beig G, Tripathi N, Jaffrey SNA (2017) Ambient particulate matter and carbon monoxide at an urban site of India: influence of anthropogenic emissions and dust storms. Environ Pollut 225:291–303. https://doi.org/10.1016/j.envpol.2017.01.038

Yadav R, Sahu LK, Tripathi N, Pal D, Beig G, Jaaffrey SNA (2019a) Investigation of emission characteristics of NMVOCs over urban site of western India. Environ Pollut 252:245–255

Yadav R, Sahu LK, Beig G, Tripathi N, Maji S, Jaaffrey SNA (2019b) The role of local meteorology on ambient particulate and gaseous species at an urban site of western India. Urban Clim 28:100449

Chapter 4
Impact of Air Pollutants on Plant Metabolism and Antioxidant Machinery

Amarjeet Singh

Abstract Accumulation of toxic substances in biosphere negatively affects the structure and function of natural ecosystem. With progress in technology, diverse array of pollutants are rapidly being added to the atmosphere by mankind without understanding their fate in nature. Therefore, it becomes essential to analyze the impact of these pollutants on organisms. It is important to understand the effect of these pollutants on plants from where it is transferred to next members of food chain ultimately affecting the mankind. Various physiological and biochemical processes such as gaseous exchange, photosynthesis, respiration, and protein synthesis are known to be affected by air pollutants. Plants respond to these abiotic stress situations by activating the stress signals leading to various physiological, genetic, and metabolic changes. This chapter highlights the effect of variety of air pollutants on plant health and activation of antioxidant system as a defense mechanism.

Keywords Urbanization · Industrialization · Abiotic stress · Crop yield reduction · Antioxidant machinery · Pollutants

4.1 Introduction

Mankind has witnessed rapid urbanization and industrialization over the last 50 years. As a consequence, a wide array of toxic substances has been introduced into the atmosphere affecting the natural processes directly or indirectly (Sirohi et al. 2018). These substances are often termed as "pollutants." Later these pollutants make their way into food chain ultimately affecting the health and well-being of humans.

Plants being sessile are continuously exposed to various types of air pollutants, which may act individually or synergistically to affect plant metabolism and growth

---

A. Singh (✉)
Department of Plant Physiology and Molecular Plant Biology, Faculty of Sciences, Eötvös Loránd University, Budapest, Hungary
e-mail: amarpc@caesar.elte.hu

P. Saxena, A. Srivastava (eds.), *Air Pollution and Environmental Health*,
Environmental Chemistry for a Sustainable World 20,
https://doi.org/10.1007/978-981-15-3481-2_4

and often leads to decline in crop yield and nutritional quality (Kulshrestha and Saxena 2016; Agrawal et al. 2003; Marshall et al. 1999). Principally, plants are not selective in gas uptake (Heagle 1989), which makes it even more challenging to recognize the stress conditions being faced by plants. This in turn adversely affects the global food security. The rate with which population is expanding, it becomes important to understand the response of plants in terms of physiology and metabolism to their external environment. Decline in crop yield in the present scenario due to any kind of stress needs to be addressed immediately for sustainable future. Therefore, understanding the impact of major air pollutants on plant physiology and metabolism will help us to look after the indirect effect of these on entire ecosystem as well as to predict the future of plant health (Saxena and Kulshrestha 2016a, b).

4.2 Air Pollutants and Their Impacts on Plant Health

It is difficult to assess the direct effect of individual pollutant on physiology and metabolism of plants because natural factors such as light, water, and mineral nutrition affects the interaction of plants with pollutants. Moreover, in a particular environment, plants are subjected to a combination of pollutants simultaneously, thereby making it more difficult to single out the causal pollutant (Kulshrestha and Saxena 2016; Mudd and Kozlowski 1975).

Some of the environmental factors that determine the rate at which morphological and physiological symptoms appear on plants are type and concentration of pollutants, duration of exposure, distance from the source of pollutant, and weather conditions. Clear, warm, still, and humid weather with high barometric pressure appears to be the perfect environmental condition when the damage caused to plants is severe (Unsworth and Ormrod 1982; Mudd and Kozlowski 1975). Injury in response to these air pollutants can occur in two forms namely chronic and acute injury. Chronic injury to plants may occur as a result of long-term exposure to pollutants at a low concentration. On the other hand, acute injury occurs when plants are fumigated with high concentration of pollutants for short duration (Saxena and Kulshrestha 2016a, b; Zeevaart 1976).

For instance, Chen et al. (2009) studied the photosynthetic response of soybean leaf toward acute (400 ppb, 6 h) and chronic (90 ppb, 8 h, 28 days) exposure to O_3. Different patterns of O_3 damage at the leaf level appeared. In chronic O_3-exposed leaves, the areas of least photosynthetic capacity were of variable size and shapes which appeared mostly in the interveinal regions. On the other hand, acute damage to leaf was characterized by small local areas of reduced photosynthetic capacity that appeared mainly in areas near the major veins.

Plants use complex recognition and response mechanisms to combat any kind of stress (Bita and Gerats 2013). Whenever stress conditions are detected by plants, first response of plants is production of reactive oxygen species (ROS). High concentrations of ROS are toxic and affect the normal functioning of cell (Saxena et al. 2017a;

Caverzan et al. 2016; Suzuki et al. 2012). Because of their highly reactive nature, ROS can damage major biomolecules such as nucleic acids, lipids, and proteins. Nucleic acids may be modified by inducing oxidation, strand breaks, deletion, and modification of nucleotides. Lipids can be damaged by inducing the chain break and increasing the fluidity of membranes or oxidation. Proteins, on the other hand, may be subjected to site-specific amino acid modification, alteration of the electric charge, and enzymes inactivation. ROS may also lead to activation of programmed cell death (PCD) that may result in death of the cells (Caverzan et al. 2016; Sharma et al. 2012).

ROS performs dual roles in cell. Under normal conditions they may act as secondary messengers involved in signal transduction networks but under oxidative stress they can potentially cause damage or even cell death. Therefore, ROS production and scavenging tightly regulates the "redox homeostasis" in cells (Sharma et al. 2012; Neill et al. 2002). ROS are by-products of aerobic metabolism and formation of various ROS such as hydrogen peroxide (H_2O_2), superoxide anion ($^{\bullet}O^{-}{}_2$), hydroxyl ion (OH^-), hydroxyl radical ($^{\bullet}OH$), and singlet oxygen ($^1O_2$) is enhanced in response to the pollutants. These ROS can act as both intra- and intercellular messengers. In absence of effective ROS detoxification strategies, conditions of "oxidative stress" are created as the levels of ROS are elevated (Saxena et al. 2017b; Sharma et al. 2012). Unavoidable leakage of electrons onto O_2 from the various electron transport activities of chloroplast, mitochondria, and other cellular compartments including plasma membrane leads to formation of ROS (Sharma et al. 2012; Heyno et al. 2011; Blokhina and Fagerstedt 2010). Figure 4.1 shows the abiotic stress-induced ROS-mediated changes in major biomolecules in plant cells.

In order to survive the deleterious effects of these ROS, plants have evolved a complex defense mechanism which maintains a balance between production and removal of ROS. Antioxidant defense system of plants consists of both enzymatic and nonenzymatic components. The enzymatic components of defense system includes superoxide dismutase (SOD), catalase (CAT), guaiacol peroxidase (GPX). A set of four enzymes are involved in ascorbate-glutathione (AsA-GSH) cycle of antioxidant defense system, viz. ascorbate peroxidase (APX), monodehydroascorbate reductase (MDHAR), glutathione-dependent dehydroascorbate reductase (DHAR) and glutathione reductase (GR) (Caverzan et al. 2016; Chew et al. 2003). Successive oxidation and reduction of Ascorbate (AsA), Glutathione (GSH), and NADPH catalyzed by the enzymes APX, MDHAR, DHAR, and GR occur in AsA-GSH cycle (Caverzan et al. 2016; Pandey et al. 2015; Sharma et al. 2012). The AsA-GSH cycle acts in different subcellular compartments (chloroplast, mitochondria, peroxisomes, cytosol, and apoplast) and multiple isoforms of enzymes of this pathway exist in cell (Pandey et al. 2015). Table 4.1 shows the major ROS scavenging antioxidant enzymes and the reactions catalyzed by them.

The nonenzymatic components of antioxidant defense machinery include major cellular redox buffers GSH and AsA. Others include carotenoids, flavonoids, alkaloids, and tocopherols (Sharma et al. 2012; Gill and Tuteja 2010; Mittler et al. 2004).

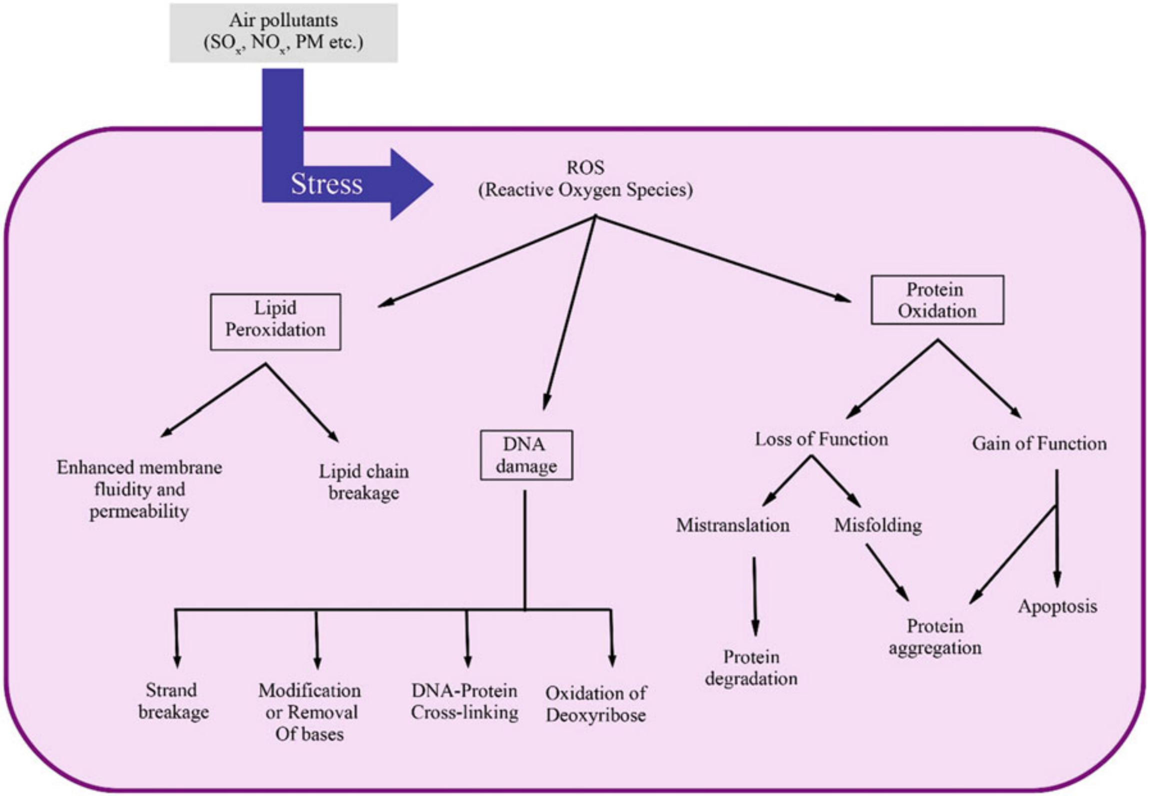


Fig. 4.1 Effect of air pollutants (abiotic stress) induced ROS on major biomolecules present in cells

Table 4.1 Major antioxidant enzymes of the plant antioxidant defense machinery and the reactions catalyzed by them (Caverzan et al. 2016; Das and Roy Choudhury 2014; Gill and Tuteja 2010)

Antioxidant enzyme	Reaction catalyzed	Enzyme commission (EC) number
Superoxide dismutase (SOD)	$O_2^{\bullet-} + O2^{\bullet-} + 2H^+ \rightarrow 2H_2O_2 + O_2$	1.15.1.1
Catalase (CAT)	$H_2O_2 \rightarrow H_2O + ½ O_2$	1.11.1.6
Guaicol peroxidase (GPX)	H_2O_2 + GSH → H_2O + GSSG	1.11.1.7
Ascorbate peroxidase (APX)	H_2O_2 + AA →2H_2O + DHA	1.11.1.11
Monodehydroascorbate reductase (MDHAR)	MDHA + NAD(P)H → AA + NAD(P)$^+$	1.6.5.4
Dehydroascorbate reductase (DHAR)	DHA + 2GSH → AA + GSSG	1.8.5.1
Glutathione reductase (GR)	GSSG + NAD(P)H → 2GSH + NAD(P)$^+$	1.6.4.2

These biomolecules can act directly to detoxify ROS and may also act by reducing the substrates for antioxidant enzymes (Caverzan et al. 2016; Mittler 2002).

The following section deals with the impact of major air pollutants on morphological, physiological, and biochemical changes within plants, and how antioxidative defense system plays a major role in acting against this abiotic stress condition.

4.3 Sulfur Dioxide (SO_2)

Sulfur dioxide is one of the most common and distinguished air pollutants across the world. It is a colorless gas with pungent odor generally released upon combustion of sulfur-based fuels and from copper smelting or released during volcanic eruption. Sulfuric acid (H_2SO_4) is the major source of acid rain, formed as a result of hydrolysis of SO_2 when dissolved in water. Over the years atmospheric level of SO_2 has increased; therefore, plants are being exposed to higher levels of SO_2 throughout the world (Saxena and Kulshrestha 2016a, b; Qu et al. 2016; Emberson et al. 2001; Robinson and Robin 1970).

A wide range of investigations has been carried out to assess the impact of SO_2 on plants (Tiwari et al. 2006; Mehlhorn et al. 1986; Riding and Percy 1985; Olszyk and Tingey 1984; Mclaughlin and Mcconathy 1983). Chronic as well as acute SO_2 exposure influences the growth of plants (Muneer et al. 2014). Sometimes small dosage of SO_2 may not have any effect on plants or may promote plant growth considering the fact that sulfur is an integral component of biomolecules such as proteins, chlorophyll, and hormones. It is also involved in efficient fixation of nitrogen by legumes (Li and Yi 2012; Mudd and Kozlowski 1975).

However, when given in large amounts, sulfur toxicity can be observed. Cereals like oats (*Avena sativa*), corn (*Zea mays*), rye (*Secale cereal*) and barley (*Hordeum* *vulgare*) and conifers such as Austrian pine (*Pinus nigra* J.F. Arnold) and Balsam fir (*Abies balsamia* L. Mill.) are somewhat sensitive to SO_2 injury. Among dicots carrot (*Daucus carota*), cabbage (*Brassica oleracea* var. *capitata*), black willow (*Salix nigra* Marsh.), beans (*Phaseolus vulgaris*), and celery (*Apium graveolens*) are sensitive (Legge and Krupa 2002; Treshow 1971). Injury on the parallel veined plants appears as necrotic streaks between the veins near the leaf tip and extends toward the base with increase in severity of injury. Conifers needle tips are the first to show injury in form of browning which extends toward the base with increase in severity of injury. Continuous exposure to injurious levels of SO_2 may then result in dark bands on the brown necrotic part of needle. Marginal or interveinal necrotic areas appear on broad leaved plants in response to acute SO_2 injury (Legge and Krupa 2002; Mudd and Kozlowski 1975; Taylor 1973). Generally young leaves are more sensitive to SO_2 than older leaves (Horsman and Wellburn 1977; Zeigler 1972). The susceptible areas affected by SO_2 become flaccid and subsequently dried becoming white to ivory in most plant leaves. Sometimes, the dead tissue may turn red, brown, or almost black (Taylor 1973). Chronic injury including brown/red leaf spots arising from leaf edges, decline in leaf area, and crop yield are some common symptoms (Kropff 1991). In monocots, acute injury symptoms appear at the tip of leaves and advances downward as necrotic and chlorotic streaks with infrequent reddish pigmentation (Rai et al. 2011).

Reduction in chlorophyll content, biomass, photosynthetic and respiration rate are common effects of excess SO_2 (Padhi et al. 2013). During the exposure to toxic levels of $SO_{2,}$ plants are not capable of assimilating the SO_2 at the same rate as it is being absorbed (Stratigakos and Ormrod 1985) which exceeds the threshold accumulation in intercellular spaces of the leaf resulting in acute injury (Thomas 1961).

Stomatas are the main facilitators of gas exchange between plants and external environment. Plants can metabolize SO_2 received in the gaseous form through stomata. Therefore, primary effect of high SO_2 exposure is the alteration of stomata. Guard cells damaged by SO_2 affect stomatal opening, thus plant water content (Bytnerowicz et al. 2007) and high-level SO_2 decrease the stomata abundance and result in aborted stomata (Olszyk and Tibbitts 1981; Koziol and Whatley 2016). Reduction in total number of stomata by plants is a mechanism to cope with the increased concentration of SO_2, preventing the entry of SO_2 inside. SO_2-induced erosion of the epicuticular wax structures around the stomata contributes to the damage of stomata (Unsworth and Black 1981; Taylor 1978). Once it enters the intercellular spaces, SO_2 dissolves in water on the cellular surfaces causing considerable cellular K^+ loss (Koziol and Whatley 2016).

SO_2 is capable of inducing apoptosis and reducing the viability of guard cells in *Tagetes erecta* in a dose-dependent manner. ROS and NO act as facilitators of SO_2-induced cell death (Wei et al. 2015). SO_2, after its entry into plants via stomata, generates more toxic forms, sulfite ($SO_3{}^{2-}$) and bisulfite ($HSO_3{}^{-}$), by dissolving in cellular cytoplasm. These toxic forms are capable of damaging plants (Lewandowska and Sirko 2008). Sulfite is 30 times more toxic than sulfate (Thomas et al. 1943). Therefore, prolonged exposure to high atmospheric concentration of SO_2 (more than 0.3 ppm) can cause severe tissue damage (Taiz and Zeiger 2002).

Stable cytoplasmic pH is an important factor for executing the important cellular activities. SO_2 causes acidification of cytoplasmic pH affecting the various metabolic processes.

Detoxification process of sulfite ($SO_3{}^{2-}$) and bisulfite ($HSO_3{}^{-}$) to less toxic form sulfate ($SO_4{}^{2-}$) leads to enhancement of ROS, such as superoxide radical ($O_2{}^{-}$), hydrogen peroxide (H_2O_2), and hydroxyl radical (OH^{-}). Increased ROS content can affect biomolecules and can cause oxidative damage to nucleic acids, proteins, and lipids. Activation of antioxidant defense machinery that consists of both enzymatic and nonenzymatic components in response to SO_2 stress takes place. In *Arabidopsis thaliana*, O^{2-} generation rate and H_2O_2 content enhances with increasing SO_2 concentration and prolonged exposure duration (Li and Yi 2012). Upregulation of defense-related genes encoding antioxidant defense enzymes viz. peroxidase (POD), Glutathione peroxidase (GPX), superoxide dismutase (SOD), cytochrome P450, heat shock proteins (Hsps) and pathogenesis-related (PR) proteins in SO_2-treated Arabidopsis shoots takes place (Li and Yi 2012). SO_2-induced upregulation of POD and other genes involved in the phenyl propanoid pathway to synthesize protective substances depicts the defense effects of the alteration of secondary metabolism in SO_2-fumigated plants (Sharma and Davis 1997).

SO_2 can affect the metabolism by either accepting or donating electrons, affecting the cellular electron transport system in plants (Osmond and Avadhani 1970). Exposure to higher concentration of SO_2 can also lead to lipid peroxidation in plants (Li and Yi 2012; Shimazaki et al. 1980). ROS produced in response to prolonged exposure to air pollutants can cause extensive damage to membranes and associated molecules, including the chlorophyll pigments and enzymes in chloroplast.

Chlorophyll content of plants serves as a biomarker parameter for air pollution levels (Darrall and Jager 1984). Chlorophyll content reduces significantly in tomato leaves with brief exposure to SO_2 (Padhi et al. 2013). SO_2 can convert chlorophyll into phaeophytin with release of Mg^{2+} by lowering the pH, changing the spectral properties (Rao and Leblanc 1965). It can also inactivate many enzymes by breaking the disulfide bridges (Cecil and Wake 1962) or may even activate some hydrolytic enzymes by inducing some conformational changes (Malhotra and Hocking 1976).

Photosynthetic CO_2 fixation is also affected by SO_2, higher concentration of SO_2 can result in inhibition of Rubisco by competing with CO_2 or bicarbonate for the binding sites in RuBP carboxylase (Zeigler 1972). Bisulfites also hold the capacity to inhibit the activity of phosphoenol pyruvate (PEP) carboxylase and malate dehydrogenase (MDH) in C4 plants (Osmond and Avadhani 1970). Mukerji and Yang (1974) also confirmed the inhibition of PEP carboxylase in spinach by sulfite in a competitive manner with respect to bicarbonate. According to different studies SO_2 has been known to affect respiration as well (Gheorghe and Ion 2011; Kropff 1991; Gilbert 1986). Ballantyne (1973), showed evidence of decreased ATP synthesis in corn and bean mitochondria in response to sodium sulfite.

Accumulation of sulfite and bisulfite in cells can disturb the existing balance between incompletely oxidized sulfur compounds and sulfhydryl groups present in glutathione and cysteine that play central role in maintaining the structural integrity of proteins (Loughman 1964; McMullen 1960). It can convert disulfide enzymes into thiosulfonates and thiols (GSH) leading to its deactivation (Bailey and Cole 1959). SO_2-damaged pine needles show 2–4 times more SH content than control leaves (Grill and Esterbauer 1973). GSH content is also greatly increased in SO_2-fumigated leaves of wheat (*Triticum aestivum*) (Navari-Izzo and Izzo 1991) indicating the activation of defense response that helps to protect cell membrane lipids against peroxidation. GSH plays important role in the process of redox buffering. It also acts as co-substrate in reaction catalyzed by GPX and DHAR as shown in Table 4.1.

Enzymes involved in antioxidant defense system such as Glutathione reductase (GR) are shown to be expressed at higher levels when exposed to SO_2. Increase in level of GR when barley plants are fumigated with SO_2 shows the activation of antioxidant defense system (Navari-Izzo and Izzo 1994). Increased levels of SOD has also been observed in spinach (*Spinacia oleracea*) leaves and *Arabidopsis* exposed to SO_2 (Li and Yi 2012; Tanaka and Sugahara 1980). Increased levels of catalase in Barley were reported (Navari-Izzo and Izzo 1994) but not in *Arabidopsis* (Li and Yi 2012). Tanaka and Sugahara (1980) stated that high level of SOD activity is correlated with increased resistance to SO_2. Also SOD activity in leaves increases with long-term exposure to low concentration of SO_2.Therefore, it can be said that SO_2 exposure results in high levels of ROS which stimulates the antioxidant defense system as it may occur in case of any abiotic stress in plants.

4.4 Nitrogen-Containing Pollutants NO_X and NHy

Nitrogen dioxide (NO_2) and nitric oxide (NO) are oxides of nitrogen and generally referred as (NOx) (Mansfield and Freer-Smith 1981). Ammonia (NH_3) and ammonium (NH_4^+) are collectively referred as "NHy." NO_2 is one of the most prevalent and harmful air pollutants in atmosphere generated by anthropogenic activities. NO_2 undergoes splitting in the presence of sunlight to form ozone (O_3) and NO. When nitrogen oxides react with moisture, they form nitric acid (HNO_3) or nitrous acid (HNO_2). Nitrate and nitrite salts are formed upon neutralization of HNO_3 and HNO_2, respectively. Therefore, NOx and derivatives exist and react either as gases in the air, as acids in droplets of water or as salts. These gases, acid gases and salts together contribute to pollution effects that have been recognized and attributed to acid rain (Park et al. 2018; Blaszczak 1999).

About half of NOx are discharged in atmosphere from automobiles exhausts (Blaszczak 1999). NOx emission from combustion is primarily in the form of NO. Other stationary sources such as industrial power plant boilers, incinerators, cement and glass manufacture, petroleum refineries and iron–steel mills, all contribute to atmospheric NOx. With increased industrialization, global NOx content in atmosphere has arisen quickly in past decades. Trace amounts of other nitrogen-containing compounds such as nitrous oxide (N_2O), nitrous acid (HONO), and ammonia (NH_3) also constitute the N-containing pollutants' emission from vehicles (Colvile et al. 2001).

Leaves are the most vulnerable to acute injury owing to their most active status in exchanging gases with the surrounding atmosphere (Taylor 1973). NOx like other gaseous pollutants enter leaf mesophyll tissue through open stomata, and upon reaction with water NOx gets converted to nitric acid or nitrous acid (Park et al. 2018), which is toxic for plant tissue. The symptoms usually appear as wound on both sides of the leaves, which initially occurs between leaf veins or alongside leaf (Law and Mansfield 1982; Taylor and Eaton 1966). However, the foliar uptake of nitrogenous compounds in the form of wet deposition is through the cuticle (WHO 2000). Exposure to gaseous NH_3 (180 $\mu g/m^3$) or NH_4^+ in rainwater (5 mmol/L) damages the crystalline framework of the epicuticular wax layer of the needles of *Pseudotsuga menziesii*. But uptake of pollutants via leaf surface is slower than uptake via stomata and is significant only when the leaf surface remains wet for longer time period such as in tropical areas (Thijsse and Baas 1990).

NO_2 can enter the intercellular cavities of the leaf and dissolves in the extracellular water to form nitrate (NO_3^-) and nitrite (NO_2^-) in equal amounts (Yan et al. 2007). When atmospheric concentration is high, it is rapidly absorbed, and susceptible areas on recently matured and rapidly expanding leaves are prone to death (Taylor 1973). Irregularly shaped, dark-pigmented lesions are formed when susceptible plants are exposed for several hours at lower atmospheric concentrations. Tip burn is common in conifers needles (Gheorghe and Ion 2011). Appearance of large yellow necrotic pockets and wilting on older leaves has been reported in several studies (Yan et al. 2007; Yu et al. 1988). In case of *Arabidopsis*, yellowing of leaves is subsequently followed by death when 18.8 mg/m^3 NO_2 is supplied (Liu et al. 2015). Similarly, chlorosis in spinach leaves appears starting from lower leaves and slowly develops upward when plants are fumigated with NO_2 at 8 ppm in the light for 36 h (Yu et al. 1988). Long-term treatment with NO_2 also leads to delay in flowering in tomato plants (Pandey and Agrawal 1994) and increased level of leaf senescence and reduced stomatal conductance in rice (Maggs and Ashmore 1998). Symptoms of acute injury on bean, tomato, and tobacco seedlings induced by exposure to high concentrations ($>$4.93 $mg/in.^3$) of NO appear as necrotic lesions similar to the lesions caused by SO_2 or by excessive concentration of ozone (Taylor and Eaton 1966).

Intrinsic plant properties, nutritional status, and environmental conditions determine the fate of N that appears on the leaf surface. When given in low concentration NOx may result in growth stimulation (WHO 2000). Low light or dark conditions increase the susceptibility of leaf tissue to NO_2 injury. Extent of accumulation of nitrite in leaves decides the acute injury to plants by NO_2 (Kato et al. 1974). Light-dependent enzyme nitrite reductase (NiR) is more or less inactive in dark; therefore, exposure of plants to NO_2 in dark results in accumulation toxic levels of nitrites causing acute injury (Zeevart 1976). In light, NiR using reducing power from the photosynthetic electron transport system reduce nitrites produced from foliar absorbed NO_2 to the ammonia that is assimilated further into N-containing compounds inside plant.

Free radical $^{\bullet}N{=}O$ is a key player in the phytotoxicity of NOx (Wellburn 1990). Phytotoxic effects of nitrogen-containing compounds (NO^{2-}, NH_3, and NH_4^+) are due to their interference with the cellular acid/base homeostasis (Raven 1988). Noncyclic electron flow in photosynthesis, ATP formation by photophosphorylation, and nitrite reduction are all pH-dependent processes inside chloroplast. Therefore, disturbance of the stromal pH by pollutants like NOx can affect the normal operation of these processes (Wellburn et al. 1980).

NOx is known to inhibit photosynthesis, reduce chlorophyll content and biomass in a variety of plants at concentrations quite below those required to produce visible leaf injury as a function of NOx concentration (Chen et al. 2010; Yan et al. 2007; Pandey and Agrawal 1994; Okano et al. 1985; Spierings 1971; Hill and Bennett 1970). Iron-nitric oxide free radical complexes have been implicated as effective inhibitors of enzymatic activity in proteins containing SH groups or histidine residues which indicates that iron-containing redox agents such as ferredoxin and cytochromes involved in photosynthesis can form a complex with NO and interfere with photosynthetic electron transport chain (Woolum et al. 1968). However, complex formation is dependent upon the concentration of NO in the cell cytoplasm. This inhibition can also be explained by competition for nicotinamide adenine dinucleotide phosphate (NADPH) between carbon assimilation and nitrite reduction in chloroplast, also the strong radical nature of NO_2 results in the generation of ROS leading to activation of antioxidant defense system (Ramge et al. 1993; Sabaratnam and Gupta 1988).

NOx being an oxidant pollutant can promote ROS formation which induces oxidative damage to biological macromolecules such as proteins, nucleic acids, and lipids, resulting in the generation of ROS (Yu et al. 1988). Upon accumulation of excess ROS, antioxidant defense system is activated to avoid oxidative damage. The induction of antioxidant enzymes is a protective action of plants against NO_2 stress (Liu et al. 2015).

Increased activities of antioxidant enzyme SOD have been reported on exposure to NO_2 (Liu et al. 2015). SOD, a metalloprotein catalyzes the dismutation of superoxide radical to H_2O_2 and molecular oxygen (O_2), providing protection against superoxide radical. In *Camphora* seedlings and *Arabidopsis* shoots SOD activity increases with increasing NO_2 concentration in a dose-dependent manner. Peroxidase (POD) and catalase (CAT) are H_2O_2 scavenging enzymes in plant cells that shows enhanced activity upon exposure to NO_2 (Liu et al. 2015; Yan et al. 2007) confirming the activation of antioxidant defense system regulated by a ROS-mediated signaling pathway.

In a recent study by Sheng and Zhu (2019) on 40 different garden plants, NO_2 showed significant increase in the activity of POD which may be linked with increased ROS generation. Sheng and Zhu (2019) also reports that increased POD not only helps in fighting against ROS directly but can also contribute to overall cellular resistance against NO_2 stress by initiating the lignification and phenolic cross-linking of cell wall. Lipids are one of the main biomolecules susceptible to damage by ROS. Peroxidation of unsaturated fatty acids in membranes results in formation of Malondialdehyde (MDA) (Labudda 2013; Price et al. 1990). Therefore, cellular MDA content is an indicator of oxidative stress induced by NO_2. Significant increase in MDA content has been reported in various species in response to NO_2

(Sheng and Zhu 2019; Liu et al. 2015; Chen et al. 2010; Yan et al. 2007) supporting the increase in cellular ROS in response to NO_2 that cause damage to membrane lipids thus affecting the membrane permeability.

ASA and glutathione are major players of antioxidant defense system in plants. Therefore, an increase in these confirms the activation of antioxidant defense system. Several studies reports change in AsA and glutathione levels upon fumigation with NOx (Liu et al. 2015; Chen et al. 2010; Yan et al. 2007).

In *Brassica campestris*, exposure to 1 $\mu L\ L^{-1}$ and higher NO_2 leads to decrease in ASA contents (Yan et al. 2007). Similarly Liu et al. (2015) also reported decrease in ASA content with increment in exposure to NO_2. According to the study by Chen et al. (2010) in Camphora, similar pattern was observed, AsA content decreased significantly during the first 30 days of exposure to the NO_2 at 0.5 $\mu L\ L^{-1}$ but it showed an increase in the following 30 days supporting the fact that ascorbate-glutathione cycle worked efficiently in the second 30 days period (Yan et al. 2007).

4.5 Ozone

Ozone is an unstable but strong oxidant with triatomic form of oxygen (O_3). In the stratosphere, it absorbs some of the sun's ultraviolet radiation. However, in troposphere it is a secondary pollutant formed in the presence of sunlight by complex photochemical reactions involving its precursors such as carbon monoxide (CO), oxides of nitrogen (NOx), and volatile organic compounds (VOC). NO_2 splits into NO and O in the presence of sunlight at wavelength of 430 nm. Free oxygen atom released from this reaction combines with molecular oxygen to form ozone. NO then reacts with free radicals formed as a result of action of UV on VOC. Free radicals then convert NO into NO_2. Newly formed NO_2 then participates in the next cycle of O_3 formation. In this way whole cycle of ozone formation is continued until the VOCs are not photoreactive anymore (Ghazali et al. 2010; Price et al. 1997).

Principal source of these precursors is from industrial emission, refineries, chemical plants, fossil fuels burning, and transport sector. These precursors can be transported to long distances before they react to form ozone in the atmosphere or O_3 may directly be transported from urban areas to agricultural lands (Singh and Agrawal 2017; Saxena and Ghosh 2011). Ozone from the stratosphere might also be transported to troposphere (Forster et al. 2007; Bell and Treshow 2002). Concentration of O_3 might be higher in rural areas than urban areas due to long distance transport of O_3 or its precursors. O_3 concentration is reported to be higher on sunny, spring and summer days when enough sunlight and primary pollutants are present in troposphere (Tiwari and Agrawal 2018; Singh and Agrawal 2017).

O_3 is phytotoxic because of strong reactive nature (Saxena et al. 2019; Mudd and Kozlowski 1975). Daily exposure to O_3 at a concentration below 120 nmol mol^{-1} for days, weeks, or months are generally considered to be chronic, whereas, exposure to O_3 at a concentration between 120 and 150 nmol mol^{-1} for even few hours is considered acute (Fiscus et al. 2005; Long and Naidu 2002). Generally O_3 exposure leads to loss in yield of agriculturally important crop plants (Pell et al. 1997; Sharma and Davis 1994; Heagle 1989). In an attempt to rescue itself from O_3 stress, biomass accumulation and allocation pattern adopted by plants dictates the yield loss and cultivar's sensitivity or resistivity toward O_3 (Tiwari and Agrawal 2018; Mills et al. 2018; Mishra et al. 2013).

Wide array of investigations have been carried out to assess the impact of continuously increasing O_3 on morphology of plants (Moura et al. 2018; Zouzoulas et al. 2009; Heagle 1989). Various factors such as duration and concentration of O_3 exposure, weather conditions, genotype, type of plants and growth stage affect the level of injury caused by O_3. Some plants are more sensitive to O_3 than others, for example soybean (*Glycine max*) is one of the most O_3-sensitive agriculturally important crops while on the other hand Sorghum (*Sorghum bicolor*) appears to be relatively insensitive to O_3. While within same plant species some cultivars/varieties maybe more sensitive than others; e.g., among two cultivars of soybean, cultivar Essex is O_3 tolerant while cultivar Forrest is sensitive to O_3 (Chernikova et al. 2000). In a study by Pleijel et al. (2006), old cultivar of wheat was found to be less affected by O_3 than the modern cultivar. Monocots as a group (e.g., grain sorghum and tall fescue) appear to be less sensitive to O_3 than dicots (cotton, peanut) (Heagle 1989).

Necrosis, interveinal chlorosis, chlorotic stippling, flecking, bronzing, and reddening of the upper leaf surface, marginal and tip burn injury are the common symptoms of O_3 injury (Ueda et al. 2013; Mishra et al. 2013; Calatayud et al. 2011; Paoletti 2009; Lee et al. 1981). Following sequence of events are supposed to be responsible for these visible symptoms: (1) O_3 interaction with cellular components in leaf tissue; (2) cell collapse and localized accumulation of cellular water at the site of interaction; (3) bleached chlorophyll of the injured cell; (4) followed by the breakdown of the leaf structure around the cell (Mudd and Kozlowski 1975).

Ozone injury symptoms generally appear between the veins on the upper surface of aged and middle-aged leaves, but can also be bifacial for some species. Young plants and middle-aged leaves are more susceptible to injury caused by O_3 than mature leaves (Gheorghe and Ion 2011). In *Arabidopsis* plants, curling of leaves and 30–48% reduction in fresh and dry weights is observed with 150 and 300 ppb O_3 for 2 weeks (Sharma and Davis 1994). Interveinal chlorosis and stippling is observed in various cultivars of wheat and mung bean upon fumigation with elevated O_3 (Mishra and Agrawal 2014; Mishra et al. 2013). Spot like necrotic lesions appears on approximately 20% of the leaf area of older leaves of tobacco whereas young leaves do not show any symptoms at all (Camp et al. 1994). Lesion formation in leaves of sensitive plants in response to O_3 exposure is comparable to the hypersensitive response against biotic stress. Rapid increase in ROS, deposition of autofluorescent phenolic compounds, and pathogenesis-related proteins (PR) expression are key plant responses against O_3 stress (Overmyer et al. 2003; Schraudner et al. 1998). Grimes et al. (1983) was able to show that phenolic compounds enhance the decomposition of O_3 in the aqueous solution primarily into $^{\bullet}OH$ radical.

Since O_3 entry through the leaf cuticle is negligible (Kerstiens and Lendzian 1989), it passes mainly through stomata and is rapidly degraded in the aqueous phase of substomatal cavity to generate various forms of ROS causing oxidative stress (Booker et al. 2009). Oxidative burst as a result of O_3 decomposition exceeds the normal ROS scavenging capacity of cells leading to activation of antioxidant defense system including antioxidant enzymes and other stress-related proteins. It also affects the nitrogen metabolism, by influencing the associated enzymes, affecting the biosynthesis of amino acids (Tiwari and Agrawal 2018).

O_3 stress also affects the common physiological processes by reducing photosynthetic proteins, reduction in the photosystem (PS) II efficiency (Feng et al. 2010; Singh et al. 2009) accelerating senescence (Feng et al. 2016) and impaired reproductive development. It also affects the stomatal conductance. All of these processes lead to decreased carbon assimilation and alteration in allocation of photosynthates. As biomass is utilized in protection and repair against stress instead of getting stored which results in reduced yield (Sarkar et al. 2015; Tripathi and Agrawal 2012; Heagle 1989).

Apoplastic and symplastic antioxidant defense system is activated upon O_3 injury. Important apoplastic antioxidants include both protein as well as nonprotein components of antioxidant defense machinery. Pool of reduced apoplastic ascorbate has been proposed to be the first line of detoxification barrier against O_3. Various studies report the correlation of total ascorbic acid (AsA) content with ozone resistance (Liu et al. 2015; Dumont et al. 2014; Conklin and Barth 2004), for instance, increased levels of apoplastic ascorbate in *Sedum album* leaves as a function of O_3 concentration (Castillo and Greppin 1988). Similarly, soybean's ozone-tolerant cultivar Essex had higher levels of total AsA than the sensitive-Forrest cv. (Robinson and Britz 2000) confirming the positive relation between the two. When the natural capacity of specific cell wall components to scavenge the ROS is limited by overproduced ROS under O_3 stress, damage to the plasma membrane eventuates (Sanmartin et al. 2003). As mentioned earlier, increase in MDA content is an indicator of stress conditions in plants. MDA content increases up to 1.4-fold in rice leaves after 24 h of fumigation with O_3 implying that increase in MDA is not caused by O_3 directly, instead it is a result of other metabolic processes affected by O_3 (Ueda et al. 2013). An increase of 314.3% and 65% in MDA content is observed in wheat during jointing and heading stages of growth, respectively, when treated with high concentration of O_3 (120 ppb) (Liu et al. 2015). Higher MDA concentrations are also observed in two different cultivars of mung bean exposed to O_3 (Mishra and Agrawal 2014). All of these findings depict the positive correlation between O_3 fumigation and increase in MDA content (Feng et al. 2016; Sarkar et al. 2015; Rai et al. 2007; Iglesias et al. 2006).

Apart from nonenzymatic components of antioxidant defense machinery, the enzymatic components play a crucial role in providing resistance against the oxidative stress due to O_3. Superoxide ($O^{-}{}_2$) generated as a product of O_3 decomposition is scavenged effectively by SOD in apoplast. H_2O_2, the product of SOD activity is further decomposed into water and oxygen by POD and CAT (Lee and Bennett 1982).

Effect of O_3 (300 ppb) has also been determined on the SOD levels in *A. thaliana* after 12 h of treatment. Cu/Zn SOD (Cytosolic/chloroplast SOD) mRNA levels increased 2-3 folds than control plants (Sharma and Davis 1994). Downregulation of organellar SOD in Japanese rice leaves is reported by Ueda et al. (2013). However, cytosolic SOD showed little sensitivity to O_3 suggesting that organellar SOD are more important in response to O_3 than cytosolic SOD (Ueda et al. 2013). A significant elevation in apoplastic SOD activity is observed in the wheat cultivar Y16 exposed to O_3 (Wang et al. 2014) showing that SOD is a component of first line of defense against O_3 stress. Similarly, an increase in activities of SOD and CAT is observed when two different cultivars of rice viz. Shivani and Malviyadhan 36 are exposed to O_3 (Sarkar et al. 2015). All of these studies highlight the importance of antioxidant enzymes involved in defense against this abiotic stress.

Ozone as an air pollutant also affects the chlorophyll content of plants. Reduction in chlorophyll is an indicator of decline in photosynthetic yield. O_3 can prevent the synthesis of chlorophyll or the ROS generated by O_3 can attack the chloroplast leading to destruction of chlorophyll. A significant decrease of 34% in chlorophyll content of sugar beet (*Beta vulgaris* cv. Loretta) during exposure period of 26 days at high concentration of O_3 is observed by Köllner and Krause (2003). Chlorophyll content decreases during both young stage as well as during flowering in *Brassica napus* (cv. Licolly). More pronounced decline is observed during flowering stage. Similarly, a decline in chlorophyll content of flag leaves of old and modern wheat varieties is observed by Pleijel et al. (2006). Total chlorophyll content of Fepagro 26 variety of *Phaseolus vulgaris*is is also affected by O_3, with a significant reduction of approximately 50% of total Chl. However, O_3 does not have any effect on the chlorophyll content of other variety, i.e., Irai (Caregnato et al. 2013). Decrease in total chlorophyll content in Aleppo pine (*Pinus halepensis*) indicated the potential damage to photosynthetic machinery (Pelloux et al. 2001). All of these studies suggest that sensitivity to O_3 is species dependent which also depends upon the stage of development.

Reduced RuBisCO activity in response to O_3 plays an important role in reduction in photosynthetic rate. Photosynthetic rates declines in five varieties of winter wheat exposed to 1.5 times the normal concentration of O_3 due to lower maximum carboxylation capacity and electron transport rates (Feng et al. 2016). Rubisco activity reduced by 40% in O_3 in Aleppo pine (*Pinus halepensis*) (Pelloux et al. 2001). In another study, it is observed that Rubisco activity and quantity decline with chronic O_3 treatment throughout the lifetime of the leaves of hybrid poplar and *Raphanus sativus* L. cv. Cherry Belle (Pell et al. 1992). It is indicated that O_3 accelerates the normal process of senescence by declining both the quantity and activity of Rubisco in leaves and chronic exposure to O_3 is necessary for continuous degradation of Rubisco (Pell et al. 1992).

4.6 Fluorides

Fluorine is the most electronegative element that belongs to halogen group of elements. It is an abundant element in earth's crust that reacts easily with most compounds. Because of its high reactivity it combines easily with other elements such as sodium, calcium, and aluminum and occurs as stable fluoride or fluorine compounds in nature (Gheorghe and Ion 2011). Fluorides are defined as any combination of elements containing the fluorine atom in the -1 oxidation state.

The primary source of fluoride in the atmosphere is the result of numerous anthropogenic activities such as aluminum and uranium smelters and industries related to steel, brick, ceramic, and glass manufacturing. Fluoride compounds are also used in toothpastes and mouthwash as a protective agent against tooth decay. Fluorine emitted from these sources is extremely reactive and easily hydrolyzes in the atmosphere to form hydrogen fluoride (HF), most hazardous gaseous fluoride pollutant. HF thereafter may react with other compounds in atmosphere to form nonvolatile stable fluorides (Walna et al. 2013). Hexafluorosilicic acid (SiF_6), tetrafluoromethane (CF_4), and hydrogen fluoride (HF) are some of the gaseous fluorides. These gaseous pollutants are highly soluble and toxic in nature. HF is a hygroscopic gas which forms the clouds acidic in nature. HF being lighter than air can harm the plants far away from the source. The nonvolatile particulate fluorides pollutants include Cryolite (Na_3AlF_6), Calcium fluoride (CaF2), and ammonium fluoride (NH_4F) (Gheorghe and Ion 2011). Solubility of these particulate fluorides is species dependent, for example, sodium fluoride is water soluble on the other hand while cryolite is insoluble in water.

Fluoride is considered as the most phytotoxic air pollutant in terms of the minimum atmospheric concentrations required to cause injury in plants which is comparable to the peroxyacetylnitrate, a nitrogen-based pollutant (Weinstein 1983). Fluoride can cause injury to sensitive plants at a concentration of 10–1000 times lesser than those of other major air pollutants (less than 1 ppb) (Unsworth and Ormrod 1982). On the other hand, the threshold concentration for O_3 and SO_2 to cause an irreversible injury is found to be generally above 0.05 ppm (Panda 2015).

Other HF-like air pollutants can enter the leaf tissue via stomata and dissolve in the water crossing the cell wall. Morphological symptom "tip burn" or marginal necrosis of the leaves is associated with fluorides-based pollutants (Fornasiero 2001; Mudd and Kozlowski 1975; Jacobson et al. 1966). Young and expanding leaves tend to be most susceptible to HF fumigation. Plant foliage can accumulate fluorides entering in its tissues for a long time which then translocates toward tip and margin of the leaves because the water in the leaf has a tendency to move toward the site of greatest evaporation, therefore appearing as marginal and tip injury (Baunthiyal et al. 2014). Due to the tendency of foliage to accumulate the fluoride, it can cause injury even at very low atmospheric concentration (Panda 2015; Gheorghe and Ion 2011). According to Baunthiyal et al. (2014), crucial factor that determines the severity of injury is the accumulation of fluoride at the active sites in toxic levels.

Chlorosis in response to fluoride injury in the affected area is generally followed by the desiccation, change in color, and finally necrosis. Fully grown and middle-aged leaves appear to be more sensitive to damage by fluoride, whereas younger and old leaves only show tip burns occasionally (Weinstein and Alscher-Herman 1982). Marginal yellowing appears earlier in the primary and older leaves of soybean which is followed by necrosis upon exposure to 15–20 nL/L fluoride concentration (Poovaiah and Wiebe 1973). Appearance of randomly scattered red areas on the entire lamina of fully expanded mature leaves of *Hypericum perforatum* is observed growing in the areas close to ceramics industry. Apparently symptoms appear on both adaxial and abaxial surface of leaves (Fornasiero 2003). Some plants are more sensitive to fluoride than others. Peach fruits, conifers, sweet corn, gladiolus, and apricot are susceptible to damage by fluoride. On the other hand, cucumber, eggplant, tobacco, and wheat show resistance to fluoride damage (Heather 2003).

All physiological and metabolic processes are affected by high internal fluoride concentration. Root–shoot length, stress tolerance index, vigor, biomass accumulation, and seed germination are all affected negatively (Baunthiyal et al. 2014). Spongy mesophyll and lower epidermis are prone to damage by fluoride upon its entry in leaf which is followed by disruption in the palisade cells (Panda 2015). According to the study of Yang and Miller (1963), fluoride fumigated leaves contain more reducing sugars and smaller amount of sucrose than the untreated leaves suggesting the inhibition of sucrose synthesis process which in turn might be related to decline in photosynthesis. Leaf necrosis can be related with various altered metabolic effects. Mcnulty and Newman (1957) report increased uptake of oxygen in beans and gladiolus exposed to HF. In a study by Hill et al. (1959), no direct effect of fluoride on the respiration is observed. However, an increase in oxygen uptake is directly correlated to leaf necrosis in gladiolus. Carbon dioxide assimilation decreases in gladiolus, barley, alfalfa, and cotton due to acute HF fumigation (Thomas 1958), indicating that destabilization of metabolic homeostasis takes place upon exposure to fluoride.

Plasma membrane and tonoplast are overly sensitive to fluoride. Plasma membrane ATPase appears to be sensitive to fluoride, thus affecting the normal metabolism (Giannini et al. 1987). Giannini studied the effects of fluoride on the plasma membrane ATPase of sugarbeet. Results indicated that fluoride inhibition of plasma membrane ATPase is at active sites of the enzyme and occurs in association with magnesium ion. Rakowski et al. (1995) studied the effect of fluoride fumigation on Eastern white pine (*Pinus strobus*) seedlings for 28 days. Significant decrease in activity of plasma membrane ATPase activity is observed after 28 days and it is suggested that plasma membrane might be primary target site for fluoride injury. Lipid peroxidation levels in response to fluoride stress also reflect the severity of damage caused. Sunflower (*Helianthus annus*) when exposed to high fluoride concentration shows increase in lipid peroxidation with increasing fluoride concentration and time of exposure (Saleh and Abdel-Kader 2003).

Concerning the activation of antioxidant machinery against fluoride stress, several studies have attempted to dissect the antioxidant defense system. But most of them focus on the fluoride received by plants via soil or hydroponics; therefore, it can be expected that plants will respond in the similar way when plants are fumigated with fluoride.

Fornasiero (2003) studied the effect of fluoride treatments on the activity of SOD in *H. perforatum.* SOD activity decreases after 2 h of fluoride fumigation which continues to decrease in the following hours as well. It is speculated that the antioxidant defense system is unable to efficiently remove the harmful ROS that results in the appearance of visible injuries. A decrease in SOD activity is observed in the sunflower (*Helianthus annus*) at the seedling growth stage (Saleh and Abdel-Kader 2003) and Li et al. (2011) showed that the activity of total SOD decreased significantly with increasing fluoride concentration in tea (*Camelllia Sinensis*) leaves. However, some studies also report increase in the SOD activity in response to the fluoride. For instance, Wilde and Yu (1998) observed an increase in mitochondrial SOD activity in mung bean seedlings. Similarly, Chakrabarti and Patra (2015) reported 75% increase in SOD activity in paddy grains and about 60% increase in roots and leaves. CAT enzyme also shows similar behavior in the presence of fluoride. CAT activity can also decline up to 80% with increasing fluoride concentration in rice (*Oryza sativa* L.) (Chakrabarti and Patra 2015). It is hypothesized that hydroxyl ions (OH^-) attached to iron atoms in catalase are replaced by low molecular weight anions causing inhibition of its activity (Kumar et al. 2009). In contrast, CAT activity increases at low fluoride concentrations and starts to decrease at higher concentrations in *C. sinensis* (Zheng et al. 2011). Further, nonenzymatic component of defense machinery, i.e., ascorbic acid also shows a tendency to decrease because of binding of F^- ion with ascorbic acid oxidase enzyme that delays the inhibition of destruction of ascorbic acid (Sharma 2018; Saleh and Abdel-Kader 2003).

4.7 Effect of Particulate Matter

Particulate matter (PM) is an air contaminant of industrial and urban areas which appears as a complex mixture of solid and liquid particles suspended in air that have different physical and chemical properties. These PMs are emitted by anthropogenic activities and natural sources. Anthropogenic activities such as coal and oil burning, motor vehicle exhaust fumes, road dust, metal and construction industrial activities are major source of introducing PM into the atmosphere. On the other hand, natural processes such as volcanic eruptions, rock weathering, forest fires, and sandstorms also release toxic PMs into the atmosphere (Prajapati 2012; Bosco et al. 2005). Another important source of PM could be nucleation, condensation, or coagulation of gaseous pollutants such as SO_2, NOx, NH_3, and VOCs (Sonwani and Kulshrestha 2018; Sonwani et al. 2016a, b; Dzierżanowski et al. 2011).

These atmospheric PMs have been classified based on their aerodynamic diameter as PM10 (<10 μm diameter), PM2.5 (<2.5 μm diameter), and ultrafine particles (UP) (<0.2 μm) (Dietz and Herth 2011; Prajapati et al. 2006; NEPC 1998; Beckett et al. 1998; Schwartz et al. 1996). Toxic PM, especially PM2.5 and UP, often comprises toxic components such as heavy metals, PAHs, polychlorinated biphenyls (PCBs), black carbon, and other carcinogenic compounds (Sonwani et al. 2016a; Dzierżanowski et al. 2011; Ariola et al. 2006; Yu et al. 2006). Sulfate, nitrate, ammonium, organic matter, and elemental carbon are regarded as the main PM contributors (Daresta et al. 2015).

Growth and development of plants can be affected by PM either by direct deposition on above ground biomass or indirectly via soil–root interactions (Žalud et al. 2012). These particle depositions onto vegetation surface occur via three major pathways: (1) wet deposition, (2) dry deposition, and (3) occult deposition. Wet deposition is a result of addition of atmospheric particles and gases into cloud droplets by nucleation followed by precipitation as rain and snow (Lovett 1994; Grantz et al. 2003). Precipitation amount and ambient particulate concentrations largely determine wet deposition. Magnitude of wet deposition is directly determined by rainfall and snowfall. Surface properties of leaves such as wettability, exposure, and roughness affect the contact with PM by strongly influencing liquid retention (Sonwani and Kulshrestha 2019; Grantz et al. 2003; Neinhuis and Barthlott 1998). Rate of dry deposition of atmospheric particles on plants and soil is considerably slower than wet or occult deposition; however, it acts constantly and affects all exposed surfaces of plants including those that are not currently physiologically active (Hicks 1986; U.S. EPA 1982). Steady deposits of grease films, dry dusts, elemental carbon encrustations, and heterogeneous secondary particles formed from gaseous precursors on vegetation can be observed (U.S. EPA 1982; Grantz et al. 2003). Leaf age, orientation, surface geometry, phyllotaxy roughness, and wettability of the leaf surface affect the dust interception and hence its retention (Prajapati 2012; Beckett et al. 2000; Neinhuis and Barthlott 1998). Occult deposition is driven by fog, cloud-water, and mist interception. Gaseous pollutant species can dissolve in the suspended water droplets of fog and clouds. Concentration of PM is usually manyfold higher in cloud or fog water than in precipitation or ambient air in the same location. Foliar surface receives PM in a hydrated form by fog and cloud water thus making it more bioavailable to plants (Grantz et al. 2003; Prajapati 2012).

Generally, the effect of PM on vegetation is associated with the reduced irradiation for photosynthesis, stomatal aperture blockage, reduction in leaf area and number, abrasion, altered pigment and mineral content and an increase in leaf temperature linked with change in surface optical properties (Rai 2016; Prajapati 2012; Kuki et al. 2008; Grantz et al. 2003; Hirano et al. 1995). Rough leaf surface receives more PM than leaves with smooth surface and inert particulate interact with plants in a purely physical manner (Hwang et al. 2011; Farmer 2002; Beckett et al. 2000). Alkaline dust material such as limestone can cause the leaf surface injury while other PM may cross the cuticle (Brandt and Rhoades 1972). Also the ionic form of chemicals can enter the mesophyll through the stomatal aperture or fissures in the cuticle by aerial deposition (Kuki et al. 2008; Watmough et al. 1999) which results in the accumulation of essential and nonessential elements in the plants (Pavlík et al. 2012; Lau and Luk 2001). Another possible root for metabolic uptake of PM is through the rhizosphere which is of great importance as large-scale sustained exposure of soil to PM occurs through wet and dry depositions of trace elements (Prajapati 2012; Voutsa et al. 1997).

Exposure of the lettuce plants to PM results in reduced leaves dry matter, increased concentration of trace elements in the above ground parts (Pavlík et al. 2012). Similarly, maize and soybean upon treatment with fly ash (a waste product of coal-fired electric generating plants) at the rate of 8 g m^{-2} day^{-1} for 30 days shows reduction in pigment content and dry matter production (Mishra and Shukla 1986).

Delayed seed germination, decreased chlorophyll content, root and shoot weight, inhibition of root elongation, and enhanced lateral root formation are observed when tomato plants are exposed to PM10 (Daresta et al. 2015). Tree species also show reduced biomass and chlorophyll content in response to dust accumulation (Younis et al. 2013; Prajapati and Tripathi 2008; Prusty et al. 2005). Similarly, cement dust pollution affects the growth and morphology. Significant delay in germination, reduced number of leaves, flower and fruits and visible marking on leaves such as chlorosis, necrosis and mottling appear as common symptoms of cement dust pollution (Katiyar et al. 2015; Rawat and Katiyar 2015; Ade-Ademilua and Obalola 2008; Kumar et al. 2008). Reduced growth of plants in response to PM can be attributed to the excessive uptake and accumulation of trace elements and alkalinity due to presence of excessive soluble salts on the leaf surface. Furthermore, the toxic effect of PM is augmented by the shading effect on leaf surface. Decrease in chlorophyll content can either be due to direct influence of PM on their biosynthesis or by indirect PM-induced destruction. Increased ROS levels in response to PM may also contribute to chlorophyll destruction (Daresta et al. 2015). Heavy metals such as Pb, Cd, and Cr accumulation by leafy vegetables, pine needles and grasses have been reported extensively (Žalud et al. 2012; Voutsa et al. 1997; Hovmand et al. 1983; Tjell et al. 1979). Trace elements in PM may contribute to decrease in chlorophyll content and chlorophyll a/b ratio of plants (Khudsar et al. 2004).

Presence of heavy metals may cause oxidative stress either by inducing ROS generation within subcellular compartments or by decreasing the enzymatic or nonenzymatic antioxidants because of their affinity with sulfur-containing group (-SH) hastening the senescence (Rai 2016; Gupta et al. 2011).

It is evident from various studies that exposure of plants to PM creates stressful conditions for plants which causes them to activate antioxidant defense system. A significant increase in ascorbic acid content in leaves of trees growing on the roadside in central Varanasi is observed in response to PM (Prajapati and Tripathi 2008). Being an antioxidant, as discussed in previous sections, it helps in combating the stress conditions and preventing leaf senescence. Atmospheric PM10 also triggers ROS production in tomato roots (Daresta et al. 2015).

Dust pH can also alter the pH of plants. For instance, cement dust on hydration releases calcium hydroxide which can increase the alkalinity of leaf surface up to pH 12. This alkaline environment can cause hydrolysis of lipids and wax components, penetrate the cuticle and denature proteins, leading to plasmolysis of leaf (Rai 2016; Guderian 1986; Czaja 1960, 1961, 1962).

Simulated acid rain and iron ore dust deposition in *Eugenia uniflora* (Surinam cherry) causes decrease in net photosynthesis, transpiration, stomatal conductance and chlorophyll a content (Neves et al. 2009). Decrease in CAT and SOD activities and injuries to membrane as depicted by high electrolyte leakage value in response to simulated acid rain in *E. uniflora* show the conditions of severe oxidative stress. Similarly, activity loss of CAT and SOD in leaves of *Clusia hilariana* (Clusia) in response to solid iron PM depicts high oxidative stress (Pereira et al. 2009) because of accumulation of toxic levels of iron that leads to inhibition of enzyme activity (Schutzendubel and Polle 2002). Decrease in total soluble protein contents in response to heavy metal toxicity in various plants such as *Platanus orientalis* (Oriental plane) and *Acer negundo* (Boxelder) can be attributed to disturbed protein synthesis mechanism and protein breakdown (Doğanlar and Atmaca 2011). Heavy metals induced lipid peroxidation and fragmentation of proteins due to highly reactive ROS may lead to reduced protein content (Solanki and Dhankhar 2011). Reduced photosynthetic rate in lettuce in response to PM has been shown to limit the supply of metabolic energy for nitrogen assimilation. Moreover, nitrate reductase activity is subjected to inhibition by trace elements (Nagajyoti et al. 2010).

PM affects the general plant growth and physiology as confirmed by extensive research from across the world. With increase in automobiles and industries plants growing near the roads and industries often face stressful conditions. Use of PM-tolerant plant species can be an effective approach to purify the ambient air. However, selection of tolerant and accumulator species depends upon the location and type of PM found in the particular area.

4.8 Summary

Implacable rise in pollutants has seriously deteriorated the quality of air and will continue to do so in coming future. This has imposed serious damaging effects on health of living organisms. In particular, plants are one of the main targets of the air pollutants causing reduction in the yield of crop plants and trees. Various air pollutants such as SO_2, NOx, ozone, PM, and fluorides have direct effect on growth and development of plants. Fluoride can cause injury in plants even at very low concentrations of less than 1 ppb in sensitive plants due to its highly electronegative nature. On the other hand, O_3 upon entry inside cell decomposes causing an oxidative burst. Nitrogen-containing compounds (NO^{2-}, NH_3, and NH_4^+) exert their phytotoxic effects by interfering with the cellular acid/base homeostasis. SO_2-induced injury is a result of generation of more toxic forms such as sulfite and bisulfite in the cellular cytoplasm which causes acidification of cytoplasmic pH affecting the various metabolic processes in cell. The negative effect of these major air pollutants results in altered morphology which is a consequence of altered metabolism. Necrosis, chlorosis, and tip burns appear to be the common symptoms in plants when exposed to these air pollutants. When exposed to pollutants, ROS production takes place that has deleterious effects on plant health which includes destruction of pigments, membrane lipids and proteins, decreased photosynthetic yield, stomatal and leaf injury. In order to protect themselves from the deleterious effects of these pollutants, plants use complex antioxidant defense system which consists of protein and nonprotein components that work together actively against ROS. Variety of studies has been conducted to understand the effect of the individual air pollutants on plant health; however, in natural environment plants are constantly being exposed to combination of these pollutants. Therefore, it becomes important to dissect the underlying mechanisms involved in protection against combination of these air pollutants.

References

Ade-Ademilua OE, Obalola DA (2008) The effect of cement dust pollution on *Celosia argentea* (Lagos Spinach) plant. J Environ Sci Technol 1(2):47–55

Agrawal M, Singh B, Rajput M et al (2003) Effect of air pollution on peri-urban agriculture: a case study. Environ Pollut 126:323–329

Ariola V, D'Alessandro A, Lucarelli F, Marcazzan G, Mazzei F, Nava S, Garcia-Orellana I, Prati P, Valli G, Vecchi R, Zucchiatti A (2006) Elemental characterization of PM10, PM2.5 and PM1 in the town of Genoa (Italy). Chemosphere 62(2):226–232

Bailey JL, Cole RD (1959) Studies on the reaction of sulfite with proteins. J Biol Chem 234 (7):1733–1739

Ballantyne DJ (1973) Sulphite inhibition of ATP formation in plant mitochondria. Phytochemistry 12(6):1207–1209

Baunthiyal M, Bhatt A, Ranghar S (2014) Fluorides and its effects on plant metabolism. Int J Agric Technol 10:1–27

Beckett KP, Freer-Smith PH, Taylor G (1998) Urban woodlands: their role in reducing the effects of particulate pollution. Environ Pollut 99(3):347–360

Beckett KP, Freer-Smith PH, Taylor G (2000) Particulate pollution capture by urban trees: effect of species and windspeed. Glob Chang Biol 6(8):995–1003

Bell JNB, Treshow M (eds) (2002) Air pollution and plant life. Wiley, Chichester

Bita C, Gerats T (2013) Plant tolerance to high temperature in a changing environment: scientific fundamentals and production of heat stress-tolerant crops. Front Plant Sci 4:273

Blaszczak RJ (1999) Nitrogen oxides (NOx): why and how they are controlled. EPA-456/F-99e006R, US Environmental Protection Agency

Blokhina O, Fagerstedt KV (2010) Reactive oxygen species and nitric oxide in plant mitochondria: origin and redundant regulatory systems. Physiol Plant 138(4):447–462

Booker F, Muntifering R, Mcgrath M et al (2009) The ozone component of global change: potential effects on agricultural and horticultural plant yield, product quality and interactions with invasive species. J Integr Plant Biol 51:337–351

Bosco ML, Varrica D, Dongarra G (2005) Case study: inorganic pollutants associated with particulate matter from an area near a petrochemical plant. Environ Res 99:18–30

Brandt CJ, Rhoades RW (1972) Effects of limestone dust accumulation on lateral growth of forest trees. Environ Pollut 3:213–217

Bytnerowicz A, Omasa K, Paoletti E (2007) Integrated effects of air pollution and climate change on forests: a northern hemisphere perspective. Environ Pollut 147(3):438–445

Calatayud V, Cerveró J, Calvo E, García-Breijo FJ, Reig-Armiñana J, Sanz MJ (2011) Responses of evergreen and deciduous Quercus species to enhanced ozone levels. Environ Pollut 159 (1):55–63

Caregnato FF, Bortolin RC, Junior AMD, Moreira JCF (2013) Exposure to elevated ozone levels differentially affects the antioxidant capacity and the redox homeostasis of two subtropical *Phaseolus vulgaris* L. varieties. Chemosphere 93(2):320–330

Castillo FJ, Greppin H (1988) Extracellular ascorbic acid and enzyme activities related to ascorbic acid metabolism in *Sedum album* L. leaves after ozone exposure. Environ Exp Bot 28 (3):231–238

Caverzan A, Casassola A, Brammer SP (2016) Reactive oxygen species and antioxidant enzymes involved in plant tolerance to stress. Abiotic and biotic stress in plants. Recent advances and future perspectives, pp 463–480

Cecil R, Wake RG (1962) The reactions of inter-and intra-chain disulphide bonds in proteins with sulphite. Biochem J 82(3):401

Chakrabarti S, Patra PK (2015) Biochemical and antioxidant responses of paddy (*Oryza sativa* L.) to fluoride stress. Fluoride 48:56–61

Chen CP, Frank TD, Long SP (2009) Is a short, sharp shock equivalent to long-term punishment? Contrasting the spatial pattern of acute and chronic ozone damage to soybean leaves via chlorophyll fluorescence imaging. Plant Cell Environ 32:327–335

Chen Z, Chen Y, Du G et al (2010) Effects of 60-day NO2 fumigation on growth, oxidative stress and antioxidative response in *Cinnamomum camphora* seedlings. J Zhejiang Univ 11:190–199

Chernikova T, Robinson JM, Lee EH, Mulchi CL (2000) Ozone tolerance and antioxidant enzyme activity in soybean cultivars. Photosynth Res 64(1):15–26

Chew O, Whelan J, Millar AH (2003) Molecular definition of the ascorbate-glutathione cycle in Arabidopsis mitochondria reveals dual targeting of antioxidant defenses in plants. J Biol Chem 278(47):46869–46877

Colvile RN, Hutchinson EJ, Mindell JS, Warren RF (2001) The transport sector as a source of air pollution. Atmos Environ 35(9):1537–1565

Conklin PL, Barth C (2004) Ascorbic acid, a familiar small molecule intertwined in the response of plants to ozone, pathogens, and the onset of senescence. Plant Cell Environ 27:959–970

Czaja AT (1960) Die Wirkung von verstäubtem Kalk und Zement auf Pflanzen. Plant Foods for Human Nutrition (Formerly Qualitas Plantarum) 7(2):84–212

Czaja AT (1961) Zementstaubwirkungen auf pflanzen: Die entstehung der zementkrusten. Qualitas plantarum et materiae vegetabiles 8(2):201–238

Czaja A (1962) Über das Problem der Zementstaubwirkung auf Pflanzen. Staub 22:228–232

Daresta BE, Italiano F, de Gennaro G, Trotta M, Tutino M, Veronico P (2015) Atmospheric particulate matter (PM) effect on the growth of *Solanum lycopersicum* cv. Roma plants. Chemosphere 119:37–42

Darrall NM, Jager HJ (1984) Biochemical diagnostic tests for the effects of air pollution on plants. In: Koziol MJ, Whatley FR (eds) Gaseous air pollutants and plant metabolism. Butterworth, London, pp 333–350

Das K, Roy Choudhury A (2014) Reactive oxygen species (ROS) and response of antioxidants as ROS-scavengers during environmental stress in plants. Front Environ Sci 2:53

Dietz K, Herth S (2011) Plant nanotoxicology. Trends Plant Sci 16:582–589

Doğanlar ZB, Atmaca M (2011) Influence of airborne pollution on Cd, Zn, Pb, Cu, and Al accumulation and physiological parameters of plant leaves in Antakya (Turkey). Water Air Soil Pollut 214:509–523

Dumont J, Keski-Saari S, Keinänen M, Cohen D, Ningre N, Kontunen-Soppela S, Jolivet Y (2014) Ozone affects ascorbate and glutathione biosynthesis as well as amino acid contents in three Euramerican poplar genotypes. Tree Physiol 34(3):253–266

Dzierżanowski K, Popek R, Gawrońska H et al (2011) Deposition of particulate matter of different size fractions on leaf surfaces and in waxes of urban forest species size fractions on leaf surfaces and in waxes. Int J Phytoremed 13:1037–1046

Emberson LD, Ashmore MR, Murray F, Kuylenstierna JC, Percy KE, Izuta T, Zheng Y, Shimizu H, Sheu BH, Liu CP, Agrawal M (2001) Impacts of air pollutants on vegetation in developing countries. Water Air Soil Pollut 130(1–4):107–118

Farmer A (2002) Effects of particulates. Air Pollut Plant Life 187

Feng Z, Pang J, Nouchi I, Kobayashi K, Yamakawa T, Zhu J (2010) Apoplastic ascorbate contributes to the differential ozone sensitivity in two varieties of winter wheat under fully open-air field conditions. Environ Pollut 158(12):3539–3545

Feng Z, Wang L, Pleijel H et al (2016) Differential effects of ozone on photosynthesis of winter wheat among cultivars depend on antioxidative enzymes rather than stomatal conductance. Sci Total Environ 572:404–411

Fiscus EL, Booker FL, Burkey KO (2005) Crop responses to ozone: uptake, modes of action, carbon assimilation and partitioning. Plant Cell Environ 28:997–1011

Fornasiero RB (2001) Phytotoxic effects of fluorides. Plant Sci 161:979–985

Fornasiero RB (2003) Fluorides effects on *Hypericum perforatum* plants: first field observations. Plant Sci 165:507–513

Forster P, Ramaswamy V, Artaxo P, Berntsen T, Betts R, Fahey DW, Haywood J, Lean J, Lowe DC, Myhre G, Nganga J (2007) Changes in atmospheric constituents and in radiative forcing. Chapter 2. In: Climate change. The physical science basis

Ghazali NA, Ramli NA, Yahaya AS, Yusof NFFM, Sansuddin N, Al Madhoun WA (2010) Transformation of nitrogen dioxide into ozone and prediction of ozone concentrations using multiple linear regression techniques. Environ Monit Assess 165(1–4):475–489

Gheorghe IF, Ion B (2011) The effects of air pollutants on vegetation and the role of vegetation in reducing atmospheric pollution. In: The impact of air pollution on health, economy, environment and agricultural sources. InTech, pp 241–280

Giannini JL, Pushnik JC, Briskin DP, Miller GW (1987) Fluoride effects on the plasma membrane ATPase of sugarbeet. Plant Sci 53(1):39–44

Gilbert OL (1986) Field evidence for an acid rain effect on lichens. Environ Pollut Ser A Ecol Biol 40(3):227–231

Gill SS, Tuteja N (2010) Reactive oxygen species and antioxidant machinery in abiotic stress tolerance in crop plants. Plant Physiol Biochem 48:909–930

Grantz DA, Garner JHB, Johnson DW (2003) Ecological effects of particulate matter. Environ Int 29:213–239

Grimes HD, Perkins KK, Boss WF (1983) Ozone degrades into hydroxyl radical under physiological conditions. Plant Physiol 72:1016–1020

Guderian R (1986) Terrestrial ecosystems: particulate deposition. In: Legge AH, Krupa SV (eds) Air pollutants and their effects on the terrestrial ecosystem, Advances in environmental science and technology, vol 18. Wiley, New York, pp 339–363

Gupta S, Nayek S, Bhattacharya P (2011) Effect of air-borne heavy metals on the biochemical signature of tree species in an industrial region, with an emphasis on anticipated performance index. Chem Ecol 27(4):381–392

Heagle AS (1989) Ozone and crop yield. Annu Rev Phytopathol 27(1):397–423

Heather G (2003) Air pollution on agricultural crops. Factsheet, No 85-002

Heyno E, Mary V, Schopfer P, Krieger-Liszkay A (2011) Oxygen activation at the plasma membrane: relation between superoxide and hydroxyl radical production by isolated membranes. Planta 234(1):35–45

Hicks BB (1986) Differences in wet and dry particle deposition parameters between North America and Europe. Aerosols: research, risk assessment, and control strategies. Lewis Publishers, Chelsea, MI, pp 973–982

Hill AC, Bennett JH (1970) Inhibition of apparent photosynthesis by nitrogen oxides. Atmos Environ 4:341–348

Hill AC, Pack MR, Transtrum LG, Winters WS (1959) Effects of atmospheric fluorides and various types of injury on the respiration of leaf tissue. Plant Physiol 34(1):11

Hirano T, Kiyota M, Aiga I (1995) Physical effects of dust on leaf physiology of cucumber and kidney bean plants. Environ Pollut 89:255–261

Horsman DC, Wellburn AR (1977) Effect of SO2 polluted air upon enzyme activity in plants originating from areas with different annual mean atmospheric SO2 concentrations. Environ Pollut (1970) 13(1):33–39

Hovmand MF, Tjell JC, Mosbæk H (1983) Plant uptake of airborne cadmium. Environ Pollut 30:27–38

Hwang HJ, Yook SJ, Ahn KH (2011) Experimental investigation of submicron and ultrafine soot particle removal by tree leaves. Atmos Environ 45(38):6987–6994

Iglesias DJ, Calatayud Á, Barreno E, Primo-Millo E, Talon M (2006) Responses of citrus plants to ozone: leaf biochemistry, antioxidant mechanisms and lipid peroxidation. Plant Physiol Biochem 44(2–3):125–131

Jacobson JS, Weinstein LH, Mccune DC et al (1966) The accumulation of fluorine by plants. J Air Pollut Control Assoc 16:412–417

Katiyar R, Rawat V, Tandon PK (2015) Responses of cement dust on morphological and biochemical parameters of mung bean (*Vigna radiata* L.). J Biol Chem Research 32(1):142–151

Kato T, Tachibana S, Inden T (1974) Studies on the injury of crops by toxic gases in covering structure (II). Environ Control Biol 12(4):103–107

Kerstiens G, Lendzian KJ (1989) Interactions between ozone and plant cuticles. 1. Ozone deposition and permeability. New Phytol 112:13–19

Khudsar T, Iqbal M, Sairam RK (2004) Zinc-induced changes in morpho-physiological and biochemical parameters in *Artemisia annua*. Biol Plant 48(2):255–260

Köllner B, Krause GHM (2003) Effects of two different ozone exposure regimes on chlorophyll and sucrose content of leaves and yield parameters of sugar beet (*Beta Vulgaris* L.) and rape (*Brassica Napus* L.) B. Water Air Soil Pollut 144:317–332

Kozioł MJ, Whatley FR (2016) Gaseous air pollutants and plant metabolism. Butterworth-Heinemann, London

Kropff MJ (1991) Long-term effects of SO2 on plants, metabolism and regulation of intracellular pH. Plant Soil 131:235–245

Kuki KN, Oliva MA, Pereira EG, Costa AC, Cambraia J (2008) Effects of simulated deposition of acid mist and iron ore particulate matter on photosynthesis and the generation of oxidative stress in *Schinus terebinthifolius Radii* and *Sophora tomentosa* L. Sci Total Environ 403 (1–3):207–214

Kulshrestha U, Saxena P (eds) (2016) Plant responses to air pollution. Springer, Singapore

Kumar SS, Singh NA, Kumar V, Sunisha B, Preeti S, Deepali S, Nath SR (2008) Impact of dust emission on plant vegetation in the vicinity of cement plant. Environ Eng Manag J7(1):31–35

Kumar KA, Varaprasad P, Rao AVB (2009) Effect of fluoride on catalase, guiacol peroxidase and ascorbate oxidase activities in two verities of mulberry leaves (*Morus alba* L.). Res J Earth Sci 1:69–73

Labudda M (2013) Lipid peroxidation as a biochemical marker for oxidative stress during drought. An effective tool for plant breeding

Lau OW, Luk SF (2001) Leaves of *Bauhinia blakeana* as indicators of atmospheric pollution in Hong Kong. Atmos Environ 35:3113–3120

Law RM, Mansfield TA (1982) Oxides of nitrogen and the greenhouse atmosphere. In: Effects of gaseous air pollution in agriculture and horticulture, vol 5. Butterworths, London

Lee EH, Bennett JH (1982) Superoxide dismutase: a possible protective enzyme against ozone injury in snap beans (*Phaseolus vulgaris* L.). Plant Physiol 69:1444–1449

Lee EH, Bennett JH, Heggestad HE (1981) Retardation of senescence in red clover leaf discs by a new antiozonant, N-[2-(2-oxo-1-imidazolidinyl) ethyl]-N′-phenylurea. Plant Physiol 67 (2):347–350

Legge AH, Krupa SV (2002) Effects of sulphur dioxide. Air Pollut Plant Life 2:135–162

Lewandowska M, Sirko A (2008) Recent advances in understanding plant response to sulfur-deficiency stress. Acta Biochim Pol 55(3):457–471

Li L, Yi H (2012) Plant physiology and biochemistry effect of sulfur dioxide on ROS production, gene expression and antioxidant enzyme activity in Arabidopsis plants. Plant Physiol Biochem 58:46–53

Li C, Zheng Y, Zhou J et al (2011) Changes of leaf antioxidant system, photosynthesis and ultrastructure in tea plant under the stress of fluorine. Biol Plant 55:563–566

Liu X, Hou F, Li G, Sang N (2015) Effects of nitrogen dioxide and its acid mist on reactive oxygen species production and antioxidant enzyme activity in Arabidopsis plants. J Environ Sci 34:93–99

Long SP, Naidu SL (2002) Effects of oxidants at the biochemical, cell and physiological levels, with particular reference to ozone. In: Bell JNB, Treshow M (eds) Air pollution and plant life. Wiley, West Sussex, pp 69–88

Loughman BC (1964) Sulphur absorption and metabolism in plants. Agrochimica 8:189

Lovett GM (1994) Atmospheric deposition of nutrients and pollutants in North America: an ecological perspective. Ecol Appl 4(4):629–650

Maggs R, Ashmore MR (1998) Growth and yield responses of Pakistan rice (*Oryza sativa* L.) cultivars to O3 and NO2. Environ Pollut 103:159–170

Malhotra SS, Hocking D (1976) Biochemical and cytological effects of sulphur dioxide on plant metabolism. New Phytol 76:227–237

Mansfield TA, Freer-Smith P (1981) Effects of urban air pollution on plant growth. Biol Rev 56:343–368

Marshall F, Bell JNB, Stonehouse J, Wildig Z, Agarwal M, Mukherjee N et al (1999) The impacts and policy implication of air pollution on agriculture in urban and peri-urban areas of developing countries: a case study from India. UK Department for International Development, London

Mclaughlin SB, Mcconathy RK (1983) Effects of SO2 and O3 on allocation of 14C-labeled photosynthate in *Phaseolus vulgaris*. Plant Physiol 73(3):630–635

McMullen AI (1960) Thiols of low molecular weight in *Hevea brasiliensis* latex. Biochim Biophys Acta 41(1):152–154

Mcnulty IB, Newman DW (1957) Effects of atmospheric fluoride on the respiration rate of bush bean and gladiolus leaves. Plant Physiol 32(2):121

Mehlhorn H, Seufert G, Schmidt A, Kunert KJ (1986) Effect of SO2 and O3 on production of antioxidants in conifers. Plant Physiol 82:336–338

Mills G, Pleijel H, Malley CS, Sinha B, Cooper OR, Schultz MG et al (2018) Tropospheric ozone assessment report: present-day tropospheric ozone distribution and trends relevant to vegetation. Elem Sci Anth 6:47

Mishra AK, Agrawal SB (2014) Biochemical and physiological characteristics of tropical mung bean (*Vigna radiata* L.) cultivars against chronic ozone stress: an insight to cultivar-specific response. Protoplasma 252(3):797–811

Mishra LC, Shukla KN (1986) Effects of fly ash deposition on growth, metabolism and dry matter production of maize and soybean. Environ Pollut Ser A Ecol Biol 42(1):1–13

Mishra AK, Rai R, Agrawal SB (2013) Field crops research differential response of dwarf and tall tropical wheat cultivars to elevated ozone with and without carbon dioxide enrichment: growth, yield and grain quality. Field Crop Res 145:21–32

Mittler R (2002) Oxidative stress, antioxidants and stress tolerance. Trends Plant Sci 7(9):405–410

Mittler R, Vanderauwera S, Gollery M, Van Breusegem F (2004) Reactive oxygen gene network of plants. Trends Plant Sci 9(10):490–498

Moura BB, Alves ES, Marabesi MA, de Souza SR, Schaub M, Vollenweider P (2018) Ozone affects leaf physiology and causes injury to foliage of native tree species from the tropical Atlantic Forest of southern Brazil. Sci Total Environ 610:912–925

Mudd JB, Kozlowski TT (1975) Responses of plants to air pollution. Academic Press, New York

Mukerji SK, Yang SF (1974) Phosphoenolpyruvate carboxylase from spinach leaf tissue. Plant Physiol 53:829–834

Muneer S, Kim TH, Choi BC et al (2014) Effect of CO, NOx and SO2 on ROS production, photosynthesis and ascorbate – glutathione pathway to induce Fragaria X annasa as a hyperaccumulator. Redox Biol 2:91–98

Nagajyoti PC, Lee KD, Sreekanth TVM (2010) Heavy metals, occurrence and toxicity for plants: a review. Environ Chem Lett 8(3):199–216

Navari-Izzo F, Izzo R (1991) Induction of enzyme activity in plants of wheat as result of SO2 fumigation. Gior Bot Ital 125:636–637

Navari-Izzo F, Izzo R (1994) Induction of enzyme activities and antioxidant production in barley plants as a result of SO2 fumigation. Plant Sci 96(1–2):31–40

Neill S, Desikan R, Hancock J (2002) Hydrogen peroxide signalling. Curr Opin Plant Biol 5 (5):388–395

Neinhuis C, Barthlott W (1998) Seasonal changes of leaf surface contamination in beech, oak, and ginkgo in relation to leaf micromorphology and wettability. The New Phytologist 138(1):91–98

NEPC (1998) Ambient air quality: National Environment Protection measure for ambient air quality. National Environment Protection Council Service Corporation, Adelaide

Neves NR, Oliva MA, Centeno C et al (2009) Science of the total environment photosynthesis and oxidative stress in the restinga plant species *Eugenia uniflora* L. exposed to simulated acid rain and iron ore dust deposition: potential use in environmental risk assessment. Sci Total Environ 407:3740–3745

Okano K, Totsuka T, Fukuzawa T, Tazaki T (1985) Growth responses of plants to various concentrations of nitrogen dioxide. Environ Pollut Ser A Ecol Biol 38:361–373

Olszyk DM, Tibbitts TW (1981) Stomatal response and leaf injury of *Pisum sativum* L. with SO2 and O3 exposures I. Influence of pollutant level and leaf maturity. Plant Physiol 67:539–544

Olszyk DM, Tingey DT (1984) Phytotoxicity of air pollutants: evidence for the photodetoxification of SO2 but not O3. Plant Physiol 72:999–1005

Osmond CB, Avadhani PN (1970) Inhibition of the β-carboxylation pathway of CO2 fixation by bisulfite compounds. Plant Physiol 45(2):228

Overmyer K, Brosché M, Kangasjärvi J (2003) Reactive oxygen species and hormonal control of cell death. Trends Plant Sci 8:335–342

Padhi SK, Dash M, Swain SC (2013) Effect of sulphur dioxide on growth, chlorophyll and sulphur contents of tomato (*Solanum lycopersicum* L.). Eur Scientific J 9(36)

Panda D (2015) Fluoride toxicity stress: physiological and biochemical consequences on plants. Int J Bio-Res Environ Agric Sci 1:70–84

Pandey J, Agrawal M (1994) Growth responses of tomato plants to low concentrations of sulphur dioxide and nitrogen dioxide. Sci Hortic (Amsterdam) 58:67–76

Pandey P, Singh J, Achary VMM, Reddy MK (2015) Redox homeostasis via gene families of ascorbate-glutathione pathway. Front Environ Sci 3:1–14

Paoletti E (2009) Ozone and urban forests in Italy. Environ Pollut 157(5):1506–1512

Park J, Ahn J, Kim K, Son Y (2018) Historic and futuristic review of electron beam technology for the treatment of SO2 and NOx in flue gas. Chem Eng J 355:351–366

Pavlík M, Pavlíková D, Zemanová V et al (2012) Trace elements present in airborne particulate matter — stressors of plant metabolism. Ecotoxicol Environ Saf 79:101–107

Pell EJ, Eckardt N, Enyedi AJ (1992) Timing of ozone stress and resulting status of ribulose bisphosphate carboxylase/oxygenase and associated net photosynthesis. New Phytol 120:397–405

Pell EJ, Schlagnhaufer CD, Arteca RN (1997) Ozone-induced oxidative stress: mechanisms of action and reaction. Physiol Plant 100:264–273

Pelloux J, Jolivet Y, Fontaine V et al (2001) Changes in Rubisco and Rubisco activase gene expression and polypeptide content in *Pinus halepensis* M. subjected to ozone and drought. Plant Cell Environ 24:123–131

Pereira EG, Oliva MA, Kuki KN, Cambraia J (2009) Photosynthetic changes and oxidative stress caused by iron ore dust deposition in the tropical CAM tree *Clusia hilariana*. Trees 23:277–285

Pleijel H, Eriksen AB, Danielsson H et al (2006) Differential ozone sensitivity in an old and a modern Swedish wheat cultivar — grain yield and quality, leaf chlorophyll and stomatal conductance. Environ Exp Bot 56:63–71

Poovaiah BW, Wiebe HH (1973) Influence of hydrogen fluoride fumigation on the water economy of soybean plants. Plant Physiol 51:396–399

Prajapati SK (2012) Ecological effect of airborne particulate matter on plants. Environ Skept Critics 1:12–22

Prajapati SK, Tripathi BD (2008) Seasonal variation of leaf dust accumulation and pigment content in plant species exposed to urban particulates pollution. J Environ Qual 37(3):865–870

Prajapati SK, Pandey SK, Tripathi BD (2006) Monitoring of vehicles derived particulates using magnetic properties of leaves. Environ Monit Assess 120(1–3):169–175

Price A, Lucas PW, Lea PJ (1990) Age dependent damage and glutathione metabolism in ozone fumigated barley: a leaf section approach. J Exp Bot 41(10):1309–1317

Price C, Penner J, Prather M (1997) NOx from lightning 2. Constraints from the global atmospheric electric circuit. J Geophys Res 102:5943–5951

Prusty BAK, Mishra PC, Azeez PA (2005) Dust accumulation and leaf pigment content in vegetation near the national highway at Sambalpur, Orissa, India. Ecotoxicol Environ Saf 60:228–235

Qu Y, An J, He Y, Zheng J (2016) An overview of emissions of SO2 and NOx and the long-range 2 transport of oxidized sulfur and nitrogen pollutants in East Asia. J Environ Sci 44:13–25

Rai PK (2016) Impacts of particulate matter pollution on plants: implications for environmental biomonitoring. Ecotoxicol Environ Saf 129:120–136

Rai R, Agrawal M, Agrawal SB (2007) Assessment of yield losses in tropical wheat using open top chambers. Atmos Environ 41(40):9543–9554

Rai R, Rajput M, Agrawal M, Agrawal SB (2011) Gaseous air pollutants: a review on current and future trends of emissions and impact on agriculture. J Sci Res 55:77–102

Rakowski KJ, Zwiazek JJ, Sumner MJ (1995) Hydrogen fluoride effects on plasma membrane composition, ATPase activity and cell structure in needles of eastern white pine (*Pinus strobus*) seedlings. Trees 9(4):190–194

Ramge P, Badeck FW, Plöchl M, Kohlmaier GH (1993) Apoplastic antioxidants as decisive elimination factors within the uptake process of nitrogen dioxide into leaf tissues. New Phytol 125:771–785

Rao DN, Leblanc F (1965) Effects of sulfur dioxide on the lichen alga, with special reference to chlorophyll. Bryologist 69:69–75

Raven JA (1988) Acquisition of nitrogen by the shoots of land plants: its occurrence and implications for acid–base regulation. New Phytol 109:1–20

Rawat V, Katiyar R (2015) A review on the effects of cement dust on vegetation. Int J Sci Innov Res Stud 3:39–45

Riding RT, Percy KE (1985) Effects of SO2 and other air pollutants on the morphology of epicuticular waxes on needles of *Pinus strobus* and *Pinus banksiana*. New Phytol 99 (4):555–563

Robinson JM, Britz SJ (2000) Tolerance of a field grown soybean cultivar to elevated ozone level is concurrent with higher leaflet ascorbic acid level, higher ascorbate-dehydroascorbate redox status, and long term photosynthetic productivity. Photosynth Res 64(1):77–87

Robinson E, Robbins RC (1970) Gaseous sulfur pollutants from urban and natural sources gaseous sulfur pollutants from urban and natural sources. J Air Pollut Control Assoc 20(4):233–235

Sabaratnam S, Gupta G (1988) Effects of nitrogen dioxide on leaf chlorophyll and nitrogen content of soybean. Environ Pollut 51:113–120

Saleh A, Abdel-Kader DZ (2003) Metabolic responses of two *Helianthus annus* cultivars to different fluoride concentration during germination and seedling growth stages. Egypt J Biol 5:43–54

Sanmartin M, Drogoudi PD, Lyons T et al (2003) Over-expression of ascorbate oxidase in the apoplast of transgenic tobacco results in altered ascorbate and glutathione redox states and increased sensitivity to ozone. Planta 216:918–928

Sarkar A, Singh AA, Agrawal SB, Ahmad A, Rai SP (2015) Cultivar specific variations in antioxidative defense system, genome and proteome of two tropical rice cultivars against ambient and elevated ozone. Ecotoxicol Environ Saf 115:101–111

Saxena P, Ghosh C (2011) Variation in the concentration of ground level ozone at selected sites in Delhi. Int J Environ Sci 1(7):1899–1911

Saxena P, Kulshrestha U (2016a) Biochemical effects of air pollutants on plants, Plant responses to air pollution. Springer, Singapore, pp 59–70

Saxena P, Kulshrestha UC (2016b) The impact of gasoline emission on plants–a review. Chem Ecol 32(4):378–405

Saxena P, Sonwani S, Kulshrestha U (2017a) Impact of tropospheric ozone and particulate matter on plant health. In: Pandey D, Sarkar A (eds) Sustaining future food security. Nova Publisher, New York, pp 19–60

Saxena P, Sharma Y, Chugh M (2017b) Assessment of ozone phytotoxic potential of dense vegetation cover of Delhi by AOT40. In: AGU Fall Meeting Abstracts, December

Saxena P, Srivastava A, Tyagi M, Kaur S (2019) Impact of tropospheric ozone on plant metabolism – a review. Pollut Res 38(1):175–180

Schraudner M, Moeder W, Wiese C, Camp WV, Inzé D, Langebartels C, Sandermann H Jr (1998) Ozone-induced oxidative burst in the ozone biomonitor plant, tobacco Bel W3. Plant J 16 (2):235–245

Schutzendubel A, Polle A (2002) Plant responses to abiotic stresses: heavy metal-induced oxidative stress and protection by mycorrhization. J Exp Bot 53(372):1351–1365

Schwartz J, Dockery DW, Neas LM (1996) Is daily mortality associated specifically with fine particles? J Air Waste Manage Assoc 46(10):927–939

Sharma R (2018) Insights into fluoride-induced oxidative stress and antioxidant defences in plants. Acta Physiol Plant 40:181

Sharma YK, Davis KR (1994) Ozone-induced expression of stress-related genes in *Ara bidopsis thaliana*. Plant Physiol 105:1089–1096

Sharma YK, Davis KR (1997) The effects of tissues resist oxidative stress: the importance of ozone on antioxidant responses in plants. Free glutathione S-transferase and glutathione peroxidase. Radicals Biol Med 23:480–488

Sharma P, Jha AB, Dubey RS, Pessarakli M (2012) Reactive oxygen species, oxidative damage, and antioxidative defense mechanism in plants under stressful conditions. J Bot 2012:1–26

Sheng Q, Zhu Z (2019) Effects of nitrogen dioxide on biochemical responses in 41 garden plants. Plants 8:45

Shimazaki K, Sakaki T, Kondo N, Sugahara K (1980) Active oxygen participation in chlorophyll destruction and lipid peroxidation in SO2 ~ fumigated leaves of spinach. Plant Cell Physiol 21:1193–1204

Singh AA, Agrawal SB (2017) Tropospheric ozone pollution in India: effects on crop yield and product quality. Environ Sci Pollut Res 24:4367–4382

Singh E, Tiwari S, Agrawal M (2009) Effects of elevated ozone on photosynthesis and stomatal conductance of two soybean varieties: a case study to assess impacts of one component of predicted global climate change. Plant Biol 11:101–108

Sirohi S, Chauhan S, Singhal S, Srivastava A (2018) Effect of air pollution on the carotenoids and ascorbic acid content in different medicinal and non-medicinal plants. J Energy Environ Carbon Credit 8(2):7–11

Solanki R, Dhankhar R (2011) Biochemical changes and adaptive strategies of plants under heavy metal stress. Biologia (Bratisl) 66:195–204

Sonwani S, Kulshrestha U (2018) Morphology, elemental composition and source identification of airborne particles in Delhi, India. J Indian Geophys Union 22(6):607–620

Sonwani S, Kulshrestha UC (2019) PM 10 carbonaceous aerosols and their real-time wet scavenging during monsoon and non-monsoon seasons at Delhi, India. J Atmos Chem 76:171–200

Sonwani S, Amreen H, Khillare PS (2016a) Polycyclic aromatic hydrocarbons (PAHs) in urban atmospheric particulate of NCR, Delhi, India. In: 41st COSPAR Scientific Assembly, vol 41

Sonwani S, Saxena P, Kulshrestha U (2016b) Role of global warming and plant signaling in BVOC emissions. In: Plant responses to air pollution. Springer, Singapore, pp 45–57

Spierings FHFG (1971) Influence of fumigations with NO2 on growth and yield of tomato plants. Neth J Plant Pathol 77:194–200

Stratigakos A, Ormorod DP (1985) Response of tomato to sulphur nutrition and SO2. Water Air Soil Pollut 247(1):19–26

Suzuki N, Koussevitzky SHAI, Mittler RON, Miller GAD (2012) ROS and redox signalling in the response of plants to abiotic stress. Plant Cell Environ 35(2):259–270

Taiz L, Zeiger E (2002) Plant physiology. Sinauer, Sunderland, MA, p 690

Tanaka K, Sugahara K (1980) Role of superoxide disxnutase in defense against SO2 toxicity and an increase in superoxide dismutase activity with SO2 fumigation. Plant Cell Physiol 21:601–611

Taylor OC (1973) Acute responses of plants to aerial pollutants. Adv Chem Ser (US) 122:9–20

Taylor C, Eaton FM (1966) Suppression of plant growth by nitrogen dioxide. Plant Physiol 41:132–135

Taylor GE Jr (1978) Plant and leaf resistance to gaseous air pollution stress. New Phytol 80 (3):523–534

Thijsse G, Baas P (1990) "Natural" and NH3-induced variation in epicuticular needle wax morphology of *Pseudotsuga menziesii* (Mirb.) Franco. Trees 4(3):11–119

Thomas MD (1958) Air pollution with relation to agronomic crops: I. General status of research on the effects of air pollution on plants. Agron J 50(9):545–550

Thomas MD (1961) Effect of air pollution on plants in "Air Pollution". W.H.O (Geneva) Monoger 46:233–278

Thomas MD, Tiendricks RH, Agrh TRC (1943) The utilization of sulphate and sulphur dioxide for the sulphur nutrition of alfalfa. Plant Physiol 18:345

Tiwari S, Agrawal M (2018) Tropospheric ozone and its impacts on crop plants: a threat to future global food security. Springer

Tiwari S, Agrawal M, Marshall FM (2006) Plants at a sub urban site using open top chambers. Environ Monit Assess 119:15–30

Tjell JC, Hovmand MF, Mossbaek H (1979) Atmospheric lead pollution of grass grown in a background area of Denmark. Nature (London) 280(5721):425–426

Treshow M (1971) Fluorides as air pollutants affecting plants. Annu Rev Phytopathol 9:21–44

Tripathi R, Agrawal SB (2012) Effects of ambient and elevated level of ozone on Brassica campestris L. with special reference to yield and oil quality parameters. Ecotoxicol Environ Saf 85:1–12

U.S. Environmental Protection Agency (1982) Air quality criteria for particulate matter and sulfur oxides. EPA Report No. EPA-600/8-82-029aF-Cf, vol 3. Environmental Criteria and Assessment Office, U.S. Environmental Protection Agency, Research Triangle Park (NC)

Ueda Y, Uehara N, Sasaki H et al (2013) Impacts of acute ozone stress on superoxide dismutase (SOD) expression and reactive oxygen species (ROS) formation in rice leaves. Plant Physiol Biochem 70:396–402

Unsworth MH, Black VJ (1981) Stomatal responses to pollutants. Stomatal Physiol 191

Unsworth MH, Ormrod DP (1982) Effects of gaseous air pollution in agriculture and horticulture (No. 32). Butterworth-Heinemann, London

Van Camp W, Willekens H, Bowler C, Van Montagu M, Inzé D, Reupold-Popp P, Sandermann H Jr, Langebartels C (1994) Elevated levels of superoxide dismutase protect transgenic plants against ozone damage. Biotechnology 12:165

Von Grill D, Esterbauer H (1973) Cystein und Glutathion in gesunden und SO2-geschädigten Fichtennadeln. Eur J For Pathol 3(2):65–71

Voutsa D, Grimanis A, Samara C (1997) Trace elements in vegetables grown in an industrial area in relation to soil and air particulate matter. Environ Pollut 94:325–335

Walna B, Kurzyca I, Bednorz E, Kolendowicz L (2013) Fluoride pollution of atmospheric precipitation and its relationship with air circulation and weather patterns. Environ Monit Assess 185:5497–5514

Wang J, Zeng Q, Zhu J, Chen C, Liu G, Tang H (2014) Apoplastic antioxidant enzyme responses to chronic free-air ozone exposure in two different ozone-sensitive wheat cultivars. Plant Physiol Biochem 82:183–193

Watmough SA, Hutchinson TC, Evans RD (1999) The distribution of 67 Zn and 207 Pb applied to white spruce foliage at ambient concentrations under different pH regimes. Environ Exp Bot 41:83–92

Wei A, Fu B, Wang Y et al (2015) Ecotoxicology and Environmental Safety Involvement of NO and ROS in sulfur dioxide induced guard cells apoptosis in *Tagetes erecta*. Ecotoxicol Environ Saf 114:198–203

Weinstein LH (1983) Effects of fluorides on plants and plant communities: an overview. Fluorides-effects on vegetation, animals and humans, pp 53–59

Weinstein LH, Alscher-Herman R (1982) Physiological responses of plants to fluoride. In: Unsworth MH, Omrmrod DP (eds) Effects of gaseous air pollution on agriculture and horticulture

Wellburn AR (1990) Tansley Review No. 24 Why are atmospheric oxides of nitrogen usually phytotoxic and not alternative fertilizers? New Phytol 115(3):395–429

Wellburn AR, Wilson J, Aldridge P (1980) Biochemical responses oxide polluted of plants to nitric atmospheres. Environ Pollut Ser A Ecol Biol 22:219–228

WHO (2000) Chapter 11. Effects of nitrogen containing air pollutants: critical levels, pp 1–28

Wilde LG, Yu M (1998) Effect of Fluoride on Superoxide Dismutase (SOD) activity in germinating mung bean seedlings. Fluoride 31(2):81–88

Woolum JC, Tiezzi E, Commoner B (1968) Electron spin resonance of iron-nitric oxide complexes with amino acids, peptides and proteins. Biochim Biophys Acta (BBA)-Protein Struct 160 (3):311–320

Yan Chun MA, Xin XU, Lid HA, Jun CAO (2007) Nitrogen dioxide-induced responses in Brassica campestris seedlings: the role of hydrogen peroxide in the modulation of antioxidative level and induced resistance. Agric Sci China 6:1193–1200

Yang SF, Miller GW (1963) Biochemical studies on the effect of fluoride on higher plants. 2. The effect of fluoride on sucrose-synthesizing enzymes from higher plants. Biochem J 88(3):509

Younis U, Bokhari TZ, Malik SA et al (2013) Variations in leaf dust accumulation, foliage and pigment attributes in fruiting plant species exposed to particulate pollution from Multan. Int J Agric Sci Res 3:1–12

Yu S, Li L, Shimazaki K (1988) Response of spinach and kidney bean plants to nitrogen dioxide. Environ Pollut 55:1–13

Yu L, Mai B, Meng X, Bi X, Sheng G, Fu J, Peng PA (2006) Particle-bound polychlorinated dibenzo-p-dioxins and dibenzofurans in the atmosphere of Guangzhou, China. Atmos Environ 40(1):96–108

Žalud P, Száková J, Sysalová J, Tlustoš P (2012) Factors influencing uptake of contaminated particulate matter in leafy vegetables. Cent Eur J Biol 7:519–530

Zeevaart AJ (1976) Some effects of fumigating plants for short periods with NO2. Environ Pollut (1970) 11(2):97–108

Zeigler I (1972) The effect of $SO_3\ ^-$ on the activity of ribulose-1,5-diphosphate carboxylase in isolated spinach chloroplasts. Planta 103:155–163

Zouzoulas D, Spyridon DK, Vassiliou G, Vardavakis E (2009) Effects of ozone fumigation on cotton (*Gossypium hirsutum* L.) morphology, anatomy, physiology, yield and qualitative characteristics of fibers. Environ Exp Bot 67:293–303

Chapter 5
Role of Global Climate Change in Crop Yield Reductions

Gyan Prakash Gupta

Abstract Uncontrolled emission of greenhouse gases (GHGs) leads to global warming and climate change. It is progressively changing at an alarming rate in the coming future. Increasing global warming is responsible for the difference in temperature, frequency of precipitation, drought events, and heat waves. By the end of the twenty-first century, the CO_2 crosses the concentration more than 600–1000 ppm, and it increases the temperature by 1–2 °C in tropical and subtropical countries. It is anticipated that food grain production would decline up to 30% depending on the plant group (C3 and C4 plant). This chapter deals with how C3 and C4 crop plant responds to elevated CO_2 and higher temperature. Increasing concentration of atmospheric CO_2 and higher temperature will promote or decrease crop growth period, development, quality, and yield. The various physiological processes like photosynthesis, respiration, and stomatal conductance are the sole mechanisms for endorsing crop growth. C3 crops grown from ambient (360 ppm) to high (720 ppm) CO_2 concentrations initially enhances the net CO_2 fixation and growth by nearly 30% but later on it reduced in photorespiration processes. Hence, CO_2 acclimation lowers down the overall shoot nitrogen concentrations. Later on, this led to a reduction in protein content and ultimately affected the plant growth rate and biomass, whereas even under the ambient CO_2, the C4 plant assimilation capability becomes saturated. The higher temperature will be responsible for heat shock injury as well as biochemical and physiological changes. Subsequently, it reduced grain production and yield depending on the geographical place. The higher temperature influences and maintains the equilibrium between C3 photosynthetic carbon assimilation and photorespiration process. It is predicted that after the interaction of atmospheric CO_2 and temperature under experimental conditions, C3 plants more favored under elevated CO_2 whereas, C4 plant more favored under higher temperature. There is a need for mitigation and adaptation strategies to improve agricultural crop production and minimizes the production risk for sustainable development.

G. P. Gupta (✉)
Department of Botany, DA-V (PG) College, CSJM University, Kanpur, Uttar Pradesh, India
e-mail: greet.gyan12@gmail.com

P. Saxena, A. Srivastava (eds.), *Air Pollution and Environmental Health*,
Environmental Chemistry for a Sustainable World 20,
https://doi.org/10.1007/978-981-15-3481-2_5

Keywords GHGs · Climate change · Global warming · C3 and C4 plant productivity · Elevated CO_2 and Temperature

5.1 Introduction

Climate change, as a result of global warming are progressively changing at an alarming rate due to augmented emission of greenhouse gases (GHGs) such as CO_2, CH_4, and N_2O from anthropogenic sources (fossil fuel combustion) (IPCC 2014). The foremost anthropogenic GHG is CO_2 which cover 76% of the total concentration and 16%, 6.2%, and 2.0% from CH_4, N_2O, and CFC gases, respectively (IPCC 2014). During the period of times, its deposition in the atmosphere alters the atmospheric concentrations of GHGs pollutants. It increases infrared absorption radiation, which is redirected from the earth's surface and imbalances the total energy of our ecological system, however progressively causing the atmosphere warmer, so that increasing global warming contributes to global climate change (i.e., increased temperature, frequency of precipitation, drought events, and heat waves) (Venkataramanan 2011; IPCC 2013). It was reported that 45%, 35%, and 20% of CO_2 emissions were produced from coal burning, oil burning, and natural gas burning, respectively (IEA 2017). In 2017, the most significant contribution of global CO_2 emissions were 27%, 15%, 10%, and 7% from China, the United States, the EU, and India, respectively, and its cover is around 59%, while 42% contributed by rest of the countries. However, the principal controlling agent is CO_2 which is responsible for global warming. Since 1950, CO_2 concentration has raised in the environment by 30%, which is a substantial increase after the industrial revolution (Fig. 5.1). Before the preindustrial revolution (1750 AD), atmospheric CO_2 was ~280 ppm (Luthi et al. 2008). In the present scenario, April 2019 registered a daily average CO_2 concentration of 412 ppm at the Mauna Loa Observatory in Hawaii. By 2050, it is anticipated to reach between 443 and 541 ppm, whereas by the end of 2100, the range will be from 421 to 936 ppm. The warming of the earth environment increases by 0.84 °C (IPCC 2014) and the global average temperature will increase by 3.7–4.8 °C (IPCC 2014; Meinshausen et al. 2011; Hartmann et al. 2013). Therefore, instant and strategic need for collective efforts from all over the world to curb emissions to keep atmospheric CO_2 at the lower end of that range (approx 421ppm). Figure 5.1 showing the global carbon budget from the year 1870 to 2017 is an accumulative contribution from various sources and sinks. Here, 202 ppm CO_2 is emitted from the burning of fossil fuels and cement industries. These emissions were 63% higher than that of 1990, with a rate of 2.7% per year (CDIAC; Le et al. 2018a, b). If this rate continues, CO_2 emissions can surpass 100 $GtCO_2$ by the end of the twenty-first century, nearly threefold the present level of 36 $GtCO_2$/year and eventually it crosses the concentration which is more than 1000 ppm (Fuss et al. 2014). Change in land use pattern is an additional factor responsible for rising atmospheric CO_2. It contributed about 88 ppm CO_2 from 1870 to 2017 (Fig. 5.1). Whereas total CO_2 emitted and released into the environment

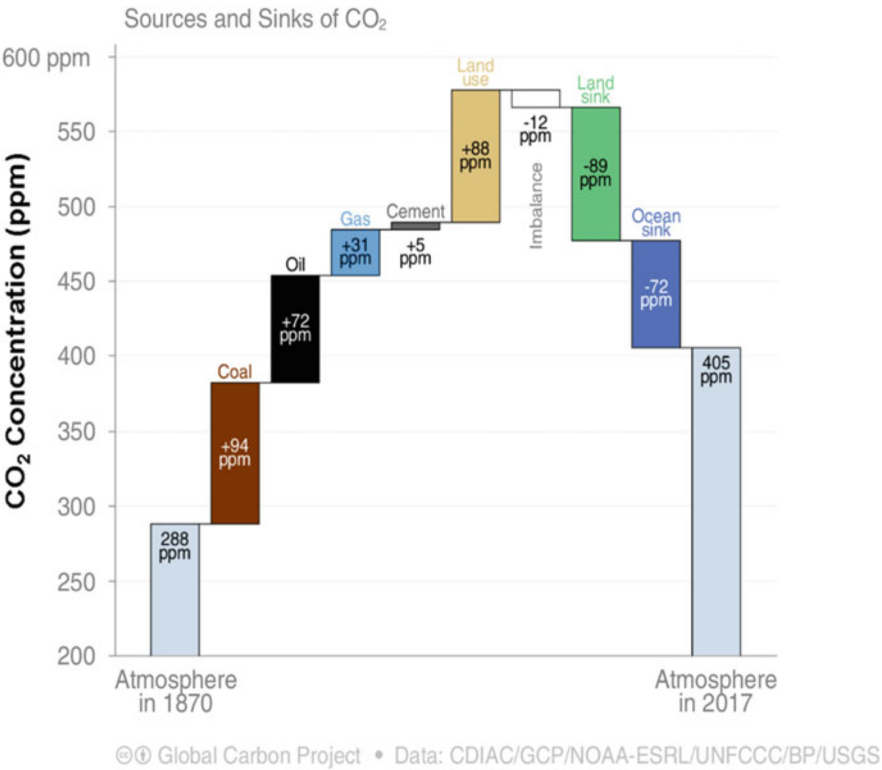


Fig. 5.1 Global carbon budget from 1870 to 2017 (Source: CDIAC; NOAA-ESRL; Houghton and Nassikas 2017; Le et al. 2018a, b)

during the last 145 years has not persisted there because of the ocean and land which act as sinks for gaseous CO_2. The land absorbs and fixes 89 ppm while oceans absorb 72 ppm CO_2 via various physiochemical processes, i.e., photosynthesis (Fig. 5.1).

Climate change raises the earth temperatures, drought and disturbed the monsoon patterns and the magnitude of air pollution that markedly affected the whole ecological function, human health as well as crop productivity of plant also (Bagley et al. 2015).

In this chapter, detailed description about C3 and C4 crop plant responses to elevated CO_2 and higher temperature is discussed. These responses are experimentally verified and tested in vitro as well as in vivo with the help of various physiological and biochemical parameters, which ultimately affected the crop yield and product quality. At the end, mitigation and adaptation strategies to improve agricultural crop production and minimize the production risk is also discussed.

5.2 Crop Response to Climate Change

In the present scenario, one of the most significant challenges is yielding ample food to meet the prerequisite of the rising world population. The issue of food security is further made intricate by climate change (Schmidhuber and Tubiello 2007; Cavagnaro et al. 2011). Elevated concentration of atmospheric CO2 at the end of the century, will promote or decrease crop growth period, development and yield which is dependent on the plant type, i.e. C3 or C4 plants (Poorter and Perez-Soba 2001; Leakey 2009). The plants are categorized into three groups based on the pathway that is used in reducing CO_2 to carbohydrate, i.e., C3, C4, and CAM plants. Photosynthesis, respiration, and stomatal conductance are the sole mechanisms for endorsing crop growth (Makino and Mae 1999). It led to morphological changes like leaf expansion, modification in shoot–root ratio, flowering, grain size, and yields (Masle 2000; Seneweera and Conroy 2005).

5.2.1 Effect of Elevated CO_2 on C3 Versus C4 Plants

By the end of the twenty-first century, atmospheric CO_2 concentrations will increase by approximately 600–1000 ppm. The response of crops to increasing atmospheric CO_2 has been experimentally studied in a control growth chamber or greenhouses, open-top chamber and also using the Free Air CO_2 Enrichment (FACE) technology.

5.2.1.1 Elevated CO_2 Affects Photosynthesis

5.2.1.1.1 C3 Versus C4 Plants

The rate of CO_2 fixation through the photosynthesis process is dependent on available intercellular CO_2 concentrations (Ci). In the plant, there are two most common pathways to fix atmospheric CO_2, i.e., C3 and C4 pathway. The key differences between these pathways are not only based on the catalyzing enzymes and intermediary products but in terms of leaf anatomical feature also. Most of the crops follow the C3 path, where CO_2 is firstly fixed by Ribulose-1,5-bisphosphate (RuBP) and catalyzed by Ribulose-1,5-bisphosphate carboxylase/oxygenase (Rubisco) enzyme (Sage et al. 1989). This carboxylation reaction produces two molecules of 3-PGA (3-phosphoglycerate), a three-carbon stable compound and hence called a C3 plant. But, Rubisco also has oxygenase activity, by fixing O_2 in a light-dependent reaction releasing CO_2 when the internal concentration of O_2 is higher than that of CO_2. This fixation cycle is known as photorespiration and evolutionary adaptation mechanism toward oxygen level (Edwards and Walker 1983). Whereas, some crops use the C4 pathway, where CO_2 is firstly reacted and fix by Phosphoenolpyruvate (PEP), formed the malate or aspartate compound. It is a

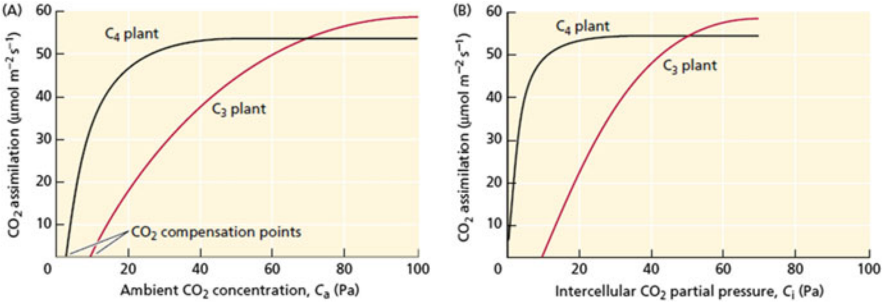


Fig. 5.2 The variation of photosynthetic carbon assimilation rate is plotted against (**a**) ambient CO_2 in air and (**b**) intercellular partial pressure of CO_2 (Ci) inside the leaf of *Tidestromia oblongifolia* (C4 plant) and *Larrea divaricata* (C3 plant) (Berry and Downton 1982; Taiz and Zeiger 2014)

four carbon stable molecule and hence called a C4 plant (Taiz and Zeiger 2014). Here, the reaction is proceeded by PEP carboxylase, which does not have bi-functionality, and it has an affinity for CO_2 only (Blanke et al. 1987). In C4 species, leaf anatomy has anatomical specialization for CO_2 fixation by spatial segregation of photosynthesis, where mesophyll cell which is located peripherally is the site of CO_2 capture whereas the C3 cycle is operated in mesophyll cells, which surrounded the bundle sheath, is called Kranz anatomy. In these cells, the C4 molecule releases the CO_2 initially fixed in outer mesophyll cell; this facilitates higher concentrations of CO_2 around Rubisco enzyme and minimizes the oxygenase activity. Therefore, C4 plants are considered more efficient than C3 plants under ambient and intercellular CO_2 level because of well-designed CO_2 fixing mechanism (Edwards and Walker 1983) (Fig. 5.2a, b).

5.2.1.1.2 Effect of Elevated CO_2 Enrichment on Crop Plant

It is reported and consistently shown that under elevated CO_2 condition it increased the photosynthetic carbon assimilation rate, therefore enhancing the growth of most plants but it depends on crops type (i.e., C3 or C4). The C3 plant has 33–40% increase as compared to C4 plant where it has only 10–15% increase in photosynthetic rate (Kimball 1983; Prior et al. 2003). But, increased CO_2 enrichment (i.e., 600–1000 ppm) can promote photosynthetic rate, especially of C3 plants. The higher atmospheric CO_2 is facilitated through the stomata and enter into the chloroplast of the mesophyll cell, where Rubisco enzyme enables carboxylation and decreases oxygenation process. Consequently, photorespiration process is hindered. Thereby, the yield of C3 crop plants is increased more than two- to threefold (Fig. 5.2a, b) (Jin et al. 2009; Lemonnier and Ainsworth 2019). Jin et al. (2009) observed that the yield of the Chinese cabbage leaf and stem lettuce increased twofold when grown between 800 and 1000 ppm CO_2. In onion, shoot and root dry mass increased under 700 ppm (Bettoni et al. 2014), whereas, in the case of Pea shoot growth and biomass production increased at 550 ppm (Butterly et al. 2016). Yields enhanced up to 44% in tomato, cucumber, and lettuce and 35% in French bean (Burkey et al. 2012; Korres et al. 2016).

In case of C4 plants, photosynthesis is saturated somewhat at the current atmospheric CO_2 level. Thus, C4 plant is not anticipated to benefit from changes in the atmospheric CO_2 levels. Leakey et al. (2009) observed that when maize plant is grown at irrespective varying concentrations of CO_2, photosynthesis rate constant (productivity) is not increased (Fig. 5.2a, b). But on the other hand, C4 crops might raise photosynthesis and yield indirectly via other factors such as anatomical adaptation, higher growth potential, and manage heat and drought stress (Leakey et al. 2009; Reich et al. 2015).

5.2.1.1.3 Implication and Mechanism of Elevated CO_2 on C3 Versus C4 Plant

C3 crops grown from ambient (360 ppm) to high (720 ppm) CO_2 concentrations initially enhance the net CO_2 fixation and growth by nearly 30% but later on they reduce photorespiration processes (Woodward 2002). However, with continuous exposures for weeks of elevated CO_2 concentrations, the net CO_2 assimilation and plant growth slowdown and limit at the rates of average 12% (Curtis 1996) and 8% (Poorter and Navas 2003), respectively, as compared to plants kept at ambient CO_2 concentrations (Bowes 1993; Moore et al. 1998; Makino and Mae 1999). Consequently, CO_2 acclimation lowers down the overall shoot nitrogen concentrations, i.e., up to 14% (Poorter and Navas 2003; Makino and Mae 1999). Later on, this led to other responses like reduction in protein content and hampering the function of photosynthesis enzymes followed by affecting the plant growth rate and biomass (Long et al. 2004; Taub and Wang 2008). The reduction of shoot nitrogen concentration is explained by Rachmilevitch et al. (2004) and Bloom et al. (2014). In their experiment, they grew *Arabidopsis*, wheat, and peas under exposure of an elevated CO_2 concentration. Initially, biomass addition accelerated in 1 week but after 3 weeks, the plant started showing N_2 deficiency symptom and prevented for flowering and within 5–7 weeks exposure plant showing significantly reduced NO_3^- reductase activity and diminishes NO_3^- assimilation in the shoots and later on plant growth got hampered. It may cause and result in catastrophic loss of C3 plant. Several experiments and observation concluded that the elevated CO_2 increases the specificity of Rubisco carboxylase or Oxygenase so that photorespiration process inhibited and decreased the yield of C3 crops because NO_3^- assimilation depends on photorespiration process (Bloom et al., 2010; Bloom et al., 2014; Kulshrestha and Saxena 2016). Thus, Rachmilevitch et al. (2004) and Bloom et al. (2014) proposed the three possible physiological mechanisms for the inhibitory effect of elevated CO_2 concentrations on NO_3^- assimilation as: (1) The photorespiration process stimulates the transport of malate from the chloroplast into the peroxisome through the cytoplasm via malate shuttle, where it formed NADH, and it reduces hydroxypyruvate, and this malate shuttle is also helpful in the production of NADH/NAD^+ ratio in the cytoplasm (Backhausen et al. 1994) and NADH plays an essential role in the conversion of NO_3^- to NO_2^- (Quesada et al. 2000); (2) transport of NO_2^- from the cytoplasm into the chloroplast stroma, and here the condition is that the stroma should be more alkaline than the cytoplasm whereas high concentrations of CO_2 somewhat increases the acidity after deposition in the stroma of the chloroplast (Shingles et al. 1996; Bloom et al. 2002); and (3) the chloroplast stroma competes for reduced ferredoxin (Fdr). It is an electron donor for the conversion of NO_2^- to NH_4^+ whereas enzyme ferredoxin-NADP reductase (FNR) has a higher affinity for Fdr than nitrite reductase (NiR) so that elevated CO_2 can be more assimilated when the production of reducing NADPH is high and therefore, NO_3^- assimilation proceeds if the availability of Fdr is more than needed for NADPH formation (Knaff 1996; Backhausen et al. 2000). Whereas, in C4 plants, NO_3^- assimilation is not affected by elevated CO_2 because the cytoplasm of mesophyll cells itself maintains a sufficient amount of malate and NADH during the first step of carboxylation reaction during the CO_2 fixation pathway. That is why NO_3^- assimilation in the shoot is independent of the elevated CO_2 (Bloom et al. 2012).

5.2.1.2 Elevated CO_2 Affects Dark Respiration

Dark respiration is explained as CO_2 release or O_2 uptake after the oxidation of substrates through the pathway of glycolysis, the oxidative pentose phosphate pathway, and the Krebs cycle pathway linked to oxidative electron transport pathways of the mitochondrion. Respiration is a catabolic pathway that generates ATP and intermediate compound to fulfill the energy requirement for plant growth and development (Wang et al. 2001; Taiz and Zeiger 2014; Saxena and Sonwani 2019a, b). It may happen in the dark or the light period (Graham 1980). At elevated CO_2, when a crop will grow at night, dark respiration will be inhibited, and daytime photosynthesis is stimulated. Hence, the photosynthesis/respiration ratio will be increased and imbalance the carbon ratio of plants as well as environment also (Mattos et al. 2014). Table 5.1 shows the direct inhibition of dark respiration rate

Table 5.1 Effect of elevated CO_2 on dark respiration in leaves of grassland species

	Dark respiration rates (nmol g^{-1} s^{-1})			
Species	*n*	360 (μmol mol^{-1})	700 (μmol mol^{-1})	% change
C3 grass				
Agropyron repens	4	15.7	16.1	2.4
Bromus inermis	5	13.5	13.5	−1.5
Koeleria cristata	4	12.4	12.4	0.1
C4 grass				
Andropogon gerardii	6	17.4	17.4	−0.3
Schizachyrium scoparium	5	15.3	14.5	−4.9
Sorghastrum nutans	6	16.8	16.5	−2.0

(+) indicates an increase, whereas (−) indicates an inhibition of respiration and their % change in leaves of grassland species after exposure of elevated and ambient CO_2, i.e., 700 and 360 μmol mol^{-1} CO_2 (Tjoelker et al. 2001).

Similarly, depending on plant species like Sugar maple (Burton et al. 1997), Douglas fir (Qi et al. 1994), and Eastern white pine (Clinton and Vose 1999), dark respiration rates are observed to decline (Peet and Wolfe 2000; Hamilton et al. 2001; Griffin et al. 2001) or remain unchanged in Citrus and French beans (Bouma et al. 1997; Jahnke 2001) while in another species like soybean, dark respiration increases (Leakey et al. 2009a).

5.2.1.2.1 Mechanism for Dark Respiration Inhibition

There are two plausible mechanisms proposed for dark respiration inhibition under elevated CO_2:

(a) Suppression of dark assimilation of CO_2 due to inhibition of PEP carboxylase enzyme (Gonzàlez-Meler et al. 1996; Van der Westhuizen and Cramer 1998).
(b) The activity of succinate dehydrogenase and cytochrome c oxidase enzymes are inhibited (Gonzàlez-Meler and Siedow 1999).

5.2.1.3 Elevated CO_2 Affects Stomatal Conductance, Water Use Efficiency, and Transpiration

Stomatal conductance (gs) measures the rate of CO_2 uptake and loss of water through the stomata. It depends on the density, size, and degree of opening of the stomata. For example, the extra open stomata permits higher conductance which leads to higher photosynthesis and transpiration rates. Bisbis et al. (2018) explain the conductance of close and open stomata under different environmental conditions. They showed stomatal conductance through the pictorial diagram of the lower surface leaf stomata and their interaction with various environmental factors like CO_2, temperature, water supply, and drought (Fig. 5.3). The stomatal conductance would be: (i) normal stomatal opening at ambient CO_2 concentration (400 ppm), i.e., regular gas exchange rate and water use efficiency (WUE); (ii) partial stomatal closure at elevated CO_2 level (500–900 ppm), i.e., decreased gas exchange resulted

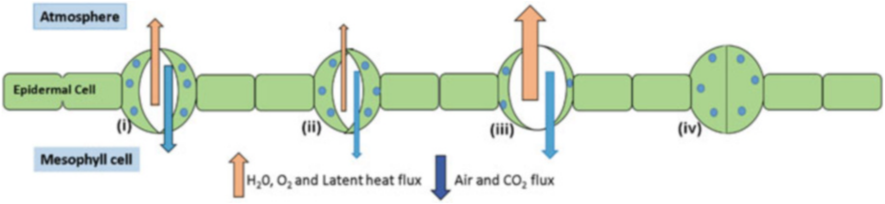


Fig. 5.3 Schematic diagram of stomatal conductance (gs) under different environmental conditions (i.e., CO_2, temperature, and water)

in enhanced WUE and reduced transpiration; (iii) maximum stomatal opening at high temperature and adequate water flow, i.e., excessive transpiration and decreased WUE; and (iv) total stomatal closure at elevated temperature and drought, i.e., minimize transpiration losses to conserve water label inside the mesophyll cell.

At elevated CO_2, gs is generally reduced in most of the plants (Wand et al. 1999; Sonwani and Saxena 2016). There are various significant physiological and ecological consequences which lead to gs reduction, e.g., lower gs may change plant water label by reducing transpiration rate, increasing photosynthesis, and promoting the WUE. Consequently, it increases the productivity of many plants, especially of arid and semiarid regions (Owensby et al. 1999; Smith et al. 2000).

The mechanism behind is that at elevated atmospheric CO_2 levels (>400 ppm), a rise in leaf intercellular CO_2 (Ci) is shown. The internal Ci rapidly increases abscisic acid (ABA) in the guard cell within minutes, and this facilitates signal for reducing stomatal conductance (Mott 2009; Engineer et al. 2015). Carrot crop reduced their stomatal conductance by 17% and 53% at 650 and 1050 ppm, respectively, but CO_2 assimilation increased by 43% and 52% at 650 and 1050 ppm, respectively, in a growth chamber (Kyei-Boahen et al. 2003). In this way, elevated CO_2 leads to increase of WUE, but it enables crops to be more susceptible to heat shock too (Engineer et al. 2015).

5.2.1.4 Elevated CO_2 Affects Product Quality and Yields

Elevated CO_2 affects the product quality as well as yields of many crop plants (Saxena and Naik 2018). Table 5.2 shows the influence of elevated CO_2 through changes in various physiological and metabolic processes that commence to the difference in the biochemical compounds like carbohydrate, protein, fatty acid, secondary metabolite, vitamins as well as a significant reduction and increase of micro- and macronutrients in different parts of the plants (Gruda 2009; Wang and Frei 2011). Becker and Klaring (2016) reported that the red leaf lettuce cultivated at elevated CO_2 (1000 ppm) has more concentration of caffeic acid, flavonoids, and sugars. Antioxidant compound (vitamin C) increased in the leaf and stem of celery, Chinese cabbage, and lettuce whereas soluble sugar increased in Chinese cabbage (Jin et al. 2009). However, several authors also reported a substantial reduction and increase in macro- and micronutrients in different crops (Pal et al. 2003; Shimono and Bunce 2009). Under elevated CO_2, the requirement and uptake of nutrients like N and P significantly increased because of vast amounts of these nutrients required in the photosynthetic and other metabolic processes (Ghannoum and Conroy 2007). In case of *Oryza*, total N uptake increased in the plant species, but N concentration decreased at the leaf level (Yang et al. 2007a; Ainsworth et al. 2007). In the case of P, the level increased (Yang et al. 2007b), lowered, or remained unaffected (Seneweera et al. 1994) whereas the level of Mg unchanged and Ca ion concentration increased in the leaf (Seneweera 2011).

Various studies also showed that elevated CO_2 substantially promoted the yield of different crops (Table 5.3). Maize yield increased by 50% (Rogers et al. 1983),

Table 5.2 Effect of elevated CO_2 on crop quality

Biochemical parameter	Experimental plant	CO_2 conc.	Effect	References
Sugars	Chinese cabbage red leaf lettuce	800–1000 ppm 1000 ppm	↑	Jin et al. (2009) Becker and Klaring (2016)
Protein	Maize	550 ± 20 ppm	↓	Abebe et al. (2016)
Antioxidant compounds				
Flavonoid, phenols	Leaf spinach, lettuce	700–1000 ppm	↑	Becker and Klaring (2016); Giri et al. (2016)
Ascorbic acid	Leaf and stem celery, lettuce, Chinese cabbage	800–1000 ppm	↑	Jin et al. (2009)
Macronutrients				
N, P, K, S, Mg	Spinach, lettuce	700 ppm	↓	Giri et al. (2016)
N, P, K	Maize	550 ± 20 ppm	↓	Abebe et al. (2016)
Ca	Rice	700 ppm	↑	Seneweera (2011)
Micronutrients (Cu, Zn)	Lettuce, spinach	700 ppm	↓	Giri et al. (2016)
NO_3 content	Leaf and stem Chinese cabbage, lettuce, celery	800–1000 ppm	↓	Jin et al. (2009)

27% (Cure and Acock 1986), and 22.9% (Meng et al. 2014) at elevated CO_2. Similarly, maize and sorghum grain yield increased by 18% at elevated CO_2 (550 ppm) (Long et al. 2006).

5.3 Effect of Higher Temperature on C3 and C4 Plants

5.3.1 *Temperature*

It is a significant factor for crop's growth and development. According to seasonal crop plants, each has specific optimum temperature range requirements. The optimum temperature for warm-season crops or cold/hot-season crops are between 20 and 25 °C and hot-season crops is 25–27 °C (Wien 1997; Sonwani and Maurya 2018). The optimal average temperature of the individual plants is 18 °C for maize, 15 °C for wheat, 25 °C for cotton, 23 °C for rice, and 22 °C for soybean. The higher temperature will be responsible for heat shock injury as well as biochemical and physiological changes. Subsequently, it reduced grain production and yield depending on the geographical place (Lobell and Field 2007; Johkan et al. 2011). The temperature increased by 1–2 °C as a consequence of higher CO_2 in tropical and subtropical countries and it is anticipated that food grain production will decline up to 30% (IPCC 2014). Experimentally, maize plants are grown at 20–25 °C (day/night) and normal photosynthesis process is observed whereas if temperature

Table 5.3 Growth yield of various crops toward elevated CO_2 concentration

Experimental plant	Crop yield (%)	References
C4 crop		
Corn	29	Cure and Acock (1986)
Sorghum	18	Long et al. (2006)
Maize	53	Abebe et al. (2016)
C3 crop		
Wheat	35	Cure and Acock (1986)
	8	Kimball et al. (1995)
Barley	70	Cure and Acock (1986)
Rice	25	Kimball (1983)
	24	Horie et al. (1996)
Soybean	22	Osborne (2016)
	29	Cure and Acock (1986)
	45	Baker et al. (1989)
	22	Fuhrer (2003)
Bean	82	Kimball (1983)
Green peas	89	Kimball (1983)
Groundnut	31	Clifford et al. (1993)
Tomato	20	Kimball (1983)
	2–26	Allen (1979)
Cucumber	30	Kimball (1983)
Lettuce	35	Kimball (1983)
Tobacco	42	Kimball (1983)
Sunflower	144	Allen (1979)
Potato	51	Kimball (1983)
	43–75	Allen (1979)
Radish	28	Kimball (1983)
Sweet potato	83	Cure and Acock (1986)
Quinoa	12–44	Bunce (2017)

increases, i.e., 25–30 °C, the photosynthesis rate decreased by 30–60% (Ben-Asher et al. 2008). Similarly, Ruiz-Vera et al. (2015) also found a 5% reduction in photosynthesis at more than 25 °C temperature.

5.3.1.1 Elevated Temperatures Affect Photosynthesis Versus Photorespiration Process

Temperature influences and maintains the equilibrium between C3 photosynthetic carbon assimilation and photorespiration process. It has mainly two methods. First one is, as the temperature increases, the solubility of CO_2 in mesophyll cell reduces as compared to O_2, hence internal concentration of CO_2 drops, and this brings about lowering the CO_2:O_2 ratio (Jordan and Ogren 1984; Taiz and Zeiger 2016; Saxena

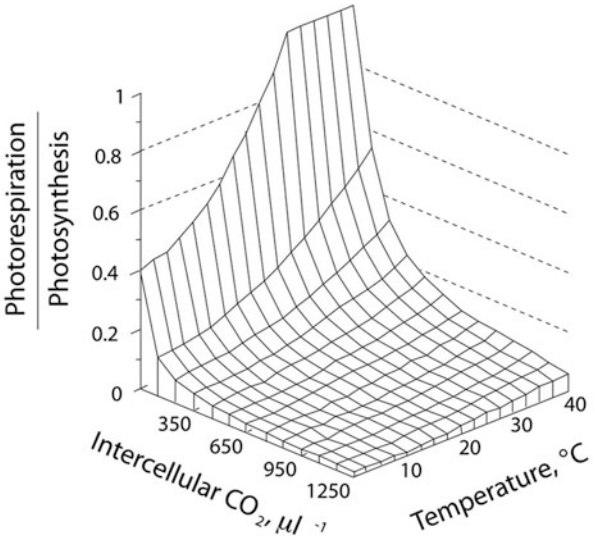


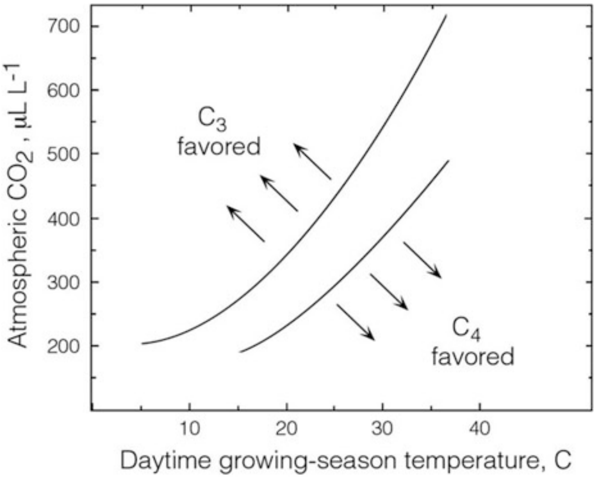
Fig. 5.4 Changes in the ratio of photorespiration to photosynthesis as a function of both CO_2 concentration and temperature for a plant with C3 photosynthesis (Ehleringer and Cerling 2001)

and Sonwani 2019a, b). The second one is that the enzymatic properties of Rubisco shifted more toward oxygenase activity as compared to carboxylase on account of increase in temperature, starting the photorespiration process over C3 carbon assimilation and initiating the fixation of O_2 to a higher degree than that of CO_2. In the C3 plant, the ratio of photorespiration to photosynthesis is dependent on increase in temperature and decrease of CO_2 or vice versa (Fig. 5.4). In this way, photosynthetic exchange of absorbed light into carbohydrate becomes less productive because there is a significant loss of CO_2 molecule in a C3 plant (Ehleringer et al. 1997; Taiz and Zeiger 2016). Based on the bi-functionality of RUBISCO (carboxylation/oxygenation) enzyme, C4 plants would be more promoted in that place where the average atmospheric temperature is higher than that of 25 °C (Taiz and Zeiger 2014; Ruiz-Vera et al. 2015) whereas, the average temperature for C3 crops is 18–25 °C. Hence, under low temperature and high atmospheric CO_2 preferred C3 plants (Wien 1997). Hence, C4 photosynthesis is preferred over C3 photosynthesis under the condition of high temperature and low atmospheric CO_2. Therefore, warmer temperature prefers C4 plant as compared to C3 plant (Fig. 5.5) because PEP carboxylase enzyme of C4 pathway is susceptible to low temperature and has good tolerance to high heat (Taiz and Zeiger 2016).

5.3.1.2 Effect of Temperature on Phenology of Crop Plants

Climate and seasonal changes actively control plant phenology. Phenology is the study of episodic biological events, such as bud break, flowering, and fruit development. It became one of the most trustworthy bioindicators for climate change (Gordo and Sanz 2010). It has been understood through various studies related to

Fig. 5.5 Interaction of atmospheric CO_2 and temperature that is predicted to favor C3 versus C4 crop plant (Ehleringer and Cerling 2001; Taiz and Zeiger 2016)



global warming which influences phenological measures for senescence, flowering, fruiting, and growth periods (Table 5.5) (Miller-Rushing and Primack 2008; Rumpff et al. 2010; Menzel et al. 2006a). There are several reasons pinpointed for the changes in the phenological features of the crops due to deviations in vernalization, photoperiodism, hormonal changes, or temperature or combinations of these aspects (Sparks et al. 2000; Rezaei et al. 2018). Only after exposure to a distinct number of days over a limited period of temperature, flowering is stimulated in determinate and indeterminate crops. After flowering, crop species terminate the vegetative growth and form fruits and end their life cycle after harvest (Peet and Wolfe 2000). In another way, global warming would fasten the growth of such crops and thereby curtail crop duration period for carbon fixation. On the one hand, it would be suitable for early maturation of plants, but on the other hand, it decreases or increases the product quality and yield (Laber and Lattauschke 2014) (Tables 5.4 and 5.5). For example, bean plant grew early but formed small seeds when grown at a temperature more than 27 or 22 °C (day/night) compared to 21 or 16 °C (Lattauschke 2015). In onions, higher temperature reduced the crop duration but yielded approximately twofold at 12 °C than that of 19 °C (Daymond et al. 1997). Elevated temperature also persuades flowering and fruit set through adverse impact on the physiological functioning of the reproductive organs. It affected double fertilization and reduction of lower husk cover and cereal development as well as fasten the sugar degradation and soften the texture of the fruit after harvest (Korner 2006;). For example, bean grown at elevated temperature triggered anomalous pollen, anther and ovule development and enhanced the flower abortion and fruit abscission (Abdelmageed and Gruda 2009).

Table 5.4 High temperature effect on crops' product quality

Parameter	Effect	Experimental plant	References
External quality			
Tipburn	↑	Lettuce, broccoli, Chinese cabbage	Saure (1998); Gruda and Tanny (2014)
Seed and fruit size	↓	Bean, pea, tomato	Siddique and Goodwin (1980); Lattauschke (2015); Gruda (2005)
Loose heads	↑	Broccoli, lettuce	Kałuzewicz et al. (2009)
Fruit coloration	↓	Tomato	Gruda and Tanny (2014)
Fruit cracking	↑	Tomato, pepper	Rosales et al. (2010); Gruda and Tanny (2014)
Internal quality			
Protein	↓	Wheat, rice	Myers et al. (2014)
Sugar content	↓	Pea, tomato, cabbage, sweet corn	Rosales et al. (2010); Gruda and Tanny (2014)
	=	Rice	Liu et al. (2017)
Starch	↓	Sweet corn	Wang and Frei (2011)
Macronutrients			
K, Mg, Fe, Ca	↓	Tomato	Rosales et al. (2010)
Fe	↑	Maize, soybean	Qiao et al. (2019)
P	↑	Maize, soybean	Qiao et al. (2019)
Ca	↓	Maize, soybean	Qiao et al. (2019)
Micronutrients			
Zn, Mn, Cu	=	Tomato	Rosales et al. (2010)
Fe, Mn	↑	Maize, soybean	Qiao et al. (2019)
Antioxidants			
Ascorbic acid (Vitamin C)	↑	Tomato, lettuce	Rosales et al. (2010)
	↓	Tomato	Wang and Frei (2011)
	=	Broccoli	Mølmann et al. (2015)
Tocopherol (Vitamin E)	↑	Lettuce	Wang and Frei (2011)
Lycopene Y	↓	Tomato	Gruda (2005); Rosales et al. (2010)
Carotene	↓	Carrot, tomato, lettuce	Ibrahim et al. (2006); Rosales et al. (2010); Wang and Frei (2011)
Anthocyanin, flavonols, phenols, glycosinolates	↑	Tomato, broccoli	Rosales et al. (2010); Gruda (2005); Mølmann et al. (2015)
Terpenes	↑	Carrot	Ibrahim et al. (2006)

5.4 Interactions of Higher Temperature and Elevated CO_2 with Product Quality and Yield

Climate change as a result of elevated CO_2 increases the temperature and affects and interacts with the physiology of the crop in various ways. Their positive or negative interaction causes an impact on yield and product quality affecting through various physio-biochemical processes (Fig. 5.6) (Reich et al. 2015; Choi et al. 2011). It

Table 5.5 Impact of elevated CO_2 and temperature on crop yield (Y, %)

Crop	CO_2 (ppm)	Temperature (°C)					References
		+1	+2	+3	+4	+5	
Wheat	660	25	16	2	–	−32	Lal et al. (1998)
	555	–	−8	–	−39	–	Siqueira et al. (1994)
	515	–	–	–	21	–	El Maayar et al. (1997)
	630	–	–	–	9 to −20	–	Adams et al. (1990)
	550	+5 to 25	−35	−10 to −50	−15 to −70	–	Rosenzweig and Tubiello (1996)
	500		−10 to −12				Cai et al. (2015)
Soybean	550	+30 to 65					Lenka et al. (2017)
	555	–	−3	–	−11	–	Siqueira et al. (1994)
	515	–	–	–	23	–	El Maayar et al. (1997)
	630	–	–	–	49 to −20	–	Adams et al. (1990)
	660	–	–	–	–	40 to −20	Adams et al. (1990)
	585	+2.4					Ruiz-Vera et al. (2015)
	700		31				Qiao et al. (2019)
Maize	550	–	−9	–	−20	–	Siqueira et al. (1994)
	515	–	–	–	–	–	El Maayar et al. (1997)
	630	–	–	–	49 to −20	–	Adams et al. (1990)
	660	–	–	–		40 to −40	Adams et al. (1990)
	550 ± 20	54		4.9			Abebe et al. (2016)
	700		25				Qiao et al. (2019)
Rice	660	4	−5	–	–	−25	Lal et al. (1998)
	500		−17 to −35				Cai et al. (2015)

increases the leaf temperature and significantly impacts the photosynthetic capability, durability as well as it promotes the senescence and curbing the growing period and yield (Van De Geijn and Goudriaan 1996; Ruiz-Vera et al. 2015; Köhler et al. 2019). Table 5.5 shows the effect of temperature and CO_2 on crop quality in terms of morphological and biochemical changes. For example, higher temperatures increased loose and puffy heads, tip burn, yellowing and storage of secondary metabolite in cabbage crop (Wien 1997). Qiao et al. (2019) observed that the

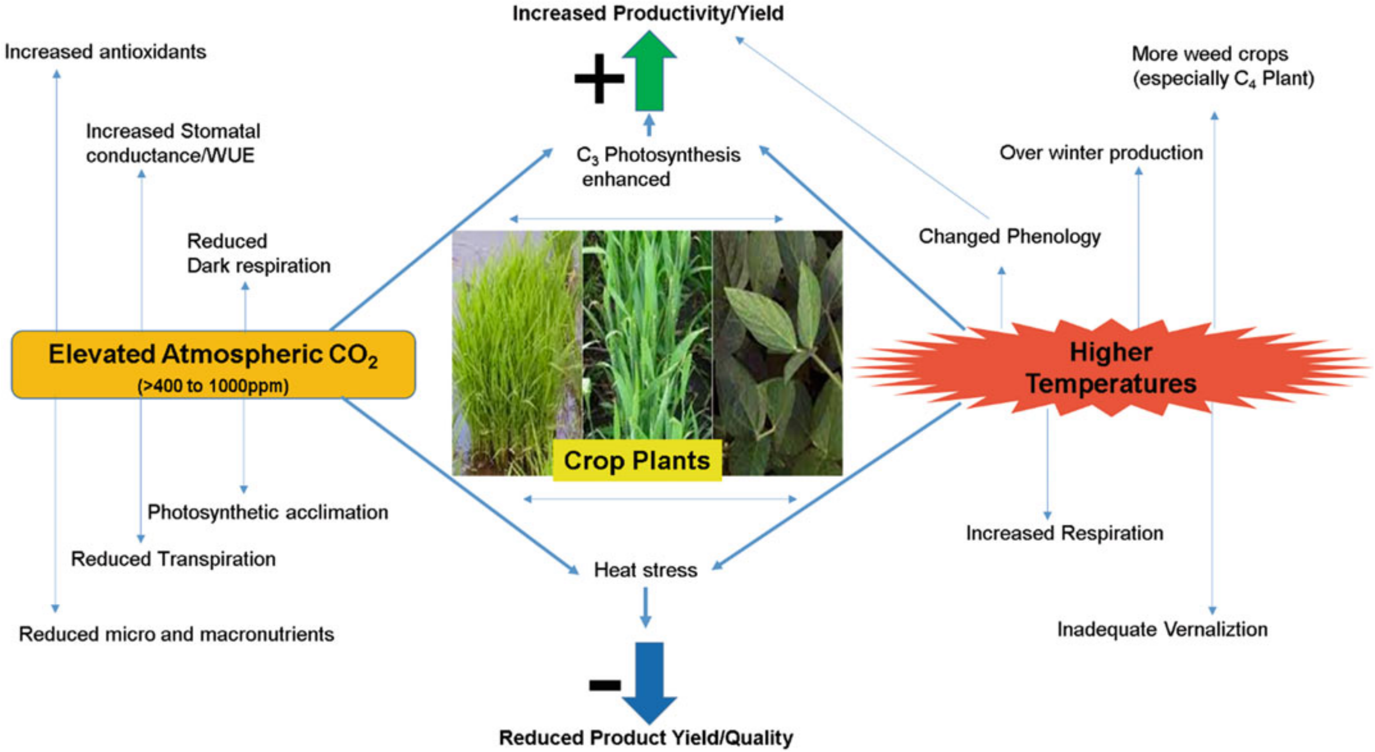


Fig. 5.6 Interaction of elevated atmospheric CO_2 concentrations and higher temperatures on crop plant

concentration of oil in soybean was 9% and recorded 14% increase under elevated temperature (eT) and elevated CO_2 (eTeCO_2) whereas in maize grain 12% and 20% higher eT and eTeCO_2 as compared to control condition. Elevated temperature and CO_2 increase the content of macronutrients (N, P, and K), expand leaf area, number of grains per row and total yield of maize crop (Abebe et al. 2016). Table 5.5 shows the impact of elevated temperature and CO_2 on various crop yields %. There are several studies that show that high temperature and CO_2 and their interaction affect the yield of maize, wheat, and rice in positive or negative way with respect to growth period, product quality, and yield of abovementioned crops (IPCC 2007; Mendelsohn and Dinar 2009; Pathak et al. 2012; Ghannoum and Conroy 2007; Leakey et al. 2004; Fang et al. 2010; Pathak et al. 2012; Vanaja et al. 2015; Tripathy et al. 2009). The adverse effects of specific elevated temperature on the plant somewhat counterbalanced by high temperature along with elevated CO_2 concentration (Qaderi et al. 2006), whereas Qiao et al. (2019) reported that average yield of soybean was increased by 31% at elevated temperature and CO_2 but not at high temperature alone. But in case of maize, elevated temperature and CO_2 and elevated temperature alone both increased the yield by nearly 25% undergrown in open-top chambers (ambient +2.1 °C, 700 ppm CO_2). On the other hand, Abebe et al. (2016) reported that the yield and total biomass increased at elevated CO_2 with ambient +1.5 °C but yield decreased at elevated CO_2 with ambient +3.0 °C temperature. Therefore, it showed that elevated CO_2 is promoting the yield, but high temperature diminished growth and development of the crop plant.

There are various ways by which temperature impacts crop physiology and yields:

(I) Increasing temperature leads to a continuous elevation in the saturation vapor pressure of air, as a result of which the vapor pressure deficit (VPD) between air and the leaf becomes increased (VPD defined as the gap between the saturation vapor pressure and the actual vapor pressure of the air). Elevated VPD leads to a decrease in water use efficiency. However, plants transpire more water per unit of carbon assimilation (Willett et al. 2007). The plants reciprocate to enhance VPD by closing their stomata leading to reduced photosynthetic rates and consequently there is an increase in the temperature of the plant body. Hence, such warming effect may elevate the heat-related impacts on the plant body.

(II) High temperature can cause heat stress. It directly impairs the plant cells and their division, flowering, and fertilization period, which can lead to infertility, lower growth period, and yields (Teixeira et al. 2011).

(III) Or elevated temperature along with elevated CO_2, it may promote the growth, survival and spread of the various pathogen and their diseases particular to crops which lead to the loss of plant (Ziska et al. 2011).

5.5 Mitigation and Adaptation Strategies

Now, there is an urgent need for strategic considerations to adopt mitigation and adaptation measures. This measure could be a more operational, economical, and practical solution to the challenge of global warming and climate change. Under mitigation practices, the focus is on decreasing the concentration of anthropogenic greenhouse gases (GHGs) and the adaptation to climate change by developing various methods (Al-Ghussain 2018; Parry et al. 2007; Saxena and Sonwani 2020). Mitigation measures are those actions that are taken to decrease and control GHGs (abatement), while adaptation measures are based on reducing susceptibility to the effects of climate change (sequestration). Some mitigation measures should be followed to mitigate the increase of pollutant emissions such as the reduction in the use of fossil fuels, replacement by green energy sources, maximum use of renewable energy, electrification of industrial unit, well-organized transport system, i.e., electric public conveyance, bicycle, pooled cars, etc. On the other hand, an adaptation measure will be required to protect the source of revenue and food security in many developing and developed countries (IPCC 2001; Adger et al. 2003). However, Howden et al. (2007) proposed significant adaptation strategies in agriculture practices toward global warming and climate change:

1. To develop the resistant varieties and species to fight with heat stress and drought, inundating and salinization
2. Reassessing and altering fertilizer dose to retain grain yield or product as well as soil quality (Adams et al. 1990)
3. Changing the irrigation method and other water harvest management, i.e., drip irrigation
4. Managing the crop activities according to time and geographical location
5. Managing river water flows for more effective supply of water for irrigation and avoiding waterlogging, erosion, and nutrient loss
6. Crop diversification
7. To promote organic farming at selected sites
8. Proper application of integrated pest and pathogen management by emerging resistant varieties and species to pests and diseases
9. To promote the practice of knowledge of climate modelling and forecasting to reduce crop production risk
10. Increasing the income of farmer through mixed farming of fish with rice fields
11. Production of pasture land, altered pasture rotation, changing the time of grazing according to livestock stocking rates as well as altering the grazing times, modification and use of adapted forage crops according to livestock also
12. To introduce and promote forest conservation, agroforestry, and forest-based venture for extra income of agrarian peoples as well as restorating the degraded ecosystem

5.6 Summary

The emission of uncontrolled GHGs and other air pollutants due to industrialization and land use change pattern causes global warming and lead to climate change. Elevated CO_2 is the main causal factor for global warming by absorbing the infrared radiation and warm the earth atmosphere. This progressive warming of atmosphere causes global climate change. The elevated CO_2 and higher temperature interact with C3 and C4 crop and impact the growth and productivity of the plant via change in various physiological and biochemical processes. At current ambient CO_2 level, C3 plant will perform well whereas C4 plant is saturated somewhat and there is little change in photosynthetic rate. But at elevated CO_2 C3 plant get benefited up to some extent in initial period but after certain period plant faces N deficiency symptom due to hindrance of NO_3 assimilation. High temperature gives benefit to C4 plant as compared to C3 plant due to temperature-insensitive enzyme and do not have enzyme bi-functionality as well as anatomical specialization. The interaction of elevated CO_2 and higher temperature affect the crop yield, and quality may be increased or decreased depending on the geographical position of plants. It affects through various physiological processes such as photosynthesis, dark respiration, stomatal conductance, water use efficiency, transpiration process, and phenological process. Hence, decline of crop quality and productivity challenge the food security issue for the coming generation. Therefore, needs to develop the mitigation and adaptation strategies to curb the pollution level to ensure the sutainable development of society. Hence, the selection of crop is now an important concern according to environmental and geographical condition so that the plants are able to maintain and reduce the crop quality and yield loss.

References

Abdelmageed AHA, Gruda N (2009) Influence of high temperatures on gas exchange rate and growth of eight tomato cultivars under controlled heat stress conditions. Eur J Hortic Sci 74:152–159

Abebe A, Pathak H, Singh SD, Bhatia A, Harit RC, Kumar V (2016) Growth, yield and quality of maize with elevated atmospheric carbon dioxide and temperature in north–west India. Agric Ecosyst Environ 218:66–72

Adams RM et al (1990) Global climate change and U.S. agriculture. Nature 345:219–224

Adger NW, Huq S, Brown K, Conway D, Hulme M (2003) Prog Dev Stud 3:179–195

Ainsworth EA, Rogers A, Leakey ADB, Heady LE, Gibon Y, Stitt M, Schurr U (2007) Does elevated atmospheric [CO_2] alter diurnal C uptake and the balance of C and N metabolites in growing and fully expanded soybean leaves? J Exp Bot 58(3):579–591

Al-Ghussain L (2018) Global warming: review on driving forces and mitigation. Environ Prog Sustain Energy. doi:10.1002/ep

Allen LH (1979) Potentials for carbon dioxide enrichment. In: Barfield BJ, Gerber JF (eds) Modification of aerial environment of plants. American Society of Agricultural Engineering Monography, Fort Collins, vol 2, pp 500–519

Backhausen JE, Kitzmann C, Sheibe R (1994) Photosynth Res 42:75–86

Backhausen JE, Kitzmann C, Horton P, Scheibe R (2000) Electron acceptors in isolated intact spinach chloroplasts act hierarchically to prevent over-reduction and competition for electrons. Photosynth Res 64:1–13

Bagley J, Rosenthal DM, Ruiz-Vera UM, Siebers MH, Kumar P, Ort DR et al (2015) The influence of photosynthetic acclimation to rising CO_2 and warmer temperatures on leaf and canopy photosynthesis models. Glob Biogeochem Cycles 29:194–206. https://doi.org/10.1002/2014GB004848

Baker JT et al (1989) Response of soybean to air temperature and carbon dioxide concentration. Crop Sci 29:98–105

Becker C, Klaring HP (2016) CO_2 enrichment can produce high red leaf lettuce yield while increasing most flavonoid glycoside and some caffeic acid derivative concentrations. Food Chem 199:736–745

Ben-Asher J, Garcia AG, Hoogenboom G (2008) Effect of high temperature on photosynthesis and transpiration of sweet corn (*Zea mays* L. var. rugosa). Photosynthetica 46(4):595–603

Berry JA, Downton JS (1982) Environmental regulation of photosynthesis. In: Govindjee (ed) Photosynthesis: development, carbon metabolism and plant productivity, vol II. Academic Press, New York, pp 263–343

Bettoni MM, Mogor AF, Pauletti V, Goicoechea N (2014) Growth and metabolism of onion seedlings as affected by the application of humic substances, mycorrhizal inoculation and elevated CO_2. Sci Hortic 180:227–235

Bisbis MB, Gruda N, Blanke M (2018) Potential impacts of climate change on vegetable production and product quality: a review. J Clean Prod 170:1602–1620

Blanke MM, Hucklesby DP, Notton BA, Lenz F (1987) Utilization of bicarbonate by apple fruit phosphoenolpyruvate carboxylase. Phytochemistry 26(9):2475–2476

Bloom AJ, Smart DR, Nguyen DT, Searles PS (2002) Nitrogen assimilation and growth of wheat under elevated carbon dioxide. Proc Natl Acad Sci U S A 99:1730–1735

Bloom AJ, Burger M, Asensio JSR, Cousins AB (2010) Carbon dioxide enrichment inhibits nitrate assimilation in wheat and Arabidopsis. Science 328:899–903

Bloom AJ, Rubio-Asensio JS, Randall L, Rachmilevitch S, Cousins AB, Carlisle EA (2012) CO_2 enrichment inhibits shoot nitrate assimilation in C3 but not C4 plants and slows growth under nitrate in C3 plants. Ecology 93:355–367

Bloom AJ, Burger M, Kimball BA, Pinter PJ Jr (2014) Nitrate assimilation is inhibited by elevated CO_2 in field-grown wheat. Nat Clim Chang 4:477. https://doi.org/10.1038/nclimate2183

Bouma TJ, Nielson KL, Eissenstat DM, Lynch JP (1997) Estimating respiration of roots in soil: interactions with soil CO_2, soil temperature, and soil water content. Plant Soil 195:221–232

Bowes G (1993) Annu Rev Plant Physiol Plant Mol Biol 44:309–332

Bunce JA (2017) Variation in yield responses to elevated CO_2 and a brief high temperature treatment in Quinoa. Plants (Basel) 6(3):26. https://doi.org/10.3390/plants6030026

Burkey K, Booker F, Ainsworth E, Nelson R (2012) Field assessment of a snap bean ozone bioindicator system under elevated ozone and carbon dioxide in a free air system. Environ pollut (Barking, Essex : 1987) 166:167–171. https://doi.org/10.1016/j.envpol.2012.03.020

Burton AJ, Zogg GP, Pregitzer KS, Zak DR (1997) Effect of measurement CO_2 concentration on sugar maple root respiration. Tree Physiol 17:421–427

Butterly CR, Armstrong R, Chen D, Tang C (2016) Free-air CO_2 enrichment (FACE) reduces the inhibitory effect of soil nitrate on N2 fixation of *Pisum sativum*. Ann Bot 117:177–185. https://doi.org/10.1093/aob/mcv140

Cai C, Yin X, He S, Jiang W, Si C, Struik PC, Luo W, Li G, Xie Y, Xiong Y, Pan G (2015) Responses of wheat and rice to factorial combinations of ambient and elevated CO_2 and temperature in FACE experiments. Glob Chang Biol 22(2):856–874. https://doi.org/10.1111/gcb.13065

Cavagnaro RT, Gleadow R, Rebecca M (2011) Plant nutrient acquisition and utilisation in a high carbon dioxide world. Funct Plant Biol 38:87–96. https://doi.org/10.1071/fp10124

CDIAC. http://cdiac.ornl.gov/trends/emis/overview_2014.html

Choi EY, Seo TC, Lee SG, Cho IH, Stangoulis J (2011) Growth and physiological responses of Chinese cabbage and radish to long-term exposure to elevated carbon dioxide and temperature. Hortic Environ Biotechnol 52(4):376–386

Clifford SC et al (1993) The effects of elevated atmospheric carbon dioxide and water stress on light interception, dry matter production and yield in stands of groundnut (*Arachis hypogaea* L.). J Exp Bot 44:1763–1770

Clinton BD, Vose JM (1999) Fine root respiration in mature eastern white pine (*Pinus strobus*) in situ: the importance of CO_2 in controlled environments. Tree Physiol 19:475–479

Cure JD, Acock B (1986) Crop response to carbon dioxide doubling: a literature survey. Agric For Meteorol 38:127–145

Curtis PS (1996) Plant Cell Environ 19:127–137

Daymond AJ, Wheeler TR, Hadley P, Ellis RH, Morison JIL (1997) The growth, development and yield of onion (*Allium cepa* L.) in response to temperature and CO_2. J Hortic Sci (UK)

Edwards G, Walker D (1983) C3 C4: mechanisms, and cellular and environmental regulations of photosynthesis. Blackwell, Oxford

Ehleringer JR, Cerling TE (2001) Photosynthetic pathways and climate. In: Schulze E-D, Heimann M, Harrison SP, Holland EA, Lloyd J, Prentice IC, Schimel D (eds) Global biogeochemical cycles in the climate system. Academic Press, San Diego, pp 267–277

Ehleringer JR, Cerling TE, Helliker BR (1997) C4 photosynthesis, atmospheric CO_2, and climate. Oecologia 112:285–299

El Maayar M et al (1997) The effects of climate change and CO_2 fertilization on agriculture in Quebec. Agric For Meteorol 85:193–208

Engineer CB, Hashimoto-Sugimoto M, Negi J et al (2015) CO_2 sensing and CO_2 regulation of stomatal conductance: advances and open questions. Trends Plant Sci. https://doi.org/10.1016/j.tplants.2015.08.014

Fang SB, Shen B, Tan K, Gao XN (2010) Effect of elevated CO_2 concentration and increased temperature on physiology and production of crops. Chin J Eco-Agric 18:1116–1124

Fuhrer J (2003) Agroecosystem response to combinations of elevated CO_2, ozone, and global climate change. Agric Ecosyst Environ 97:1–20

Fuss S, Canadell JG, Peters GP et al (2014) Commentary: betting on negative emissions. Nat Clim Chang 4:850–853

Ghannoum O, Conroy JP (2007) Phosphorus deficiency inhibits growth in parallel with photosynthesis in a C-3 (*Panicum laxum*) but not two C-4 (*P. coloratum* and *Cenchrus ciliaris*) grasses. Funct Plant Biol 34(1):72–81

Giri A, Armstrong B, Rajashekar CB (2016) Elevated carbon dioxide level suppresses nutritional quality of lettuce and spinach. Am J Plant Sci 7(1):246

Gonzàlez-Meler MA, Siedow JN (1999) Direct inhibition of mitochondrial respiratory enzymes by elevated CO_2: does it matter at the tissue or wholeplant level? Tree Physiol 19:253–259

Gonzàlez-Meler MA, Ribas-Carbó M, Siedow JN, Drake BG (1996) Direct inhibition of plant mitochondrial respiration by elevated CO_2. Plant Physiol 112:1349–1355

Gordo O, Sanz JJ (2010) Impact of climate change on plant phenology in Mediterranean ecosystems. Glob Chang Biol 16:1082–1106

Graham D (1980) Effects of light on “dark” respiration. In: Metabolism and respiration: a comprehensive treatise, pp 525–579

Griffin KL, Tissue DT, Turnbull MH, Schuster W, Whitehead D (2001) Leaf dark respiration as a function of canopy position in *Nothofagus fusca* trees grown at ambient and elevated CO_2 partial pressures for 5 years. Funct Ecol 15:497–505

Gruda N (2005) Impact of environmental factors on product quality of greenhouse vegetables for fresh consumption. Crit Rev Plant Sci 24(3):227–247

Gruda N (2009) Do soilless culture systems have an influence on product quality of vegetables? J Appl Bot Food Qual 82:141–147

Gruda N, Tanny J (2014) Protected crops. In: Dixon GR, Aldous DE (eds) Horticulture: plants for people and places, vol 1. Springer, Netherlands, pp 327–405

Hamilton JG, Thomas RB, Delucia EH (2001) Direct and indirect effects of elevated CO_2 on leaf respiration in a forest ecosystem. Plant Cell Environ 24:975–982

Hartmann DL, Klein Tank AMG, Rusticucci M et al (2013) Observations: atmosphere and surface. In: Stocker TF, Qin D, Plattner G-K et al (eds) Climate change 2013: The physical science basis. Contribution of Working Group I to the Fifth Assessment Report of the Intergovernmental Panel on Climate Change. Cambridge University Press, Cambridge, pp 159–254

Horie T, Matsui T, Nakagawa H, Omasa K (1996) Effects of elevated CO_2 and global climate change on rice yield in Japan. In: Climate change and plants in East Asia. Springer, Tokyo

Houghton RA, Nassikas AA (2017) Global and regional fluxes of carbon from land use and land cover change 1850–2015. Global Biogeochem Cycles 31:456–472. https://doi.org/10.1002/2016GB005546

Howden M, Soussana JF, Tubiello FN (2007) Adaptation strategies for climate change. Proc Natl Acad Sci 104:19691–19698

Ibrahim MA, Nissinen A, Prozherina N, Oksanen EJ, Holopainen JK (2006) The influence of exogenous monoterpene treatment and elevated temperature on growth, physiology, chemical content and headspace volatiles of two carrot cultivars (*Daucus carota* L.). Environ Exp Bot 56 (1):95–107

IEA (2017) CO_2 emissions from fuel combustion – highlights. Int Energy Agency 1:1–162. https://doi.org/10.1787/co2_fuel-2017-en

IPCC (2001) Climate change 2001: Mitigation. Contribution of working group III to the third assessment report of the Intergovernmental Panel on Climate Change

IPCC (2007) Climate change—impacts, adaptation and vulnerability technical summary of working group II. Fourth Assessment Report Inter-governmental Panel on Climate Change. Cambridge University Press, Cambridge, p 976

IPCC (2013) Summary for policymakers. In: Stocker TF, Qin D, Plattner G-K, Tignor M, Allen SK, Boschung J, Nauels A, Xia Y, Bex V, Midgley PM (eds) Climate change 2013: the physical science basis. Contribution of Working Group I to the Fifth Assessment Report of the Intergovernmental Panel on Climate Change. Cambridge University Press, Cambridge

IPCC (2014) Climate change 2014: Impacts, adaptation, and vulnerability. Part A: Global and sectoral aspects. In: Field CB, Barros VR, Dokken DJ, Mach KJ, Mastrandrea MD, Bilir TE, Chatterjee M, Ebi KL, Estrada YO, Genova RC, Girma B, Kissel ES, Levy AN, Mac Cracken S, Mastrandrea PR, White LL (eds) Contribution of Working Group II to the Fifth Assessment Report of the Intergovernmental Panel on Climate Change. Cambridge University Press, Cambridge, 1132 p

Jahnke S (2001) Atmospheric CO_2 concentration does not directly affect leaf respiration in bean or poplar. Plant Cell Environ 24:1139–1151

Jin C, Du S, Wang Y, Condon J, Lin X, Zhang Y (2009) Carbon dioxide enrichment by composting in greenhouses and its effect on vegetable production. J Plant Nutr Soil Sci 172(3):418–424

Johkan M, Oda M, Maruo T, Shinohara Y (2011) Crop production and global warming impacts. Case studies on the economy, human health, and on urban and natural environments. http://www.intechopen.com

Jordan DB, Ogren WL (1984) The CO_2/O_2 specificity of ribulose 1,5-bisphosphate carboxylase/oxygenase. Planta 1618(1):308–313

Kałuzewicz A, Krzesinski W, Knaflewski M (2009) Effect of temperature on the yield and quality of broccoli heads. Veg Crops Res Bull 71:51–58

Kimball BA (1983) Carbon dioxide and agricultural yield: an assemblage and analysis of 770 prior observation. USDA/ARS Water Conservation Laboratory Report Number 14. Phoenix, AZ

Kimball BA et al (1995) Productivity and water use of wheat under free-air CO_2 enrichment. Glob Chang Biol 1:429–442

Knaff DB (1996) In: Ort DR, Yocum CF (eds) Oxygenic photosynthesis: the light reactions, vol 4. Kluwer, Dordrecht, pp 333–361

Köhler IH, Huber SC, Bernacchi CJ, Baxter IR (2019) Increased temperatures may safeguard the nutritional quality of crops under future elevated CO_2 concentrations. Plant J. https://doi.org/10.1111/tpj.14166

Korner C (2006) Plant CO_2 responses: an issue of definition, time and resource supply. New Phytol 172:393–411

Korres NE, Norsworthy JK, Tehranchian P, Gitsopoulos TK, Loka DA, Derrick MO, Gealy DR, Moss SR, Burgos NR, Ryan Miller M, Palhano M (2016) Cultivars to face climate change effects on crops and weeds: a review. Agron Sustain Dev 36:12

Kulshrestha U, Saxena P (eds) (2016) Plant responses to air pollution. Springer, Singapore

Kyei-Boahen S, Astatkie T, Lada R, Gordon R, Caldwell C (2003) Gas exchange of carrot leaves in response to elevated CO_2 concentration. Photosynthetica 41(4):597–603

Laber H, Lattauschke G (2014) Vegetable production, 2nd edn. Eugen Ulmer Verlag, Stuttgart. (In German)

Lal M et al (1998) Vulnerability of rice and wheat yields in NW India to future changes in climate. Agric For Meteorol 89:101–114

Lattauschke G (2015) Extreme heat caused massive yield and quality reductions in medium and late peas. In: Versuche im deutschen Gartenbau 2015. Sachsisches Landesamt für Umwelt. Landwirtschaft und Geologie, Dresden. (in German)

Le QC et al (2018a) Global carbon budget. 2017. Earth Syst Sci Data 10:405–448

Le QC et al (2018b) Global carbon budget. 2018. Earth Syst Sci Data. https://doi.org/10.5194/essd-2018-120

Leakey ADB (2009) Rising atmospheric carbon dioxide concentration and the future of C-4 crops for food and fuel. Proc Roy Soc B Biol Sci 276:2333–2343

Leakey ADB, Bernacchi CJ, Dohleman WFG, Ort DR, Long SP (2004) Will photosynthesis of maize (*Zea mays*) in the US Corn Belt increase in future [CO_2] rich atmospheres? An analysis of diurnal courses of CO_2 uptake under free-air concentration enrichment (FACE). Glob Chang Biol 10:951–962

Leakey AD et al (2009) Elevated CO_2 effects on plant carbon, nitrogen, and water relations: six important lessons from FACE. J Exp Bot 60:2859–2876

Leakey ADB, Ainsworth EA, Bernacchi CJ, Rogers A, Long SP, Ort DR (2009a) Elevated CO_2 effects on plant carbon, nitrogen, and water relations, six important lessons from FACE. J Exp Bot 60:2859–2876

Lemonnier P, Ainsworth EA (2019) In: Yadav SS, Redden RJ, Hatfield JL, Ebert AW, Hunter D (eds) Crop responses to rising atmospheric [CO_2] and global climate change. Food security and climate change, 1st edn. Wiley

Lenka NK, Lenka S, Thakur JK, Elanchezhian R, Aher SB, Simaiya V, Yashona DS, Biswas AK, Agrawal PK, Patra AK (2017) Interactive effect of elevated carbon dioxide and elevated temperature on growth and yield of soybean. Curr Sci 113:12

Liu S, Waqas MA, Wang S-h, X-y X, Wan Y-f (2017) Effects of increased levels of atmospheric CO_2 and high temperatures on rice growth and quality. PLoS One 12(11):e0187724. https://doi.org/10.1371/journal.pone.0187724

Lobell DB, Field CB (2007) Global scale climate–crop yield relationships and the impacts of recent warming. Environ Res Lett 2:014002. 7 pp

Long SP, Ainsworth EA, Rogers A, Ort DR (2004) Rising atmospheric carbon dioxide: plants face the future. Annu Rev Plant Biol 55:591–628

Long SP, Ainsworth EA, Leakey ADB, Nösberger J, Ort DR (2006) Food for thought: lower-than-expected crop yield stimulation with rising CO_2 concentrations. Science 312:1918–1921

Luthi D, Le Floch M, Bereiter B, Blunier T, Barnola J-M, Siegenthaler U, Raynaud D, Jouzel J, Fischer H, Kawamura K, Stocker TF (2008) High-resolution carbon dioxide concentration record 650,000-800,000 years before present. Nature 453:379–382. https://doi.org/10.1038/nature06949

Makino A, Mae T (1999) Photosynthesis and plant growth at elevated levels of CO_2. Plant Cell Physiol 40(10):999–1006

Masle J (2000) The effects of elevated CO_2 concentrations on cell division rates, growth patterns, and blade anatomy in young wheat plants are modulated by factors related to leaf position, vernalization, and genotype. Plant Physiol 122(4):1399–1415

Mattos L, Moretti C, Jan S, Sargent S, Lima C, Fontenelle M (2014) Chapter 19. climate changes and potential impacts on quality of fruit and vegetable crops. https://doi.org/10.1016/B978-0-12-800876-8.00019-9

Meinshausen M, Smith SJ, Calvin K et al (2011) The RCP greenhouse gas concentrations and their extensions from 1765 to 2300. Clim Chang 109:213–241

Mendelsohn R, Dinar A (2009) Climate change and agriculture: an economic analysis of global impacts, adaptation and distributional effects. Edward Elgar, Cheltenham, p 256

Meng F, Zhang J, Yao F, Hao C (2014) Interactive effects of elevated CO_2 concentration and irrigation on photosynthetic parameters and yield of maize in Northeast China. PLoS One 9(5): e98318. https://doi.org/10.1371/journal.pone.0098318

Menzel A, Sparks TH, Estrella N et al (2006a) European phonological response to climate change matches the warming pattern. Glob Chang Biol 12:1969–1976

Miller-Rushing AJ, Primack RB (2008) Global warming and flowering times in Thoreau's Concord: a community perspective. Ecology 89:332–341

Mølmann JA, Steindal AL, Bengtsson GB, Seljåsen R, Lea P, Skaret J, Johansen TJ (2015) Effects of temperature and photoperiod on sensory quality and contents of glucosinolates, flavonols and vitamin C in broccoli florets. Food Chem 172:47–55

Moore T, Roulet N, Waddington J (1998) Uncertainty in predicting the effect of climatic change on the carbon cycling of Canadian peatlands. Clim Chang 40(2):229–245

Mott KA (2009) Opinion: stomatal responses to light and CO_2 depend on the mesophyll. Plant Cell Environ 32:1479–1486

Myers SS et al (2014) Increasing CO_2 threatens human nutrition. Nature 510:139–142. https://doi.org/10.1038/nature13179

NOAA. http://www.esrl.noaa.gov/gmd/ccgg/trends/global.html

Osborne CP (2016) Crop yields: CO_2 fertilization dries up. Nat Plants 2(9):1–2

Owensby CE, Ham JM, Knapp AK, Auen LM (1999) Biomass production and species composition change in a tall grass prairie ecosystem after long-term exposure to elevated atmospheric CO_2. Glob Chang Biol 5:497–506

Pal M, Rao LS, Srivastava AC, Jain V, Sengupta UK (2003) Impact of CO_2 enrichment and variable nitrogen supplies on composition and partitioning of essential nutrients of wheat. Biol Plant 47 (2):227–231

Parry M, Canziani O, Palutikof J, van der Linden P, Hanson C (2007) Climate change 2007: impacts, adaptation and vulnerability. Contribution of Working Group II to the Fourth Assessment Report of the Intergovernmental Panel on Climate Change. Cambridge University Press, Cambridge. https://doi.org/10.1256/004316502320517344

Pathak H, Aggarwal PK, Singh SD (2012) Climate change impacts, adaptations and mitigation in agriculture: methodology for assessment and application. Indian Agricultural Research Institute, New Delhi, p xix+302

Peet MM, Wolfe DW (2000) Crop ecosystem responses to climatic change: vegetable crops. In: Reddy KR, Hodges HF (eds) Climate change and global crop productivity. CAB International, Oxon, pp 213–243

Poorter H, Navas ML (2003) New Phytol 157:175–198

Poorter H, Perez-Soba M (2001) The growth response of plants to elevated CO_2 under non-optimal environmental conditions. Oecologia 129(1):1–20

Prior SA, Torbert HA, Runion GB, Rogers HH (2003) Implications of elevated CO_2-induced changes in agroecosystem productivity. J Crop Prod 8:217–244

Qaderi MM, Kurepin LV, Reid DM (2006) Growth and physiological responses of canola (*Brassica napus*) to three components of global climate change: temperature, carbon dioxide and drought. Physiol Plant 128:710–721

Qi J, Marshall JD, Mattson KG (1994) High soil carbon dioxide concentrations inhibit root respiration of Douglas fir. New Phytol 128:435–442

Qiao Y, Shujie M, Qi L, Jian J, Xiaosan L, Caixian T (2019) Elevated CO_2 and temperature increase grain oil concentration but their impacts on grain yield differ between soybean and maize grown in a temperate region. Sci Total Environ 666:405–413

Quesada A, Gomez-Garcia I, Fernandez E (2000) Involvement of chloroplast and mitochondria redox valves in nitrate assimilation. Trends Plant Sci 5:463–464

Rachmilevitch S, Cousins AB, Bloom AJ (2004) Nitrate assimilation in plant shoots depends on photorespiration. PNAS 101(31):11506–11510

Reich M, Meerakker AN, Parmar S, Hawkesford MJ, De Kok LJ (2015) Temperature determines size and direction of effects of elevated CO_2 and nitrogen form on yield quantity and quality of Chinese cabbage. Plant Biol 18(S1):63–75

Rezaei EE, Siebert S, Hüging H, Ewert F (2018) Climate change effect on wheat phenology depends on cultivar change. Sci Rep 8(1):4891. https://doi.org/10.1038/s41598-018-23101-2

Rogers HH, Bingham GE, Cure JD, Smith JM, Surano KA (1983) Responses of selected plant species to elevated carbon dioxide in the field. J Environ Qual 2(4):569–574

Rosales MA, Cervilla LM, Sanchez-Rodríguez E, Rubio-Wilhelmi MDM, Blasco B, Ríos JJ, Ruiz JM (2010) The effect of environmental conditions on nutritional quality of cherry tomato fruits: evaluation of two experimental Mediterranean greenhouses. J Sci Food Agric 91(1):152–162

Rosenzweig C, Tubiello FN (1996) Effects of changes in minimum and maximum temperature on wheat yields in the Central US: a simulation study. Agric For Meteorolo 80:215–230

Ruiz-Vera UM, Siebers MH, Drag DW, Ort DR, Bernacchi CJ (2015) Canopy warming caused photosynthetic acclimation and reduced seed yield in maize grown at ambient and elevated [CO_2]. Glob Chang Biol 21(11):4237–4249

Rumpff L, Coates F, Morgan JW (2010) Biological indicators of climate change: evidence from long-term flowering records of plants along the Victorian coast, Australia. Austral J Bot 58:428. https://doi.org/10.1071/BT10053

Sage RF, Sharkey TD, Seemann JR (1989) Acclimation of photosynthesis to elevated CO_2 in 5 C-3 species. Plant Physiol 89(2):590–596

Saure MC (1998) Causes of the tipburn disorder in leaves of vegetables. Sci Hortic 76:131–147

Saxena P, Naik V (eds) (2018) Air pollution: sources, impacts and controls. CABI, Oxford

Saxena P, Sonwani S (2019a) Primary criteria air pollutants: environmental health effects, Criteria air pollutants and their impact on environmental health. Springer, Singapore, pp 49–82

Saxena P, Sonwani S (2019b) Criteria air pollutants: chemistry, sources and sinks, Criteria air pollutants and their impact on environmental health. Springer, Singapore, pp 7–48

Saxena P, Sonwani S (2020) Criteria air pollutants and their impact on environmental health. Springer, Singapore

Schmidhuber J, Tubiello FN (2007) Global food security under climate change. Proc Natl Acad Sci U S A 104:19703–19708

Seneweera S (2011) Effects of elevated CO_2 on plant growth and nutrient partitioning of rice (*Oryza sativa* L.) at rapid tillering and physiological maturity. J Plant Interact 6(1):35–42

Seneweera SP, Conroy JP (2005) Enhanced leaf elongation rates of wheat at elevated CO_2: is it related to carbon and nitrogen dynamics within the growing leaf blade? Environ Exp Bot 54 (2):174–181

Seneweera S, Milham P, Conroy J (1994) Influence of elevated CO_2 and phosphorus-nutrition on the growth and yield of a short-duration rice (*Oryza Sativa* l cv Jarrah). Austral J Plant Physiol 21(3):281–292

Shimono H, Bunce JA (2009) Acclimation of nitrogen uptake capacity of rice to elevated atmospheric CO_2 concentration. Ann Bot 103:87–94

Shingles R, Roh MH, McCarty RE (1996) Plant Physiol 112:1375–1381

Siddique MA, Goodwin PB (1980) Seed vigour in bean (*Phaseolus vulgaris* L. cv. Apollo) as influenced by temperature and water regime during development and maturation. J Exp Bot 31 (1):313–323

Siqueira OJF et al (1994) Potential effects of global climate changes for Brazilian agriculture and adaptive strategies for wheat, maize and soybean. Revista Brasileira de Agrometorologia 2:115–129

Smith SD, Huxman TE, Zitzer SF, Charlet TN, Housman DC, Coleman JS, Fenstermaker LK, Seemann JR, Nowak RS (2000) Elevated CO_2 increases productivity and invasive species success in an arid ecosystem. Nature 408:79–82

Sonwani S, Maurya V (2018) Impact of air pollution on the environment and economy. In: Air pollution: sources, impacts and controls. CABI, Oxford. ISBN 9781786393890

Sonwani S, Saxena P (2016) Identifying the sources of primary air pollutants and their impact on environmental health: a review. IJETR 6(2):111–130

Sparks TH, Jeffree EP, Jeffree CE (2000) An examination of the relationship between flowering times and temperature at the national scale using long-term phenological records from the UK. Int J Biometeorol 44:82–87

Taiz L, Zeiger E (2014) Plant physiology, 6th edn. SAGE, Los Angeles

Taiz L, Zeiger E (2016) Plant physiology, 7th edn. SAGE, Los Angeles

Taub DR, Wang XZ (2008) Why are nitrogen concentrations in plant tissues lower under elevated CO_2? A critical examination of the hypotheses. J Integr Plant Biol 50:1365–1374. https://doi.org/10.1111/j.1744-7909.2008.00754.x

Teixeira E, Fischer G, Velthuizen H, Walter C, Ewert F (2011) Global hot-spots of heat stress on agricultural crops due to climate change. Agric For Meteorolo 170:206–215. https://doi.org/10.1016/j.agrformet.2011.09.002

Teixeira EI, Fischer G, van Velthuizen H, Walter C, Ewert F (2012) Global hot-spots of heat stress on agricultural crops due to climate change. Agric For Meteorol. (in press)

Tjoelker MG, Oleksyn J, Lee TD, Reich PB (2001) Direct inhibition of leaf dark respiration by elevated CO_2 is minor in 12 grassland species. New Phytol 150:419–424

Tripathy R, Ray SS, Singh AK (2009) Analyzing the impact of rising temperature and CO_2 on growth and yield of major cereal crops using simulation model. In: Panigrahy S et al (eds) ISPRS Archives XXXVIII-8/W3 Workshop Proceedings: Impact of Climate Change on Agriculture, India

Van De Geijn SC, Goudriaan J (1996) The effects of elevated CO_2 and temperature change on transpiration and crop water use. In: Bazzaz F, Sombroek W (eds) Global climate change and agricultural production. FAO and Wiley, New York, pp 101–122

Van der Westhuizen MM, Cramer MD (1998) The influence of elevated rhizosphere dissolved inorganic carbon concentrations on respiratory O_2 and CO_2 efflux in tomato roots. J Exp Bot 49:1977–1985

Vanaja M, Maheswari M, Jyothi Lakshmi N, Sathish P, Yadav SK, Salini K, Vagheera P, Vijay Kumar G, Razak A (2015) Variability in growth and yield response of maize genotypes at elevated CO_2 concentration. Adv Plants Agric Res 2:42. https://doi.org/10.15406/apar.2015.02.00042

Venkataramanan S (2011) Causes and effects of global warming. Indian J Sci Technol 4:226–229. https://doi.org/10.17485/ijst/2011/v4i3/29971

Wand SJE, Midgley GF, Jones MH, Curtis PS (1999) Responses of wild C4 and C3 grass (Poaceae) species to elevated atmospheric CO_2 concentration: a meta-analytic test of current theories and perceptions. Glob Chang Biol 5:723–741

Wang Y, Frei M (2011) Stressed food – The impact of abiotic environmental stresses on crop quality. Agric Ecosyst Environ 141(3):271–286

Wang X, Lewis JD, Tissues DT, Seemann JR, Griffin KL (2001) Effects of elevated atmospheric CO_2 concentration on leaf dark respiration of *Xanthium strumarium* in light and in darkness. PNAS 98(5):2479–2484

Wien HC (1997) The physiology of vegetable crops. CAB International, Oxon-New York

Willett KM, Gillett NP, Jones PD, Thorne PW (2007) Attribution of observed surface humidity changes to human influence. Nature 449:710–712

Woodward FI (2002) Curr Opin Plant Biol 5:207–211

Yang LX, Huang HY, Yang HJ (2007a) Seasonal changes in the effects of free-air CO_2 enrichment (FACE) ion nitrogen (N) uptake and utilization of rice at three levels of N fertilization. Field Crop Res 100:189–199

Yang LX, Wang YL, Huang JY (2007b) Seasonal changes in the effects of free-air CO_2 enrichment (FACE) on phosphorus uptake and utilization of rice at three levels of nitrogen fertilization. Field Crop Res 102:141–150

Ziska LH, Blumenthal DM, Runion GB, Hunt ER, Diaz-Soltero H (2011) Invasive species and climate change: an agronomic perspective. Clim Change 105:13–42

Chapter 6
Air Pollution and Its Role in Stress Physiology

Deepti Goyal, Alpa Yadav, and Tanvi Vats

Abstract Environment plays a crucial role in the physiological processes of plants. The numerous biotic and abiotic stresses in the plant habitat trigger complex responses in vital processes like photosynthesis, respiration and stomatal function. In this chapter we discuss the effect of various air pollutants on the stress physiological parameters. These studies are crucial because one of the major responses to plant pollutants is the inhibition of photosynthesis. This inhibition of photosynthesis not only alters the growth pattern and longevity but also changes plant phenology. Besides, assimilation of pollutants into the plant processes ultimately leads to their inclusion in the animal community. All this leads to a vicious cycle wherein the ecological factors suppress plant growth and in turn plants hamper the ecology. In this chapter we have also reviewed and highlighted the mechanistic aspect of the pollutants on the vital physiological parameters. The major pollutants which are emphasized are sulphur dioxide (SO_2), nitrogen oxides (NO_x) and ozone (O_3) while physiological parameters reviewed are stomatal conductance, photosynthesis and respiration and photorespiration. These physiological processes are important parameters in governing growth and health of plants. Because all the natural processes are cyclic in nature, it is pertinent to observe that the stress in plants caused by the pollutants also directly and indirectly affects the human population.

Keywords Environmental stress · Physiological processes · Air pollutants · Mechanistic pathway

---

D. Goyal · A. Yadav · T. Vats (✉)
Gautam Buddha University, Greater Noida, Uttar Pradesh, India
e-mail: tanvi@gbu.ac.in

P. Saxena, A. Srivastava (eds.), *Air Pollution and Environmental Health*,
Environmental Chemistry for a Sustainable World 20,
https://doi.org/10.1007/978-981-15-3481-2_6

6.1 Introduction

The equilibrium between ecosystem stability and socio-economic upgradation is dwindling day by day. The ecosystem stability and its dynamics are key parameters which determine the structure and function of any ecosystem. These parameters are maintained by various biotic and abiotic factors. The factors which negatively affect the agricultural and natural ecosystems include global climate change, deforestation, shifts in land use pattern and air pollution. It is important to note that these factors are interrelated and are not exclusive of each other.

In comparison to other factors, air pollution mostly affects flora and fauna. There are two ways by which airborne pollutants affect ecosystem, directly by toxicity and indirectly by altering soil nutrient availability. It is well established that a single factor does not affect terrestrial ecosystems but a multitude of factors result in chronic exploitation of ecosystem (Taylor et al. 1994). Increased concentrations of CO_2, elevated ultraviolet B (UV-B) radiation, high nitrogen deposition, nutrient deficiencies, drought or temperature extremes are the most emphatic stresses that degrade and hamper the plant characteristics.

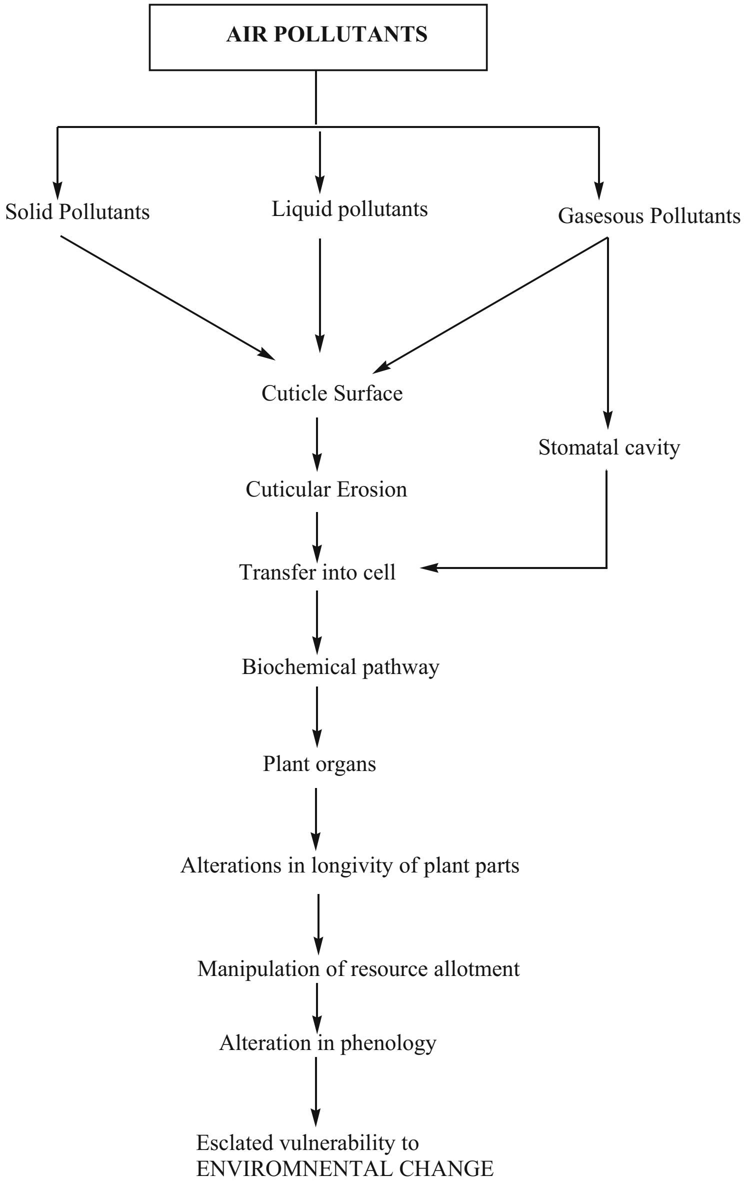
One can safely assume that the air pollutants concern the above-ground parts of the plants in a greater manner than the roots because they are directly exposed to the pollutants. The air pollutants including gaseous pollutants, dust particles and aerosols are adsorbed directly on the large leaf surfaces of vegetation and impact plant function and structure (Mukherjee and Agrawal 2018). The most important plant processes affected by the air quality deterioration are altering of species composition and structure, rate of decomposition, growth and morphology, physiological processes like photosynthesis, respiration, photorespiration and stomatal conductance, leaf functional traits and bioaccumulation of toxic chemicals. The pollutants penetrate from environment into the cells and act as an important carrier in the chain. This is represented in Scheme 6.1.

The pollutants affect the different physiological processes to different extent. The general parameters used to quantify these processes are tabulated in Table 6.1.

Air pollutants are classified as (a) primary pollutants and (b) secondary pollutants. The primary pollutants are the pollutants which are directly released from stationary and mobile sources. Figure 6.1 gives the provenance of primary pollutants.

The primary pollutants undergo chemical changes and reactions to generate secondary pollutants. The formation of secondary pollutants is depicted in Scheme 6.2.

Although there are several pollutants which generate stress in plant physiology, in this chapter we will be discussing only SO_2, NO_x and O_3. The deleterious effects of harmful atmospheric pollutant such as sulphur dioxide, ozone, oxides of nitrogen, peroxyacetyl nitrate, and fluoride on the physiological, morphological and biochemical aspects of flora have been widely reviewed (Baek and Woo 2010). These pollutants mainly disturb the biochemical and physiological processes and cellular structure of the plants (Saxena and Kulshrestha 2016a, b). It is also believed that the pollutants initially disturb the biochemical processes (photosynthesis, respiration,



Scheme 6.1 Pathway of air pollutants: From air to the plant

lipid and protein biosynthesis, etc.), and then attack the ultrastructural level (disorganization of cellular membranes), and cellular level (cell wall, mesophyll and nuclear breakdown) (Saxena and Kulshrestha 2016a, b).

Table 6.1 Quantification of physiological parameters

Process	Tested parameter
Photosynthesis	Chlorophyll fluorescence CO_2 Fixation Gas exchange/stomatal function Photosynthetic functions like ATP production, electron transport, enzymes and metabolites involved in Calvin Cycle
Respiration	Gas exchange Mitochondrial functions Enzymes involved in respiration
Water parameters	Transpiration Stomatal function Water potential Permeability of cuticle
Nutrients	N, P, K, S, Mg, Ca Iron contents
Biorhythms of plants	Electrical conductance in leaves

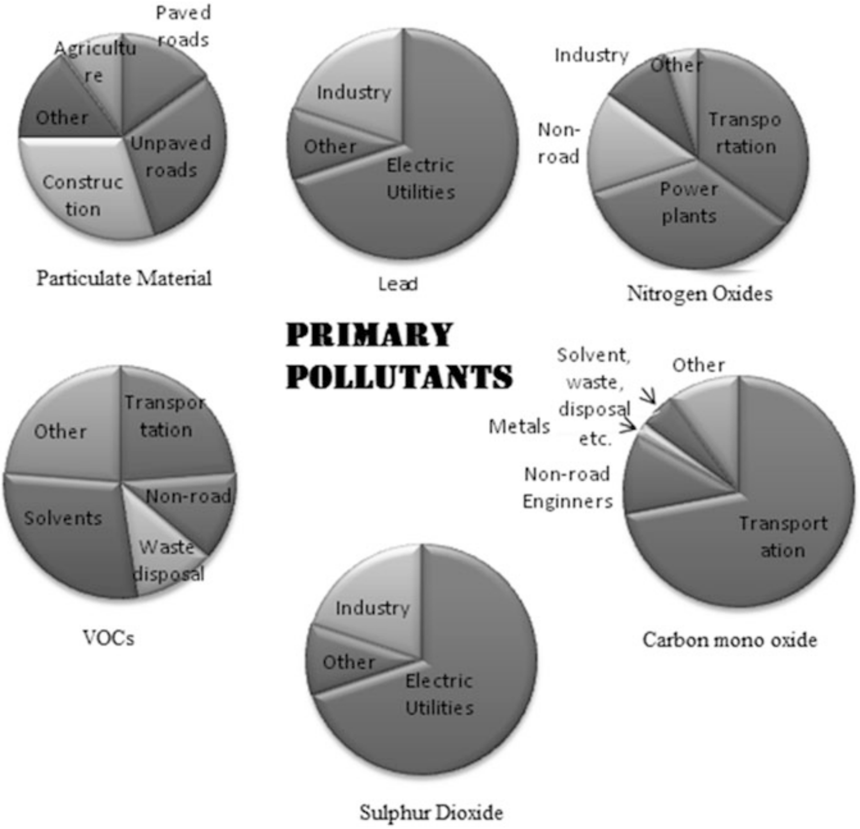
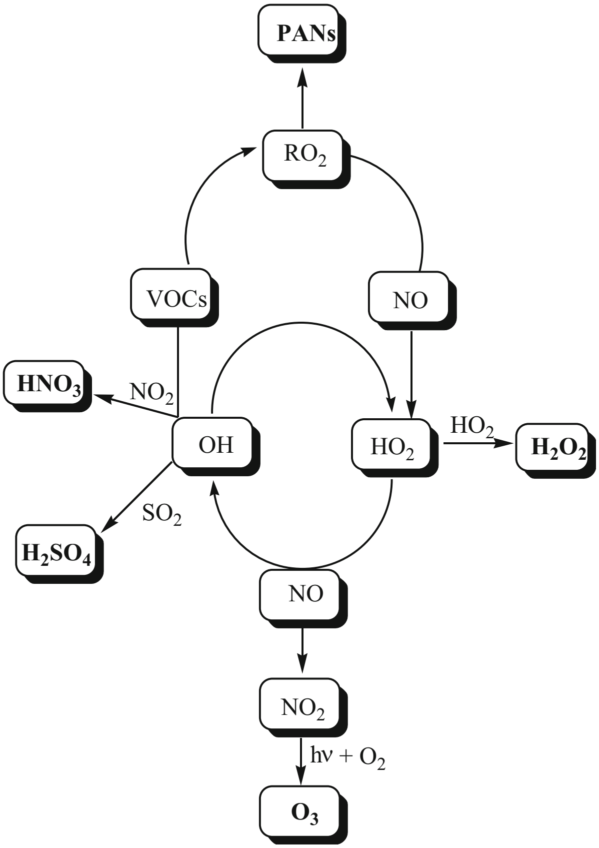


Fig. 6.1 Sources of primary pollutants



Scheme 6.2 Formation of secondary pollutants

6.2 SO_2 and Its Effect on Plant Physiology

One of the most widespread and dangerous air pollutants is sulphur dioxide (SO_2). The main sources of its origin include the burning of sulphur containing fossil fuels and smelting of sulphur containing metals. Another prominent source of SO_2 in winters is crop cultivation using a greenhouse. Greenhouses are meant to keep warm by burning fuels like diesel oil, heavy oil, kerosene and by-product oil, all of which have high sulphur content and their combustion leads to high SO_2 emission (Park et al. 2010).

SO_2 affect the environment both in gaseous as well as aqueous form. In aqueous form, SO_2 in the atmosphere results in acid rain, which is very damaging for plants, trees and forests. Acid rain leaches essential nutrients like calcium and magnesium from soil, which results in the plantation getting more prone to infection and damage by cold weather and insects. Not only this, aluminium also is removed from the soils which hinders the water up-taking capacity of the trees. Besides, acid rain destroys the outer coating of leaves, hampering the photosynthesis. In human beings even the trace amount of acid particles leads to respiratory problems like asthma, chronic bronchitis and pneumonia. In the aquatic system, increase in acid content reduces the pH of water bodies leading to fish mortality.

In gaseous form, SO_2 affects the human health by entering through the respiratory tract. It causes irritation in the skin, and mucous membranes of eyes, nose, throat and lungs, which is responsible for throat irritation, coughing, wheezing and breathing difficulty. High concentration of SO_2 can affect lung function, worsen asthma attacks and heart disease in sensitive groups.

Apart from living organisms, SO_2 is equally hazardous to man-made materials. It severely damages a variety of carbonate-containing building materials like limestone, marble and mortar.

SO_2 attacks on leather causing disintegration of leather goods. In case of metals though aluminium is almost noble to SO_2 attack, other metals like iron and steel get highly corroded by it. Various other materials like paper, wool, cotton etc. also deteriorate on SO_2 exposure leading to embrittlement and eventual loss of strength (Saxena et al. 2019).

Sulphur being a key constituent of amino acids, proteins and a few vitamins, is essential for plant metabolism. A low concentration of SO_2 is necessary for physiological growth of plants (Darrall 1989), especially in sulphur-deficient plants in which sulphate might be metabolized to sulphur to fulfill nutrition in plants (DeKok 1990). But at higher concentration of SO_2, general disruption of photosynthesis, respiration and other fundamental cellular processes can occur. This can be understood as a chain of events wherein an increased uptake of SO_2 leads to a buildup of sulphites and sulphates which in turn are cytotoxic and stop the growth and productivity of plants (Darrall 1989; Agrawal and Verma 1997). SO_2 toxicity has a rather adverse effect on plant pigments and therefore SO_2 exposure reduces photosynthetic activities. SO_2 exposure also leads to tissue damage and the most affected areas are stomata, cell membranes and leaves while the most affected functions are transpiration, membrane transport and permeability. These all, in unison leads to reduced plant growth and a diminished yield (Crittenden and Read 1978; Unsworth and Ormrod 1982).

SO_2 uptake can be both from root as well as shoot system. Sulphur is taken in the form of sulphate ions by the root and is assimilated into organic sulphur compounds. These sulphur compounds are employed in various biochemical processes and thus eventually become a part and parcel of the ecosystem (Omasa et al. 2002; De Kok et al. 2002).

The SO_2 uptake by the plant's shoot system can be shown as in the schematic representation in Fig. 6.2.

Plants do not show a uniformity in responses to SO_2 exposure due to their different absorption efficiency towards the gas as well as their capability to remove the excessive sulphur and detoxify the pollutants. Once the SO_2 enters in the plant through leaf, it dissolves in the moisture present in mesophyll cell and converts into sulphite and bisulphate (Kulshrestha and Saxena 2016). These toxic elements (sulphite and bisulphate) are then translocated to other parts of the plant. Several studies have been done during the past two decades to understand the effect of SO_2

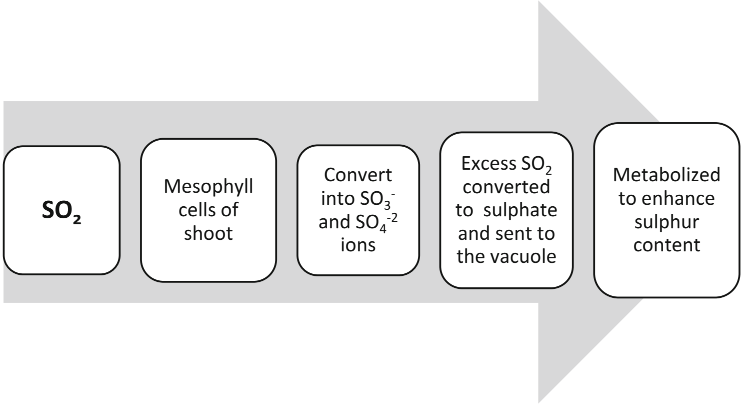


Fig. 6.2 SO_2 uptake by shoot of plants

Table 6.2 Classification of plants on the basis of extent of SO_2 exposure effects

Type of Plants	Species	Exposure effect
Herbs	Wheat, Barley, Pea, Beet, Bean, Carrot, Chilli, Petunea, Oat, Potato, Tobaco.	Immediate deterioration
	Croton, Opuntia, Nerium, Vinca	Slow, chronic effect
Shrubs and Trees	Mango, Arjun, Sisso, Jamun	Immediate deterioration
	Neem, Banyan, Bougainvillea, Chatim, Jarul, Sims, Pongamia pinnata	Slow, chronic effect

on various plants species. On that basis, a list of acute and chronic effects on plants is given in Table 6.2.

Exposure to SO_2 at even low concentrations may have several damaging effects on plants, such as:

- Reduction in photosynthetic and transpiration rate
- Increase in respiration rate
- Increase in stomatal conductance
- Reduction in chlorophyll content
- Membrane lipid peroxidation

6.2.1 SO_2 and Its Effect on Stomata

The pollutants enter into the plant through the leaf having abundant stomata on its surface. The response of stomata towards SO_2 depends upon species of plant, concentration of SO_2, plant age and environmental conditions. It is also found that exposure time of SO_2 affects opening and closing of stomata (Saxena and Sonwani 2019a, b, c). When a leaf gets exposed to SO_2 for a short time it causes stomatal opening, while for long-time exposure it causes stomatal closing (Abeyratne and Ileperuma 2006; Raschk 1975; Rao et al. 1983; Verma and Singh 2006; Robinson et al. 1998; Bytnerowicz et al. 2007). The effect of SO_2 concentration on stomatal opening is different in different plants (Biggs and Davis 1980). In one plant species it can cause stomatal opening while in another stomatal closing (Mudd 1975).

The SO_2 uptake depends upon the pore size and quantity of stomata, which affect the turgidity of guard cells. Long-term exposure of high concentration of SO_2 reduces the ability of guard cells to collect sulphur and open or close the stomata (Guderian 2012; Knabe 1976), which then alters the fabrication and supply of photosynthates (Khan and Khan 1993). There is decrease in stomata abundance (Olszyk and Tibbitts 1981; Kumari and Prakash 2015; Koziol and Whatley 2016), which is a necessary action to avoid entrance of high-level SO_2 into the leaf, due to which damaging of plant tissues can occur (Kumari and Prakash 2015).

Abeyrante and Ileperuma (2006) have studied the effect of SO_2 on stomatal pore width of *Argyreia populifolia* leaves at the three sampling sites of the Peradeniya University Park, Sri Lanka. Sampling site 1 was reported at high SO_2 concentration and other two locations (sampling site 2 and 3) were with moderate SO_2 concentration. A reduction (almost 50%) in the values of both length and width of stomatal pore were observed at sampling site number 1, whereas sampling sites 2 and 3 gave almost identical values of pore length and width (Abeyratne and Ileperuma 2006).

A decrease in cellular pH responsible for stomatal closure is also reported due to sulphur dioxide fumigation. This is due to the fact that SO_2 reacts with cellular water content and produces sulphuric acid according to the following reaction:

$$\left(SO_2 + H_2O \rightarrow [SO_2 \cdot H_2O] \rightarrow HSO_3^{-} + H^+ \rightarrow SO_3^{2-} + 2H^+\right)$$

This may lead to inhibition of K^+ pump, responsible for stomatal closure (Dhir 2016) which thus affects the photosynthetic yield. Another reason for closing of stomata is the presence of abscisic acid (AbA) hormone in the leaf which is produced due to exposure to SO_2 (Hu et al. 2014). In case of high SO_2 exposure, stomatal conductance also gets reduced which affect the physiological processes of photosynthesis (Choi et al. 2014a; Liu et al. 2017).

Majemik and Mansfield (1971) found that SO2 does not affect the normal diurnal cycle of opening and closing of stomata, but increases the apertures during the day in plants (Majernik and Mansfield 1971). Similar results were found in another study on a plant species *Vicia faba*. The stomatal conductance was increased by 20–25% on exposure to low SO_2 concentrations (Black and Black 1979). This enhanced opening was responsible for damage of epidermal cells adjoining to the stomata.

In another study stomatal abundance and increase in epidermal cells in leaves of *Azadirachta indica* and *Polyalthia longifolia* (Pal et al. 2000), *Cassia siamea* (Aggarwal 2000), and *Nyctanthes arbortristis*, *Quisqualis indica* and *Terminalia arjuna* (Rai and Kulshreshtha 2006) on SO_2 exposure have been found. Along with this, reduced stomata and epidermal cells size with exposure to SO_2 has also been found from other researchers' works (Aggarwal 2000; Kaur 2004; Dineva 2006). This may be due to inhibited cell elongation, leaf area and increase in cell occurrence (Rai and Kulshreshtha 2006).

6.2.2 SO_2 and Its Effect on Photosynthesis

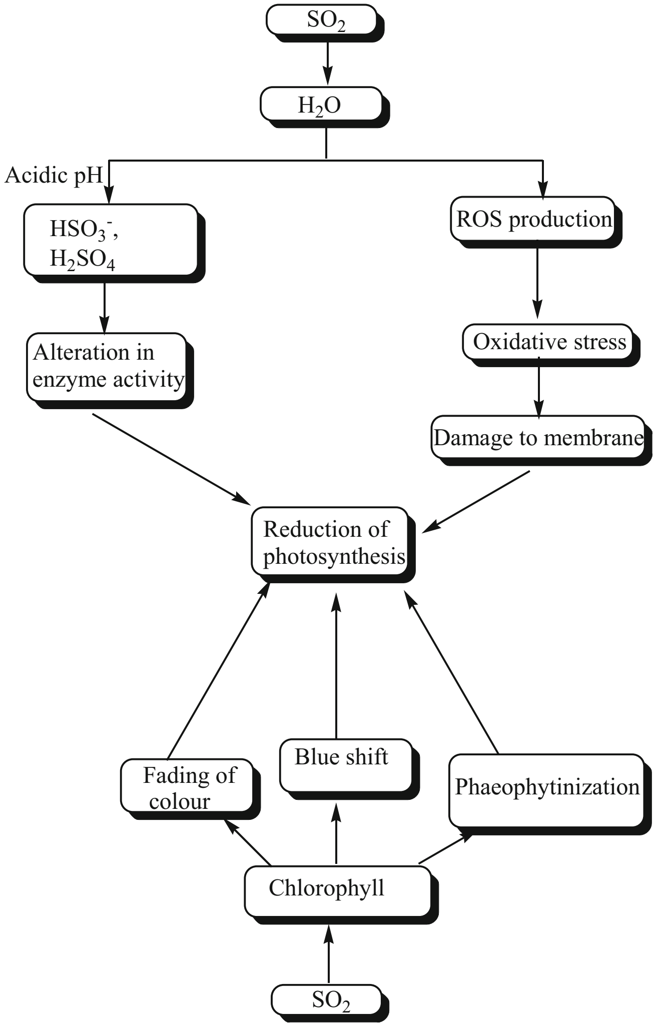
The vital physiological process of photosynthesis is highly sensitive to SO_2 concentration and duration of exposure (Saxena and Sonwani 2020). In various studies it was found that short-time exposure to low SO_2 concentrations generally stimulates photosynthesis, whereas long-time exposure even at low concentration of SO_2 was responsible for inhibition of photosynthesis (Gheorghe and Ion 2011).

SO_2 destroys electron transport between photosystems, which decreases the rate of electron transport throughout the chain. The overall result of this is the reduced rate of photosynthesis (Gheorghe and Ion 2011). Another reason for reduced photosynthetic rate on SO_2 exposure may be due to reduced amount of chlorophyll (Aminifar and Ramroudi 2014; Hetherington and Woodward 2003). SO_2 exposure effect on chlorophyll can be trifurcated in three ways:

1. Fading of chlorophyll color
2. Phaeophytinization wherein chlorophyll molecules get degraded to phaeophytin (less active molecule))
3. Blue shift in pigment spectrum of lichens (Hetherington and Woodward 2003)

In a study on oak species, *Gracia* and coworkers found that decrease in photosynthetic rate could be due to reduction in protein contents and decreased carboxylation efficiency resulting in a reduced CO_2 uptake besides the chlorophyll factors (Farage et al. 1991). Because of its acidifying properties, a very high concentration of CO_2 acts as inhibitor. Reduction in leaf area and CO_2-induced shift in the timing of the leaf ontogenetic processes (Miller et al. 1997; Rey and Jarvis 1998) may also be the additional factors for reduced photosynthesis. Similar results were found for rice and spinach.

SO_2 deteriorates the height and girth of plant axis. It is well documented in literature that the photochemical efficiency of photosystem II in a healthy leaf ranges between 0.74 to 0.85, which gets drastically reduced when exposed to SO_2 (Choi et al. 2014b; Seyyednejad and Koochak 2011a; Lichtenthaler et al. 2005; Sobrado 2011). Furthermore, SO_2 exposure also inhibits the activity of essential Calvin cycle enzymes like Fructose bisphosphatase and Ribulose bisphosphate carboxylase (Chung et al. 2011). Reduction in the total chlorophyll content upon exposure to the gaseous SO_2 has also been documented in the literature. This may be due to the negative impact of SO_2 on chlorophyll metabolism (Choi et al. 2014b; Seyyednejad and Koochak 2011b). In addition to this, Seyyednejad and Koochak demonstrated that in *Prosopis juliflora*, the concentration of photosynthesis pigments like chlorophyll carotenoids Fwas decreased when leaf was exposed to SO_2. The reason behind



Scheme 6.3 Reduction of photosynthesis on SO_2 exposure

this is the deposition of suspended particulate on leaf surface (Seyyednejad and Koochak 2011b).

The reduction in photosynthesis on SO_2 exposure is represented in Scheme 6.3.

6.2.3 SO_2 and Its Effect on Respiration and Photorespiration

Respiration also called dark respiration is a metabolic pathway which produces energy-rich molecules by the breaking of larger molecules like carbohydrates

(Sonwani and Saxena 2016). In general, the rate of respiration increases when a plant is exposed to gaseous pollutants viz. SO_2, O_3, HF and NO_2. Some researchers have found that the rate of dark respiration increased when exposed to 35–380 ppb concentration of sulphur dioxide, while some other haves reported no change at 20–4000 ppb concentration of SO_2 (either short term, i.e., less than 8 h or long term, i.e., more than 1 day). Black and Unsworth (1979) studied the effect of SO_2 on one species, *Vicia faba* and they observed that the increase in the rate of dark respiration was not affected by the SO_2 concentration from 35 to 175 ppb (Black and Unsworth 1979). In addition to this, SO_2 exposure also affects the respiration rate in lichens and bryophytes. In some cases when certain lichens such as *C. impexa, Hypogymnia physodes, and Usnea fragiliscence* were exposed to a low concentration of aqueous solution of SO_2 with 23–27 ppm concentration, a decrease in rate of respiration was observed. On the basis of these findings, we can conclude that the change in respiration rate mainly depends on the concentration of SO_2.

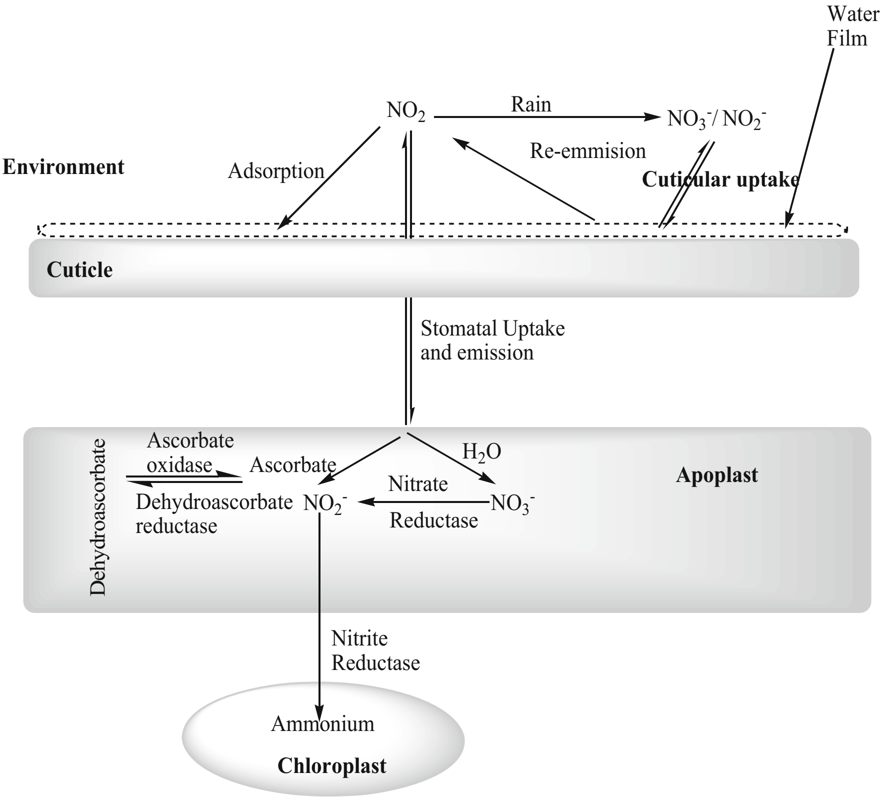
Photorespiration, also known as light respiration, occurs in chlorophyll tissues of plants in the presence of light and at higher O_2 concentration (Saxena and Naik 2018). It is a distinguished aspect of C_3 plants and essentially absent in C_4 plants. Generally, air pollutant has little effect on photorespiration. At high SO_2 concentration (1000 ppb), inhibition in photorespiration occurred. The effect of sulphur dioxide on photorespiration is also found in higher plants, due to which their productivity is greatly influenced (Black 1984).

6.3 NO_X and Its Effect on Plant Physiology

NOx, a primary air pollutant, enters into the environment through fuel combustion processes. The increase in automobile exhaust emissions from industrialized areas is responsible for increase in NO_x concentration (Munzi et al. 2009; Hu et al. 2015; Hultengren et al. 2004). NO_x is mainly composed of NO (>90%) and NO_2, which can covert into each other in sunlight and in the presence of some gases like O_3. NO_2 also releases some harmful pollutants in the environment like O_3 and particulate matters (Rahmat et al. 2013; Bermejo-Orduna et al. 2014; Marais et al. 2017).

Research studies have adopted two assumptions for plant response to NO_2 exposure. In one assumption it is proposed that NO_2 can produce nitrogenous compounds by its metabolism and incorporation in the nitrate assimilation pathway, which does not cause any visible injury (Stulen et al. 1998). Some other studies anticipated that the majority of the plants show evidence of fewer amounts of NO_2 (Middleton et al. 1958).

On the basis of a general hypothesis, it is believed that when NO_2 is present in high concentration it can cause extreme accumulations of NO_2^- (Okano and Totsuka 1986) and cell acidification (Schmutz et al. 1995). Due to this, deterioration of plant growth occurs by production of reactive oxygen species (ROS) and inhibition of absorption of N (Sonwani and Maurya 2018). On the other hand, some different physiological responses were obtained on NO_2 exposure. Therefore, there is a



Scheme 6.4 Assimilation of NO_2 into chloroplast via stomata

disagreement on the effects of NO_2 exposure on plants and a united conclusion has not been reached.

Various environmental factors (Gebler et al. 2000; Chaparro-Suarez et al. 2006), stomatal dimension and conductance (Chaparro-Suarez et al. 2011; Breuninger et al. 2013) affect the foliar NO_2 uptake. NO_x is a free radical gas which transfers electrons crossways biological membranes due to which reactive oxygen species (ROS), a by-product of other biological reactions is generated (Xu et al. 2010). The NO_2 uptake in plants may be done directly through the stomata and/or through roots. The assimilation of NO_2 into chloroplast via stomatal route is given in Scheme 6.4.

The mechanism of absorption of gaseous NO_2 into leaf through stomata is proposed in two ways.

1. Either it is disproportionated to nitrate and nitrite in the apoplast

$$NO_2 + H_2O \rightarrow H^+ + NO_3^- + NO_2^-$$

2. Or absorbed in leaf apoplast by ascorbate

$$\mathrm{Asc} - \mathrm{OH} + \mathrm{NO_2} \rightarrow \mathrm{Asc} - \mathrm{O} + \mathrm{H^+NO_2^-}$$

In apoplast NO_2^- is converted to NO_3^- where it is metabolized by enzyme nitrate reductase (NaR) producing NO_2,where it is taken up from the apoplast and then transported into the chloroplast (Eller et al. 2011). At high concentration NO_2 is capable of being stimulated upon nitrate reductase and responsible for more intense reduction of nitrite to ammonia and amino acids (Erisman et al. 2007)

It is pertinent to note that there are relatively few reports on effect of NO_x on plant physiology, perhaps because of the fact that it is less toxic as compared to SO_2. It is also found that the effects of NO_x are most prominent when it is combined with SO_2 (Carlson 1983; Whitmore and Mansfield 1983; Freer-Smith 1985). In uncombined form NO_x is damaging only at high concentrations (Reinert et al. 1982). Furthermore, NO has less toxic effect than NO_2, which may be attributed to its less solubility in water which in turn leads to lower uptake (Mansfield and Freer-Smith 1981). Another reason could be that NO gets easily converted to NO_2; therefore, the effects of NO are difficult to quantify. Nowadays, the concentration of NO_2 is steadily increasing and in some countries it exceeds the concentration of SO_2 (Lane and Bell 1984a; Martn and Barber 1984). It is also found that in rural areas the concentration of NO and NO_2 may be same, but in urban areas the NO concentration is high (Lane and Bell 1984b).

6.3.1 Effect of NO_2 on Stomatal Conductance

On foliar surface, the flow of NO_2 into the leaf has been a matter of intense discussion (Rogers et al. 1979; Fatima et al. 2018; Thoene et al. 1996; Teklemariam and Sparks 2006; Gebler et al. 2000). The exhaustive studies lead to the conclusion that the NO_2 deposition was much more than the cuticular deposition through stomatal contribution to the total leaf. Some studies suggested that cuticular contribution is upto 5% in most of the cases (Saxena and Sonwani 2019a, b, c). In 1900, Wellburn introduced that at 140 ppb NO_2 concentration, deposition through the stomata was higher than the deposition on the cuticle (Wellburn 1990).

The NO_2 uptake process is highly influenced by the water films. It is proposed that absorption of water occurs through water film of plant surface. High humidity leads to deposition of water films on the leaf surface which in turn serves as sink for atmospheric NO_2 (Burkhard and Eiden 1994). Some researchers suggested that NO_2 consumption is solely depended on stomatal opening and that cuticular expulsion was entirely ruled out.

The stomatal dynamics as well as stomata-related physiological and biochemical processes are affected by the corrosive and oxidizing attributes of NO_2 (Takagi and Gyokusen 2004; Mazarura 2012). In a recent study on populous trees, Yanbo Hu et al. (2015) showed that NO_2 gas has remarkable adverse influence on stomata connected with physiological processes of *Populus alba* and *P. berolinensis* leaves (Hu et al. 2015).

6.3.2 Effect of NO_x on Photosynthesis

Reduced photosynthesis is observed in various plants when exposed to gaseous NO_x, even at concentrations that do not produce visible injury (Hill and Bennett 1970; Capron and Mansfield 1976). It was also been observed that the effect of NO was much more rapid than the effect of NO_2 (Hill and Bennett 1970). In another study it was found that NO_2 concentration and exposure time were responsible for reduced photosynthesis (Srivastava et al. 1975). The effect of NO_x on photosynthesis is much less than other pollutants. Short-term exposure (< 8 h) of NO_2 between 500 to 700 ppb and continuing exposure (⍰20-h period) at 250 ppb, can cause changes in photosynthetic rate (Hill and Bennett 1970; Capron and Mansfield 1976). In variation to above, nitric oxide disrupted four times like NO_x gets reduced to dioxide at 1000 ppb in a 4d ventilation of a variety of greenhouse plants (Saxe and Murali 1989).

Reduction of NO_2, into nitrite and ammonia was found when NO_2 entered into the plant, by reduced ferredoxin or by reduced nicotinamide adenine dinucleotide phosphate (NADPH) Reduction of NO_2 could be explained in the rate of photosynthesis as on the basis of presence of NADPH for nitrite reduction and absorb carbon in the chloroplast. Furthermore, the acidic behaviour of NO_2 can change the electron movement and photophosphorylation. As photoelectron systems are associated with chloroplast membranes, any changes in their structures would influence activities of the photosystems (Hill and Bennett 1970; Srivastava et al. 1975).

NO_2 exposure is also responsible for swelling of chloroplast membranes (Wellburn 1990). This may result if NO_2 is reduced into ammonia, which is not rapidly incorporated into amino-forms and thus responsible for inhibition of photosynthesis by uncoupling electron transport (Avron 1960). Similarly, in some lichen species, reduced chlorophyll content was observed on NO_2 fumigation (Nash 1976). The inhibition in pigment synthesis on NO_2 exposure is also documented in the literature. This may be due to inhibition in photooxidative processes, which may affect the synthesis of chlorosis. Moreover, rise in percentage of chlorophyll by about 10% occurred in *Pisum sativum* with NO_2 exposure in some other study (Horsman and Wellburn 1975).

In some investigations, researchers found the effects of NO on photosynthesis rate of glasshouse crops, particularly the tomato (*Lycopersicon esculentum*). From the results, they concluded that in controlled fumigation, some NO is oxidized to NO_2. So, it is difficult to interpret the effect of NO on photosynthesis since atmosphere will contain a blend of the oxides (Saxena and Sonwani 2019a, b, c). It is also found that it is not clear that which oxide is the more toxic. In a latest research it has been reported that with NO rate of photosynthesis decreases rapidly as compared to NO_2 (Hill and Bennett 1970).

In comparison with the effect of NO_2 alone, spraying with a mixture of NO_2 and SO_2 has been found to show adverse effect on the rate of photosynthesis. At lower concentration (200-250 ppb), the combined effect of these gases on inhibition of net photosynthesis was much higher than with these gases individually. The study was done on various plant species like *Medicago sativa*, *alfalfa*, and *Glyeine max* (Carlson 1983). Thus, nitrogen dioxide and nitric oxide had reported good results only at high concentrations, that is 500–700 ppb and above, but when it combines with sulphur dioxide, effect of inhibition is high than that single gas.

6.3.3 Effect of NO_2 on Respiration and Photorespiration

Respiration is an important process for plant metabolism and growth, and also for rebuild and neutralization of the toxics (Koziol and Whatley 2016). Currently, there is no compatible way to recognize the effects of nitrogen dioxide on respiration. At concentrations between 40 and 400 ppb of nitrogen dioxide or nitric oxide, no effect was found on inhibition and stimulation, but high concentrations of these two gases, that is 1000–7000 ppb, showed effective behaviour for the same. Bengtson et al. (1982) have been studying the effect of NO and NO_2 on *Pinus sylvestris* at 40–400 ppb for 6 h. They found ineffective behaviour of NO_x on respiration in the absence of light at this concentration and time. In another study on pot plant cultivators, it was demonstrated that on 1000 ppb of NO fumigation for 4 h, there was inhibition in one cultivator (5.1%), while under similar conditions of NO_2 fumigation, there was an increase in two cultivators (8.2%) (Grennfelt et al. 1983).

Sabaratnam and Gupta investigated the effect of NO_2 on one-month mature soybean plants. The plants were treated with different specified limits of NO_2 concentrations from 0.1 μl $liter^{-1}$ to 0.5 μl $liter^{-1}$ for 5 days (7 hour per day), under controlled environment. The results showed that above the concentration from 0.3 μl $liter^{-1}$ of NO_2, the rate of dark respiration was rapidly increased; this may be due to elevated activity of cellular physiology to metabolize the pollutant to non-toxic forms caused by NO_2 (Sabaratnam and Gupta 1988).

Oxides of nitrogen (NO and NO_2) are sometimes referred to as total reactive nitrogen oxides, which includes NO, NO_2, nitrous oxide (N_2O), nitric acid (HNO_3), nitrous acid (HNO_2), peroxyacetyl nitrate (PAN), organic nitrates, and other forms of oxidized nitrogen (Weller et al. 2002). Nitric oxide free radical is unstable and not easily deposited to surfaces in remarkable amounts (Horii et al. 2004). NOx species, mainly NO_2 a free radical, and a potent oxidant is related to deposition studies. It is principal constituent of urban air pollution (Jacobson et al. 2004). In atmosphere NO_2 is produced by the oxidation of nitric oxide (NO) which is formed by the oxidation of N_2 at high temperatures during combustion processes in energy production, burning of fossil fuels in automobiles by tropospheric ozone (O_3). O_3

rapidly converted NO to $NO_2$by oxidation process. NO_2 levels are used as an overall indicator of the atmospheric NOx status for U.S. EPA.

6.4 O_3 and Its Effect on Plant Physiology

The troposphere ozone is formed under sunlight via chain of chemical reactions with different intermediates of nitrogen oxides (NOx) and volatile organic compounds. Depending upon where it is found, ozone can be good or adverse. Good ozone forms a safety layer to shield us from harmful UV rays of sun (Sonwani and Kulshrestha 2016). The process of formation of ozone occurs naturally in upper atmosphere. Unfavourable ozone is formed in lower atmosphere, that is troposphere, where pollutants come in this level from man-made pollutants and from chemical reaction occuring in the presence of sunlight. Ozone at lower level is a destructive air pollutant. Ozone is a secondary pollutant formed in the presence of sunlight with nitrogen oxides (NOx), which comes mainly from automobiles and biomass burning, in the presence of volatile organic compounds as shown in Fig. 6.2.

Ozone in tropospheric region considered as a highly reactive pollutant, which produces adverse effects on plant development (Betzelberger et al. 2012; Wilkinson et al. 2012). Ozone goes into the plant through the stomata and reacts with different compounds connected with cell walls and membranes. The effect of ozone on plant development is determined with the concentration of ozone and the exposure time. Long-time exposures of ozone pollution on plant can change plant physiology, leading to changes in plant activities that can ultimately affect climate and atmospheric chemistry via transpiration, biogenic emissions, dry deposition, etc. Reduction of photosynthesis by dry deposition onto leaves is a major sink for ozone, but ozone exposure is also detrimental for the following phenomenon:

- Plant tissues → impact on ecosystems and crops.
- Reduces stomatal conductance (damage).
- Surface ozone is a major air pollutant (causing ~0.7 M deaths/year).
- Reduces leaf Area Index (damage).
- Toxicity of ozone on plants shows symptoms comprising foliar damage, premature leaf erosion, decrease in growth and limited belowground of proportion of carbon.

The effect of O_3 exposure on plants and their eventual influence on ecosystem is given in Fig. 6.3.

6.4.1 Effects of Ozone Stress on Stomatal Conductance

The mechanistic pathway of ozone influence on stomatal conductance is by injuring the epidermal cells and causing them to break and open wide (Sonwani et al. 2016).

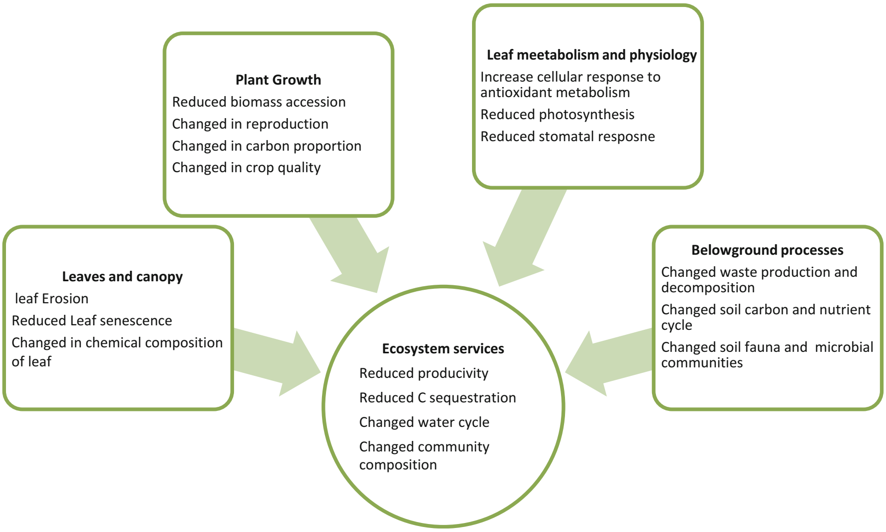


Fig. 6.3 Effect of O_3 exposure on plant ecosystem

This leads to closing of stomata and decrease in stomatal conductance. Besides stomatal functions, ozone exposure damages photosynthetic tissues and reduces photosynthetic pigments (Saxena et al. 2017).

The O_3 exposure goes hand in hand with the weather conditions. Stagnant weather conditions limit the dose of ozone absorption. Also, it is interesting to note that low humidity leads to low stomatal conductance.

On studying the effect of O_3 exposure on *B. nigra*, it was observed that even a short-term exposure reduced stomatal conductance and leaf transpiration, resulting in low quantities of intracellular CO_2. A long-term exposure, on the other hand, increased intracellular CO_2. The bleaching and chlorosis of the leaves was observed and was attributed to increase in CO_2 concentration (Paoletti and Grulke 2005).

O_3 stress also brings about the appearance of *MYB44* gene. This gene takes a major role in responses to abioitc and biotic stress. It is believed that the appearance of *MYB44* elevates tolerance to low rainfall, by improving stomatal closure, leaf senescence and ROS scavenging. This is indicative of the extreme stress in plants because of O_3 exposure, matching draught- or famine-like situation (Baldoni et al. 2015; Jung et al. 2008; Jaradat et al. 2013).

6.4.2 *Effects of Ozone on Photosynthesis*

The plants exposure to ozone is the main internal factor which influences the rate of photosynthesis through multiple ways. The change in the photosynthetic rate on O_3 exposure depends on variety of plant, leaf age, O_3 concentration and exposure time, and other environmental conditions (Moldau et al. 1993; Dizengremel 2001). Along with these factors, reduced carboxylation efficiency is also found as an important factor for reduced photosynthesis (Pell et al. 1992, 1994; Farage and Long 1999). Due to the strong oxidizing nature of O_3, it significantly reduces photosynthesis and therefore responsible for reduction in ribulose 1.5-bisphosphate carboxylase/oxygenase (Rubisco) activity (Dizengremel 2001), and decreases the CO_2 uptake of leaf (Farage et al. 1991).

Many researchers have found that ozone generally decreases photosynthetic rate of plants (Bagard et al. 2008) and also reduces plant productivity (Dizengremel 2001). Along with this, O_3 is also responsible for chlorophyll degradation and early leaf ageing (Bergmann et al. 1999; Ranieri et al. 2001). In a similar study on soyabean leaves, a reduction in chloroplast content, photolysis and oxygen of water was found when soyabean leaves was exposed to O_3. This is due to the fact that this increased ozone accounts for reduced phosphorylation (Julia and Kangasjärvi 2015). Moreover, persistent O_3 exposure above 40 nL L^{-1} concentration produces reactive oxygen species (ROS) which prevent uptake of O_3 through leaf by closing the stomata (Bergmann et al. 1999; Ranieri et al. 2001; Vahisalu et al. 2010).

Many researchers have studied the effect of concentrations and exposure time of O_3 on photosynthesis to know the effect of threshold concentrations and dose–response relationships. Some studies show reduction in photosynthesis when short-term exposed at 100 ppb, while others show reductions at 200–500 ppb. On long-term exposure to O_3 (<1 d) at 35–45 ppb also effects photosynthesis in crop plants (Reich and Amundson 1985).

Some studies reveal that the effect of O_3 exposure on tree species was only at higher concentrations or at long-time exposure than in herbaceous plants (Barnes 1972). Other species were more tolerant. In some cases, it was established that the O_3 exposure affects the newly enlarged foliage much more than mature foliage. It was evident when newly enlarged foliage of white pine was exposed to 150 ppb of O_3 for 19 days; it did not survive for 77 days long exposure.

The effect of O_3 on photosynthetic rate in another plant species was calculated at 85 and 125 ppb ozone exposure, and it was found that this affect the quantum yield, light dispersion value and the light damages point of the photosynthesis responses.

The O_3 exposure also affects photosystem I and II in plants. When *Spinacia oleracea* chloroplast was exposed to O_3, the electron transport system in both photosystems was inhibited; however, photosystem I was found much more effected than photosystem II (Reich and Amundson 1985; Coulson and Robert 1974). This may be due to the reduced photophosphorylation which resulted from reduction in electron transport. O_3 affected the chlorophyll content of the treated plants (Leffler and Cherry 1974). In another research a strong connection was observed between chlorophyllioss and visible necrosis (Knudson et al. 1977). The reduction comparison of Chlorophyll a to Chlorophyll b was also observed on O_3 exposure. This may be due to the fact that chlorophyll a has much more affinity towards O_3 than chlorophyll b.

Schreiber et al. (1978) reported the effect of O_3 on the fluorescence characteristics in *Phaseolus vulgaris*. The results showed that a long-time exposure at low concentration was much more injurious than a short-time exposure at high concentration. On this basis it was concluded that the effect of O3 on fluorescence is due to its action on the donor site of Photosystem II. On further increase in O_3 exposure, it reduces the electron transfer from Photosystem II to Photosystem I (Schreiber et al. 1978).

6.4.3 Effects of Ozone on Respiration and Photorespiration

The respiration in plants is generally found to increase when exposed to ozone above a threshold concentration. The O_3 exposure can either raise (Todd 1958; Barnes 1972) or reduce respiration rate in plants. In a study, researchers observed no immediate effect on respiration after O_3 exposure on *Phaseolus vulgaris*, but after long exposure for about 24 h some adverse effects occurred (Pell and Brennan 1973). Furthermore, at 150 ppb concentration of ozone, decreased level of respiration occurred. Ozone exposure also inhibited respiration in *Nicotiana tabacum* leaf mitochondria (Lee 1967).

In several researches it is observed that the rate of photosynthesis also affects the rate of respiration in plants. If photosynthetic rate is very high, then a small change in the rate of respiration will not alter carbon balance of the plant, while low photosynthetic rate can cause changes in respiration and thus can affect the development of the plant.

6.4.3.1 Photorespiration

The photorespiratory pathway arises through the oxygenation of ribulose-1,5 bisphosphate (RuBP) by Rubisco that produced one molecule of 3-phosphoglycerate and one molecule of the 2-carbon compound phosphoglycolate. The photorespiratory cycle permits the process of changing of this compound into 3-phosphoglycolate through a number of reactions that controls across three different compartments—chloroplasts, peroxisomes and mitochondria and releases CO_2 and NH_3 (Mouillon et al. 1999). Lots of studies show that photorespiration adversely affected leaf phenology in ozone-treated leaves during photosynthesis process (Bagarda et al. 2008).

Stromal CO_2 concentration reduced when low stomatal conductance will promote photorespiration, thus reducing the C:O ratio. In the light one of the factors activating stomatal closure, dry period and salt/osmotic stress are noted. Despite that, many bacterial pathogens that capture on the leaf through the stomata, such as *P syringae*, can also activate this response (Melotto et al. 2008).

6.5 Conclusion and Future Recommendations

The present review concludes that several morphological parameters do get affected by the air pollutant-induced stress. Stress caused by air pollution results in decreased photosynthetic rate, chlorophyll content, stomatal conductance, net photosynthetic carbon dioxide assimilation and carboxylation effectiveness. These parameters primarily include leaf characteristics like cuticle, stomata, etc. The leaf stats in turn influence gaseous exchanges including respiration and photorespiration in plants which further increase environmental stress. It is imperative to understand that effect of individual pollutants is quite different from each other and also from species to species. For instance, NO_x in low concentration acts as a growth promoter.

Environmental stress along with the plant response towards them leads to an eventual slow-down of total biomass growth rate. The need of the hour is to address the knowledge gap in quantification of effect of individual pollutants as well as in combined form on stress physiology. Even the changes observed are small, yet they play a critical role in existence of plant in stress. It should remain in mind that the overall effects of air pollutants on physiological parameters are not exclusive of each other. All of them are inter dependent and the remedial actions should include the holistic measures to balance the ecosystem stability and socio-economic upgradation.

References

Abeyratne VDK, Ileperuma OA (2006) Impact of ambient air pollutants on the stomatal aperture of Argyreiapopulifolia. Ceylon J Sci 35(1):9–15

Aggarwal P (2000) The effect of auto-exhaust pollution on leaf surface of *Cassia siamea* (L.), a road side tree. Acta Ecol 22:101–106

Agrawal M, Verma M (1997) Amelioration of sulphur dioxide phytotoxicity in wheat cultivars by modifying NPK nutrients. J Environ Manag 49(2):231–244

Aminifar J, Ramroudi M (2014) Ecophysiological aspects of envir-onmental pollutions on growth of plants. Appl Sci Rep 8:99–102

Avron M (1960) Photophosphorylation by swiss-chard chloroplasts. Biochim Biophys Acta 40:257–272

Baek SG, Woo SY (2010) Physiological and biochemical responses of two tree species in urban areas to different air pollution levels. Photosynthetica 48:23–29

Bagard M, Le Thiec D, Delacote E, Hasenfratz-Sauder MP, Banvoy J, Gérard J, Dizengremel P, Jolivet Y (2008) Ozone-induced changes in photosynthesis and photorespiration of hybrid poplar in relation to the developmental stage of the leaves. Physiol Plant 134:559–574

Bagarda M, Le Thiecb D, Delacotea E, Saudera MPH, Banvoya J, Gérard J, Dizengremela P, Jolivet Y (2008) Ozone-induced changes in photosynthesis and photorespiration of hybrid poplar in relation to the developmental stage of the leaves. Physiol Plant 134:559–574

Baldoni E, Genga A, Cominelli E (2015) Plant MYB transcription factors: their role in drought response mechanisms. Int J Mol Sci 16:15811–15851

Barnes RL (1972) Effects of chronic exposure to ozone on photosynthesis and respiration of pines. Environ Pollut 3:133–138

Bengtson C, Grennfelt P, Bostrom C-A, Troeng E, Skarby L, Sjodin A, Peterson K (1982) Deposition and uptake of nitrogen oxides in Scots Pine needles (Pinus sylvestris L.). Report lVL-B 647. Institute for Water and Air Pollution Research, Gothenburg, Sweden

Bergmann E, Bender J, Weigel HJ (1999) Ozone threshold doses and exposure-response relationships for the development of ozone injury symptoms in wild plant species. New Phytol 144 (3):423–435

Bermejo-Orduna R, McBride JR, Shiraishi K, Elustondo D, Lasheras E, Santamaría JM (2014) Biomonitoring of traffic-related nitrogen pollution using Lethariavulpina (L.) Hue in the Sierra Nevada, California. Sci. Total Environ 490:205–212

Betzelberger AM, Yendrek CR, Sun J, Leisner CP, Nelson RL, Ort DR, Ainsworth EA (2012) Ozone exposure response for U.S. soybean cultivars: linear reductions in photosynthetic potential, biomass, and yield. Plant Physiol 160:1827–1839

Biggs AR, Davis DD (1980) Stomatal response of three birch species exposed to varying doses of SO_2. J Am Soc Hortic Sci 100:514–516

Black VJ (1984) The effect of air pollutants on apparent respiration. In: Koziol MJ, Whatley FR (eds) Gaseous air pollutants and plant metabolism. Butterworths, London, pp 231–248

Black CR, Black VJ (1979) The effects of low concentrations ofsulphur dioxide on stomata1 conductance and epidermal cell survi-val in field bean (Viciafiba L.). J Exp Bot 30:291–298

Black VJ, Unsworth MH (1979) Resistance analysis of sulfur dioxide fluxes to Viciafaba. Nature 282:68–69

Breuninger C, Meixner FX, Kesselmeier J (2013) Field investigations of nitrogen dioxide (NO2) exchange between plants and the atmosphere. Atmos Chem Phys 13:773–790

Burkhard J, Eiden R (1994) Thin water films on coniferous needles. Atmos Environ 28:2001–2017

Bytnerowicz A, Omasa K, Paoletti E (2007) Integrated effects of air pollution and climate change on forests: a northern hemisphere perspective. Environ Pollut 147:438–445

Capron TM, Mansfield TA (1976) Inhibition of net photosynthesis in tomato in air polluted with NO and NO_2. J Exp Bot 27:1181–1186

Carlson RW (1983) Interaction between SO_2 and NO_2 and their effects on photosynthetic properties of soybean *Glycine max*. Environ Pollut Ecol Biol 32:11–38

Chaparro-Suarez IG, Thielmann A, Meixner FX, Kesselmeier J (2006) Re-investigation of the nitrogen dioxide (NO_2) uptake by tree species. Geophys Res Abstr 8:706–716

Chaparro-Suarez IG, Meixner FX, Kesselmeier J (2011) Nitrogen dioxide (NO_2) uptake by vegetation controlled by atmospheric concentrations and plant stomatal aperture. Atmos Environ 45:5742–5750

Choi D, Toda H, Kim Y (2014a) Effect of sulfur dioxide (SO_2) on growth and physiological activity in Alnussieboldiana at Miyakejima Island in Japan. Ecol Res 29:103–110

Choi D, Toda H, Kim Y (2014b) Effect of sulfur dioxide (SO_2) on growth and physiological activity in Alnussieboldiana at Miyakejima Island in Japan. Ecol Res 29:103–110

Chung YC, Chung PL, Liao SW (2011) Carbon fixation efficiency of plants influenced by sulfur dioxide. Environ Monit Assess 173:701–707

Coulson CL, Robert L (1974) Heath, Inhibition of the photosynthetic capacity of isolated chloroplasts by ozone. Plant Physiol 53:32–38

Crittenden PD, Read DJ (1978) The effects of air pollution on plant growth with special reference to sulphur dioxide. New Phytol 80(1):49–62

Darrall NM (1989) The effect of air pollutants on physiological processes in plants. Plant Cell Environ 12:1–30

De Kok LJ, Westerman S, Stuiver CEE, Weidner W, Stulen I, Grill D (2002) Interaction between atmospheric hydrogen sulphide deposition and pedosphericsulphate nutrition in *Brassica oleracea* L. Phyton 42:35–44

DeKok LJ (1990) Sulphur metabolism in plants exposed to atmospheric sulphur. In: Rennenberg H, Brunold CH, de vacuoles Kok LJ, Stulen I (eds) Sulphur nutrition and sulphur assimilation in higher plants agricultural aspects. SPB Academic, The Hague, pp 111–130

Dhir B (2016) Air pollutants and photosynthetic efficiency of plants. In: Kulshrestha U, Saxena P (eds) Plant responses to air pollution. Springer, Singapore

Dineva S (2006) Development of leaf blades of *Acer platanoides* in industrially contaminated environment. Dendrobiology 55:25–32

Dizengremel P (2001) Effects of ozone on the carbon metabolism of forest trees. Plant Physiol Biochem 39:729–742

Eller ASD, McGuire KL, Sparks JP (2011) Responses of sugar maple and hemlock seedlings to elevated carbon dioxide under altered above- and belowground nitrogen sources. Tree Physiol 31:391–401

Erisman JW, Bleeker A, Galloway J, Sutton MS (2007) Reduced nitrogen in ecology and the environment. Environ Pollut 150:140–149

Farage PK, Long SP (1999) The effects of O3 fumigation during leaf development on photosynthesis of wheat and pea: an in vivo analysis. Photosynth Res 59:1–7

Farage PK, Long SP, Lechner EG, Baker NR (1991) The sequence of change within the photosynthetic apparatus of wheat following short-term exposure to ozone. Plant Physiol 95:529–535

Fatima N, Akram M, Shahid M, Abbas G, Hussain M, Nafees M, Wasaya A, Tahir M, Amjad M (2018) Germination, growth and ions uptake of moringa (*Moringa oleifera* L.) grown under saline condition. J Plant Nutr 41(12):1555–1565

Freer-Smith PH (1985) The influence of SO_2 and NO_2 on the growth, development and gas exchange of *Betula pendula* Roth. New Phytol 99:417–430

Gebler A, Rienks M, Rennenberg H (2000) NH_3 and NO_2 fluxes between beech trees and the atmosphere—correlation with climatic and physiological parameters. New Phytol 147:539–560

Gheorghe IF, Ion B (2011) The Effects of air pollutants on vegetation and the role of vegetation in reducing atmospheric pollution. In: The impact of air pollution on health, economy, environment and agricultural sources. Intech Publisher, Shanghai, pp 256–259

Grennfelt P, Bengtson C, Skarby L (1983) Dry deposition of nitrogen dioxide to Scots pine needles. In: Pruppacher HR, Semonin RG, WGN S (eds) Precipitation scavenging, dry deposition, and resuspension. Volume 2. Dry deposition and resuspension proceedings of the fourth international conference, November–December 1982, Santa Monica, CA. Elsevier, New York, NY, pp 753–762

Guderian R (2012) Air pollution: phytotoxicity of acidic gases and its significance in air pollution control, vol 92. Springer, Berlin

Hetherington AM, Woodward FI (2003) The role of stomata in sensing and driving environmental change. Nature 424:901–908

Hill AC, Bennett JH (1970) Inhibition of apparent photosynthesis by nitrogen oxides. Atmos Environ 4:341–348

Horii CV, Munger JW, Wofsy SC, Zahniser M, Nelson D, McManus JB (2004) Fluxes of nitrogen oxides over a temperate deciduous forest. J Geophys Res 109:D08305

Horsman DC, Wellburn AR (1975) Synergistic effect of SO_2 and NO_2 polluted air upon enzyme activity in pea seedlings. Environ Pollut 8:122–133

Hu KD, Tang J, Zhao DL, Hu LY, Li YH, Liu YS, Jones R, Zhang H (2014) Stomatal closure in sweet potato leaves induced by sulfur dioxide involves H_2S and NO signaling pathways. Biol Plant 58:676–680

Hu YB, Bellaloui N, Tigabu M, Wang JH, Diao J, Wang K, Yang R, Sun GY (2015) Gaseous NO_2 effects on stomatal behavior, photosynthesis and respiration of hybrid poplar leaves. Acta Physiol Plant 37:39

Hultengren S, Gralen H, Pleijel H (2004) Recovery of the epiphytic lichen flora following air quality improvement in south-west Sweden. Water Air Soil Pollut 154:203–211

Jacobson MZ, Seinfeld JH, Carmichael GR, Streets DG (2004) The effect on photochemical smog of converting the U.S. fleet of gasoline vehicles to modern diesel vehicles. Geophys Res Lett 31: L02116. https://doi.org/10.1029/2003GL018448

Jaradat MR, Feurtado JA, Huang D, Lu Y, Cutler AJ (2013) Multiple roles of the transcription factor AtMYBR1/AtMYB44 in ABA signaling, stress responses, and leaf senescence. BMC Plant Biol 13:192

Julia P, Kangasjärvi VJ (2015) Plant signalling in acute ozone exposure. Plant Cell Environ 38:240–252

Jung C, Seo JS, Han SW, Koo YJ, Kim CH, Song SI, Nahm BH, Choi YD, Cheong JJ (2008) Overexpression of AtMYB44 enhances stomatal closure to confer abiotic stress tolerance in transgenic Arabidopsis. Plant Physiol 146:623–635

Kaur S (2004) Stomatal responses of lemon (*Citrus medica*) to exhaust emissions from vehicles using different types of fuel. Pollut Res 23:451–454

Khan MR, Khan MW (1993) The interaction of SO_2 and root-knot nematode on tomato. Environ Pollut 81:91–102

Knabe W (1976) Effects of sulfur dioxide on terrestrial vegetation. J Hum Environ 5:213–218

Knudson LL, Tibbitts TW, Edwards GE (1977) Measurement of ozone injury by determination of leaf chlorophyll concentration. Plant Physiol 60:606–608

Koziol MJ, Whatley FR (2016) Gaseous air pollutants and plant metabolism. Butterworth-Heinemann, Oxford

Kulshrestha U, Saxena P (eds) (2016) Plant responses to air pollution. Springer

Kumari S, Prakash I (2015) Changes in micromorphology of plant Sidaveronicaefolia in response to air pollution stress in Meerut city. Proceeding of the UGC Sponsored National Seminar on "The Role of Biology in Bringing Second Green Revolution", India

Lane PI, Bell JNB (1984a) The effects of simulated urban air pollution on grass yield: Part 1 – Description and simulation of ambient pollution. Environ Pollut Ser B 8:245–263

Lane PI, Bell JNB (1984b) The effects of simulated urban air pollution on grass yield: Part 2 – Performance of Loliumperenne, Phleumpratense and Dactylisglomerate fumigated with SO_2, NO_2 and/or NO. Environ Pollut Ser A 35:97–12

Lee TT (1967) Inhibition of oxidative phosphorylation and respiration by ozone in tobacco mitochondria. Plant Physiol 42:691–696

Leffler HR, Cherry JH (1974) Destruction of enzymatic activities of corn and soybean leaves exposed to ozone. Can J Bot 52(6):1233–1238

Lichtenthaler HK, Buschmann C, Knapp M (2005) How to correctly determine the different chlorophyll fluorescence parameters and the chlorophyll decrease ratio RFd of leaves with the PAM fluorometer. Photosynthetica 43:379–393

Liu Y, Li Y, Li L, Zhu Y, Liu J, Li G, Lin H (2017) Attenuation of sulfur dioxide damage to wheat seedlings by coexposure to nitric oxide. Bull Environ Contam Toxicol 99:146–151

Majernik O, Mansfield TA (1971) Direct effect of SO_2 pollution on the degree of opening of stomata. Nature 227:377

Mansfield TA, Freer-Smith PH (1981) Effects of urban air pollution on plant growth. Biol Rev 56:343–368

Marais EA, Jacob DJ, Choi S, Joiner J, Belmonte-Rivas M, Cohen RC, Ryerson TB, Weinheimer AJ, Volz-Thomas A (2017) Nitrogen oxides in the global upper troposphere interpreted with cloud-sliced NO_2 from the Ozone Monitoring Instrument. EGU Gen Assem 19:121–156

Martn A, Barber FR (1984) Acid gases and acid in rain monitored for over 5 years in rural east-central England. Atmos Environ 18:1715–1724

Mazarura U (2012) Effect of sequences of ozone and nitrogen dioxide on plant dry matter and stomatal diffusive resistance in radish. Afr Crop Sci J 20:371–384

Melotto M, Underwood W, He SY (2008) Role of stomata in plant innate immunity and foliar bacterial diseases. Annu Rev Phytopathol 46:101–122

Middleton JT, Darley EF, Brewer RF (1958) Damage to vegetation from polluted atmospheres. J Air Pollut Control Assoc 1958(8):9–15

Miller A, Tsai CH, Hemphill D, Endres M, Rodermel S, Spalding M (1997) Elevated CO_2 effects during leaf ontogeny. Plant Physiol 115:1195–1200

Moldau H, Sober J, Sober A (1993) Impact of acute ozone exposure on CO2 uptake by two cultivars of *Phaseolus vulgaris* L. Photosynthetica 28:133–141

Mouillon JM, Aubert S, Bourguignon J, Gout E, Douce R, Rebeille F (1999) Glycine and serine catabolism in nonphotosynthetic higher plant cells: their roll in C1 metabolism. Plant J 20 (2):197–205

Mudd JB (1975) Sulfur dioxide. In: Responses of plants to air pollution. Academic Press, New York

Mukherjee A, Agrawal M (2018) Use of GLM approach to assess the responses of tropical trees to urban air pollution in relation to leaf functional traits and tree characteristics. Ecotoxicol Environ Saf 152:42–54

Munzi S, Pisani T, Loppi S (2009) The integrity of lichen cell membrane as a suitable parameter for monitoring biological effects of acute nitrogen pollution. Ecotoxicol Environ Saf 72:2009–2012

Nash TH (1976) Sensivity of lichenes of NO_2 fumigation. Bryologist 79:103–106

Okano K, Totsuka T (1986) Absorption of nitrogen dioxide by sunflower plants grown at various levels of nitrate. New Phytol 102:551–562

Olszyk DM, Tibbitts TW (1981) Stomatal response and leaf injury of Pisumsativum L. with SO_2 and O_3 exposures I. Influence of pollutant level and leaf maturity. Plant Physiol 67:539–544

Omasa K, Saji H, Youssefian S, Kondo N (2002) Air pollution and plant biotechnology—prospects for phytomonitoring and phytoremediation. In: De Kok LJ, Stuiver CEE, Westerman S, Stulen I (eds) Elevated levels of hydrogen sulphide in the plant environment: nutrient or toxin. Springer, Tokyo, pp 201–219

Pal A, Kulshreshtha K, Ahmad KJ, Yunus M (2000) Changes in leaf surface structures of two avenue tree species caused by auto exhaust pollution. J Environ Biol 21:15–21

Paoletti E, Grulke NE (2005) Does living in elevated CO2 ameliorate tree response to ozone? A review on stomatal responses. Environ Pollut 137(3):483–493

Park JS, Shin JW, Ahn TT, Son JE (2010) Analysis of CO_2 and harmful gases caused by using burn-type CO_2 generators in greenhouses. J Bio-Environ Control 19:177–183

Pell EJ, Brennan E (1973) Changes in respiration, photosynthesis, adenosine 5′-triphosphate, and total adenylate content of ozonated Pinto Bean foliage as they relate to symptom expression. Plant Physiol 51:378–381

Pell EJ, Eckardt N, Enyedi AJ (1992) Timing of ozone stress and resulting status of ribulose bisphosphatase carboxylase/oxygenase and associated net photosynthesis. New Phytol 120:397–405

Pell EJ, Eckardt NA, Glick RE (1994) Biochemical and molecular basis for impairment of photosynthetic potential. Photosynth Res 39:453–462

Rahmat M, Maulina W, Rustami E, Azis M, Budiarti DR, Seminar KB, Yuwono AS, Alatas H (2013) Performance in real condition of photonic crystal sensor based NO_2 gas monitoring system. Atmos Environ 79:480–485

Rai A, Kulshreshtha K (2006) Effect of particulates generated from automobile emission on some common plants. J Food Agric Environ 4:253–259

Ranieri A, Castagna A, Gian BB, Soldatini F (2001) Iron deficiency differently affects peroxidase isoforms in sunflower. J Exp Bot 52(354):25–35

Rao IM, Amundson RG, Alscher-Herman R, Anderson LE (1983) Effects of SO_2 on stomatal metabolism in Pisumsativum L. Plant Physiol 72:573–577

Raschk K (1975) Stomatal action. Annu Rev Plant Physiol 26:309–340

Reich PB, Amundson RG (1985) Ambient levels of ozone reduce net photosynthesis in tree and crop species. Science 230(4725):566–570

Reinert RA, Shriner DS, Rawlings JO (1982) Responses of radish to all combinations of three concentrations of nitrogen dioxide, sulfur dioxide, and ozone. J Environ Qual 11:52–57

Rey A, Jarvis PG (1998) Long-term photosynthetic acclimation to increased atmospheric CO_2 concentration in young birch (*Betula pendula*) trees. Tree Physiol 18:441–450

Robinson MF, Heath J, Mansfield TA (1998) Disturbances in stomatal behaviour caused by air pollutants. J Exp Bot 49:461–469

Rogers HH, Jeffries HE, Witherspoon AM (1979) Measuring air pollutions uptake by plants: nitrogen dioxide. J Environ Qual 18:551e557

Sabaratnam S, Gupta G (1988) Effects of nitrogen dioxide on biochemical and physiological characteristics of soybean. Environ Pollut 55:149–158

Saxe H, Murali NS (1989) Diagnostic parameters for selecting against novel spruce (*Picea abies*) decline: 11. Response of photosynthesis and transpiration to acute NO_2 exposures. Physiol Plant 76:349–355

Saxena P, Kulshrestha U (2016a) Biochemical effects of air pollutants on plants. In: Plant responses to air pollution. Springer, Singapore, pp 59–70

Saxena P, Kulshrestha UC (2016b) The impact of gasoline emission on plants–a review. Chem Ecol 32(4):378–405

Saxena P, Naik V (eds) (2018) Air pollution: sources, impacts and controls. CABI

Saxena P, Sonwani S (2019a) Primary criteria air pollutants: environmental health effects. In: Criteria air pollutants and their impact on environmental health. Springer, Singapore, pp 49–82

Saxena P, Sonwani S (2019b) Criteria air pollutants: chemistry, sources and sinks. In: Criteria air pollutants and their impact on environmental health. Springer, Singapore, pp 7–48

Saxena P, Sonwani S (2019c) Secondary criteria air pollutants: environmental health effects. In: Criteria air pollutants and their impact on environmental health. Springer, Singapore, pp 83–126

Saxena P, Sonwani S (2020) Criteria air pollutants and their impact on environmental health. Springer, Singapore

Saxena P, Sharma Y, Chugh M (2017, December). Assessment of ozone phytotoxic potential of dense vegetation cover of Delhi by AOT40. In AGU fall meeting abstracts

Saxena P, Srivastava A, Tyagi M, Kaur S (2019) Impact of tropospheric ozone on plant metabolism – a review. Pollut Res 38(1):175–180

Schmutz P, Tarjan D, Günthardt-Goerg MS, Matyssek R, Bucher JB (1995) Nitrogen dioxide – a gaseous fertilizer of Poplar trees. Phyton 35:219–232

Schreiber U, Vidaver W, Runeckles VC, Rosen P (1978) Chlorophyll fluorescence assay for ozone injury in intact plants. Plant Physiol 61:80–84

Seyyednejad SM, Koochak H (2011a) Some morphological and biochemical responses due to industrial air pollution in Prosopisjuliflora (Swartz) DC plant. Afr J Agric Res 8(18):1968–1974

Seyyednejad SM, Koochak H (2011b) Some morphological and biochemical responses due to industrial air pollution in *Prosopis juliflora* (Swartz) DC plant. Afr J Agric Res 8 (18):1968–1974

Sobrado MA (2011) Leaf pigment composition and fluorescence signatures of top canopy leaves in species of the upper Rio Negro forests. Res J Bot 6:141–149

Sonwani S, Kulshreshtha U (2016) Particulate matter levels and it's associated health risks in East Delhi. Proceedings of Indian aerosol science and technology association conference on aerosol and climate change: insight and challenges. IASTA Bull 22(1–2). ISSN 09714510

Sonwani S, Maurya V (2018) Impact of air pollution on the environment and economy. In: Air pollution: sources, impacts and controls. CABI Publisher, Oxford. ISBN 9781786393890

Sonwani S, Saxena P (2016) Identifying the sources of primary air pollutants and their impact on environmental health: a review. IJETR 6(2):111–130

Sonwani S, Saxena P, Kulshrestha U (2016) Role of global warming and plant signaling in BVOC emissions. In: Plant responses to air pollution. Springer, Singapore, pp 45–57

Srivastava HS, Jolliffe PA, Runeckles VC (1975) The influence of nitrogen supply during growth on the inhibition of gas exchange and visible damage to leaves by NO_2. Environ Pollut 9:35–47

Stulen I, Perez-Soba M, De Kok LJ, Van der Eerden L (1998) Impact of gaseous nitrogen deposition on plant functioning. New Phytol 139:61–70

Takagi M, Gyokusen K (2004) Light and atmospheric pollution affect photosynthesis of street trees in urban environments. Urban For Urban Green 2:167–171

Taylor GE, Johnson DW, Andersen CP (1994) Air pollution and forest ecosystems: a regional to global perspective. J Ecol Appl 4:662–689

Teklemariam TA, Sparks JP (2006) Leaf fluxes of NO and NO_2 in four herbaceous plant species: The role of ascorbic acid. Atmos Environ 40:2235–2244

Thoene B, Rennenberg H, Weber P (1996) Absorption of atmospheric NO2 by spruce (Piceaabies) trees: II. Parameterization of NO_2 fluxes by controlled dynamic chamber experiments. New Phytol 134:257–266

Todd GW (1958) Effect of ozone and ozonated I -hexene on respiration and photosynthesis of leaves. Plant Physiol 33:416–420

Unsworth MH, Ormrod DP (eds) (1982) Air pollution in agriculture and horticulture. Book chapter – Godzik S, Krupa SV, Effects of sulfur dioxide on growth and productivity of crop plants (pp 247–265). Butterworths, London

Vahisalu T, Puzo I, Brosche M, Valk E, Lepiku M, Moldau H, Pechter P, Wang YS, Lindgren O, Salojarvi J, Loog M, Kangasjarvi J, Kollist H (2010) Ozone-triggered rapid stomatal response involves the production of reactive oxygen species, and is controlled by SLAC1 and OST1. Plant J 62:442–453

Verma A, Singh SN (2006) Biochemical and ultrastructural changes in plant foliage exposed to auto-pollution. Environ Monit Assess 120:585–602

Wellburn AR (1990) Why are atmospheric oxides of nitrogen usually phytotoxic and not alternative fertilizers? New Phytol 115:395–429

Weller R, Jones AE, Wille A, Jacobi HW, McIntyre HP, Sturges WT, Huke M, Wagenbach D (2002) Seasonality of reactive nitrogen oxides (NOy) at Neumayer Station, Antarctica, 107 (D23), ACH 2-1–ACH 2-11

Whitmore ME, Mansfield TA (1983) Effects of long-term exposure to SO_2 and NO_2 on Poapratensis and other grasses. Environ Pollut Ser A 31:217–235

Wilkinson S, Mills G, Illidge R, Davies WJ (2012) How is ozone pollution reducing our food supply? J Exp Bot 63:527–536

Xu J, Yin H, Liu X (2010) Nitric oxide is associated with long term zinc tolerance in Solnumnigrum. Plant Physiol 154(3):1319–1334

Chapter 7
Air Pollution Exposure Studies Related to Human Health

Neha Singh

Abstract Air is a complex amalgamation of gases, water vapor, and dust particles. With development in civilized human society and later on boom in industrialization, there is drastic change in the air quality levels. Levels of different gaseous pollutants, heavy metals, particulate matter, volatile organic compounds, and other pollutants are constantly rising up in the air majorly due to combustion of solid fuels and fossil fuels. All these pollutants greatly diverge in their chemical composition, reaction time and mechanism of action. Depending on the exposure time and concentration, these pollutants can exert various toxicological impacts on human health such as respiratory, neurological, and cardiovascular disorder and other life-threatening diseases like cancer. Air pollution has also been connected with premature child birth, morbidity, mortality and reduced life expectancy. In this chapter, the sources of air pollution their effects on different organs and systems in human body as well as their mechanism of action will be discussed.

Keywords Air pollution · Pollutants · Particulate matter · Heavy metals · Human health · Cardiovascular · Pulmonary · Digestive system · Nervous system · Skin · Cancer

7.1 Introduction

One of the major conundrums with which mankind is struggling today is air pollution. Air pollution is the heterogeneous concoction of the gases, water vapor, and particulate matter (PM) in the air. Nowadays, air quality is significantly getting deteriorated mainly because of rapid industrialization and urbanization that can impact the human health and environment. The impact of air quality on human health is at an alarming stage where nine out of ten people breathe polluted air which

---

N. Singh (✉)
Eötvös Loránd University, Budapest, Hungary
e-mail: neha.singh@ttk.hu

P. Saxena, A. Srivastava (eds.), *Air Pollution and Environmental Health*,
Environmental Chemistry for a Sustainable World 20,
https://doi.org/10.1007/978-981-15-3481-2_7

sums up to 7 million deaths (one out of eight deaths globally) in 2012 solely due to polluted air exposure (WHO 2014). Substances which are responsible for causing deleterious impacts on the environment are categorized as pollutants. Pollutants are mainly divided into two categories as primary pollutants, which mainly consists of particles or particulate matter (PM), sulfur oxides (SO_X), nitrogen oxides (NO_X), volatile organic compounds (VOCs), and oxides of carbon and secondary pollutants such as ground-level ozone (O_3) which are derived from primary air pollutants (Saxena and Sonwani 2019a, b; Türk and Kavraz 2011).

Every living being is exposed to the air pollution every day, some to a more and some to a less extent depending on their lifestyle and socioeconomic status (Rückerl et al. 2011). The effect of air quality impacts the human health directly as on an average a person inhales around 500 million liters of air (12,000–16,000 liters of air daily) (Saxena and Sonwani 2019a, b; Türk and Kavraz 2011). The first study about the effects of air pollution was performed in the Meuse Valley, Belgium, where more than 60 people died because of smog in 1930 (Firket 1936). Mechanistic, epidemiological, clinical, and experimental studies have provided numerous evidences supporting the adverse and detrimental effects of long-term and short-term exposure to air pollution on human health. The aim of this chapter concentrates on identifying different sources of air pollution, types of pollutants, and mainly their impact on overall human health and functioning of different organs and systems in human body independently and in combined manner.

7.2 Types of Air Pollution Impacting Human Health

Indoor Air Pollution (IAP)

Indoor air quality (IAQ) is a parameter that refers to the quality of air inside and the surrounding residential area, shopping complexes, schools and colleges, gym etc.

IAP is ubiquitous and of major concern globally. Both urban and rural societies are directly affected by the indoor air pollution but the source and the type of pollutant to which each society is exposed varies differently. The risk of getting affected by air pollution is more profound in underdeveloped or developing countries especially because of the smoke emitted from the combustion of solid fuels such as coal and biomass (wood, crop residue, animal dung, etc.) (Sonwani and Kulshreshtha 2016). Combustion of coal and other solid fuels can produce variant pollutants such as CO, NO_x, SO_x, PM, benzene, and polycyclic aromatic hydrocarbons (PAH) as well as various toxic element such as mercury, selenium, arsenic, lead, and fluorine (Sonwani et al. 2016; Kurt et al. 2016). Whereas, developed societies are mostly exposed to the semi-volatile and volatile organic compounds. Another source of IAP is Tobacco Smoking (TS). It has been known for centuries that TS is injurious for the health of the smoker but recently the health concern has been raised for those who do not smoke directly and instead inhale the smoke from the primary smoker, hence are known as passive or involuntary smokers, especially the children and infants. Tobacco smoke is usually similar to other types of smokes,

Table 7.1 Comparison of number of deaths caused by outdoor and indoor pollution (shown in percentage) WHO 2014

Diseases	Outdoor air pollution (in percentage)	Indoor air pollution (in percentage)
Ischemic heart disease	40	26
Stroke	40	34
Chronic obstructive pulmonary disease (COPD)	11	22
Lung cancer	6	6
Acute lower respiratory infection (in children)	3	12

a heterogeneous mixture of gases, tiny particles, and chemical carcinogens (Saxena and Ghosh 2012).

Recent literatures have established direct and strong correlation between the exposure of smoke from solid fuels and occurrence of various acute and chronic infections and diseases such as acute respiratory tract infections (ARI), chronic obstructive pulmonary disease (COPD), and lung cancer (Zhang and Smith 2003; Torres-Duque et al. 2008; Smith-Sivertsen et al. 2009; Perez-Padilla et al. 2010; Sood 2012; Chen and Modrek 2018).

Outdoor Air Pollution (OAP)

The sources of OAP are varied unlike the IAP, both natural and anthropogenic activities play crucial role in determining the quality of the air. Nevertheless, the pollutants present in outdoor air are mostly similar to indoor air but their concentrations are different and usually higher. Natural sources of air pollution include volcanic eruption, lightning, forest and brush fires, pollen grains, etc. Though natural sources has always been around us but in most of the cases it has never been any threat to mankind. Perhaps, pollution caused by anthropogenic activities is of major concern in today’s time. Unstoppable urbanization and modernization of societies are highly responsible for worsening the air quality. Burning of fossil fuels and garbage, smoke emission from transport, power generation and industries are some of the major sources of outdoor air pollution.

Another study has raised an imperative concern about the transport of PM, ozone, and the toxic compounds over large distances, such as movement of dust particles from Mongolia to Western United States or the deposition of the dust from Saharan region to the Caribbean countries (Kurt et al. 2016).

Both indoor and outdoor air pollution are of equal concern and threat for human health as both affect the different sections of the society. Table 7.1 outlines the breakdown of the number of deaths because of various cardiopulmonary diseases caused by outdoor and indoor air pollution independently.

7.3 Pollutants Are Categorized into Four Major Categories Depending on Their Physical and Chemical Nature

1. Gaseous pollutants (e.g., SO_x, NO_x, CO_x, O_3, volatile organic compounds, such as Benzo(a)pyrene, BaP)
2. Persistent organic pollutants (POPs)
3. Heavy metals (e.g., lead, mercury, arsenic, chromium, etc.)
4. Respirable particulate matter ($PM_{2.5}$ and PM_{10})

7.3.1 Gaseous Pollutants (e.g., SO_2, NO_x, CO_x, O_3, Volatile Organic Compounds (VOCs), Such as Benzo(a)pyrene (BaP)

Gaseous pollutants are very common and most ubiquitous source of air pollution and are mainly produced by burning of fossil fuels in industries, vehicles, and homes resulting in the emission of many noxious gases such as carbon monoxide (CO), sulfur oxides (SO_x), nitrogen oxides (NO_x), volatile organic compounds (VOCs), and soot particles (Sonwani and Saxena 2016).

Out of all the pollutants, CO is considered as one of the major toxic pollutants present in the environment. Gases like SO_x and NO_x are toxic when inhaled in high concentrations, besides that they are involved in secondary particle formation when they react with ammonia (used mainly in agriculture) (Wang et al. 2015). Ground-level ozone is a secondary pollutant and is formed because of the photochemical reaction involving the interaction of nitrogen oxides and organic compounds in the presence of sunlight (Schultz et al. 2017).

VOCs are mostly released from the combustion process during the energy production and road transport. VOCs are mostly organic in nature such as benzene; when inhaled in large quantity they can cause hematological problems and cancer along with serious respiratory system damage (Kampa and Castanas 2008; Sonwani et al. 2016; Saxena and Ghosh 2015, 2019; Saxena 2015).

7.3.2 Persistent Organic Pollutants (POPs) (e.g., Dioxins)

POPs are composed of organic chemicals (polyhalogenated hydrocarbons that contain chlorine, bromine, or fluorine) that remain in the environment for a long period of time. POPs are lipophilic in nature and have certain other characteristics on the basis of which they are identified, such as their toxicity, bioaccumulation potential, persistence capabilities, and their ability for long distance transport (Beyer et al. 2000). They enter into the environment through pesticides and certain industrial chemicals and are ubiquitous; because of their long persistence potential they tend to bioaccumulate and biomagnify, eventually entering into the food chain (WHO 2010). Their distribution is not regional or confined to a place; they are globally distributed through air, water, ocean currents, and polluted living organisms. Human can be exposed to POPs in a variety of ways including inhalation, oral ingestion, or direct contact with skin (Kim et al. 2013; Wang et al. 2012). Long-term exposure to POPs can cause serious concern to human health including cancer, neurodegenerative disease, birth defects, and sterility and immune defects. Children are more vulnerable to be affected by exposure to different pollutants compared to the adults (WHO 2010; Ruzzin 2012).

7.3.3 Heavy Metals

Heavy metals usually refer to the metals with relatively high atomic weight and density (more than 4 g/cm^3) which is usually 5 times greater than water and are generally toxic to human health even when present in low concentration, along with the density, chemical properties of metal are of equal concern. Some of the heavy metals commonly found in environment as contaminants are lead (Pb), copper (Cu), manganese (Mn), magnesium (Mg), cadmium (Cd), mercury (Hg), iron (Fe), zinc (Zn), chromium (Cr), cobalt (Co), arsenic (as), molybdenum (Mo), and selenium (Se) (Duruibe et al. 2007; Tchounwou et al. 2012). They are present in trace amounts in the environment and hence also known as trace elements (ppb range to less than 10 ppm) (Tchounwou et al. 2012).

Both natural and anthropogenic activities contribute to the high concentrations of heavy metals in the environment (Aksu 2015). With advancement in industries and the large-scale application of heavy metals in medical, technology, and defense lead to the wide distribution of heavy metals in the environment (Tchounwou et al. 2012). Exposure to heavy metals are known to affect cellular components (such as nucleic acid, proteins, and lipids) and its organelles such as nucleus, mitochondria, endoplasmic reticulum, lysosome, cell membrane, and enzymes involved in metabolism, detoxification, and damage repair (Squibb and Fowler 1981; Wang and Shi 2001). The toxicity caused by heavy metals such as arsenic, cadmium, chromium, and nickel can trigger DNA damage by base pair mutations and insertions–deletions or attack of reactive oxygen species on DNA (Landolph 1994). They can cause toxicity by inhalation, ingestion, and direct dermal contact (Tchounwou et al. 2012).

7.3.4 Respirable Particulate Matter ($PM_{2.5}$ and PM_{10})

Particulate matter (PM) is also known as particle pollution, and is one of the major forms of airborne pollutants found in both modern as well as underdeveloped countries. It is a complex amalgam of very tiny solid and liquid particles of varying chemistry and size present in the air around us (Brook 2007). PMs are very fine particles which are classified on the basis of their aerodynamic diameter: coarse (diameter $< 10\ \mu m$; PM_{10}), fine (diameter $< 2.5\ \mu m$; $PM_{2.5}$), and ultrafine (nanoparticles diameter $< 0.1\ \mu m$; $PM_{0.1}$) (Mannuccio et al. 2015; Sonwani and Kulshrestha 2019).

Coarse particles are usually of high pH and basic in nature whereas smaller particles are often acidic in nature (Dockery and Pope 1994). Major sources for fine particles are vehicles, power plants, industrial and residential plants. It can have exacerbated effects on human health. These fine particles travel with air and get deposited into the respiratory tract. Different PM has different effect depending on its size and aerodynamic diameter (Lee et al. 2014; Sonwani and Kulshrestha 2018). PM_{10} particles usually originate from the dust from the road and agriculture, combustion of wood and solid fuels such as charcoal, demolition of buildings and mining activities (Block and Calderón-Garcidueñas 2009). $PM_{2.5}$ particles are very tiny and emerge from the gas or the condensation of the vapors during combustion of fossil fuels or industrial emission and are composed of both organic and inorganic compounds (Block and Calderón-Garcidueñas 2009). $PM_{2.5}$ is alone known to be responsible for nearly 3.3 million deaths per year globally and long-term exposure with $PM_{2.5}$ by10 $\mu g/m^3$ can increase the rate of cardiovascular mortality up to 11% (Kurt et al. 2016; Saxena and Sonwani 2019a, b).

The complexity of pollutants present in both outdoor and indoor air is mostly similar in composition; it only varies in the concentration of different pollutants in the air because of the different sources. Hence, Table 7.2 summarizes the source of all the major pollutants in outdoor and indoor air.

7.4 Impact of Air Pollution on Human Health

Numerous studies have established a correlation between air quality and human health. Deterioration in the quality of air is usually associated with increase in the cardiopulmonary mortality cases and exacerbation in the morbidity and mortality. Air pollution can affect our health directly in many ways depending upon various factors such as exposure time, intensity of pollution, type of pollutants exposed to, and general health status of the population. Different pollutants can affect our bodies in many different ways by altering the normal functioning of different body parts. Heart attacks, respiratory disease, and lung cancer rates are significantly higher in the population who breathe polluted air compared to those who live in comparatively cleaner environment (Mabahwi et al. 2014; Sonwani and Kulshrestha 2016). Figure 7.1 summarizes the effect of air pollution on different body systems and organs.

Table 7.2 Different pollutant sources contaminating outdoor and indoor air

Pollutants	Indoor air pollution source	Outdoor air pollution source
Fine particles ($PM_{2.5}$, PM_5, PM_{10})	Fuel or tobacco combustion, cleaning, cooking	Industries, agriculture, construction and demolition activities
Carbon monoxide	Fuel or tobacco combustion	Vehicle exhaust, industries, power plants
Polycyclic aromatic hydrocarbons	Fuel or tobacco combustion, cooking, residential heating.	Vehicles exhaust, petroleum product spillage, sewage sludge and tarry or creosote waste materials
Nitrogen oxides	Fuel combustion	Vehicle exhaust, power plants
Sulfur oxides	Coal combustion	Industries and automobiles
Arsenic and fluorine	Coal combustion	Agricultural chemicals, mining, metal smelting, burning of fossil fuels
Volatile and semi-volatile organic compounds	Fuel or tobacco combustion, consumer products, furnishing, construction materials, cooking	Automobile emission, gasoline marketing and storage tanks, petroleum and chemical industries, dry cleaning and natural gas combustion
Aldehydes	Fuel or tobacco combustion, consumer products, furnishing, construction materials, cooking	Industries, chemical production, power plants
Pesticides	Consumer products, dust from outside	Agricultural chemicals
Lead	Remodeling and demolition of painted surfaces	Vehicle combustion, industries, power plants
Biological pollutant	Moist areas, ventilation system, furnishings	Pollen grains, spores from fungi
Radon	Soil under building, construction materials	Construction materials
Free radicals and other short-lived, highly reactive compounds	Indoor chemistry	Vehicle exhaust, combustion of fossil fuel, power plants, industries

7.5 Respiratory System

Air pollution has direct effect on respiratory system. Increase in the number of cases and exacerbation in the diseases like chronic obstructive pulmonary disease (COPD), asthma, respiratory infection, and lung cancer has been observed with increase in the levels of pollutant in the air (Sunyer et al. 1997; Chen et al. 2014; Jiang et al. 2016). PM present in air enters into the respiratory system during breathing and tends to get deposited (Ni et al. 2015). Particles with size greater than 5 μm get deposited in the upper airways or lower larger airways, instead of smaller particles, less than 5 μm in size enters into the smaller areas of the respiratory system such as trachea, bronchioles, and alveoli (Dockery and Pope 1994). Although human body has devised several mechanisms to eliminate these particles when found in small quantities. Particles deposited in the trachea and alveoli are usually

Fig. 7.1 Air pollution affecting different body systems and organs

expelled by coughing whereas particles deposited beyond the terminal bronchioles are taken care by phagocytic cells such as macrophages (Dockery and Pope 1994). Gaseous pollutants have direct and immediate impact on the normal functioning of respiratory system. SO_2 can cause irritation in eyes, nose, and throat upon short-term exposure, where in long-term exposure of SO_2 can cause diseases including asthma, emphysema, bronchitis, and lung cancer (Kulle et al. 1986; Liu et al. 2016; Chen et al. 2014). Exposure of the pollutants such as PM, CO, SO_x, NO_x, and O_3 during the prenatal and postnatal developmental period results in decreased lung function, increasing the risk of developing respiratory infection and diseases later in the life (Wang and Pinkerton 2007; Mortimer et al. 2008).

7.5.1 Asthma and Respiratory Disorder

Air pollution is known to increase the incidence of asthma and can also exacerbate preexisting asthma. It is the most common chronic disease found among children and adolescents (Asher and Pearce 2014). Two major characteristic features known as the symptoms of asthma are: inflammation and hyperresponsiveness; in the airway and lungs they are usually triggered by alteration in the levels of different pollutants such as O_3, NO_2, and $PM_{2.5}$ (Aris et al. 1993; Frampton et al. 2002; Guarnieri and Balmes 2014). Excessive exposure to ozone can cause hole in lung tissues which can cause decrement in lung function, asthma, and other respiratory disorders, whereas inhalation of excess SO_2 is mainly responsible for causing more prominent bronchoconstriction (Seltzer et al. 1986; Johns and Linn 2011; Shrivastava et al. 2017). Samoli et al. (2011) studied the acute effect of PM_{10}, SO_2, NO_2, and O_3 exposure on the emergency admission of children due to exacerbation of asthma among age group of 0–14 years, from the year 2001 to 2004 where 10 μg/m^3 increase in the level of PM_{10} and SO_2 was associated with 2.5% increase (95% CI: 0.1–5.0) and 6% increase (95% CI: 0.9–11.3), respectively. From the year 1992 to 1998 the reduction in levels of pollution have reduced the asthma rate significantly upto14% and levels of CO significantly affected the rate of hospitalization because of asthma among children of 1–18 years of age groups (Neidell 2004). Another study conducted in Copenhagen, Denmark, examined the acute effect of different air pollutants affecting the children of age group 0–18 year by monitoring the increase in the number of emergency admission in hospital due to asthma. They found a significant association between hospital admission of children and different pollutants, NO_x (OR—1.11; 95% CI 1.05 to 1.17), NO_2 (OR—1.10; 95% CI 1.04 to 1.16), PM_{10} (OR—1.07; 95% CI 1.03 to 1.12), and $PM_{2.5}$ (OR—1.09; 95% CI 1.04 to 1.13) (Iskandar et al. 2012). Another study conducted in four major cities of Europe, during the year 1986–1992 found that there is significant increase in the hospital admission of adults with increase in the ambient levels of NO_2 (relative risk (RR) per 50 μg/m^3 increase 1.03, 95% CI 1.003 to 1.055) (Sunyer et al. 1997). In a controlled study where eight asthmatic patients were expose to 0.4 ppm of NO_2 for 1 hr. have shown the early bronchoconstrictor response (Belanger et al. 2006).

Heavy metals are known to exacerbate the existing asthma and kindle the inflammatory response in lungs and airway passage. Strong association has been found between the exposure of heavy metals and prevalence and exacerbation of asthma (Novey et al. 1983). Exposure to high levels of chromium (IV) can result in breathing problems and asthma (Das et al. 2011). Higher concentration of lead in blood (>5 μg/dL) is positively correlated with asthma (Zeng et al. 2016; Wu et al. 2019) whereas higher concentration of chromium and manganese in blood is related with more coughing and wheezing (Zeng et al. 2016). Exposure to the heavy metal fumes of zinc and mercury can cause temporary respiratory problems, bronchitis and asthma (Kawane et al. 1988; Jaishankar et al. 2014).

7.5.2 Chronic Obstructive Pulmonary Disease (COPD)

COPD is responsible for 2% of total global diseases and is the third major cause of deaths among women in developing countries as they are exposed to the smoke emitted from the burning of the solid fuels while cooking and heating (Smith-Sivertsen et al. 2009). Smoking is considered as another major factor for causing COPD (Fortoul et al. 2011). PM and NO_x have shown strong association with the development and worsening of the existing COPD (Salvi and Barnes 2009). PM matter released after the combustion of fuel enters into the lungs via airways causing inflammation and impairment of the lung function by increasing the permeability of endothelial cells, activating macrophages, lymphocytes $CD8^+$ and neutrophils (Fortoul et al. 2011). PM can also carry microorganisms which get adhered on the surface of air passage cavity and lungs which may exacerbate the prevalence of COPD (Sethi and Murphy 2001; Sze et al. 2014; Ni et al. 2015).

10 $\mu g/m^3$ increase in the concentration of PM_{10}, O_3, NO_2, and SO_2 in ambient air also elevates the risk for prevalence and hospital admission because of COPD (Ghozikali et al. 2015; Khaniabadi et al. 2018). Næss et al. (2007) have shown that increase in NO_2 levels of more than 40 $\mu g/m^3$ increases the prevalence of COPD in individuals of two different age groups: 51–70 and 71–90 years.

Heavy metals are also known to worsen the COPD (Leem et al. 2015; Heo et al. 2017). Studies performed in animal models of COPD or emphysema have shown that to trigger the inflammatory response in lungs along with the progression of emphysema upon intratracheal injection or inhalation of cadmium, similar symptoms are observed in COPD patients (Thurlbeck and Foley 1963; Tuder et al. 2003; Kirschvink et al. 2005, 2009). High concentration of various trace metals such as cadmium, cobalt, iron, and mercury is found in high concentration of the serum of COPD patients compared to the control (Asker et al. 2018; Heo et al. 2017). Another large-scale study performed in the United States found positive correlation between cadmium and lead concentration in serum with the occurrence of obstructive lung disease (OLD) and reduced lung function (Rokadia and Agarwal 2013).

7.5.3 Pulmonary Carcinogenesis and Mutagenesis

Lung cancer is one of the major causes of death across the globe (Torre et al. 2015). Earlier, consumption of tobacco in any form, either chewing or smoking was considered as prime cause of cancer. It has been known that 90% of lung cancer deaths in male and 75–80% lung cancer deaths in female in the United States are solely attributed to tobacco smoking (Hecht 1999). Although high number of lung cancer cases are also found among nonsmokers as well (Beelen et al. 2008; Raaschou-Nielsen et al. 2010), this indicates that there are other factors apart from tobacco consumption which are responsible for lung cancer. These factors can be environmental tobacco smoke, occupational and environmental exposure to carcinogens or mutagens (such as radon, arsenic, lead, or mercury), exposure to smoke emission from industries and traffic (Nyberg et al. 2000). A number of studies have been performed independently and they established a strong correlation between air pollution and lung cancer mortality. On the basis of one of the largest studies performed in Europe, urban air pollution is responsible for contributing as high as 11% in lung cancer cases (Molina et al. 2008). Another study performed in six different states of the United States found positive association between the air pollution and the mortality rate because of lung cancer and cardiopulmonary disease (Dockery et al. 1993). Increase in the ambient concentration of $PM_{2.5}$ for more than 10 $\mu g/m^3$ increases the risk of lung cancer mortality up to 8% (Perez-Padilla et al. 2010; Pope et al. 2002).

PMs generated from heavy metals such as lead (Pb), mercury (Hg), nickel (Ni), cadmium (Cd), cobalt (Co), and chromium (Cr) are known to have carcinogenic effects on human and animal models. They are considered as driving forces for lung carcinomas (Salnikow and Zhitkovich 2008). These heavy metals interfere the vital functions of human body at the cellular and molecular level by deregulating the cell proliferation, differentiation, or apoptosis. Cadmium modulates the gene expression and signal transduction and alters the levels of the different proteins involved in the cellular defense mechanism such as DNA repair and cell death (Matés et al. 2010). Cadmium and cobalt both trigger the lung carcinomas in animal models upon inhalation (Beyersmann and Hartwig 2008). Nickel has also been recognized as a potent carcinogen. In several animal studies inhalation of particulate nickel caused tumor development in lungs (Dunnick et al. 1995). It causes epigenetic status of the DNA which in turn alters the gene expression pattern, disrupting the iron homeostasis in cells and the function by binding with the iron-dependent enzymes, generating Reactive Oxygen Species (ROS) and hypoxic-signaling pathways into the cells (Salnikow and Zhitkovich 2008). Cr is another heavy metal known to possess carcinogenic and mutagenic abilities. Upon entering into the cells the reduction of Cr takes place from Cr(IV) to Cr(III) which can cross-link between different macromolecules such as DNA–DNA or DNA–Protein (Fortoul et al. 2011). Feng et al. (2003) found that lung cancer from workers exposed to Cr(IV) at occupational site carries a very high percentage of Guanine (G) to Thymine (T) transversion mutation in the non-transcribed strand of p53 gene in lung tissues. p53 is a tumor-suppressor gene. Direct and long-term exposure to cadmium elevates the risk of developing lung cancer (Tchounwou et al. 2012).

Gaseous pollutants such as SO_x and NO_x have been associated with lung cancer. Inhalation of these pollutants such as ultrafine and fine particulate, SO_x can cause DNA damage which can ultimately lead to lung cancer (Cao et al. 2011). Compare to the 30 $\mu g/m^3$ (lowest exposure level) of NO_x exposure, increase in the incidence rate ratio of lung cancer were 1.30 (95% CI; 1.07-1.57) for 30-72 $\mu g/m^3$ (Intermediate exposure level) and 1.45 (95% CI; 1.12-1.88) for >72 $\mu g/m^3$ (highest exposure level) of NO_x. Corresponding to that, there is 37% (95% CI; 6-76%) elevation in the lung cancer incidence rate ratio per 100 $\mu g/m^3$ of NO_x exposure has been observed (Raaschou-Nielsen et al., 2010).

7.6 Cardiovascular System

Out of the total deaths caused by air pollution, around 60–80% is because of cardiovascular diseases (Lelieveld et al. 2015). Exposure to different pollutants can cause different types of damage and affect the cardiovascular health differently. Numerous studies have established a strong correlation between particle pollution and blood pressure and it is well known that increase in blood pressure increases the risk of cardiovascular diseases (Ibald-Mulli et al. 2001; An et al. 2018). Short-term exposure to air pollution can provoke acute effects such as fluctuation in blood pressure and long-term exposure can escalate the risk of cardiovascular disease and can also reduce the life span by a few years (An et al. 2018). Cardiovascular health is mainly affected by oxidative stress, systemic inflammation, and autonomic nervous system imbalance that affect arterial walls (Krishnan et al. 2012).

7.6.1 Blood Pressure and Hypertension

A number of studies assessing the effect of air pollution on cardiovascular morbidity and mortality had also monitored the fluctuation in the blood pressure. It has shown that there is increase in systolic blood pressure (SBP) and diastolic blood pressure (DBP) by 2.8/2.7 mm Hg in patients after rise in the levels of $PM_{2.5}$ per 10 μg/m^3 (Brook 2007). Exposing 23 non-smoking, healthy individuals to $PM_{2.5}$ and ozone (O_3) for 2 hrs. showed a significant rise of 6 mmHg in DBP (Urch et al. 2005). A study carried out with random population of more than 3000 sample size found that there is 0.74 mmHg (95% CI: 0.08–1.40) increase in SBP with an elevation in the level of SO_2 by 80 μg/m^3 (Ibald-Mulli et al. 2001). A study conducted by Guo et al. (2010) in Beijing, China indicates that 10 μg/m^3 increase in the concentration of SO_2 and NO_2 is associated with increase in the number of Emergency Hospital Visits (EHVs), hence concluding that increase in the gaseous pollutant is associated with hypertension-related EHVs.

The association between exposure to heavy metals such as mercury (Torres et al. 2000; Clarkson et al. 2003; Houston 2011), lead (Harlan 1988; Bogden et al. 1991; Vaziri et al. 1999), cadmium (Gallagher and Meliker 2010), arsenic (Abhyankar et al. 2012; Alissa and Ferns 2011) and increase in the blood pressure or hypertension has been well established. Study performed in Bangladesh has established the dose-dependent relationship between the high prevalence of hypertension among the locals living in the endemic areas and high arsenic levels in the water bodies compare to those living in non-endemic areas (Rahman et al. 1999). It has been shown that high levels of cadmium in blood is associated with modest elevation in the blood pressure; however, no association was found between urinary cadmium levels and blood pressure (Tellez-Plaza et al. 2008). Association between lead exposure and hypertension is well established by numerous studies (Pirkle et al. 1985; Harlan et al. 1985). A meta-analysis performed by Nawrot et al. (2002)

included more than 58,000 subjects; twice the increase in the concentration of lead in blood is associated with increase in SBP 0.5–1.4 mmHg and DBP 0.4–0.8 mmHg. Various independent studies have not found any consistency between different levels of mercury exposure and fluctuations in the levels of blood pressure, thereby made it difficult establish any direct relation between them (Kobal et al., 2004; Pedersen et al., 2005; Solenkova et al., 2014).

7.6.2 Systemic Inflammation and Oxidative Stress

As a result of various studies it has been known that pollutant can induce the inflammation directly in the respiratory tract which can cause systemic inflammation, hence increasing the risk of cardiovascular morbidity and mortality (An et al. 2018; Salvi et al. 1999). Exposure of individuals with concentrated ambient particles (CAP_S) (Ghio et al. 2000) and diesel exhaust (Salvi et al. 1999) initiates the local inflammation and oxidative stress with increase in the concentration of inflammatory markers such as neutrophils, B-lymphocytes, histamines, T cells (CD4+ and CD8+) in airway and bronchoalveolar lavage. With increase in the concentration of pollutants (mainly NO_2, CO, and PM_{10}) increase in the levels of plasma fibrinogen and viscosity has been observed, possibly due to the inflammatory reaction (Pekkanen et al. 2000; Peters et al. 1997). A study performed in animals where adult mice were co-exposed with different concentration of SO_2, NO_2, and $PM_{2.5}$ stimulates the inflammatory response by increasing the levels of cyclooxygenase-2 (COX-2), tumor necrosis factor-α (TNF-α), inducible nitric oxide synthase (iNOS), and interleukin-6 (IL-6) (Zhang et al. 2015). A control study was performed in which the healthy individuals who spent 5 days near the steel mill where the concentration of CO, SO_x, UFP was higher have shown increase in the levels of pro-inflammatory cytokines such as IL-4, IL-6, and endothelins (Kumarathasan et al. 2018).

7.6.3 Vascular Endothelial Dysfunction and Atherosclerosis

Endothelium is a single-layered wall made up of endothelial cells which line all our blood vessels, such as arteries, veins, arterioles, venules, and capillaries. It plays a critical role in maintaining cardiovascular health by regulating blood pressure, atherogenesis, and thrombosis (An et al. 2018). Short-term exposure to $PM_{2.5}$, NO_2, and SO_2 in mice causes endothelial dysfunction (Zhang et al. 2015), apoptosis in endothelial cells, increases the levels of circulating monocytes and T cells as well as antiangiogenic profile (Pope et al. 2016). A cohort study conducted in different cities indicates that increase in long-term exposure to $PM_{2.5}$ (by 3 $\mu g/m^3$) is strongly associated with 0.3% decrease in flow-mediated dilation (FMD) (95% CI: -0.6 to -0.03; $p = 0.03$) and vasoconstriction, hence affecting the endothelial function (Krishnan et al. 2012).

Studies have linked air pollution events with atherosclerosis, mechanism including lipid peroxidation and inflammation in vascular wall (Künzli et al. 2005). Vascular endothelial dysfunction is now considered as an early maker for atherosclerosis and can prognosticate future cardiovascular events (Davignon and Ganz 2004; Araujo 2011). Increase in the ambient level of $PM_{2.5}$ and traffic is positively correlated with the progression of CIMT (common carotid artery intima-media thickness), which is a biomarker and subclinical form of atherosclerosis (Künzli et al. 2010; Lorenz et al. 2014).

Paraoxonase, a HDL-associated enzyme mediated the cardioprotective antioxidant activity by hydrolyzing the oxidized lipids and trace metals such as lead, and can inhibit the paraoxonase and thus kindle the LDL oxidation and atherosclerosis development (Li et al. 2006). Animal studies have confirmed similar results where exposure of heavy metals such as lead and cadmium to the model animal leads to the progression of atherosclerosis (Revis et al. 1981; Messner et al. 2009).

7.6.4 Myocardial Infarction, Heart Failure, and Anemia

Myocardial infarction (MI) is a medical term used for heart attack, in which the flow of blood is either decreased or completely stops causing damage to the cardiac muscle. Studies have established a parallel and significant association with different pollutants such as SO_2, NO_2, PM_{10}, and $PM_{2.5}$ except O_3 and the risk of heart failure and MI (Wellenius et al., 2005b; Mustafić et al. 2012). Similar result has been observed where hospital admission and death because of heart failure is linked with the exposure of different pollutants such as SO_2, NO_2, $PM_{2.5-10}$ except O_3 (Shah et al. 2013). In a large-scale prospective cohort study and meta-analysis, annual increase of $PM_{2.5}$ and PM_{10} by 5 $\mu g/m^3$ and 10 $\mu g/m^3$ is associated with 13% and 12% increase in the incidence of acute coronary events (MI and unstable angina) (Cesaroni et al. 2014).

Accumulation of mercury also occurs because of the consumption of nonfatty fresh water fish which is associated with MI as well as death from coronary heart disease (Salonen et al. 1995).

Heavy metals such as lead can be efficiently absorbed in the circulatory system that can damage the membrane of the red blood cells (RBC) by interfering with the enzymes responsible for their formation which can result in clinical anemia (Nikolić and Nikić 2008). Chronic exposure to air pollution can cause anemia in humans (WHO 2000). A meta-analysis study reported about 13% increase in the cardiovascular mortality rates after 10 $\mu g/m^3$ increase in the NO_2 concentration annually (Faustini et al. 2014).

7.7 Nervous System

Correlation between air pollution and respiratory health has been well described by countless independent studies across the globe. From last few years, studies have shown that retrograding quality of air causes oxidative stress and inflammation which is then transmitted beyond lungs and vascular system causing damage to the central nervous system (CNS) (Sunyer 2008). Air pollution assaults the nervous system in many different ways either by causing the damage at cellular and molecular level, activating multiple pathways involved in inflammation and oxidative stress which leads to neurological disorders. Pollutants such as PAH, $NO_{x,}$ lead, and mercury are considered as major neurotoxicant. Long-term exposure to such pollutants can lead to neuro-developmental defects. Animal studies have shown that exposing the animals to high levels of pollution, such as PM, combustion, and smoke from diesel engine resulted in accumulation of pro-inflammatory cytokines in brain tissue (Guxens and Sunyer 2012). There is a strong correlation between inflammation and neurodegradation. While breathing tiny nanosized particles can move into the CNS causing neuro-inflammation, oxidative stress and neurotoxicity (Block and Calderón-Garcidueñas 2009; Levesque et al., 2011). Air pollution is known to affect *in utero* growth and postnatal development.

Two hypotheses explain how air pollution affects the nervous system (Sunyer 2008; Guxens and Sunyer 2012):

1. *Indirect affect*: Inflammation in the respiratory tract alters the levels of cytokines, e.g., TNF-α and IL-1, which upregulate the expression of two important inflammatory genes in brain COX2 and IL-1β.
2. *Direct affect*: It has been observed that ultrafine particles containing metals translocate directly into the brain without entering into the respiratory tract.

7.7.1 Neuro-inflammation, Oxidative Stress, and Neurotoxicity

In vitro experiments performed on cells, where they are dosed with laboratory-generated or filter-collected ambient UFPs showed varied degree of pro-inflammatory and oxidative stress-related cellular response and decreased cell growth and viability (Oberdörster et al. 2005). Another in vitro study has established a dose-dependent relationship between the exposures of astroglial cells with oxygen-ozone. 0–40 μg/mL of oxygen-ozone gas per mL of complete medium were not toxic to astroglial cells, while higher concentrations 60 μg/mL or more severely decreased cell viability (Zhou et al. 2008). Oberdörster and Utell (2002) suggested that brain may be vulnerable with the exposure to UFP.

A study was conducted in which rats were exposed to diesel exhaust that demonstrated the uplifted levels of various inflammatory cytokines such as IL-6, IL-1β, MIP-1 (macrophage inflammatory factor), and TNF-α (Levesque et al. 2011). Another study has shown that young adults aged 25.1 ± 1.5 years from high exposure to air pollution have upregulated cyclooxygenase-2 (COX-2), IL-1β, and CD14 in olfactory bulb, substantia nigrae, frontal cortex, and vagus nerve as well as disrupting the blood–brain barrier, generating oxidative stress, and inflammatory cell trafficking compared to the group who were less exposed to air pollution (Calderón-Garcidueñas et al. 2008).

Heavy metals such as lead, mercury, and manganese are strongly implicated in neurotoxicity and nervous system damage (Block et al. 2012). Short-term exposure to lead during perinatal period and childhood has been associated with impaired intellectual function and attention, whereas high doses of lead exposure can cause encephalopathy and convulsions (Mendola et al. 2002). Chronic exposure to manganese leads to the deposition of nanosized manganese particle which causes toxicity from globus pallidus to the basal ganglia region, including the substantia nigra pars compacta involved in the Parkinson's disease (Lucchini et al. 2009). Chronic low dose exposure of ozone can induce oxidative damage and neurodegeneration in the striatum, substantia nigra, and hippocampus in rodents (Pereyra-Muñoz et al. 2006; Rivas-Arancibia et al. 2010). Another study in rats has shown that when exposed to carbon monoxide there is elevation of oxidative stress in cochlear blood vessels which in turn destroys the spiral ganglia neuron and inner hair cells (Lopez et al. 2008).

7.7.2 Neurological Disorder and Neuropsychological Effects

New evidences are emerging from different studies suggesting that increase in the ambient air pollutants increases the risk of getting different neurological disorder such as stroke, Multiple Sclerosis (MS), Alzheimer's disease (AD), and Parkinson's disease (PD) in adults (Genc et al. 2012). AD is one of the most prevalent causes of dementia and chronic neurodegenerative disorder which gradually worsens with time. It is characterized by either the deposition of amyloid-beta (Aβ) peptide fibrils known as amyloid plagues or the tangles of another twisted protein called Tau built up inside the cells (Ballard et al. 2011). The second most common neurodegenerative disorder is Parkinson's disease. PD is associated with gradual loss of dopaminergic loss of neurons in the substantia nigra which results in the dysfunction of basal ganglia that is responsible for the initiation and execution of the movements (Shulman et al. 2011). The occurrence of neurodegenerative disorder is governed by multiple factors including genetic predisposition, nutritional factors, and environmental factors such as exposure to heavy metals and chemicals (insecticides, pesticides) and air pollution (Migliore and Coppedè 2009). There are some common factors which can trigger or contribute in the onset of neurological disorders by damaging the specific neurons such as protein aggregation and amyloid formation, oxidative stress and inflammation in neuronal cells, mitochondrial dysfunction, cellular apoptosis, and microglial activation (Ballard et al. 2011; Shulman et al. 2011).

Many epidemiological studies have shown adverse effects of air pollution on the cognitive behavior in elderly adults (Kramer et al. 2009; Power et al. 2011). Long-term exposure to $PM_{2.5}$ and PM_{10} is cognitively equivalent to brain aging by 1–2 years and is associated with significant decline in the cognitive function in older women (Weuve et al. 2012; Chen et al. 2015). It has been found that young adults and children living in cities with high air pollution levels have altered brain innate immune response and accumulation of Aβ42 and α-synuclein in their neurons (Calderón-Garcidueñas et al. 2008). Exposure to mercury can affect the CNS differently depending on the exposure. Individuals exposed to mercury vapor have impaired cognitive abilities, mainly affecting memory, attention, and psychomotor functioning (Milioni et al. 2017).

Another animal study in which rats were exposed to ozone for a short period resulted in the development of cerebral edema (Crețu et al. 2010). Behavioral studies in rats showed that exposure to ozone can alter the behavior depending upon the dose, time of exposure, and stage of development (Rivas-Arancibiaa et al. 2003; Santucci et al. 2006; Sorace et al. 2001).

7.7.3 Stroke

A stroke is known to occur when the blood supply to different parts of brain is disrupted or reduced, depriving the brain from essential components to function such as oxygen and nutrients. Depending on the cause, strokes have been classified into two categories: Ischemic stroke and Hemorrhagic stroke. Ischemic stroke is solely responsible for 80% of total deaths caused by stroke in which blood supply to the brain tissues gets impaired because of the clot formation and for the rest 20% of stroke deaths is because of hemorrhagic stroke, in which there is a leakage of blood from vessels around the brain (Villeneuve et al. 2006). It has been clearly understood that increase in the ambient air pollution causes inflammation which elevates the level of various cytokines in the blood that promote blood coagulation and thrombosis (Seaton et al. 1995). From the last few years many epidemiological studies have been performed independently providing compelling evidences between the quality of air and the stroke mortality.

While breathing, CO enters into the lungs where it binds with hemoglobin in the blood instead of oxygen and forms carboxy-hemoglobin (COHb), which interferes with the transport of oxygen which may result in hypoxia, neurological disorder, and neuropsychological alterations (Schwela 2000). Villeneuve et al. (2006) showed that CO and NO_2 exposure is associated with the increased risk of hemorrhagic stroke (ORs 1.32, 95% CI = 1.09–1.60) and (ORs 1.26, 95% CI = 1.09–1.46). In 2003 Tsai et al., found a positive correlation between different pollutants and stroke incidences, from the data collected in the span of 4 years from 1997 to 2000. In the single-pollutant model, a positive relationship is established between levels of different pollutants such as SO_2, NO_2, CO, O_3, and PM_{10} and hospital admissions because of both ischemic stroke and primary intracerebral hemorrhage whereas in the 2-pollutant models, only PM_{10} and NO_2 are significantly associated and correlated with hospital admissions for both the types of stroke wherein the effects of CO, SO_2, and O_3 gaseous pollutants were mostly nonsignificant when either NO_2 or PM_{10} levels were controlled independently (Tsai et al. 2003). Another study that describes the similar finding where, increase in the stroke mortality cases for each interquartile range in the same day was 1.5% [95% CI, 1.3–1.8%] for PM10 and 2.9% (95% CI, 0.3–5.5%) for O3 wherein, increase for each interquartile range in a 2 day lag was 3.1% (95% CI, 1.1–5.1%) for NO2, 2.9% (95% CI, 0.8–5.0%) for SO2, and 4.1% (95% CI, 1.1–7.2%) for CO (Hong et al. 2002a). Many other independent studies have confirmed similar findings, stating that stroke cases are significantly associated with prevailing levels of NO and O_3 (Wellenius et al. 2005a; Maheswaran et al. 2005; Hong et al. 2002b). Symptoms of stroke and other cerebrovascular diseases can be instigated by the pollutants in much lower concentration than given as guidelines in many different countries and the world health organization (WHO) (Ponka and Virtanen 1996).

7.8 Digestive System

Effect of air pollution on digestive system has been least studied when compared with a number of studies performed to analyze the effect of ambient air pollution on respiratory system or cardiovascular system. Oral route is usually exposed to more pollutants present in air, as both food and water are affected significantly by the quality of air. All the particles >6 μm get clear from the lungs with mucus and transported to the intestine. Pollutant in the air can cause intestinal injury by directly affecting the epithelial cell layer, increase in the immune response and amending the gut flora (Opstelten et al. 2016). Systemic inflammation caused upon exposure to different pollutants affects intestine as well as is responsible for other gastric disorders.

7.8.1 Inflammatory Bowel Disease (IBD) and Appendicitis

New studies have found a positive correlation between the air pollution levels and gastrointestinal disorders such as Inflammatory Bowel Disease (IBD) and appendicitis. With increase in industrialization, there is increase in the incidence of Ulcerative Colitis (UC) and Crohn's disease; despite this fact, fewer studies are available which studied the direct effect of air pollution and IBD.

It has been seen that there is increase in the incidences of UC and Crohn's disease in the developed nations of North America and Western Europe (Richard F.A. Logan 1998). However, there were rare cases of IBD that were observed in developing nation such as India and China, but with the boom in industrialization, there is increase in the incidences of IBD (Desai and Gupte 2005; Zheng et al. 2005). Both UC and Crohn's disease are considered as two major forms of IBD. On the whole, the incidence of the IBD occurrence is not related directly with air pollution but children and young adults living in areas with high concentration of SO_2 and NO_2 are more likely to suffer from ulcerative colitis and Crohn's disease (Kaplan et al. 2010). Another study performed in 2011 by Ananthakrishnan et al. found that there is a strong association between the hospitalization cases due to the IBD (3890 cases, mean of 81.3 IBD hospitalizations/100,000 people per year) with increase in the pollutants.

There is a dramatic surge in the cases of appendicitis recorded during the nineteenth and early part of the twentieth century when the industrialization of the society was at its peak (Williams 1983). Later in the middle of the twentieth century many countries such as the United States and the United Kingdom passed legislations to improve the quality of air, which resulted in significant decrease in the number of cases registered for appendicitis (Addiss et al. 1990; Chen et al. 2007; Williams et al. 1998). The strong connection between the short-term exposure to the air pollution and occurrence of appendicitis has been established (Kaplan et al. 2009). The rise in the number of appendicitis has been seen mostly during the summers, when most individuals spend their time outside exposed to pollutants like NO_2 and ozone which are considered as primary risk factors when compared to other pollutants (Kaplan et al. 2010).

7.8.2 Gastrointestinal Tumor

Gastric cancer is another most common type of cancer found globally with about twice the rate in men (Torre et al. 2015). Occupational exposure of metals and other pollutants along with dust and high temperature is associated with higher risk of developing different types or gastric/digestive system tumors (Santibanez et al. 2012; Bevan et al. 2012). The exposure of biomass smoke is associated with the frequency of gastric and oesophageal cancer (Kayamba et al. 2017). A study performed by ESCAPE (European Study of Cohorts for Air Pollution Effects) found that elevated level of $PM_{2.5}$ particles suspended in the air by 5 $\mu g/m^3$ is associated with inflation in the hazard ratio of gastric cancer by 1.38 (95% CI 0.99; 1.92) and for upper aero digestive tract cancer (UDAT) by 1.05 (95% CI 0.62; 1.77); no correlation is observed with the exposure of NO_x (Nagel et al. 2018).

A study was performed by Lopez-Abente et al. (2012) for over the period of 10 years (1997–2006), where colorectal cancer mortality cases were examined. "Near vs far" analysis of RR was estimated in those towns (total 8098) which were situated within 2 km of distance from different industrial installations. A strong and significant RR is observed in the towns situated near different industries such as mining industry (RR—1.07; 95% CI 1.00–1.14), food and beverage sector (RR—1.069; 95% CI 1.02–1.11), metal production and processing (RR—1.065; 95% CI

1.01–1.12) and ceramic industry (RR—1.05; 95% CI 1.00–1.09). Another study was performed where the risk of bladder cancer was estimated among those who work in metal industry and a positive correlation was observed (Colt et al. 2014). Similar study was performed in Spain where positive correlation was found between the risk of developing gastric tumor (colorectal and liver cancer) and residential proximity to the metal production and processing industry (García-pérez et al. 2010).

A study was performed in rats, where 7 groups of rats were fed with different concentration of DEP (0, 0.2, 0.8, 2, 8, 20, or 80 mg DEP/kg) for 21 days and DNA leisons and oxidative stress were observed causing DNA strand breakage, uplifted repair capacity of oxidative base damage, protein oxidation and cellular apoptosis in colon mucosa cells and in liver (Dybdahl et al. 2003). Exposure to cadmium and lead is positively associated with the development of prostrate, kidney, liver, and stomach cancer (Tchounwou et al. 2012; Rousseau et al. 2007; Steenland and Boffetta 2000).

7.9 Effect on Fertility, Reproduction, and Pregnancy

From the last two decades scientists have been debating over the impact of air pollution and infertility. There are many studies performed which claimed that there is no association between the amount of pollutants in the ambient air whereas in retrospect many other studies found a positive correlation between infertility, childbirth, and quality of air. WHO has concluded that the increase in the levels of air pollution has adverse effects on pregnancy (WHO 2005). Although different pollutants exert varied effects when exposed to them either independently or in combinatorial manner.

7.9.1 Fertility

The first study in human relates to the increase in the levels of high traffic-related air pollution, particularly the concentration of coarse particle $PM_{2.5-10}$ and reduction in the fertility rates (IRR = 0.87, 95% CI 0.82, 0.94) (Nieuwenhuijsen et al. 2014). Data from epidemiological studies suggests that air pollutant damages and causes defects during the gametogenesis process which ultimately is responsible for reduction in the reproductive potentials among the exposed population (Carré et al. 2017).

Young men exposed to high concentration of SO_2 were found to have sperm abnormalities originated during the spermatid formation (Dejmek et al. 2000). With increase in concentration of $PM_{2.5}$ by 10 μg/m^3 each was associated with the decrease in fecundability of about 22% (95% CI: 6–35%), as well as high concentration of NO_2 was also associated with reduced fecundability (Slama et al. 2013). More than 40 μg/dL of inorganic lead in the blood can impede male reproductive function by impairment in spermatogenesis, reduction in sperm count, volume and density as well as change in sperm mobility and morphology (Apostoli et al. 1998).

In a prospective cohort of nurses, small increase in the risk of infertility was observed among individuals living close to the major roads compared to those who are living far away from the major road (multivariable hazard ratio 1.11, 95% CI 1.02–1.20) (Mahalingaiah et al. 2016).

7.9.2 Fetus Development, Child Birth, and Development

Fetus development is a critical stage that is more vulnerable to the toxic pollutant because of rapid cell division and proliferation, development of organs and less pronounced metabolic pathways (Srám et al. 2005). A study carried out in Sydney stipulate that 10% reduction in the ambient concentration of $PM_{2.5}$ for over 10 years resulted in about 650 (95% CI: 430–850) fewer premature deaths (Broome et al. 2015). A time-series study conducted in Vancouver analyzed the effect of gaseous pollutant on different stages of pregnancy and child development. They found that the exposure to the elevated level of SO_2 during the first month of pregnancy resulted in low birth weight of the child whereas exposure to SO_2 and CO in the last month of pregnancy resulted in the preterm birth and along with NO_2 was also responsible for intrauterine growth retardation (IUGR) of the developing fetus (Liu et al. 2003). A strong association was observed between SO_2 and preterm birth and low birth weight, whereas levels of total suspended matter (TSP) and NO_x show weak correlation and marginal correlation (Bobak 2000). Another attempt was made in Southern California to study the effect of pollutants such as CO, SO_2, NO_2, O_3, and PM_{10}. With increase in the concentration of PM_{10} by 50 μg over 6 weeks results in 20% preterm birth although the levels of CO and number of preterm birth was not consistent, and hence, no pattern was observed (Ritz et al. 2000). Pre- or postnatal exposure to PAH, $PM_{2.5}$, and NO_x can delay or wield the negative effect on the neuropsychological development in children (Frutos et al. 2015). Exposure to lead is strongly associated with decline in intelligence, less IQ (Intelligent Quotient), retardation in neurobehavioral development, impairment of hearing and speech, and poor attention and growth (Amodio-cocchieri et al. 1996; Kaul et al. 1999; Tchunwou et al. 2012).

7.9.3 In Vitro Fertilization (IVF), Clinical Pregnancy, and Implantation Rates

There are very few studies in literature demonstrating the effects of air pollution on IVF (Frutos et al. 2015). Studies have reported the pernicious effect of elevated level of air pollutants on clinical pregnancy and implantation rates (Perin et al. 2010a, b).

Legro et al. (2010) studied the effect of different pollutants at different stages of IVF and embryo transfer. Increase in the concentration of NO_2 is significantly related to the reduction in the probability of conceiving pregnancy and live birth during all the stages of an IVF cycle with OR—0.76 (95% CI: 0.66–0.86), per 0.01 ppm increase. Increase in the concentration of O_3 was negatively associated with live birth but the association was not significant. No correlation was observed between the levels of SO_2 and PM_{10} and live birth rate, number of oocytes retrieved and embryo transfer to the women undergoing their first IVF cycle.

7.9.4 Miscarriage

Green et al. (2009) concluded that pregnant women living within 50 m of road are at high risk of spontaneous abortion because of traffic and vehicle emission. Another epidemiological study performed in Labin (Croatia) found significant association between the pollutants released from the combustion of coal and complications during pregnancy, miscarriages, and stillbirths (Mohorovic et al. 2010). It was also observed that there is more than twofold increase in the risk of miscarriage when there was a brief exposure to high levels of PM_{10} during the preconceptional period irrespective of the method of conception (Perin et al. 2010a, b). Another study performed by the same group confirms the above finding where women exposed to high concentration of PM (>56 μg/m^3) during the follicular phase of conception cycle had high risk of miscarriage (OR—5.05; 95% CI: 1.04–25.51) (Perin et al. 2015). Faiz et al. (2012), examining the rate of stillbirth associated with air pollution levels from 1998 to 2004, confirmed that increase in the levels of different gaseous pollutants such as NO_2, SO_2, and CO during pregnancy appeared to increase the relative odds of stillbirth. They showed that 10-ppb increase in concentration of NO_2 during the first trimester (OR = 1.16, 95% CI: 1.03–1.31), 3-ppb increase in the concentration of SO_2 during the first (OR = 1.13, 95% CI: 1.01–1.28) and third (OR = 1.26, 95% CI: 1.0–1.37) trimesters, and each 0.4-ppm increase in concentration of CO during the second (OR = 1.14, 95% CI: 1.01–1.28) and third (OR = 1.14, 95% CI: 1.06–1.24) trimesters is associated with relative odds of stillbirth.

Exposure to heavy metals such as lead from low to moderate concentration (0–30 μg/dL) elevates the risk of spontaneous abortion (Hertz-Picciotto 2000).

7.10 Effect on Skin

Skin is the first line of defense in the body of an organism; being the largest and outermost organ, it is exposed to all the pollutants present in the environment. Air pollutants exist in many different forms and hence can directly cross the physical barrier and be absorbed by the subcutaneous tissue and sebaceous glands present beneath the skin. Prolonged exposure of the skin to the pollutants can alter its normal defense mechanism and exert a detrimental effect which may result in various skin diseases (Puri et al. 2017). There are four mechanisms through which air pollutants can cause adverse effects on skin health (Mancebo and Wang 2015):

- Free radical generations
- Induction of inflammatory signaling cascade and subsequent impairment of skin barrier
- Aryl hydrocarbon receptor (AhR) activation
- Alteration in skin microflora

Encounter of various pollutants with the skin can cause oxidative stress which destroys the homeostasis by depleting the cellular pool of redox enzymes (glutathione peroxidase, glutathione reductase, superoxide dismutase, catalase) and antioxidants (Vitamin E, Vitamin C, and glutathione) (Valacchi et al. 2012). Oxidative stress can cause alteration in the activation status of a number of transcription factors (AP1, NF-kB, cJUN, etc.) and rewiring various generic signaling pathways such as ERK-1/2/5, p38, and JNK (Wlaschek et al. 2001; Dhar et al. 2002; Kim et al. 2005) resulting in the damage of macromolecules (DNA, proteins, or lipids), extrinsic aging of the skin, inflammatory or allergic reactions such as dermatitis (contact or atopic), psoriasis, acne, and skin cancer (Kampa and Castanas 2008; Baudouin et al. 2002; Kohen and Gati 2000; Halliwell and Cross 1994).

7.10.1 Oxidative Stress, Skin Aging, and Pigmentation

PAH gets converted into quinones, redox-cycling chemicals that can generate reactive oxygen species (ROS) (Penning 1993). PAH can bind to the combustion-derived PM in the air which can be readily absorbed in the skin by hair follicle or trans-epidermal absorption and result into oxidative stress and skin aging (Menichin 1992; Vierkotter et al. 2010). A direct correlation between prevalence and severity of acne and the number of cigarettes smoked among smokers has been observed (Schäfer et al. 2001). PAH is known to be associated with skin aging and pigmentation (Puri et al. 2017). Long-term exposure of murine skin to ozone diminishes the levels of antioxidants such as tocopherol (vitamin E) and ascorbic acid (vitamin C) and elevates the level of lipid peroxidation product (malondialdehyde) which impair the normal functioning of skin cells and causes inflammation (Thiele et al. 1997).

Exposing normal human epidermal keratinocytes (NHEKs) to 0.8 ppm ozone for 30 min will result into DNA damage, depletion of ATP levels, NAD-dependent histone deacetylase, sirtuin3. (McCarthy et al. 2013). Similar levels of ozone's exposure to human participants resulted in 70% decrease in the endogenous levels of vitamin E and increase in the levels of lipid hydroperoxide by up to 230% in stratum corneum (He et al. 2006). Direct exposure of skin to dust particles, cigarette smoke, oxides (SO_x, NO_x, O_3) and VOCs results in oxidative damage, significant increase in the levels of pro-inflammatory genes and also the genes responsible for wrinkle formation, pigmentation and aging (Choi et al. 2011; Krutmann et al. 2014)

Long-term exposure to particulate matter can lead to pigmentation, which is the sign of premature skin aging (Vierkotter et al. 2010; Krutmann et al. 2014).

7.10.2 Dermatitis, Psoriasis, and Eczema

Schultz-Larsen (1993) found that the pervasiveness of atopic dermatitis has increased over the past few decades in Europe. A comparative study was performed in different parts of Germany examining different parts and levels of pollution such as East Germany (industrial; sulfurous) and West Germany (urban; oxidizing), which showed that the prevalence of atopic dermatitis was greatest in East Germany when compared to West Germany as well as many other direct and indirect parameters of air pollution were measured and the greatest association of the atopic eczema was found with NOx exposure (indoor use of gas without the cooker top and close proximity to roads with heavy traffic) (Schäfer and Ring 1997).

A study performed in children when shifted from one building to another during study indicated that there is increase in the risk of atopic dermatitis aggravation due to high levels of VOCs in the building (Kim et al. 2015). Exposure of keratinocytes cultures with VOCs are known to elevate the concentration of various cytokines such as IL-8 and IL-1B causing atopic dermatitis and eczema (Puri et al. 2017). NO_2, SO_2, and SO_3 were associated with exacerbation of eczema and atopic dermatitis whereas $PM_{2.5}$ has higher heavy metal ratio (such as arsenic, copper, cadmium, nickel, vanadium, lead, and zinc) compared to PM_{10}, and is associated with prevalence of eczema and atopic dermatitis (Kathuria and Silverberg 2016). An increase in the concentration of ozone by 10 $\mu g/m^3$ in 7 days average resulted in significant increase in the urticaria (3.84%), eczema (2.86%), contact dermatitis (3.22%), and rash (2.72%) (Xu et al. 2011).

Kramer et al. (2009) showed that children of age 6 years living in traffic-related, highly polluted areas are at greater risk for the prevalence of eczema (risk of 1.69 per 90% range of soot concentration).

7.10.3 Skin Cancer

Melanoma, basal cell carcinoma (BCC), and squamous cell carcinoma (SCC) are the most common types of skin cancer in people worldwide. In an animal study VOCs (hexachlorobenzene) have seen to cause precancerous lesion on the skin of rats (Michielsen et al. 1999). Activated PAH can produce epoxides and diols which can bind to the DNA and are responsible for causing carcinogenesis (Baudouin et al. 2002; Kelfkens et al. 1991). A meta-analysis showed that smoking was significantly associated with cutaneous squamous cell carcinoma and basal cell carcinoma (OR, 1.52; 95% CI, 1.15–2.01 and OR, 0.95; 95% CI, 0.82–1.09) or nonmelanoma skin cancer (OR, 0.62; 95% CI, 0.21–1.79) (Leonardi-Bee et al. 2012). Rezazadeh and

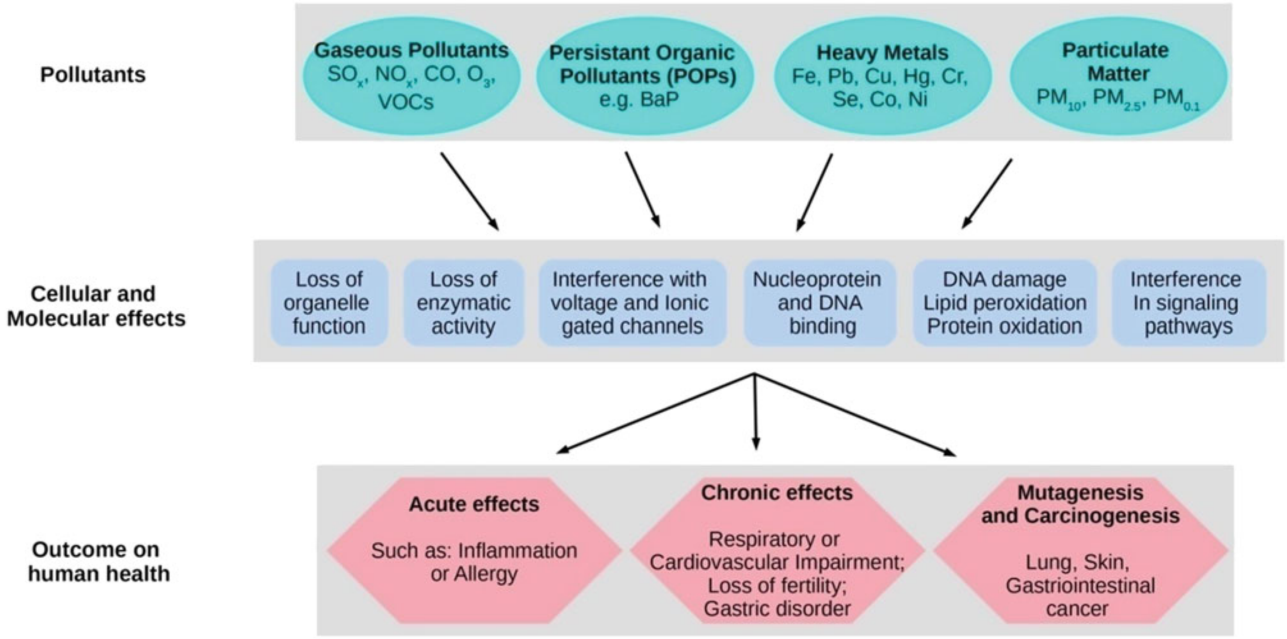


Fig. 7.2 Mechanism by which pollutants affect human health

Athar (1997) showed that the iron overload on 7,12-dimethylbenz[a]anthracene promotes genotoxic stress and initiates skin tumorigenesis in mice. Ingesting arsenic alone or in combination with other risk factors such as UV-B, it is known to cause SCC and BCC (Fabbrocini et al. 2010).

7.11 Summary

This chapter reviews the adverse effects of different air pollutants on human health. Pollutants in air can trigger and exacerbate many diseases which may affect many different organs and systems by impairing their ability to perform normal function, ultimately leading to high mortality and morbidity. As mentioned, concentration of pollutants and exposure time cause different acute and chronic effects. Briefly, Fig 7.2 summerizes different pollutants and their effects of different systems and organs of human body.

It has been over five decades when the first study about the effect of environmental pollutants on human health was performed; it is still one of the major topics of research in the area of environmental science and human health. Dose-dependent effects of many pollutants have been studied extensively for different body systems and organs, such as pulmonary or cardiovascular system. Other systems and organs such as digestive system, nervous system or in skin, the effects of air pollution has been overlooked, though few studies were performed which established a strong and direct correlation between them. Controlled exposure studies of toxic air pollutants solely cannot explain the effects of air pollutants on human health; along with that animal toxicology, occupational and epidemiology studies together can conclude the effect of a single pollutant and complex pollutants on human health.

References

Abhyankar LN, Jones MR, Guallar E, Navas-Acien A (2012) Arsenic exposure and hypertension: a systematic review. Environ Health Perspect 120:494–500

Addiss DG, Shaffer N, Fowler BS, Tauxe RV (1990) The epidemiology of appendicitis and appendectomy in the United States incidental appendectomies are commonly performed at the time of other abdominal or pelvic surgery to prevent future appendicitis (16). Because the epidemiology of incidental ap. Am J Epidemiol 132:910–925

Aksu A (2015) Sources of metal pollution in the urban atmosphere (A case study: Tuzla, Istabul). J Environ Health Sci Eng 13:79. https://doi.org/10.1186/s40201-015-0224-9

Alissa EM, Ferns GA (2011) Heavy metal poisoning and cardiovascular disease. J Toxicol. https://doi.org/10.1155/2011/870125

Amodio-Cocchieri R (1996) LEAD in human blood from children living in CAMPANIA, ITALY. J Toxicol Environ Health 47(4):311–320

An Z, Jin Y, Li J, Li W, Wu W (2018) Impact of particulate air pollution on cardiovascular health. Curr Allergy Asthma Rep 18(3):15

Ananthakrishnan AN, Mcginley EL, Binion DG (2011) Ambient air pollution correlates with hospitalizations for inflammatory bowel disease: an ecologic analysis. Inflamm Bowel Dis 17:1138–1145. https://doi.org/10.1002/ibd.21455

Apostoli P, Kiss P, Porru S et al (1998) Male reproductive toxicity of lead in animals and humans. Occup Environ Med 55:364–374

Araujo JA (2011) Particulate air pollution, systemic oxidative stress, inflammation, and atherosclerosis. Atmos Health 4:79–93. https://doi.org/10.1007/s11869-010-0101-8

Aris RM, Christian D, Hearne PQ et al (1993) Ozone-induced airway inflammation in human subjects as determined by airway lavage and biopsy. Am J Respir Crit Care Med 148:1363–1372

Asher I, Pearce N (2014) Global burden of asthma among children. Int J Tuberc Lung Dis 18:1269–1278

Asker S, Asker M, Yeltekin AC, Aslan M, Ozbay B, Demir H, Turan H (2018) Serum levels of trace minerals and heavy metals in severe COPD patients with and without pulmonary hypertension. Int J Chron Obstruct Pulmon Dis 13:1803–1808

Ballard C, Gauthier S, Corbett A et al (2011) Alzheimer' s disease. Lancet 377:1019–1031. https://doi.org/10.1016/S0140-6736(10)61349-9

Baudouin C, Charveron M, Tarroux R et al (2002) Environmental pollutants and skin cancer. Cell Biol Toxicol 18:341–348

Beelen R, Hoek G, Van Den Brandt PA et al (2008) Long-term exposure to traffic-related air pollution and lung cancer risk. Epidemiology 19:702–710. https://doi.org/10.1097/EDE.0b013e318181b3ca

Belanger K, Gent JF, Triche EW et al (2006) Association of indoor nitrogen dioxide exposure with respiratory symptoms in children with asthma. Am J Respir Crit Care Med 173:297–303. https://doi.org/10.1164/rccm.200408-1123OC

Bevan R, Young C, Holmes P et al (2012) Occupational cancer in Britain Gastrointestinal cancers: liver, oesophagus, pancreas and stomach. Br J Cancer 107:S33–S40. https://doi.org/10.1038/bjc.2012.116

Beyer A, Mackay D, Matthies M, Wania F, Webster E (2000) Assessing long-range transport potential of persistent organic pollutants. Environ Sci Technol 4(4):699–703

Beyersmann D, Hartwig A (2008) Carcinogenic metal compounds: recent insight into molecular and cellular mechanisms. Arch Toxicol 82:493–512. https://doi.org/10.1007/s00204-008-0313-y

Block ML, Calderón-Garcidueñas L (2009) Air pollution: mechanisms of neuroinflammation and CNS disease. Trends Neurosci 32:506–516. https://doi.org/10.1016/j.tins.2009.05.009

Block ML, Elder A, Auten RL et al (2012) The outdoor air pollution and brain health workshop. Neurotoxicology 33:972–984. https://doi.org/10.1016/j.neuro.2012.08.014

Bobak M (2000) Outdoor air pollution, low birth weight, and prematurity. Environ Health Perspect 108:173–176

Bogden JD, Gertner SB, Kemp FW et al (1991) Dietary lead and calcium: effects on blood pressure and renal neoplasia in Wistar rats. Minerals Trace Elements 121:718–728

Brook RD (2007) Why physicians who treat hypertension should know more about air pollution. J Clin Hypertens 9:629–635

Broome RA, Fann N, Navin TJ et al (2015) The health benefits of reducing air pollution in Sydney, Australia. Environ Res 143:19–25. https://doi.org/10.1016/j.envres.2015.09.007

Calderón-Garcidueñas L, Solt AC, Carlos Henríquez-Roldán RJ, Bryan Nuse L, Herritt R-C et al (2008) Long-term air pollution exposure is associated with neuroinflammation, an altered innate immune response, disruption of the blood-brain barrier, ultrafine particulate deposition, and accumulation of amyloid β −42 and α-synuclein in children and young. Toxicol Pathol 36:289–310. https://doi.org/10.1177/0192623307313011

Cao J, Yang C, Li J et al (2011) Association between long-term exposure to outdoor air pollution and mortality in China: a cohort study. J Hazard Mater 186:1594–1600. https://doi.org/10.1016/j.jhazmat.2010.12.036

Carré J, Gatimel N, Moreau J et al (2017) Does air pollution play a role in infertility?: a systematic review. Environ Health 16:82. https://doi.org/10.1186/s12940-017-0291-8

Cesaroni G, Stafoggia M, Galassi C et al (2014) Long term exposure to ambient air pollution and incidence of acute coronary events: prospective cohort study and meta-analysis in 11 European cohorts from the ESCAPE project. BMJ 348:f7412. https://doi.org/10.1136/bmj.f7412

Chen C, Modrek S (2018) Gendered impact of solid fuel use on acute respiratory infections in children in China. BMC Public Health 18(1):1170. https://doi.org/10.1186/s12889-018-6035-z

Chen T, Shofer S, Gokhale J, Kuschner WG (2007) Outdoor air pollution: overview and historical perspective. Med Sci 333:230–234. https://doi.org/10.1097/MAJ.0b013e31803b8c91

Chen G, Wan X, Yang G, Zou X (2014) Traffic-related air pollution and lung cancer: a meta-analysis. Thorac Cancer 6:307–318. https://doi.org/10.1111/1759-7714.12185

Chen JC, Wang X, Wellenius GA, Serre ML, Driscoll I, Casanova R, McArdle JJ, Manson JE, Chui HC, Espeland MA (2015) Ambient air pollution and neurotoxicity on brain structure: evidence from women's health initiative memory study. Ann Neurol 78:466–476. https://doi.org/10.1002/ana.24460.Ambient

Choi H, Wook D, Kim W et al (2011) Asian dust storm particles induce a broad toxicological transcriptional program in human epidermal keratinocytes. Toxicol Lett 200:92–99. https://doi.org/10.1016/j.toxlet.2010.10.019

Clarkson TW, Magos L, Myers GJ (2003) The toxicology of mercury — current exposures and clinical manifestations. N Engl J Med 349:1731–1737

Colt JS, Friesen MC, Stewart PA et al (2014) A case-control study of occupational exposure to metal working fluids and bladder cancer risk among men. Occup Environ Med 71:667–674. https://doi.org/10.1136/oemed-2014-102362.221.A

Crețu DI, Sovrea A, Ignat RM, Filip A, Bidian C, Crețu AURICA (2010) Morpho-pathological and physiological changes of the brain and liver after ozone exposure. Rom J Morphol Embryol 51:701–706

Das S, Grewal A, Banerjee M (2011) A brief review: heavy metal and their analysis. Int J Pharm Sci Rev Res 11:13–18

Davignon J, Ganz P (2004) Role of endothelial dysfunction in atherosclerosis. Circulation 109: III27. https://doi.org/10.1161/01.CIR.0000131515.03336.f8

Dejmek J, Jelínek R, Solansky I, Benes I, Srám RJ (2000) Fecundability and parental exposure to ambient sulfur dioxide. Environ Health Perspect 108:647–654

Desai HG, Gupte PA (2005) Increasing incidence of Crohn's disease in India: is it related to improved sanitation? Indian J Gastroenterol 24(1):23

Dhar A, Young MR, Colburn NH (2002) The role of AP-1, NF-κB and ROS/NOS in skin carcinogenesis: the JB6 model is predictive. Mol Cell Biochem 234:185–193

Dockery DW, Pope CA (1994) Acute respiratory effects of particulate air pollution. Annu Rev Public Health 15:107–132

Dockery DW, Pope CA, Xu X et al (1993) An association between air pollution and mortality in six US cities. N Engl J Med 329:1753–1175

Dunnick JK, Elwell MR, Radovsky AE et al (1995) Comparative carcinogenic effects of nickel subsulfide, nickel oxide, or nickel sulfate hexahydrate chronic exposures in the Lung1. Cancer Res 55:5251–5256

Duruibe JO, Ogwuegbu MOC, Egwurugwu JN (2007) Heavy metal pollution and human biotoxic effects. Int J Phys Sci 2:112–118

Dybdahl M, Risom L, Mùller P et al (2003) DNA adduct formation and oxidative stress in colon and liver of Big Blue Ò rats after dietary exposure to diesel particles. Carcinogenesis 24:1759–1766. https://doi.org/10.1093/carcin/bgg147

Fabbrocini G, Triassi M, Mauriello MC et al (2010) Epidemiology of skin cancer: role of some environmental factors. Cancers (Basel) 2:1980–1989. https://doi.org/10.3390/cancers2041980

Faiz AS, Rhoads GG, Demissie K et al (2012) Ambient air pollution and the risk of stillbirth. Am J Epidemiol 176:308–316. https://doi.org/10.1093/aje/kws029

Faustini A, Rapp R, Forastiere F (2014) Nitrogen dioxide and mortality: review and meta-analysis of long-term studies. Eur Respir J 44:744–753. https://doi.org/10.1183/09031936.00114713

Feng Z, Hu W, Rom WN et al (2003) Chromium (VI) exposure enhances polycyclic aromatic hydrocarbon-DNA binding at the p53 gene in human lung cells Chromium (VI) exposure enhances polycyclic aromatic hydrocarbon-DNA binding at the p53 gene in human lung cells. Carcinogenesis 24:771–778. https://doi.org/10.1093/carcin/bgg012

Firket BYJ, March RI (1936) Fog along the Meuse valley. Trans Faraday Soc 32:1192–1196

Fortoul TI, Rojas-Lemus M, Rodriguez-Lara V, Cano-Gutierrez G, Gonzalez-Villalva A, Ustarroz-Cano M, . . . Gonzalez-Rendon ES (2011) Air pollution and its effects in the respiratory system. The Impact of Air Pollution on Health, Economy, Environment and Agricultural Sources 41

Frampton MW, Boscia J, Roberts NJ et al (2002) Nitrogen dioxide exposure: effects on airway and blood cells. Am J Physiol Cell Mol Physiol 2982:L155–L165

Frutos V, González-Comadrá M, Solà I et al (2015) Impact of air pollution on fertility: a systematic review. Gynecol Endocrinol 31(1):7–13. https://doi.org/10.3109/09513590.2014.958992

Gallagher CM, Meliker JR (2010) Blood and urine cadmium, blood pressure, and hypertension: a systematic review and meta-analysis. Environ Health Perspect 118:1676–1684. https://doi.org/10.1289/ehp.1002077

García-pérez J, López-cima MF, Pérez-gómez B, Aragonés N (2010) Science of the total environment mortality due to tumours of the digestive system in towns lying in the vicinity of metal production and processing installations. Sci Total Environ 408:3102–3112. https://doi.org/10.1016/j.scitotenv.2010.03.051

Genc S, Zadeoglulari Z, Fuss SH, Genc K (2012) The adverse effects of air pollution on the nervous system. J Toxicol 2012:782462. https://doi.org/10.1155/2012/782462

Ghio AJ, Kim C, Devlin RB (2000) Concentrated ambient air particles induce mild pulmonary inflammation in healthy human volunteers. Am J Respir Crit Care Med 162:981–988

Ghozikali MG, Mosaferi M, Safari GH, Jaafari J (2015) Effect of exposure to O3, NO2, and SO2 on chronic obstructive pulmonary disease hospitalizations in Tabriz, Iran. Environ Sci Pollut Res 22:2817–2823. https://doi.org/10.1007/s11356-014-3512-5

Green RS, Malig B, Windham GC et al (2009) Residential exposure to traffic and spontaneous abortion. Environ Health Perspect 117:1939–1944. https://doi.org/10.1289/ehp.0900943

Guarnieri M, Balmes JR (2014) Outdoor air pollution and asthma. Lancet 383(9928):1581–1592

Guo Y, Tong S, Li S et al (2010) Gaseous air pollution and emergency hospital visits for hypertension in Beijing, China: a time-stratified case-crossover study. Environ Health 9:57

Guxens M, Sunyer J (2012) A review of epidemiological studies on neuropsychological effects of air pollution. Swiss Med Wkly 141(1):w13322

Halliwell B, Cross CE (1994) Oxygen-derived species: their relation to human disease and environmental stress. Environ Health Perspect 102:5–12

Harlan WR (1988) The relationship of blood lead levels to blood pressure in the U.S. population. Environ Health Perspect 78:9–13

Harlan WR, Landis JR, Schmouder RL et al (1985) Blood lead and blood pressure relationship in the adolescent and adult US population. JAMA 253:530–514

He QC, Tavakkol A, Wietecha K et al (2006) Effects of environmentally realistic levels of ozone on stratum corneum function. Int J Cosmet Sci 28:349–357

Hecht SS (1999) Tobacco smoke carcinogens and lung cancer. J Natl Cancer Inst 91:1194–1210

Heo J, Park HS, Hong Y et al (2017) Serum heavy metals and lung function in a chronic obstructive pulmonary disease cohort. Toxicol Environ Health Sci 9:30–35. https://doi.org/10.1007/s13530-017-0300-x

Hertz-Picciotto I (2000) The evidence that lead increases the risk for spontaneous abortion. Am J Ind Med 38(3):300–309

Hong Y, Lee J, Kim H et al (2002a) Effects of air pollutants on acute stroke mortality. Environ Health Perspect 110:187–191

Hong Y-C, Lee J-T, Kim H, Kwon H-J (2002b) Air Pollution. Stroke 33(9):2165–2169

Houston MC (2011) Role of mercury toxicity in hypertension, cardiovascular disease, and stroke. J Clin Hypertens 13:621–627. https://doi.org/10.1111/j.1751-7176.2011.00489.x

Ibald-mulli A, Stieber J, Koenig W, Peters A (2001) Effects of air pollution on blood pressure: a population-based approach. Am J Public Health 91:571–577

Iskandar A, Andersen ZJ, Bønnelykke K, Ellermann T, Andersen KK, Bisgaard H (2012) Coarse and fine particles but not ultrafine particles in urban air trigger hospital admission for asthma in children. Thorax 67(3):252–257

Jaishankar M, Tseten T, Anbalagan N et al (2014) Toxicity, mechanism and health effects of some heavy metals. Interdiscip Toxicol 7:60–72. https://doi.org/10.2478/intox-2014-0009

Jiang XQ, Mei XD, Feng D (2016) Air pollution and chronic airway diseases: what should people know and do? J Thorac Dis 8:E31–E40

Johns DO, Linn WS (2011) A review of controlled human SO2 exposure studies contributing to the US EPA integrated science assessment for sulfur oxides. Inhal Toxicol 23:33–43. https://doi.org/10.3109/08958378.2010.539290

Kampa M, Castanas E (2008) Human health effects of air pollution. Environ Pollut 151:362–367. https://doi.org/10.1016/j.envpol.2007.06.012

Kaplan GG, Dixon E, Panaccione R, Fong A, Chen L, Szyszkowicz M et al (2009) Effect of ambient air pollution on the incidence of appendicitis. CMAJ 181(9):591–597

Kaplan GG, Hubbard J, Korzenik J et al (2010) The inflammatory bowel diseases and ambient air pollution: a novel association. Am J Gastroenterol 105:2412–2419. https://doi.org/10.1038/ajg.2010.252

Kathuria P, Silverberg JI (2016) Association of pollution and climate with atopic eczema in US children. Pediatr Allergy Immunol 27:478–485. https://doi.org/10.1111/pai.12543

Kaul B, Sandhu RS, Depratt C et al (1999) Follow-up screening of lead-poisoned children near an auto battery recycling plant, Haina, Dominican Republic. Environ Health Perspect 107:917–920

Kawane H, Soejima R, Umeki S, Niki Y (1988) Metal fume fever and asthma. Chest 93:1116–1117. https://doi.org/10.1378/chest.93.5.1116b

Kayamba V, Heimburger DC, Morgan DR et al (2017) Exposure to biomass smoke as a risk factor for oesophageal and gastric cancer in low-income populations: a systematic review. Malawi Med J 29:212–217

Kelfkens G, De Gruijl FR, Van Der Leun JC (1991) Tumorigenesis by short-wave ultraviolet A: papillomas versus squamous cell carcinomas. Carcinogenesis 12:1377–1382

Khaniabadi YO, Daryanoosh M, Sicard P et al (2018) Chronic obstructive pulmonary diseases related to outdoor PM10, O3, SO2, and NO2 in a heavily polluted megacity of Iran. Environ Sci Pollut Res 25:17726–17734

Kim AL, Labasi ÃJM, Zhu Y et al (2005) Role of p38 MAPK in UVB-induced inflammatory responses in the skin of SKH-1 hairless mice. J Invest Dermatol 124:1318–1325. https://doi.org/10.1111/j.0022-202X.2005.23747.x

Kim KH, Jahan SA, Kabir E, Brown RJ (2013) A review of airborne polycyclic aromatic hydrocarbons (PAHs) and their human health effects. Environ Int 60:71–80. https://doi.org/10.1016/j.envint.2013.07.019

Kim E, Kim S, Lee JH et al (2015) Indoor air pollution aggravates symptoms of atopic dermatitis in children. PLoS One 10:e0119501. https://doi.org/10.1371/journal.pone.0119501

Kirschvink N, Fi L, Wirth D et al (2005) Repeated cadmium nebulizations induce pulmonary MMP-2 and MMP-9 production and enphysema in rats. Toxicology 211(1–2):36–48. https://doi.org/10.1016/j.tox.2005.02.012

Kirschvink N, Martin N, Fievez L et al (2009) Airway inflammation in cadmium-exposed rats is associated with pulmonary oxidative stress and emphysema. Free Radic Res 40:241–250. https://doi.org/10.1080/10715760500494657

Kobal AB, Horvat M, Preze M et al (2004) The impact of long-term past exposure to elemental mercury on antioxidative capacity and lipid peroxidation in mercury miners. J Trace Elem Med Biol 17:261–274

Kohen R, Gati I (2000) Skin low molecular weight antioxidants and their role in aging and in oxidative stress. Toxicology 148:149–157

Kramer U, Sugiri D, Ranft U, Krutmann J et al (2009) Eczema, respiratory allergies, and traffic-related air pollution in birth cohorts from small-town areas. J Dermatol Sci 56:99–105. https://doi.org/10.1016/j.jdermsci.2009.07.014

Krishnan RM, Adar SD, Szpiro AA, Jorgensen NW, Hee V et al (2012) Vascular responses to long- and short-term exposure to fine particulate matter: MESA Air (Multi-Ethnic Study of Atherosclerosis and Air Pollution). J Am Coll Cardiol 60(21):2158–2166

Krutmann J, Liu W, Li L et al (2014) Pollution and skin: from epidemiological and mechanistic studies to clinical implications. J Dermatol Sci 76:163–168. https://doi.org/10.1016/j.jdermsci.2014.08.008

Kulle TJ, Sauder LR, Hebel JR, Miller WR, Green DJ, Shanty F (1986) Pulmonary effects of sulfur dioxide and respirable carbon aerosol. Environ Res 41:239–250

Kumarathasan P, Vincent R, Blais E et al (2018) Cardiovascular and inflammatory mechanisms in healthy humans exposed to air pollution in the vicinity of a steel mill. Part Fibre Toxicol 15 (1):34

Künzli N, Jerrett M, Mack WJ et al (2005) Ambient air pollution and atherosclerosis in Los Angeles. Environ Health Perspect 113:201–206. https://doi.org/10.1289/ehp.7523

Künzli N, Jerrett M, Garcia-Esteban R et al (2010) Ambient air pollution and the progression of atherosclerosis in adults. PLoS One 5(2):e9096. https://doi.org/10.1371/journal.pone.0009096

Kurt OK, Zhang J, Pinkerton KE (2016) Pulmonary health effects of air pollution. Curr Opin Pulm Med 22(2):138

Landolph JR (1994) Molecular mechanisms of transformation of C3H/1OT1/2 Cl 8 mouse embryo cells and diploid human fibroblasts by carcinogenic metal compounds. Environ Health Perspect 102:119–125

Larsen FS (1993) Atopic dermatitis: a genetic-epidemiologic study in a population-based twin sample. J Am Acad Dermatol 28:719–723. https://doi.org/10.1016/0190-9622(93)70099-F

Lee B, Kim B, Lee K (2014) Air pollution exposure and cardiovascular disease. Toxicol Res 30:71–75

Leem AY, Kim SK, Chang J et al (2015) Relationship between blood levels of heavy metals and lung function based on the Korean National Health and nutrition examination survey IV–V. Int J Chron Obstruct Pulmon Dis 10:1559

Legro RS, Sauer MV, Mottla GL et al (2010) Effect of air quality on assisted human. Hum Reprod 25:1317–1324. https://doi.org/10.1093/humrep/deq021

Lelieveld J, Evans JS, Fnais M et al (2015) The contribution of outdoor air pollution sources to premature mortality on a global scale. Nature 525:367–371. https://doi.org/10.1038/nature15371

Leonardi-Bee J, Ellison T, Bath-Hextall F (2012) Smoking and the risk of nonmelanoma skin cancer: systematic review and meta-analysis. Arch Dermatol 148(8):939–946

Levesque S, Taetzsch T et al (2011) Diesel exhaust activates and primes microglia: air pollution, neuroinflammation, and regulation of dopaminergic neurotoxicity. Environ Health Perspect 119:1149–1155. https://doi.org/10.1289/ehp.1002986

Li W, Pan M, Chung M et al (2006) Lead exposure is associated with decreased serum Paraoxonase 1 (PON1) activity and genotypes. Environ Health Perspect 114:1233–1236. https://doi.org/10.1289/ehp.9163

Liu S, Krewski D, Shi Y et al (2003) Association between gaseous ambient air pollutants and adverse pregnancy outcomes in Vancouver, Canada. Environ Health Perspect 111:1773–1778. https://doi.org/10.1289/ehp.6251

Liu SK, Cai S, Chen Y, Xiao B, Chen P, Xiang XD (2016) The effect of pollutional haze on pulmonary function. J Thoracic Dis 8(1):E41–E56

Logan RF (1998) Inflammatory bowel disease incidence: up, down or unchanged? Gut 42:309–311

Lopez IA, Acuna D, Beltran-parrazal L et al (2008) Oxidative stress and the deleterious consequences to the rat cochlea after prenatal chronic mild exposure to carbon monoxide in air. Neuroscience 151:854–867. https://doi.org/10.1016/j.neuroscience.2007.10.053

Lopez-abente G, Garcia-perez J, Boldo E, Ramis R (2012) Colorectal cancer mortality and industrial pollution in Spain. BMC Public Health 12:589. https://doi.org/10.1186/1471-2458-12-589

Lorenz MW, Polak JF, Kavousi M et al (2014) Carotid intima-media thickness progression to predict cardiovascular events in the general population (the PROG-IMT collaborative project): a meta-analysis of individual participant data. Lancet 379:2053–2062. https://doi.org/10.1016/S0140-6736(12)60441-3.Carotid

Lucchini RG, Martin CJ, Doney BC (2009) From manganisum to manganese-induced parkinsonism: a conceptual model based on the evolution of exposure. NeuroMolecular Med 11:311–321. https://doi.org/10.1007/s12017-009-8108-8

Mabahwi NAB, Ling O et al (2014) Human health and wellbeing: human health effect of air pollution. Proce Soc Behav Sci 153:221–229. https://doi.org/10.1016/j.sbspro.2014.10.056

Mahalingaiah S, Hart JE, Laden F et al (2016) Adult air pollution exposure and risk of infertility in the Nurses' Health Study II. Hum Reprod 31(3):638–647

Maheswaran R, Haining RP, Brindley P et al (2005) Outdoor air pollution and stroke in sheffield, United Kingdom a small-area level geographical study. Stroke 36:239–243. https://doi.org/10.1161/01.STR.0000151363.71221.12

Mancebo SE, Wang SQ (2015) Recognizing the impact of ambient air pollution on skin health. J Eur Acad Dermatol Venereol 29:2326–2332. https://doi.org/10.1111/jdv.13250

Mannuccio P, Sergio M, Ida H, Massimo M (2015) Effects on health of air pollution: a narrative review. Intern Emerg Med 10:657–662. https://doi.org/10.1007/s11739-015-1276-7

Matés JM, Segura JA, Alonso FJ, Márquez J (2010) Free radical biology & medicine roles of dioxins and heavy metals in cancer and neurological diseases using ROS-mediated mechanisms. Free Radic Biol Med 49:1328–1341. https://doi.org/10.1016/j.freeradbiomed.2010.07.028

McCarthy JT, Pelle E, Dong K et al (2013) Letter to the editor effects of ozone in normal human epidermal keratinocytes. Exp Dermatol 22:360–361. https://doi.org/10.1111/exd.12125

Mendola P, Selevan SG, Gutter S, Rice D (2002) Environmental factors associated with a spectrum of neurodevelopmental deficits. Ment Retard Dev Disabil Res Rev 8:188–197. https://doi.org/10.1002/mrdd.10033

Menichin E (1992) Urban air pollution by polycyclic aromatic hydrocarbons: levels and sources of variability. Sci Total Envivron 116(1–2):109–135

Messner B, Knoflach M, Seubert A et al (2009) Cadmium is a novel and independent risk factor for early atherosclerosis mechanisms and in vivo relevance. Arterioscler Thromb Vasc Biol 29:1392–1398. https://doi.org/10.1161/ATVBAHA.109.190082

Michielsen CCPPC, Van Loveren AH, Vos JG (1999) The role of the immune system in hexachlorobenzene-lnduced toxicity. Environ Health Perspect 107:783–792

Migliore L, Coppedè F (2009) Environmental-induced oxidative stress in neurodegenerative disorders and aging. Mutat Res Toxicol Environ Mutagen 674:73–84. https://doi.org/10.1016/j.mrgentox.2008.09.013

Milioni ALV, Nagy BV et al (2017) NeuroToxicology Full length article Neurotoxic impact of mercury on the central nervous system evaluated by neuropsychological tests and on theautonomic nervous system evaluated by dynamic pupillometry. Neurotoxicology 59:263–269. https://doi.org/10.1016/j.neuro.2016.04.010

Mohorovic L, Petrovic O, Haller H, Micovic V (2010) Pregnancy loss and maternal methemoglobin levels: an indirect explanation of the association of environmental toxics and their adverse effects on the mother and the fetus. Int J Environ Res Public Health 7:4203–4212. https://doi.org/10.3390/ijerph7124203

Molina JR, Yang P, Cassivi SD et al (2008) Non-small cell lung cancer: epidemiology, risk factors, treatment, and survivorship. Mayo Clin Proc 83(5):584–594

Mortimer K, Neugebauer R, Lurmann F et al (2008) Air pollution and pulmonary function in asthmatic children: effects of prenatal and lifetime exposures. Epidemiology 19:550–557

Mustafić H, Jabre P, Caussin C et al (2012) Main air pollutants and myocardial infarction: a systematic review and meta-analysis. JAMA 307:713–721

Næss Ø, Nafstad P, Aamodt G et al (2007) Relation between concentration of air pollution and cause-specific mortality: four-year exposures to nitrogen dioxide and particulate matter pollutants in 470 neighborhoods in Oslo, Norway. Am J Epidemiol 165:435–443. https://doi.org/10.1093/aje/kwk016

Nagel G, Stafoggia M, Pedersen M et al (2018) Air pollution and incidence of cancers of the stomach and the upper aerodigestive tract in the European Study of Cohorts for Air Pollution Effects (ESCAPE). Int J Cancer 143:1632–1643. https://doi.org/10.1002/ijc.31564

Nawrot TS, Thijs L, Den Hond EM et al (2002) An epidemiological re-appraisal of the association between blood pressure and blood lead: a meta-analysis. J Hum Hypertens 16:123–131. https://doi.org/10.1038/sj/jhh/1001300

Neidell MJ (2004) Air pollution, health, and socio-economic status: the effect of outdoor air quality on childhood asthma. J Health Econ 23(6):1209–1212

Ni L, Chuang C, Zuo L (2015) Fine particulate matter in acute exacerbation of COPD. Front Physiol 6:294. https://doi.org/10.3389/fphys.2015.00294

Nieuwenhuijsen MJ, Basagaña X, Dadvand P et al (2014) Air pollution and human fertility rates. Environ Int 70:9–14. https://doi.org/10.1016/j.envint.2014.05.005

Nikolić MD, Nikić AS (2008) Effects of air pollution on red blood cells in children. Polish J Environ Stud 17:267–271

Novey S, Wells ID, Tech BS (1983) Asthma and IgE antibodies induced by chromium and nickel salts. J Allergy Clin Immunol 72:407–412

Nyberg F, Gustavsson P, Ja L et al (2000) Urban air pollution and lung cancer in stockholm. Epidemiology 11:487–495

Oberdörster G, Utell MJ (2002) Ultrafine particles in the urban air: to the respiratory tract- and beyond? Environ Health Perspect 110(8):A440–A441

Oberdörster G, Oberdörster E, Oberdörster J (2005) Review nanotoxicology: an emerging discipline evolving from studies of ultrafine particles. Environ Health Perspect 113:823–839. https://doi.org/10.1289/ehp.7339

Opstelten JL, Beelen RMJ, Leenders M, Hoek G (2016) Exposure to ambient air pollution and the risk of inflammatory bowel disease: a European nested case – control study. Dig Dis Sci 61:2963–2971. https://doi.org/10.1007/s10620-016-4249-4

Pedersen EB, Jørgensen ME, Pedersen MB et al (2005) Blood and 24-h ambulatory blood. Am J Hypertens 18:612–618. https://doi.org/10.1016/j.amjhyper.2004.11.022

Pekkanen J, Brunner EJ, Anderson HR et al (2000) Daily concentrations of air pollution and plasma fibrinogen in London. Occup Environ Med 57:818–822

Penning TM (1993) Dihydrodiol dehydrogenase and its role in polycyclic aromatic hydrocarbon metabolism. Chem Biol Interact 89:1–34

Pereyra-Muñoz N, Rugerio-Vargas C, Angoa-Pérez M et al (2006) Oxidative damage in substantia nigra and striatum of rats chronically exposed to ozone. J Chem Neuroanat 31:114–123. https://doi.org/10.1016/j.jchemneu.2005.09.006

Perez-Padilla R, Schilmann A, Riojas-Rodriguez H (2010) Respiratory health effects of indoor air pollution. Int J Tuberc Lung Dis 14:1079–1086

Perin PM, Maluf M, Czeresnia CE, Januário DANF, Saldiva PHN (2010a) Impact of short-term preconceptional exposure to particulate air pollution on treatment outcome in couples undergoing in vitro fertilization and embryo transfer (IVF/ET). J Assist Reprod Genet 27(7):371–382

Perin PM, Maluf M, Czeresnia CE et al (2010b) Effects of exposure to high levels of particulate air pollution during the follicular phase of the conception cycle on pregnancy outcome in couples undergoing in vitro fertilization and embryo transfer. Fertil Steril 93(1):301–303

Peters A, Döring A, Wichmann H, Koenig W (1997) Increased plasma viscosity during an air pollution episode: a link to mortality? Lancet 349(349):1582–1587

Pirkle JL, Schwartz J, Landis JR et al (1985) The relationship between blood lead levels and blood pressure and its cardiovascular risk implications. Am J Epidemiol 121:246–258

Ponka A, Virtanen M (1996) Low-level air pollution and hospital admissions for cardiac and cerebrovascular diseases in Helsinki. Am J Public Health 86:1273–1280

Pope CAP III, Burnett RT, Thun MJ et al (2002) Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. JAMA 287:1132–1141

Pope CA III, Bhatnagar A, McCracken JP et al (2016) Exposure to fine particulate air pollution is associated with endothelial injury and systemic inflammation. Circ Res 119(11):1204–1214

Power MC, Weisskopf MG, Alexeeff SE et al (2011) Traffic-related air pollution and cognitive function in a cohort of older men. Environ Health Perspect 119:682–687. https://doi.org/10.1289/ehp.1002767

Puri P, Hospital S, Kumar SN (2017) Effects of air pollution on the skin: a review. Indian J Dermatol Venereol Leprol 83:415. https://doi.org/10.4103/0378-6323.199579

Raaschou-Nielsen O, Bak H, Sørensen M et al (2010) Air pollution from traffic and risk for lung cancer in three Danish cohorts. Cancer Epidemiol Prev Biomarkers 19:1284–1292. https://doi.org/10.1158/1055-9965.EPI-10-0036

Rahman M, Tondel M, Ahmad SA (1999) Hypertension and arsenic exposure in Bangladesh. Hypertens Arsen Expo Bangladesh Hypertens 33:74–78. https://doi.org/10.1161/01.HYP.33.1.74

Revis NW, Zinsmeister AR, Bull R (1981) Atherosclerosis and hypertension induction by lead and cadmium ions: an effect prevented by calcium ion. Proc Natl Acad Sci 78:6494–6498

Rezazadeh H, Athar M (1997) Evidence that iron-overload promotes induced skin tumorigenesis in mice. Redox Rep 3:303–309. https://doi.org/10.1080/13510002.1997.11747127

Ritz B, Yu F, Chapa G, Fruin S (2000) Effect of air pollution on preterm birth among children born in Southern California between 1989 and 1993. Epidemiology 11:502–511

Rivas-Arancibia S, Guevara-Guzma R, Lo Y et al (2010) Oxidative stress caused by Ozone exposure induces loss of brain repair in the hippocampus of adult rats. Toxicol Sci 113:187–197. https://doi.org/10.1093/toxsci/kfp252

Rivas-Arancibiaa S, Dorado-Martíneza C, Colin-Barenquea L et al (2003) Effect of acute ozone exposure on locomotor behavior and striatal function. Pharmacol Biochem Behav 74:891–900. https://doi.org/10.1016/S0091-3057(03)00011-X

Rokadia HK, Agarwal S (2013) Serum heavy metals and obstructive lung disease results from the national health and nutrition examination survey. Chest 143:388–397. https://doi.org/10.1378/chest.12-0595

Rousseau MC, Parent ME, Nadon L et al (2007) Occupational exposure to lead compounds and risk of cancer among men: a population-based case-control study. Am J Epidemiol 166:1005–1014. https://doi.org/10.1093/aje/kwm183

Rückerl R, Schneider A, Breitner S et al (2011) Health effects of particulate air pollution: a review of epidemiological evidence. Inhal Toxicol 23(10):555–592

Ruzzin J (2012) Public health concern behind the exposure to persistent organic pollutants and the risk of metabolic diseases. BMC Public Health 12(1):298. https://doi.org/10.1186/1471-2458-12-298

Salnikow K, Zhitkovich A (2008) Genetic and epigenetic mechanisms in metal carcinogenesis and cocarcinogenesis: nickel, arsenic and chromium. Chem Res Toxicol 21:28–44. https://doi.org/10.1021/tx700198a.Genetic

Salonen JT, Seppänen K, Nyyssönen K et al (1995) Intake of mercury from fish, lipid peroxidation, and the risk of myocardial infarction and coronary, cardiovascular, and any death in eastern Finnish men. Circulation 91

Salvi SS, Barnes PJ (2009) Chronic obstructive pulmonary disease in non-smokers. Lancet 374:733–743. https://doi.org/10.1016/S0140-6736(09)61303-9

Salvi S, Blomberg A, Rudell B et al (1999) Acute inflammatory responses in the airways and peripheral blood after short-term exposure to diesel exhaust in healthy human volunteers. Am J Respir Crit Care Med 159(3):702–709

Samoli E, Nastos PT, Paliatsos AG et al (2011) Acute effects of air pollution on pediatric asthma exacerbation: evidence of association and effect modification. Environ Res 111(3):418–424

Santibanez M, Alguacil J, Garcı M et al (2012) Occupational exposures and risk of stomach cancer by histological type. Occup Environ Med 69:268–275. https://doi.org/10.1136/oemed-2011-100071

Santucci D, Sorace A, Francia N et al (2006) Prolonged prenatal exposure to low-level ozone affects aggressive behaviour as well as NGF and BDNF levels in the central nervous system of CD-1 mice. Behav Brain Res 166:124–130. https://doi.org/10.1016/j.bbr.2005.07.032

Saxena P (2015) Seasonal variation of isoprene emissions from tropical roadside plant species and their possible role in deteoriating air quality. Environ Skeptics Critics 4(2):67

Saxena P, Ghosh C (2012) A review of assessment of benzene, toluene, ethylbenzene and xylene (BTEX) concentration in urban atmosphere of Delhi. Int J Phys Sci 7(6):850–860

Saxena P, Ghosh C (2015) A sustainable approach towards minimizing atmospheric benzene by the use of plants. Int J Agric Crop Sci 8(3):495–502

Saxena P, Ghosh C (2019) Establishing correlation between abiotic stress and isoprene emission of selected plant species. In: Emerging issues in ecology and environmental science. Springer, Cham, pp 43–65

Saxena P, Sonwani S (2019a) Primary criteria air pollutants: environmental health effects. In: Criteria air pollutants and their impact on environmental health. Springer, Singapore, pp 49–82

Saxena P, Sonwani S (2019b) Secondary criteria air pollutants: environmental health effects. In: Criteria air pollutants and their impact on environmental health. Springer, Singapore, pp 83–126

Schäfer T, Ring J (1997) Epidemiology of allergic diseases. Allergy 52:14–22

Schäfer T, Nienhaus A, Vieluf D et al (2001) Epidemiology of acne in the general population: the risk of smoking. Br J Dermatol 145(1):100–104

Schultz MG, Schröder S, Lyapina O, Cooper OR, Galbally I, Petropavlovskikh I, . . . Seguel RJ (2017) Tropospheric Ozone assessment report: database and metrics data of global surface ozone observations. Elementa: Science of the Anthropocene 5

Schwela D (2000) Air pollution and health in urban areas. Rev Environ Health 15:13–42

Seaton A, Godden D, MacNee W, Donaldson K (1995) Particulate air pollution and acute health effects. Lancet 345:176–178

Seltzer J, Bigby BG, Stulbarg M et al (1986) O3-induced change in bronchial reactivity to methacholine and airway inflammation in humans. J Appl Physiol 60:1321–1326

Sethi S, Murphy TF (2001) Bacterial infection in chronic obstructive pulmonary disease in 2000: a state-of-the-art review. Clin Microbiol Rev 14:336–363. https://doi.org/10.1128/CMR.14.2.336

Shah ASV, Langrish JP, Nair H et al (2013) Global association of air pollution and heart failure: a systematic review and meta-analysis. Lancet 382:1039–1048. https://doi.org/10.1016/S0140-6736(13)60898-3

Shrivastava D, Nayak B, Kant S, Panda J (2017) A review on health impacts of air pollutants with a special mention to Eastern Parts of India, a growing hub for diseases. Int J Earth Atmos Sci 4:21–33

Shulman JM, De Jager PL, Feany MB (2011) Parkinson's disease: genetics and pathogenesis. Annu Rev Pathol Mech Dis 6:193–224. https://doi.org/10.1146/annurev-pathol-011110-130242

Slama R, Bottagisi S, Solansky I, Lepeule J (2013) Short-term impact of atmospheric pollution on fecundability. Epidemiology 24:871–879. https://doi.org/10.1097/EDE.0b013e3182a702c5

Smith-Sivertsen T, Diaz E, Pope D et al (2009) Original contribution effect of reducing indoor air pollution on women' s respiratory symptoms and lung function: the respire randomized trial, Guatemala. Am J Epidemiol 170:211–220. https://doi.org/10.1093/aje/kwp100

Solenkova NV, Newman JD, Berger JS et al (2014) Metal pollutants and cardiovascular disease: mechanisms and consequences of exposure. Am Heart J 168:812–822. https://doi.org/10.1016/j.ahj.2014.07.007

Sonwani S, Kulshreshtha U (2016) Particulate matter levels and it's associated health risks in East Delhi. Proceedings of Indian aerosol science and technology association conference on aerosol and climate change: insight and challenges. IASTA Bull 22(1–2). ISSN 09714510

Sonwani S, Kulshrestha U (2018) Morphology, elemental composition and source identification of airborne particles in Delhi, India. J Indian Geophys Union 22(6):607–620

Sonwani S, Kulshrestha UC (2019) PM 10 carbonaceous aerosols and their real-time wet scavenging during monsoon and non-monsoon seasons at Delhi, India. J Atmos Chem 76:171–200

Sonwani S, Saxena P (2016) Identifying the sources of primary air pollutants and their impact on environmental health: a review. IJETR 6(2):111–130

Sonwani S, Amreen H, Khillare PS (2016) Polycyclic Aromatic Hydrocarbons (PAHs) in urban atmospheric particulate of NCR, Delhi, India. In 41st COSPAR Scientific Assembly (vol. 41)

Sood A (2012) Indoor fuel exposure and the lung in both developing and developed countries: an update. Clin Chest Med 33(4):649–665. https://doi.org/10.1016/j.ccm.2012.08.003

Sorace A, De Acetis L, Alleva E et al (2001) Prolonged exposure to low doses of ozone: short- and long-term changes in behavioral performance in mice. Environ Res 85:122–134. https://doi.org/10.1006/enrs.2000.4097

Squibb KS, Fowler BA (1981) Relationship between metal toxicity to subcellular systems and the carcinogenic response. Environ Health Perspect 40:181–188

Srám RJ, Binková B, Dejmek J, Bobak M (2005) Ambient air pollution and pregnancy outcomes: a review of the literature. Environ Health Perspect 113:375–382. https://doi.org/10.1289/ehp.6362

Steenland K, Boffetta P (2000) Lead and cancer in humans: where are we now? Am J Ind Med 38:295–299

Sunyer J (2008) The neurological effects of air pollution in children. Eur Respir Soc 32:535–537. https://doi.org/10.1183/09031936.00073708

Sunyer J, Spix C, Que P et al (1997) Urban air pollution and emergency admissions for asthma in four European cities: the APHEA Project. Thorax 52:760–765

Sze MA, Hogg JC, Sin DD (2014) Bacterial microbiome of lungs in COPD. Int J Chron Obstruct Pulmon Dis 9:229–238

Tchounwou PB, Yedjou CG, Patlolla AK, Sutton DJ (2012) Heavy metal toxicity and the environment. In: Luch A (ed) Molecular, clinical and environmental toxicology. experientia supplementum, vol 101. Springer, Basel

Tellez-plaza M, Navas-acien A, Crainiceanu CM et al (2008) Cadmium exposure and hypertension in the 1999–2004 National Health and Nutrition Examination Survey (NHANES). Environ Health Perspect 116:51–56. https://doi.org/10.1289/ehp.10764

Thiele JJ, Podda M, Packer L (1997) Tropospheric Ozone: an emerging environmental stress to skin. Biol Chem 378:1299–1305

Thurlbeck WM, Foley FD (1963) Experimental pulmonary emphysema: the effect of intratracheal injection of cadmium chloride solution in the guinea pig. Am J Pathol 42(4):431–441

Torre LA, Bray F, Siegel RL, Ferlay J (2015) Global cancer statistics, 2012. CA Cancer J Clin 65:87–108. https://doi.org/10.3322/caac.21262

Torres AD, Rai AN, Hardiek ML (2000) Mercury intoxication and arterial hypertension: report of two patients and review of the literature. Pediatrics 105:e34–e34

Torres-Duque C, Maldonado D, Pérez-Padilla R, Ezzati M, Viegi G (2008) Biomass fuels and respiratory diseases: a review of the evidence. Proc Am Thorac Soc 5(5):577–590. https://doi.org/10.1513/pats.200707-100RP

Tsai SS, Goggins WB, Chiu HF, Yang CY (2003) Evidence for an association between air pollution and daily stroke admissions in Kaohsiung, Taiwan. Stroke 34(11):2612–2616

Tuder RM, Mcgrath S, Neptune E (2003) The pathobiological mechanisms of emphysema models: What do they have in common? Pulm Pharmacol Ther 16:67–78. https://doi.org/10.1016/S1094-5539(02)00099-8

Türk YA, Kavraz M (2011) Air pollutants and its effects on human healthy: the case of the city of Trabzon. In Advanced topics in environmental health and air pollution case studies. InTech

Urch B, Silverman F, Corey P et al (2005) Acute blood pressure responses in healthy adults during controlled air pollution exposures. Environ Health Perspect 113:1052–1055. https://doi.org/10.1289/ehp.7785

Valacchi G, Sticozzi C, Pecorelli A et al (2012) Cutaneous responses to environmental stressors. Ann N Y Acad Sci 1271:75–81. https://doi.org/10.1111/j.1749-6632.2012.06724.x

Vaziri ND, Liang K, Ding Y (1999) Increased nitric oxide inactivation by reactive oxygen species in lead-induced hypertension. Kidney Int 56:1492–1498. https://doi.org/10.1046/j.1523-1755.1999.00670.x

Vierkotter A, Schikowski T, Ranft U et al (2010) Airborne particle exposure and extrinsic skin aging. J Invest Dermatol 130:2719–2726. https://doi.org/10.1038/jid.2010.204

Villeneuve PJ, Chen L, Stieb D, Rowe BH (2006) Associations between outdoor air pollution and emergency department visits for stroke in Edmonton, Canada. Eur J Epidemiol 21:689–700. https://doi.org/10.1007/s10654-006-9050-9

Wang L, Pinkerton KE (2007) Air pollutant effects on fetal and early postnatal development. Birth Defects Res C Embryo Today 81(3):144–154

Wang S, Shi X (2001) Molecular mechanisms of metal toxicity and carcinogenesis. Mol Cell Biochem 222:3–9

Wang Y, Tian Z, Zhu H, Cheng Z, Kang M, Luo C, Zhang G (2012) Polycyclic aromatic hydrocarbons (PAHs) in soils and vegetation near an e-waste recycling site in South China: concentration, distribution, source, and risk assessment. Sci Total Environ 439:187–193. https://doi.org/10.1016/j.scitotenv.2012.08.018

Wang S, Nan J, Shi C, et al (2015) Atmospheric ammonia and its impacts on regional air quality over the megacity of Shanghai, China. Nat Publ Gr 1–13. https://doi.org/10.1038/srep15842

Wellenius GA, Schwartz J, Mittleman MA (2005a) Air pollution and hospital admissions for ischemic and hemorrhagic stroke among medicare beneficiaries. Stroke 36:2549–2553. https://doi.org/10.1161/01.STR.0000189687.78760.47

Wellenius GA, Bateson TF, Mittleman MA et al (2005b) Particulate air pollution and the rate of hospitalization for congestive heart failure among medicare beneficiaries in Pittsburgh, Pennsylvania. Am J Epidemiol 161(11):1030–1036

Weuve J, Puett RC, Schwartz J et al (2012) Exposure to particulate air pollution and cognitive decline in older women. Arch Intern Med 172:219–227

Williams GR (1983) Presidential address: a history of appendicitis: with anecdotes illustrating its importance. Ann Surg 197(5):495

Williams NM, Jackson D, Everson NW et al (1998) Is the incidence of acute appendicitis really falling? Ann R Coll Surg Engl 80(2):122

Wlaschek M, Tantcheva-Poór I, Naderi L (2001) Solar UV irradiation and dermal photoaging. J Photochem Photobiol B Biol 63(1–3):41–51

World Health Organization (WHO) (2000) Air quality guidelines for Europe 2nd edition

World Health Organization (WHO) (2005) Indoor air pollution from solid fuels and risk of low birth weight and stillbirth

World Health Organization (WHO) (2010) Persistent organic pollutants: impact on child health

World Health Organization (WHO) (2014). https://www.who.int/mediacentre/news/releases/2014/air-pollution/en/

Wu K, Chang C, Yen C, Lai C (2019) Associations between environmental heavy metal exposure and childhood asthma: a population-based study. J Microbiol Immunol Infect 52(2):352–362. https://doi.org/10.1016/j.jmii.2018.08.001

Xu F, Yan S, Wu M et al (2011) Ambient ozone pollution as a risk factor for skin disorders. Br J Dermatol 165(1):224–225

Zeng X, Xu X, Zheng X et al (2016) Heavy metals in PM 2.5 and in blood, and children's respiratory symptoms and asthma from an e-waste recycling area. Environ Pollut 210:346–353. https://doi.org/10.1016/j.envpol.2016.01.025

Zhang JJ, Smith KR (2003) Indoor air pollution: a global health concern. Br Med Bull 68:209–225. https://doi.org/10.1093/bmb/ldg029

Zhang Y, Ji X, Ku T, Sang N (2015) Inflammatory response and endothelial dysfunction in the hearts of mice co-exposed. Environ Toxicol 31:1996–2005. https://doi.org/10.1002/tox

Zheng JJ, Zhu XS, Huangfu Z et al (2005) Crohn's disease in mainland China: a systematic analysis of 50 years of research. Chinese J Dig Dis 6(4):175–181

Zhou N, Fu Z, Sun T (2008) Neuroscience letters effects of different concentrations of oxygen ozone on rat's astrocytes in vitro. Neurosci Lett 441:178–182. https://doi.org/10.1016/j.neulet.2008.06.036

Chapter 8
Impacts of Air Pollution on Epidemiology and Cardiovascular Systems

Ram Raj Prasad and Sandeep Paudel

Abstract Our health is closely related to our environment, such that a healthy environment brings healthy living and vice versa. Pollution due to air is a prime environmental aspect contributing to the burden of different diseases in human and also has considerable economic impact. The total air pollution accounts approximately 7 million deaths globally. Pollutants produced as combustion of particulate matter have demonstrated a time-series effect on human health. The size of inhalable particulate matter (PM10 and PM2.5) affects the mortality and morbidity upon short- and long-term exposure among all population, with highest effect on elderly individuals. Exposure to these pollutants produces the pathological alteration, such as increased inflammatory response, systemic oxidative stress, cardiovascular stress, and change in pulmonary autonomous nervous system activity. These molecular pathological events trigger several pulmonary and cardiovascular manifestations in human. From epidemiology point of view, it has been explored that among different air pollutants, particulate matter, ozone, carbon monoxide, nitrogen dioxide and sulfur dioxide are the major ones. The highest mortality is mainly observed in Asian populations as compared to Europeans and Americans. The top ten countries with the highest mortality are China, India, Pakistan, Bangladesh, Nigeria, the United States, Russia, Brazil, and Philippines, respectively. In this chapter, we reviewed different PM exposure-based epidemiological studies with more focus on high ambient Total Suspended Particulate (TSP) levels. It has also been found that overall absolute risk for mortality due to PM exposure is higher for cardiovascular compared to pulmonary disorders in case of both acute and chronic exposures.

Keywords Air pollution · Particulate matter · Cardiovascular disease

---

R. R. Prasad (✉) · S. Paudel
Anschutz Medical Campus, University of Colorado, Denver, CO, USA

Jawaharlal Nehru University, New Delhi, India
e-mail: ram.prasad@cuanschutz.edu

P. Saxena, A. Srivastava (eds.), *Air Pollution and Environmental Health*,
Environmental Chemistry for a Sustainable World 20,
https://doi.org/10.1007/978-981-15-3481-2_8

8.1 Introduction

Human health is closely associated to the environment of its habitat and healthy habitat leads to healthy living (Seymour 2016). The damage done to the current environmental condition of our planet is quite noticeable. Climate change and air pollution due to different pollutants in the air is one of the major concerns all around the world. It has been estimated that a large number leading to premature deaths and adverse health effects are linked to air pollution. According to WHO reports, air pollution is the global, massive, single, and most significant environmental health risk. Health complications as a result of frequent exposure to air pollutants have high considerable economic impact, consequently increasing health care costs and premature deaths (Kelly and Fussell 2015). Pollutants that subsidize to air pollution can be divided into primary and secondary. Primary air pollutants are those which have their own origin such as carbon monoxide, nitrogen oxides, volatile organic compounds (VOC), sulfur dioxide, and particulate matter. Secondary air pollutants are those which are the outcome of primary air pollutants such as ozone and smog (Admassu et al. 2005).

Due to a drastic increase in urbanization and industrialization all over the world, there is a steep rise in the concentrations of various air pollutants. This has led to adverse impact on human existence in various forms like climate change, global warming, and flooding (Admassu et al. 2004). Inefficient pollution control methods, inadequacy of regulations enforcement, uncontrolled vehicular emissions, and increasing deforestation are components responsible for rising environmental air pollutants (McCarthy et al. 2009; Aggarwal and Jain 2015). Inhalation of fine particles causes severe lung disorders due to its high ambient TSP levels along with elevated levels of particulate matters (PM) PM10 and PM2.5 (Carrico et al. 2003; Begum et al. 2008). The most significant contributor of particulate matter is mainly vehicle exhaust (Agarwal et al. 2006). In most of the developing countries and cities, a distinct population of particulate matter is mostly found that are having deleterious impact on different categories of populations. The effect of particulate matter on human health can only be reduced when there is sharp decline in vehicular or construction-based activities (World Bank 2004). The adverse health impact of PM is directly proportional to its size, where PM2.5 and PM0.1 have the most hazardous impact as compared to PM10 (Dockery et al. 1993; Pope et al. 2002). Premature death from heart and lung disease, chronic bronchitis, asthma attacks and respiratory illness are topmost health effects of PM (Smith 2002). Various epidemiological studies have estimated the possible health impacts resulting from different categories of particulate matter locally, regionally, and globally. World health report (2016) also states that ambient particulate matter pollution is capable of 1.4% total annual global mortality with an estimated death of about 0.8 million people.

8.2 Air Pollution and Human Health

Air pollution is known as contagion mixture of both indoor and outdoor surroundings caused by any chemical, biological, or physical agents which tend to alter the air quality (Ji et al. 2017). The substances, either natural or anthropogenic, that contaminate the environment are known as pollutant. Air pollution could be categorized as natural or man-made and stationary or mobile on the basis of source of emissions. Natural sources include volcanic eruption, fires, storm, fog, and mist while man-made sources include vehicular exhaust, industrial emissions, crop residue burning, biomass burning, mining, and domestic activities (Barnes et al. 1999). On the basis of origin, air pollution sources can be of different types like point, area, and line. Point sources are those which have single known source of pollution from which pollutants are discharged such as factory smokestack, pipe, and ditch. Area sources are those sources of pollution which emit a substance from a particular area, for example, open burning and forest fires and evaporation losses from large spills of volatile liquids. Line sources are idealized geometric emitter that can be represented by an emission source consisting of straight line which may be of finite or infinite length, for example, roadway air pollution, aircraft emissions, noise pollution, etc. In 2005, WHO released "WHO Air quality guidelines" that has set up various permissible limits for different air pollutants that directly impact the human health. These guidelines help to reduce air pollutants to a certain level by regular monitoring and evaluation of their concentrations in the air. With the implementation of PM10 guidelines by WHO, there is significant improvement in its concentration from 70 to 20 μg/m^3 and subsequently mortality rates were reduced to approximately 15%. This guide code applied worldwide is established through expert appraisal of scientific evidence for particulate matter (PM): limit for PM2.5: 10 μg/m^3 annual mean; 25 μg/m^3 24-h mean, limits for PM10: 20 μg/m^3 annual mean; 50 μg/m^3 24-h mean (WHO 2016). Comprehensive infinite risk for mortality due to PM vulnerability is greater for cardiovascular disease (CDV) compared to pulmonary diseases in respect to short- as well as long-term exposures. Even at identical acute relative risk elevations estimated between cardiovascular and pulmonary mortality, CVD account rises by 69%.

Air pollution is regarded as one of the dominant killers worldwide, responsible for 7 million deaths per year, out of which 600,000 are children (Yang et al. 2017). WHO reported that around 300 million children have been affected due to outdoor air pollution at global level. Air pollution is also spreading at a very higher rate in both urban and rural areas, mainly on low and middle income countries. South East Asia and Western Pacific regions had population-based burden of 3.3 million and 2.6 million death toll rate due to indoor and outdoor pollution, respectively (WHO 2012) (Table 8.1). Stronger link between both indoor and outdoor air pollution is associated with increased cardiovascular disease (Endes et al. 2017; Kelly and Fussell 2017; Bhatia et al. 2017), hypertension, diabetes (Vora et al. 2014), rheumatoid arthritis (De Roos et al. 2014), pulmonary diseases like asthma, lung cancer (Dominici et al. 2006; Machado et al. 2014; Bai et al. 2018) oxidative stress, physical

Table 8.1 Air pollution-related diseases and its respective death percentage count

Air pollution	Related disease	Death (%)
Outdoor (2.6 million deaths)	Ischemic heart disease	40.0
	Stroke	40.0
	Chronic obstructive pulmonary disease (COPD)	11.0
	Lung cancer	6.0
	Acute lower respiratory infections in children	3.0
Indoor (3.3million deaths)	Ischemic heart disease	26.0
	Stock	34.0
	Chronic obstructive pulmonary disease (COPD)	22.0
	Lung cancer	6.0
	Acute lower respiratory infections in children	12.0

Table 8.2 Major emission estimates and its source categories

Category	Air pollutants	Ambient concentration	Source
Particulate matter (PM)	Coarse (PM10)	150 μg/m^3	Area sources (agricultural tilling, dry cleaners, open burning, wildfires) and non-electricity generating units—EGUs (boilers, cement kilns, process heaters, turbines)
	Fine (PM2.5)	65 μg/m^3	Area sources (agricultural tilling, dry cleaners, open burning, wildfires) and non-electricity generating units—EGUs (boilers, cement kilns, process heaters, turbines)
Gases matter (GS)	NO_2	100 μg/m^3 (0.05 ppm)	Area sources, non-electricity generating units (Non-EGUs), EGUs (electricity producing utilities), on-road motor vehicles (buses, cars, trucks) and non-road vehicles and engines (aircraft, construction equipment, locomotives, marine engines)
	O_3	160 μg/m^3 (0.08 ppm)	Ultraviolet light transformed molecules, NO_x, on-road motor vehicles and non-road vehicles emitted
	SO_2	365 μg/m^3 (0.14 ppm)	Non-electricity generating units (Non-EGUs) and electricity generating units (EGUs)
	CO	10 μg/m^3 (9 ppm)	On-road motor vehicles (buses, cars, trucks) and non-road vehicles and engines (aircraft, construction equipment, locomotives, marine engines)

disability (Weuve et al. 2016) and inflammation (Møller et al. 2014). Among all the air pollution-associated diseases, cardiovascular tops the chart with the highest incidence of 69% death toll rate. WHO also suggested that the concentrations of particulate matter can be reduced by adopting right policy measures due to which 15% of the lives can be saved at worldwide level.

Among different air pollutants, PM, ozone (O_3), carbon monoxide (CO), nitrogen dioxide (NO_2), and sulfur dioxide (SO_2) are the major ones (Fröhlich 2017) (Table 8.2). The total air pollution including particulate matter (PM), O_3, CO,

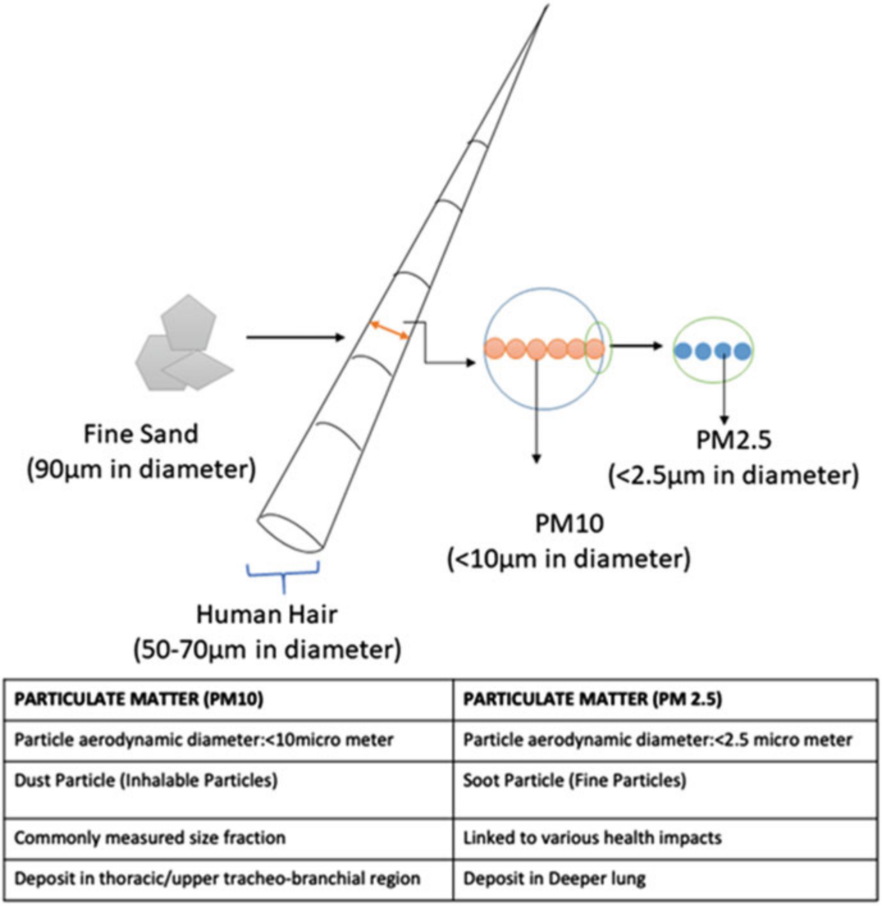
SO_2, NO_x, and VOCs accounts for loss of approximately 7 million lives globally. The highest mortality is mainly in Asian countries. Top ten countries with the highest mortality rate due to air pollution are China followed by India, Pakistan, Bangladesh, Nigeria, the United States, Russia, Brazil, and Philippines, respectively.

8.2.1 Particulate Matter (PM)

Particulate matter (PM) is the mixture of solid and liquid droplets suspended in the air and differentiated on the basis of different particle sizes (Manigrasso et al. 2017; Olatunji et al. 2018; Voidazan et al. 2018). PM not only evolves from natural sources such as volcanic eruptions but also through human actions such as burning of fossil fuels, waste disposal, and metal extraction activities. Both natural and artificial sources may emit either precursors like SO_2, NO_2, VOCs along with favorable meteorological conditions forming PM or can directly emit in the atmosphere through vehicular exhaust and industrial emissions (Olopade et al. 2017; Rahman et al. 2017). Coarse dust particles (PM10) are those having particle size of less than or equal to 10 μm in diameter and are inhalable particles (Fig. 8.1). The main sources for coarse dust particles are large-scale crushing or grinding activities during construction and suspension of road dust due to vehicular activities. It can deposit in thoracic or upper trachea-bronchial region causing severe health problems (Yin et al. 2017). Fine particles (PM2.5) are those having aerodynamic diameter size of 2.5 μm or smaller which are generally invisible and can only be observed using high-resolution microscopes. The prime source includes various types of combustion, motor vehicles, wood burning, and industrial processes (You et al. 2017b) as shown in Fig. 8.1. These are soot particles and are linked to various health impacts and are deposited in deeper lungs.

(a) Coarse Particles (PM10)

Coarse particles are one of the primary air pollutants (solid and liquid particles floating) that are respirable and thus can penetrate deep into the respiratory system. These PM are 5–7 times smaller in diameter compared to human hair (50–70 μm diameter) (Fig. 8.1). The particular matter composition, shape, size, presence of additional pollutants, and metrological factors determine its toxicity and concentration (Patton et al. 2014; Kelly and Fussell 2015). PM10 is one of the significant air pollutants which is the biggest threat to all living organisms. When compared from the era of industrialization to the present time, coarse particles have become a significant air pollutant in urban, suburban, rural, and remote parts of the globe (Fang et al. 2013; Li et al. 2015; Yang et al. 2019). In urban cities of most countries, PM10 level is above the WHO and their respective countries standard (WHO 2016). PM10 lead to inflammatory responses, congenital heart failure, ischemic heart disease, respiratory and circulatory fatality, birth risk, and cancer risk (Hemminki and Pershagen 1994; Zhang et al. 2014; Agay-Shay et al. 2013; Silbajoris et al. 2011). Larger particular matter of and around 10 μm endures solely in the nose and



PARTICULATE MATTER (PM10)	PARTICULATE MATTER (PM 2.5)
Particle aerodynamic diameter:<10micro meter	Particle aerodynamic diameter:<2.5 micro meter
Dust Particle (Inhalable Particles)	Soot Particle (Fine Particles)
Commonly measured size fraction	Linked to various health impacts
Deposit in thoracic/upper tracheo-branchial region	Deposit in Deeper lung

Fig. 8.1 Particulate matter size classification: Comparative analysis of PM10 and PM2.5. Size determines the behavior of atmospheric particle, where PM10 has quintuple diameter to hair and PM2.5 quaternary smaller than PM10

throat part and smaller particles of and around 1 μm easily accumulate in the lower regions of the lungs. The intermediate sizes greater than 1 μm and less than 10 μm reside between the upper part of the respiratory tract. Setiawan et al. (2014) reported the positive strong correlation between higher concentrations of PM10 and their harmful impacts on human beings. As per the global data statistics, prominent higher concentration of PM10 is reported in Asia, followed by Africa and European countries. Moreover, according to 2016 data, top ten polluted countries around the globe are Cameron, Egypt, Mauritania, Niger, Nigeria, Pakistan, Qatar, Saudi Arabia, Tajikistan, and United Arab Emirates, respectively, as shown in Fig. 8.2.

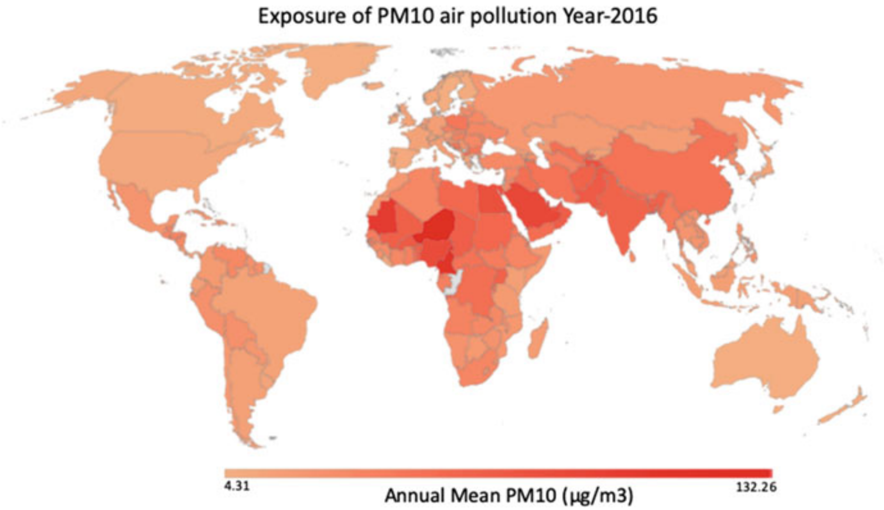


Fig. 8.2 Exposure to PM10 air pollution (2016): World Map showing annual mean exposure of population weighted exposure to ambient PM10 pollution throughout the globe on country basis

(b) Fine Particles (PM2.5)

Very tiny particles in the atmosphere of and around 2.5 mm wide are denoted as fine particles or PM2.5. These particles originate from dust, dirt, soot, and smoke, very tiny enough and are easily inhaled (Luo et al. 2018). According to Liu et al. (2018), it has been found that PM2.5 have shown stronger correlation with respect to their size range and human health effects. Infants and elderly population are at highest risk due to the inhalation of PM2.5 particles and most susceptible to pulmonary and heart diseases. Vulnerability to fine particulate matter (2.5 μm aerodynamic diameter) contributes to a predicted loss of 915,900 lives in China alone each year. Among these number of deaths, around 15% are attributable to PM2.5 due to transportation activities (Arashiro et al. 2018; Luyts et al. 2018). Severe health risks are associated with ultrafine particles (<0.1 μm) and composing PM2.5 and their main source is vehicular exhaust (Li et al. 2017; Hou et al. 2018; Kim et al. 2018; Kumar et al. 2018). Ultrafine particles can travel extensively deep into the lungs (Baldauf et al. 2016; Fonceca et al. 2018; Boogaard and van Erp 2019) as well as associated with the biomarkers of cardiovascular diseases leading to DNA hypomethylation and mortality (Atkinson et al. 2016; Kim et al. 2016; Simonetti et al. 2018; He et al. 2018; Louwies et al. 2018). PM2.5 components such as black carbon (BC) also have been associated with cardiorespiratory impacts (Donaldson et al. 2001; He et al. 2018). Since research on UFP and BC is not sufficient compared to PM as a whole, their effects are usually not included in mortality estimates (You et al. 2017a; Vreeland et al. 2017).

The annual risk of PM2.5 is increasing with years resulting in 3.0 million deaths (5.2% of global deaths in 2016). More than 50% occur in China and India. Comparative analysis showed that PM2.5 attributed to the increase in deaths by 68% from

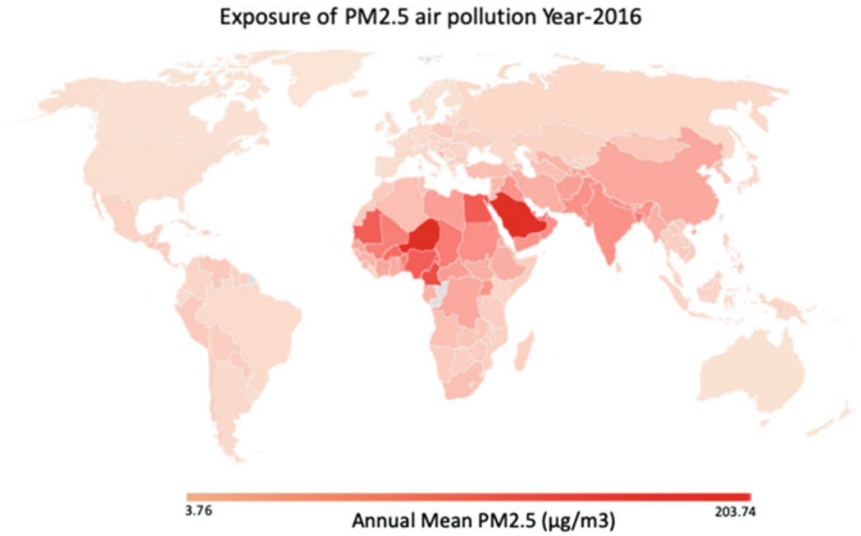


Fig. 8.3 Exposure to PM2.5 air pollution (2016): World Map showing annual mean exposure to PM2.5 pollution throughout the globe on country basis

1990 to 2016. World data clearly indicates that prominent higher concentration of PM10 is found in Asian followed by African and European countries. According to 2016 data, top ten polluted countries around the globe are Burkina Faso, Cameron, Egypt, Kuwait, Mauritania, Niger, Nigeria, Qatar, Saudi Arabia, and United Arab Emirates, respectively, as shown in Fig. 8.3.

The nature of particulate matter in the atmosphere determines the entry, absorption potential, and deposition of particles in the lungs (Clark et al. 2010). Particles which are larger than PM10 never reach the lungs as they get trapped within the nose and throat. Particles ranging from 10 µm in diameter or less become the most burdensome to the human health. Particles size smaller than 5 µm can be transported to bronchial tubes, while particles size of 2.5 µm or smaller can reside into the lung's deepest portion (Prospero 1999; Mahowald et al. 2014).

8.2.2 *Ozone (O_3)*

Ozone is the important component of photochemical smog and one of the greenhouse gases (Shi et al. 2016; Tham et al. 2017). It is a highly reactive, oxidative gas responsible for severe lung diseases, cardio-attacks and strokes all around the globe (Nuvolone et al. 2018). Ozone concentrations are mostly higher in summer season due to its photochemical nature and favorable meteorological conditions like high temperature and high solar radiation. Ozone is a secondary pollutant which is formed by its precursors like nitrogen oxides, carbon monoxide, and volatile organic compounds present in the atmosphere (Kan et al. 2012). Moreover, when hydroxyl radical (OH) reacts with trace gases, it leads to high concentration of ozone and hence becomes responsible for global warming. The increasing O_3 level gives rise to serious health outcomes like inflammation, respiratory injury, and decreased lung activity (Kinney 1999; Koken et al. 2003).

8.2.3 Carbon Monoxide (CO)

CO is an outcome of carbon-containing fuels formed by mechanism of incomplete combustion (Yang et al. 2016). CO has the property to readily combine with hemoglobin due to its high affinity (200–250 times) as compared to oxygen and forms carboxyhemoglobin (COHb) (Gorman et al. 2003). When concentrations of COHb increases rapidly, oxygen level also sharply decreases and leads to morbidity and mortality (Graber et al. 2007; Pissuwan et al. 2016). CO is regarded as the most toxic inhalable gas which is both outdoor as well as indoor air pollutant and leads to sudden illness and death (Von Burg 1999). CO arises mainly from oxidation of fossil fuels like coal, kerosene, wood, and natural gases. Various forms of tobacco smoke and automobile exhaust also contain large concentration of CO. The level of CO ranges from 0.5 to 100 parts per million (ppm) both in indoor and outdoor environment. The standard range of indoor CO levels ranges from 0.5 to 5 ppm. This value exceeds the standard range in the presence of tobacco smoke in environment, incompetent heating, and ventilation up to 100 ppm (Penney et al. 2010), whereas, in urban zones, levels are typically from 20 to 40 ppm. In the periphery around highways and heavy construction site, the range of CO becomes much higher (Raub 1999). As it reaches to peak or exceeds standard range, the chance of CO poisoning initially shows the acute symptoms of headache, dizziness, and shortness of breath (Gorman et al. 2003; Piantadosi 1999). Further increase in exposure of level from acute to chronic leads to neurotoxicity, unconsciousness, cognitive and visual impairment and ultimately death. Research around years have determined chronic CO exposure as a risk factor for cardiovascular disease (Samoli et al. 2007). The underlining mechanisms for cardiotoxicity are associated with chronic CO risk identified to be directly or indirectly associated with activation and modulation of various intracellular signaling mechanisms. These mechanisms probably include CO-dependent regulation of cell proliferation, lipid-raft associated signaling protein caveolin-1, and modulation of NADPH oxidase (Taillé et al. 2005).

8.2.4 Nitrogen Oxides

The primary source of NO is fossil fuel combustion in motor vehicles and industrial emissions particularly from power generation plants. NO has the ability to react with ammonia, moisture, and other compounds to give rise to small particles (Ibeneme et al. 2016). These tiny particles can aggravate existing severe cardiovascular disease and asthma (Latza et al. 2009; Zhang et al. 2016). NO_2 is a one of the criteria pollutant, which is the outcome of lightning, volcanic eruption, oxidation of NO to NO_2, power houses, motor vehicles, bacterial source and combustion of fossil fuels (more than 70% of NO_2 Production) (WHO). NO_2 can affect human health mostly at higher or even at low concentrations. Epidemiological studies show that atmospheric NO_2 attributes about 5–7% lungs cancer disease in both smokers and nonsmokers (Goudarzi et al. 2012; Omidi et al. 2016). It has severe acute effects followed by chronic effects like pulmonary as well as chronic obstructive pulmonary diseases (COPD), infant mortality chronic cough, bronchitis, infant mortality, and visibility (Barnett et al. 2005). According to WHO, the annual mean value of NO_2 is 40 $\mu g/m^3$ with respect to human health; however, even short-term exposure to NO_2 leads to cardiovascular-related risks to exposed population (Brook 2008).

8.2.5 Sulfur Dioxide (SO_2)

The main source of SO_2 is from combustion of sulfur-containing fuels, especially in power plants and diesel engines, vehicular exhaust, and fossil fuel combustion (Silva et al. 2017). Sulfur dioxide majorly produces cytotoxic effect on retinal pigment of epithelial cells (Rall 1974; Bose et al. 2016). Short-term effect of SO_2 on human health results in respiratory diseases in children (asthma, mainly in winter) (Pikhart et al. 2001; Heinrich et al. 2002) and registered a large number of both morbidity and mortality cases (Katsouyanni et al. 1997). SO_2 leads to respiratory aggravation, bronchoconstriction, and most importantly cardiovascular disorders. Initial phase of cardiovascular abnormalities includes reduced heart rate vulnerability (Tunnicliffe et al. 2001). SO_2 also causes respiratory symptoms such as shortness of breath, increased risk of asthma, and respiratory mortality (Clark et al. 2010; Chen et al. 2007; Zhao et al. 2008).

8.3 Air Pollution-Induced Cardiovascular Index and Their Consequences to Morbidity and Mortality

Pope and Dockery have highlighted the relationship between cardiovascular manifestation and air pollution. Their findings suggest that short-term daily exposure to particulate matter shows higher risk of cardiovascular-related mortality than all other diseases (Pope and Dockery 2006). Acute PM exposure contributes to 69% CVD-related deaths and 28% pulmonary-related manifestation (Brook et al. 2010). Some more critical studies have also been highlighted and give the addition prospective on exposure time and airborne pathogenesis.

Short-Term Exposure Shorter exposure with a high level of pollutants is proportionally liked to the CVD-associated mortality. Increase in PM10 quantity by 10 μg/m^3 promotes 1–2% surge in cardiovascular and pulmonary disease cases (Schwartz 2001). However, elevated exposure to PM2.5 by 10 μg/m^3 increases the risk of myocardial infraction around 2.8%. Particulate matter-based morbidity and mortality are independent of other gaseous co-pollutants (i.e., NO_2, SO_2, O_3, and CO). Acute exposure to O_3 causes cardiopulmonary defect in middle-age person with no previous CVD (Bell et al. 2007). 1.0 μg/m^3 increase of CO leads to 1.25% cardiovascular-related deaths, and short-term exposure to NO_2 also promotes CVD pathogenesis (Samoli et al. 2006, 2007). The particulate matter-associated CVD risks appeared to be more in elderly person, while the younger population are more susceptible to NO_2 exposure (Argacha et al. 2016).

Long-Term/Chronic Exposure High PM level with prolonged exposure shows a greater magnitude than acute exposure. World Health Organization highlighted that constant exposure to PM2.5 leads to 800 thousand premature deaths per year and it ranked as the 13th leading cause of worldwide death (WHO 2002). Several other reports also suggest a strong association between chronic exposure to PM and cardiovascular mortality (Pope et al. 2004a). The annual increase of PM10 by 10 μg/m^3 and PM2.5 by 5 μg/m^3 promotes the risk of myocardial infarction by 10–13% (Cesaroni et al. 2014). Long-term exposure to PM2.5 causes premature arteriosclerosis and with traffic-related pollutants, it promotes coronary artery calcification (Kaufman et al. 2016). Chronic effect of airborne contaminants on mortality is examined in cohort studies. These studies involve exposure of pollutants and a large number of volunteers, and provide the data related to the impact of the pollutants on human life span (Künzli et al. 2001).

Air pollutants cause considerable alterations in cardiovascular indexes like heart rate, heart rate variability, arrhythmia, heart failure, cardiac arrest, ischemic heart disease, cerebrovascular disease (Stroke), blood pressure, vascular tone atherosclerosis, thrombosis, and coagulation (Table 8.3). These air pollution-induced cardiovascular indexes affect the viability of human life and increase mortality.

8.3.1 Heart Rate (HR) and Heart Rate Variability (HRV)

The increase of PM2.5 by 15.5 μg/m^3 reduces the heart rate in old-aged people, while PM10 exposure (100 μg/m^3) increases the heart rate by 5–10 beats per minute (POPE et al. 1999; Gold et al. 2000). Heart rate viability is strongly associated with the rate of exposure to particulate matter in both acute and chronic condition. Effect on HRV is correlated with the presence of nickel and iron in airborne particulate matter (Chang et al. 2007).

Table 8.3 Air pollution components and cardiovascular manifestation

Air pollution components	Cardiovascular effect	Short-term exposure	Long-term exposure
Particulate matter	Ischemic heart disease	↑↑↑	↑↑↑
	Heart failure	↑↑	↑
	Cerebrovascular disease/stroke	↑↑	↑
	Thrombosis and coagulation	↑↑	↑
	Blood pressure	↑↑	–
	Vascular tone	↑	↑↑
	Atherosclerosis	–	↑↑↑
	Hypertension	↑	↑↑
Ozone	Stroke	↑↑	↑
	Cardiac arrest	↑	↑
	Hypertension	↑	–
Nitrogen oxides	Heart failure	↑↑	↑
	Transient ischemic attack and stroke	↑↑	↑
	Myocardial infraction risk	↑↑	↑
Carbon monoxide	Heart failure	↑↑	↑
	Cardiac arrest	↑	↑
	Myocardial infraction risk	↑↑	↑
Sulfur oxides	Heart failure	↑↑	↑
	Myocardial infraction risk	↑↑	↑

Arrow indicates epidemiological evidence of cardiovascular consequences on exposure to different air pollutants. ↑↑↑ strong, ↑↑ moderate, ↑ weak evidences

8.3.2 *Arrhythmia, Heart Failure, and Cardiac Arrest*

Cohort studies exhibit that arrhythmia, heart failure, and cardiac arrest are associated with increased exposure to PM2.5. Rise of PM2.5 by 10 $\mu g/m^3$ increases heart failure cases by 1.28% (Dominici et al. 2006). Air pollutants also increase ventricular size and myocardial fibrogenesis that are the causes of cardiac arrhythmia and arrest (Wold et al. 2012).

8.3.3 *Ischemic Heart Disease*

Several studies have shown the relation between increased level of PM2.5 and ischemic heart disease. Coronary heart disease is a major cause of cardiovascular-related mortality and increase of PM2.5 by 10 $\mu g/m^3$ promotes coronary heart disease cases (Pope et al. 2004a). PM2.5 along with PM10, NO_2, and O_3 are key causative factors for myocardial infarction, which is the first manifestation of ischemic heart disease. Patients aged 75 years or above acquire more ST-elevated myocardial infraction on exposure to PM10. However, younger population are more susceptible to fine and ultrafine particulate matter and NO_2 exposure (Argacha 2017).

8.3.4 Thrombosis and Coagulation

Exposure of air pollutants to the cardiovascular system promotes hypercoagulability of blood. This physiological effect is either due to the entry of particulate matter into bloodstream or release of PM-induced circulating factors (Brook et al. 2010). Recent study reveals that exposure of concentrated ambient particulates (CAPs) to mice activates its pulmonary autonomous nervous system (ANS), which further stimulates the inflammatory responses and release of cytokines from alveolar macrophages to disseminate prothrombotic state (Chiarella et al. 2014).

8.3.5 Cerebrovascular Disease/Stroke

Data of 188 countries between 1990 and 2013 from Global Burden of Disease database shows 29% of air pollutant-induced cardiac manifestation are contributed only by stroke (Feigin et al. 2016). Many studies have found a positive association between air pollution, stroke, and mortality. Annual increase of air pollution by 5 μg/m^3 enhances the risk of stroke up to 19% (Stafoggia et al. 2014). Time-series experiment has highlighted that increased air pollutants (notably PM, NO_2, CO, and O_3) level is associated with cerebrovascular disease. Air pollution-induced cardiac stroke has good correlation with ischemic, but not exactly with hemorrhagic stroke (Hong et al. 2002). Similar studies have also revealed that PM2.5 and CO-induced stroke mortality is predominant in warm season than the cold season (Kettunen et al. 2007). The short-term elevated level of the gaseous pollutants like O_3, NO_2, and CO also promotes the risk of stroke (Henrotin et al. 2010).

8.3.6 Blood Pressure, Vascular Tone, and Atherosclerosis

Increase exposure of PM2.5 by 10.5 μg/m^3 enhances the diastolic pressure from 2.7 to 2.8 Hg in cardiac patients, and further increase of PM2.5 by 13.9 μg/m^3 enhances diastolic pressure by 6.95 Hg without affecting systolic pressure (Zanobetti et al. 2004). Exposure to concentrated ambient particulates (CAPs) by 150 μg/m^3 along with O_3 (120 parts/billion) causes significant constriction of bronchial artery (up to 0.09 mm) (Brook et al. 2002). Higher exposure to PM2.5 and carbon particulates is correlated with increase in vascular constriction (O'Neill et al. 2005). However, chronic exposure to the fine and ultrafine particulate matter causes severe atherosclerosis. In human, 10 μg/m^3 of PM2.5 promotes the carotid artery thickness by 5.9% (Suwa et al. 2002; Künzli et al. 2005). Long-term exposure to traffic-related PM2.5 (22.8 μg/m^3) also promotes coronary artery calcification.

8.3.7 Hypertension

Long-term exposure to PM promotes the vascular oxidative stress, activation of ANS, inflammatory responses, increased sensitivity, and release of angiotensin II and endothelin which in turn develop systemic hypertension in animal (Brook et al. 2010; Ying et al. 2014). Controlled acute exposure to the concentrated ambient particles with ozone causes diastolic hypertension due to ANS imbalance (Urch et al. 2005).

8.4 Mechanism of Cardiovascular Toxicity Induced by Air Pollutants

The number of epidemiological studies strongly shows that air pollutants exposure causes a significant impact on the cardiovascular system. Association between cardiovascular complication and air pollution is now supported and asserted by the vast number of studies; however, the exact causative factors of pollutants and the molecular physiology by which airborne pollutants trigger cardiovascular abnormalities are understood to only some extent. Air pollutants which cause cardiovascular stress are gaseous and particulate in nature. Fine (PM2.5) and ultrafine (PM0.1) particulate matter are major components for manifestation of cardiovascular disease (Brook et al. 2010). The acute exposure to air pollutants leads to the development of myocardial infarction and change in heart viability, heart rate, blood pressure, blood coagulability, and vascular tone while prolonging exposure (chronic exposure) disseminates the progression of atherosclerosis (Brook et al. 2010).

Accumulating a number of evidences show that PM causes the chemical modification of oligonucleotides, resulting in the genetic and epigenetic changes which lead to aberrant genomic expression. Along with this it also induces oxidative stress and abnormal secretion of the stress-related hormone. To explain the mechanism of airborne pollutants-induced pathogenesis, three notable hypotheses were proposed, which explain the association between cardiovascular disease and air pollutants exposure (Fig. 8.4):

PM-induced pulmonary inflammatory responses promote oxidative and cardiovascular stress.

Exposure to PM leads to autonomic nervous system (ANS) imbalance and pathological alterations.

Inhaled air pollutants directly translocate in the circulatory system and perturb the distant cell and tissue components of the cardiovascular system.

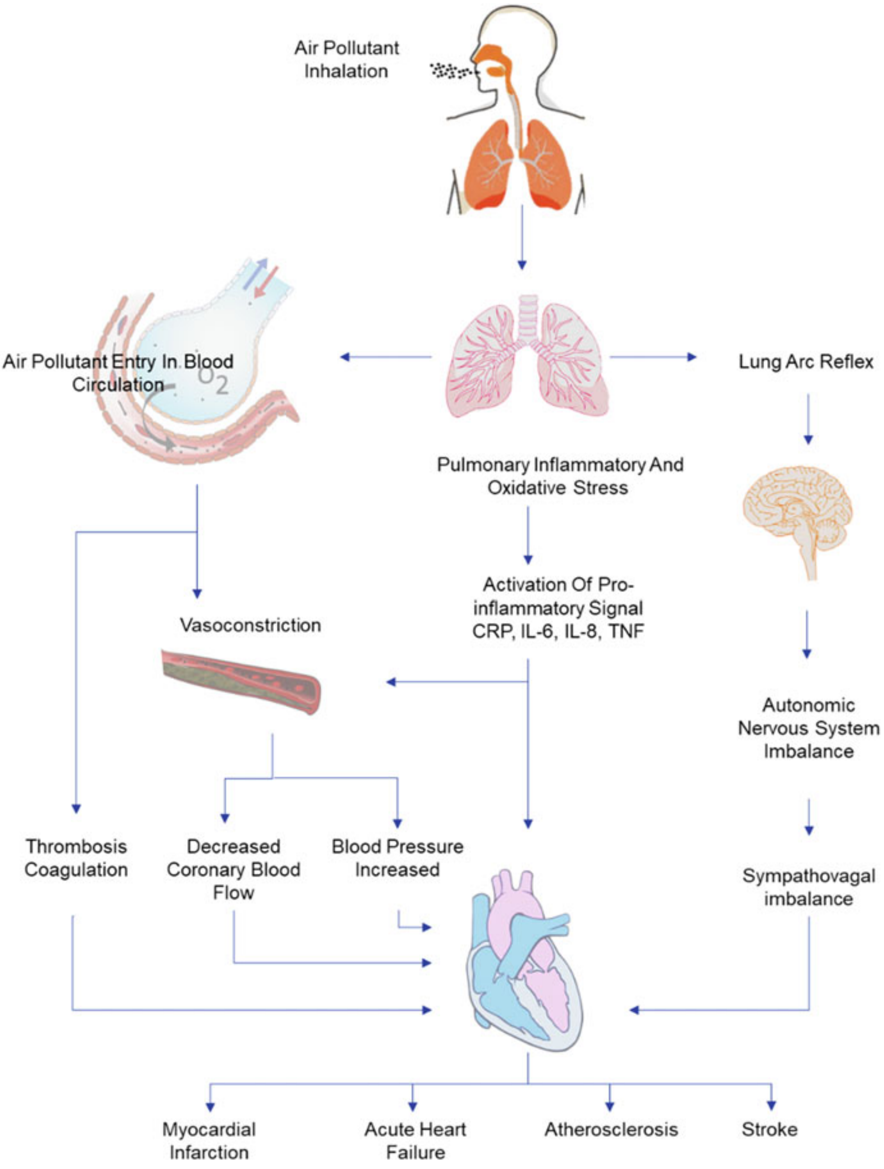


Fig. 8.4 Pathological mechanism of cardiovascular toxicity associated with increasing air pollutants

8.4.1 *PM-induced Pulmonary Inflammatory Responses Promote Oxidative and Cardiovascular Stress*

Air pollutants initiate a number of inflammatory reactions as soon as they come in contact with the lungs (GHIO et al. 2000). Particulate matter is a mixture of a wide range of particles (0.1–10 μm); these particles are typically composed of combustion originated carbon center, peripheral metal, and hydrocarbon. Larger and fine particulate matter (PM10 and PM2.5) are the major cause of pulmonary stress. PM induces the oxidative stress, inflammation, and pulmonary cell toxicity in the lungs (Donaldson and Stone 2003). Administration of aqueous PM induces the generation of reactive oxygen species (ROS), which increases bronchoalveolar localization of macrophages. Alveolar-localized macrophages release cytokines, interleukins, and TNF and start the inflammatory response. These PM also increase the bronchoalveolar lavage alpha-1 antitrypsin and fibronectin level that are the symptoms of pulmonary necrosis (GHIO and DEVLIN 2001). Prolonged exposure to polycyclic aromatic hydrocarbon-related PM2.5 rises the level of 8-hydroxy-2-deoxyguanosine and etheno-DNA adducts, which are the biomarker for oxidative DNA damage. These DNA adducts are predominant in road traffic workers (Bagryantseva et al. 2010). Air pollutant-induced elevated oxidize plasma LDL, IL-1&6 and TNF modulate the signaling of the cardiovascular system, which leads to the sympathovagal imbalance (Jacobs et al. 2011; Bind et al. 2012).

8.4.2 *Exposure of PM Leads to Autonomic Nervous System (ANS) Imbalance and Pathological Alterations*

Diesel exhaust (DE) particulate matter increases the heart rate and reduces the heart rate viability; it extends the ventricular depolarization and shortens repolarization, which lead to the development of arrhythmia. DE air pollutants alter the activity of TRP channels of the airway sensory neurons, present in the respiratory system. Functional alteration in these channels stimulates the midbrain which further excites the cortex and preganglionic autonomic neuron, resulting in the imbalance of autonomic nervous system, blood pressure, and heart rate (HR) (Hazari et al. 2011). A recent study has showed that exposure to the concentrated ambient particles (CAPs) stimulates the sympathetic nervous system and induces the secretion of catecholamine which further activates β2-adrenergic receptor of alveolar macrophages. Active β2-adrenergic receptor promotes the release of IL-6 that initiates the hypercoagulability of blood (prothrombotic state) (Chiarella et al. 2014).

8.4.3 Inhaled Air Pollutants Directly Translocate in the Circulatory System and Perturb the Distant Cell and Tissue Components of the Cardiovascular System

Ultrafine particulate matter (UFP) (PM0.1) and gaseous pollutants are directly translocated in the circulatory system and affect the distance organs (e.g., heart) (Nemmar et al. 2002). Injection of isolated UFP from ambient air promotes the left ventricle ejection fraction (LVEF) without affecting the heart rate (HR). UFP exhibits the potentially harmful impact on preexisting coronary disease patients (Wold et al. 2006). UFP-mediated direct acute response triggers the production of ROS, which gives rise to myocardial and endothelial dysfunction (Przyklenk and Kloner 1989). Exposure to PM0.1 also induces the level of circulatory c-Protein and inflammatory factor, which are responsible for increasing blood coagulability, vasoconstriction, and myocardial ischemia (Peters et al. 1997; Brook et al. 2002; Pope et al. 2004b).

8.5 Epidemiology of Air Pollution and Cardiovascular Disease

Ninety-one percent of the global population is living in the area where the air index is below the WHO recommended limits. Level of air pollutants like SO_2, NO_x, O_3, CO, and particulate matter (PM2.5 and PM10) increased with the time, which created a range of harmful effect over human health, vegetation, ecosystem, and climate. Industrial development has been marked as a key transition for the increase in SO_2 emission; sulfur pollution was first viewed in Europe, followed by North America. These two continents contribute highest to the emission of sulfur dioxides in the atmosphere; North America's SO_2 emission pick was around 37.7 million tons in 1970 and around 71.2 million tons in Europe in 1980. Though industrial development in Asia and Africa started much later, these regions began SO_2 emission in the late twentieth century (Klimont et al. 2013). Another air pollutant NO_x (nitrogen oxide), emitted from the combustion of fossil fuel and biomass burning, also contributes to air pollution-induced health problem; NO_x emission has been seen high in developing countries. North America and Europe were emitting around 7.0 metric tons of NO_2 in the early nineteenth century. However, Asian and African continents had started the emission of NO in the late nineteenth century (Dignon and Hameed 1989). Ozone is another noxious atmospheric pollutant, and its global emission level increased by 7.2% from 1990 to 2015 (0.0568 ppm to 0.0609 ppm). In the most polluted country, i.e., Bangladesh, Brazil, China, India, and Pakistan, the level of ozone was enhanced from 14% to 25% between 1990 and 2015, although exposure of ozone has decreased in the United States and Indonesia by 0.0037 ppm and 0.0065 ppm, respectively (Cohen et al. 2017). These gaseous pollutants work as a precursor for the creation of particulate matter. Level of particulate matter (PM2.5) has increased from 31.5 μg/m^3 (1990) to 35.02 μg/m^3

(2016), and its concentration has increased more rapidly in highly polluted cities. Level of PM2.5 has remained stable in India and Pakistan, but its concentration in another South Asian country (Bangladesh) has increased since 2010. The highest surge of PM2.5 has been found in Nigeria (African country) after 2015. However, there has been a significant reduction in PM2.5 level reported in China, and a slight decrease in Brazil, Russia, and the United States. Higher concentration of PM2.5 in 2016 was reported in Nigeria (203 μg/m^3), Saudi Arabia (187·8 μg/m^3), Qatar (148·2 μg/m^3), Egypt (126·7 μg/m^3), Bangladesh (101·04 μg/m^3), Mauritania (123.6 μg/m^3), Nepal (78·0 μg/m^3), India (75·3 μg/m^3), and China (56·4 μg/m^3), whereas the lowest PM2.5 exposure in 2016 is reported in Pacific Island countries and territories (PM2.5 $\leq$ 8·0 μg/m^3). The analysis of global PM2.5 air pollution data to different geographical regions shows that the African continent has the highest PM2.5 burden than Asia and North America. However, Europe has the lowest level of PM2.5 (Fig. 8.5).

About 7 million deaths (4.2 million outdoor pollution and 3.8 million household pollution) had happened in the year 2016 due to low air quality. Air pollutants attribute to many diseases like cardiovascular disease, lung cancer, and pulmonary disease, which cause premature death in the population. Most of the airborne-associated mortalities are due to cardiovascular pathogenesis. Particulate matter, SO_2 and NO_x are the major contaminants, causing the deleterious effect on human cardiovascular system. Studies done in more than 30 different European cities show that elevated level of SO_2 and NO_x are associated with increased cardiovascular abnormalities and mortality (Katsouyanni et al. 1997; Samoli et al. 2006). Increase in short-term exposure of SO_2 and NO_2 by 10 ppb enhance the risk of heart disease by 2.36 and 1.70, respectively (Newby et al. 2015). Myocardium is the most sensitive organ for SO_2 and its exposure affects the cytochrome c oxidase activity of myocardial mitochondria, which leads to cardiovascular manifestation (Qin et al. 2016). American Heart Association in 2010 had stated that an increase of cardiovascular manifestation is associated with long-term exposure to PM2.5. Increased level of PM2.5 has elevated the mortality rate by 0.7 million (3.5 M to 4.2 M) between 1990 and 2015. Air pollution contributes to 7.5% global mortality and is ranked as the fifth global risk factor for deaths. Air pollutants exposure causes cardiovascular disease (58.4%), lung cancer (16.5%), and lower respiratory infection (24.7%). Cardiovascular disease is the major contributor to death associated with air pollution. PM2.5 exhibits sever effect on male compared to female (1018.6 deaths per 100,000 males, and 703.4 deaths per 100,000 females), and it also displays more impact on older people (aged >70 years) than younger ones (aged ≤5 years) (GBD 2016).

Cardiovascular disease (CVD) is the illness of the heart and blood vessels. It is one of the leading diseases in global health burden, which contributes around 31% of all global deaths. CVD plays a part in deaths of approximately 17.9 million people every year and out of this 80% of CVD deaths occurred due to heart attack and stroke. Air pollution is the major factor for cardiovascular-related abnormalities, and alone it had contributed to around 7 million deaths in 2016. More than 90% of these deaths had occurred in lower-income or middle-income countries. Asian countries (i.e., China, India, Pakistan, and Bangladesh) had highest pollutant-related mortality

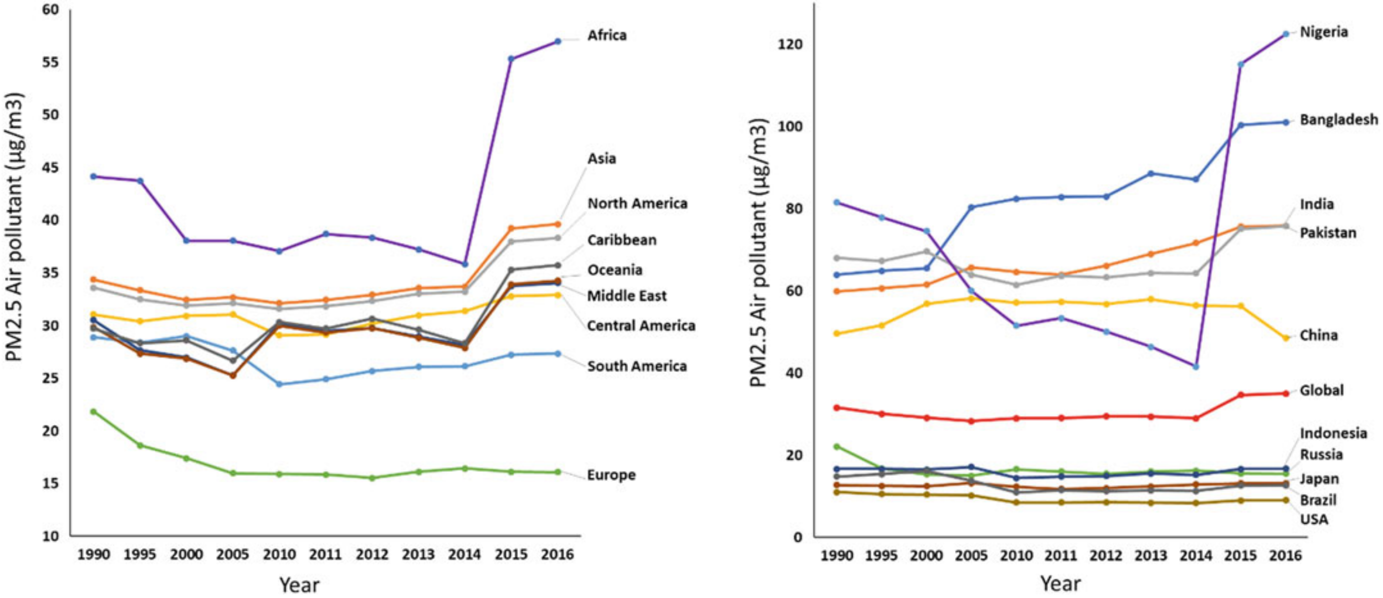


Fig. 8.5 Trends of PM2.5 exposure in different geographical regions and globally polluted countries

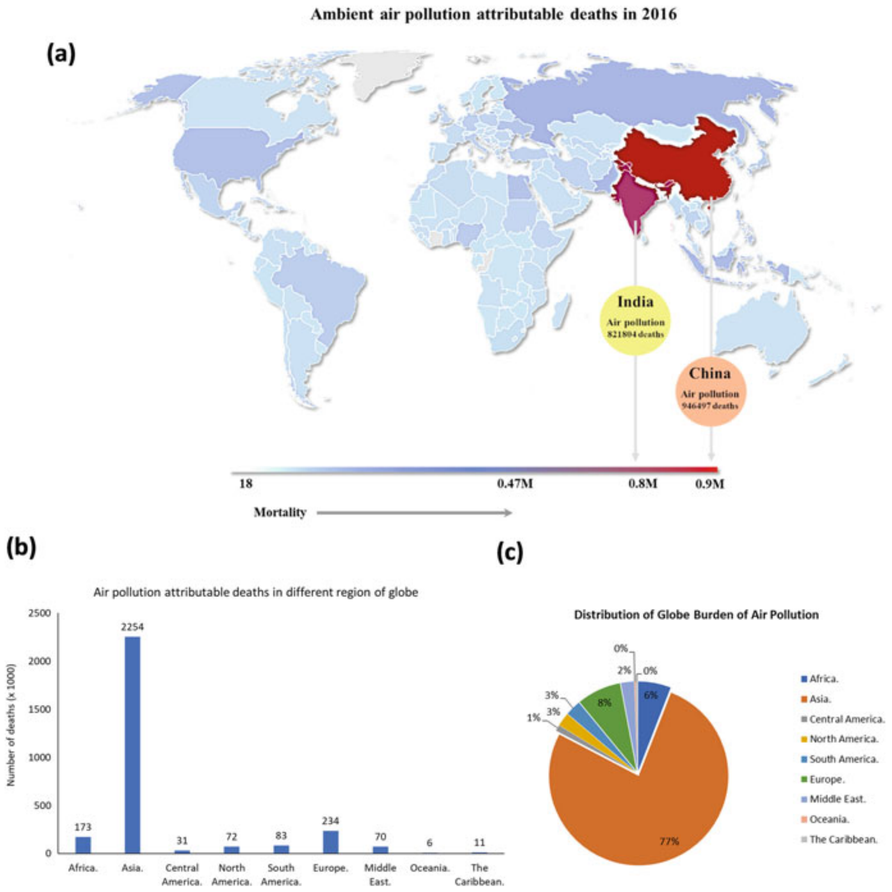


Fig. 8.6 (**a**) Total deaths attribution associated with air pollution in all the countries of the globe. (**b**) Graphical representation of death number in different regions of the world and (**c**) represent percentage distribution of mortality in the same

(2.25 million deaths), in which China was the top contributor with 0.94 million followed by India with 0.84 million deaths. The African region had 0.17 million CVD-associated deaths, in which Nigeria had the highest incident rate. American continent had 0.18 million air pollution-related causalities in 2016, in which the United States, Mexico, and Brazil were the major contributors. In Europe around 0.23 million deaths were reported in 2016 due to air pollution, in which Ukraine had carried the higher burden. Remaining casualties had occurred in Middle East (70,096), Oceania (5872), and Caribbean region (11,484) (Fig. 8.6) (WHO 2016).

8.6 Summary and Recommendations

This chapter summarized the association between prevalence of major air pollutants and cardiovascular manifestation in the population. As per WHO report, four major components of air pollution are PM, O_3, NO_x, and SO_2. Particulate matter is a critical component of air pollution and it contains all the hazardous organic and inorganic constituents. PM exposure causes more sever morbidity and mortality than other pollutants at any concentration. Even though WHO has recommended threshold level of 20 $\mu g/m^3$ for PM10 and 10 $\mu g/m^3$ for PM2.5 annually, other gaseous air pollutants like O_3, NO_2, and SO_2 are also very crucial for increased airborne illness. Combination of these gaseous pollutants with particulates matter creates more harmful impact. WHO has also recommended the threshold level for these gaseous pollutants as 100 $\mu g/m^3$ of O_3 for 8 h, 40 $\mu g/m^3$ of NO_x annually, and 20 $\mu g/m^3$ of SO_2 for 24 h. Air pollution is a crucial environmental risk factor for human health and by reducing the air pollution level, countries can reduce disease and socioeconomic burdens. Outdoor pollution in both urban and rural areas caused around 4.2 million deaths in 2016. The case-crossover study done in Germany, the United States, Europe, and China shows that increased exposure to PM10 by 10 $\mu g/m^3$ induces the 1.0–1.50% increase of cardiovascular cases. In further study on teen and adult population (0.5 million) with 16 years of follow-up shows that consequence of cardiovascular mortality increased by 8–18% for every 10.5 $\mu g/m^3$ increase of PM2.5 level. Similarly, case-crossover studies done in China and the United States show that increase exposure to SO_2 and NO_2 by 10 $\mu g/m^3$ elevates 2.0–3.0% arrhythmia and blood pressure cases. Ozone is also an important component of air pollution; several European studies reported that daily heart-related mortality increases by 0.4 per 10 $\mu g/m^3$ rise of ozone. To tackle this issue many environment protection and health associated agencies have given the recommendation and guidelines. WHO has given the recommendation to the industries, power generation plant, municipal, agricultural waste management and transport to use clean technology that reduces the industrial emission, use of clean and heavy duty diesel vehicle, fuel with reduced sulfur content, renewable combustion free power source (light, water, and wind) and strict system to reduce the open garbage and biomass burning. Other agencies like EPA have set the limit for the emission of air pollutant from chemical plant, vehicle, and other industries. The European Environment Agency, with a network of 33 countries, gathered the data and made the assessment and policy for environment protection.

As specific therapy for air pollution illness is difficult to pinpoint due to diverse health consequences. The detrimental health impact of pollutants is not confined to the source site, since fine particles can travel further and can therefore affect the health of people living and working in the surrounding areas. Basic and fundamental new directions can enable better predictions for future conditions and scenarios. Joining hands among individuals along with NGOs in coordination with government can implement strong policy to reduce pollutant exposure using environmental education, safe clean technologies, and limiting pollution exposure strategies.

References

Admassu M, Wuhshet M, Tilaye T (2004) Sanitary survey in Gondar town. Ethiop J Heal Dev 18:39–42. https://doi.org/10.4314/ejhd.v18i1.9864

Admassu M, Wubshet M, Gelaw B (2005) A survey of bacteriological quality of drinking water in North Gondar. Ethiop J Heal Dev 18:112–115. https://doi.org/10.4314/ejhd.v18i2.9946

Agarwal R, Jayaraman G, Anand S, Marimuthu P (2006) Assessing respiratory morbidity through pollution status and meteorological conditions for Delhi. Environ Monit Assess 114:489–504. https://doi.org/10.1007/s10661-006-4935-3

Agay-Shay K, Friger M, Linn S et al (2013) Air pollution and congenital heart defects. Environ Res 124:28–34. https://doi.org/10.1016/j.envres.2013.03.005

Aggarwal P, Jain S (2015) Impact of air pollutants from surface transport sources on human health: a modeling and epidemiological approach. Environ Int 83:146–157. https://doi.org/10.1016/j.envint.2015.06.010

Arashiro M, Lin Y-H, Zhang Z et al (2018) Effect of secondary organic aerosol from isoprene-derived hydroxyhydroperoxides on the expression of oxidative stress response genes in human bronchial epithelial cells. Environ Sci Process Impacts 20:332–339. https://doi.org/10.1039/C7EM00439G

Argacha J-F (2017) Air pollution and myocardial infarction. Eur Heart J 38:141–141. https://doi.org/10.1093/eurheartj/ehw622

Argacha JF, Collart P, Wauters A et al (2016) Air pollution and ST-elevation myocardial infarction: a case-crossover study of the Belgian STEMI registry 2009–2013. Int J Cardiol 223:300–305. https://doi.org/10.1016/J.IJCARD.2016.07.191

Atkinson RW, Analitis A, Samoli E et al (2016) Short-term exposure to traffic-related air pollution and daily mortality in London, UK. J Expo Sci Environ Epidemiol 26:125–132. https://doi.org/10.1038/jes.2015.65

Bagryantseva Y, Novotna B, Rossner P et al (2010) Oxidative damage to biological macromolecules in Prague bus drivers and garagemen: impact of air pollution and genetic polymorphisms. Toxicol Lett 199:60–68. https://doi.org/10.1016/J.TOXLET.2010.08.007

Bai K-J, Chuang K-J, Chen J-K et al (2018) Investigation into the pulmonary inflammopathology of exposure to nickel oxide nanoparticles in mice. Nanomedicine 14:2329–2339. https://doi.org/10.1016/j.nano.2017.10.003

Baldauf RW, Devlin RB, Gehr P et al (2016) Ultrafine particle metrics and research considerations: review of the 2015 UFP workshop. Int J Environ Res Public Health 13:1054. https://doi.org/10.3390/ijerph13111054

Barnes J, Bender J, Lyons T, Borland A (1999) Natural and man-made selection for air pollution resistance. J Exp Bot 50:1423–1435. https://doi.org/10.1093/jxb/50.338.1423

Barnett AG, Williams GM, Schwartz J, Neller AH, Best TL, Petroeschevsky AL, Simpson RW (2005) Air pollution and child respiratory health. Am J Respir Crit Care Med 171(11):1272–1278

Begum BA, Biswas SK, Hopke PK (2008) Assessment of trends and present ambient concentrations of PM2.2 and PM10 in Dhaka, Bangladesh. Air Qual Atmos Heal 1:125–133. https://doi.org/10.1007/s11869-008-0018-7

Bell ML, Kim JY, Dominici F (2007) Potential confounding of particulate matter on the short-term association between Ozone and mortality in multisite time-series studies. Environ Health Perspect 115:1591–1595. https://doi.org/10.1289/ehp.10108

Bhatia S, Bhatia S, Mears J et al (2017) Seasonal periodicity of ischemic heart disease and heart failure. Heart Fail Clin 13:681–689. https://doi.org/10.1016/j.hfc.2017.05.004

Bind M-A, Baccarelli A, Zanobetti A et al (2012) Air pollution and markers of coagulation, inflammation, and endothelial function: associations and epigene-environment interactions in an elderly cohort. Epidemiology 23:332–340. https://doi.org/10.1097/EDE.0b013e31824523f0

Boogaard H, van Erp AM (2019) Assessing health effects of air quality actions: what's next? Lancet Public Heal 4:e4–e5. https://doi.org/10.1016/S2468-2667(18)30235-4

Bose K, Lakshminarasimhan H, Sundar K, Kathiresan T (2016) Cytotoxic effect of ZnS nanoparticles on primary mouse retinal pigment epithelial cells. Artif Cells Nanomed Biotechnol 44:1764–1773. https://doi.org/10.3109/21691401.2015.1102739

Brook RD (2008) Cardiovascular effects of air pollution. Clin Sci 115(6):175–187

Brook RD, Brook JR, Urch B et al (2002) Inhalation of fine particulate air pollution and ozone causes acute arterial vasoconstriction in healthy adults. Circulation 105:1534–1536

Brook RD, Rajagopalan S, Pope CA et al (2010) Particulate matter air pollution and cardiovascular disease. Circulation 121:2331–2378. https://doi.org/10.1161/CIR.0b013e3181dbece1

Carrico CM, Kus P, Rood MJ et al (2003) Mixtures of pollution, dust, sea salt, and volcanic aerosol during ACE-Asia: radiative properties as a function of relative humidity. J Geophys Res 108:8650. https://doi.org/10.1029/2003JD003405

Cesaroni G, Forastiere F, Stafoggia M et al (2014) Long term exposure to ambient air pollution and incidence of acute coronary events: prospective cohort study and meta-analysis in 11 European cohorts from the ESCAPE Project. BMJ 348:f7412. https://doi.org/10.1136/bmj.f7412

Chang C-C, Hwang J-S, Chan C-C, Cheng T-J (2007) Interaction effects of ultrafine carbon black with iron and nickel on heart rate variability in spontaneously hypertensive rats. Environ Health Perspect 115:1012–1017. https://doi.org/10.1289/ehp.9821

Chen T-M, Kuschner WG, Gokhale J, Shofer S (2007) Outdoor air pollution: nitrogen dioxide, sulfur dioxide, and carbon monoxide health effects. Am J Med Sci 333(4):249–256

Chiarella SE, Soberanes S, Urich D et al (2014) β2-Adrenergic agonists augment air pollution–induced IL-6 release and thrombosis. J Clin Invest 124:2935–2946. https://doi.org/10.1172/JCI75157

Clark NA, Demers PA, Karr CJ et al (2010) Effect of early life exposure to air pollution on development of childhood asthma. Environ Health Perspect 118:284–290. https://doi.org/10.1289/ehp.0900916

Cohen AJ, Brauer M, Burnett R et al (2017) Estimates and 25-year trends of the global burden of disease attributable to ambient air pollution: an analysis of data from the Global Burden of Diseases Study 2015. Lancet (London, England) 389:1907–1918. https://doi.org/10.1016/S0140-6736(17)30505-6

De Roos AJ, Koehoorn M, Tamburic L et al (2014) Proximity to traffic, ambient air pollution, and community noise in relation to incident rheumatoid arthritis. Environ Health Perspect 122:1075–1080. https://doi.org/10.1289/ehp.1307413

Dignon J, Hameed S (1989) Global emissions of nitrogen and sulfur oxides from 1860 to 1980. JAPCA 39(2):180–186

Dockery DW, Pope CA, Xu X et al (1993) An association between air pollution and mortality in six U.S. cities. N Engl J Med 329:1753–1759. https://doi.org/10.1056/NEJM199312093292401

Dominici F, Peng RD, Bell ML et al (2006) Fine particulate air pollution and hospital admission for cardiovascular and respiratory diseases. JAMA 295:1127. https://doi.org/10.1001/jama.295.10.1127

Donaldson K, Stone V (2003) Current hypotheses on the mechanisms of toxicity of ultrafine particles. Ann Ist Super Sanita 39:405–410

Donaldson K, Stone V, Seaton A, MacNee W (2001) Ambient particle inhalation and the cardiovascular system: potential mechanisms. Environ Health Perspect 109:523–527. https://doi.org/10.1289/ehp.01109s4523

Endes S, Schaffner E, Caviezel S et al (2017) Is physical activity a modifier of the association between air pollution and arterial stiffness in older adults: the SAPALDIA cohort study. Int J Hyg Environ Health 220:1030–1038. https://doi.org/10.1016/j.ijheh.2017.06.001

Fang Y, Mauzerall DL, Liu J et al (2013) Impacts of 21st century climate change on global air pollution-related premature mortality. Clim Chang 121:239–253. https://doi.org/10.1007/s10584-013-0847-8

Feigin VL, Roth GA, Naghavi M et al (2016) Global burden of stroke and risk factors in 188 countries, during 1990–2013: a systematic analysis for the Global Burden of Disease Study 2013. Lancet Neurol 15:913–924. https://doi.org/10.1016/S1474-4422(16)30073-4

Fonceca AM, Zosky GR, Bozanich EM et al (2018) Accumulation mode particles and LPS exposure induce TLR-4 dependent and independent inflammatory responses in the lung. Respir Res 19:15. https://doi.org/10.1186/s12931-017-0701-z

Fröhlich E (2017) Hemocompatibility of inhaled environmental nanoparticles: potential use of in vitro testing. J Hazard Mater 336:158–167. https://doi.org/10.1016/j.jhazmat.2017.04.041

GBD (2016). https://vizhub.healthdata.org/gbd-compare

GHIO AJ, DEVLIN RB (2001) Inflammatory lung injury after bronchial instillation of air pollution particles. Am J Respir Crit Care Med 164:704–708. https://doi.org/10.1164/ajrccm.164.4.2011089

GHIO AJ, KIM C, DEVLIN RB (2000) Concentrated ambient air particles induce mild pulmonary inflammation in healthy human volunteers. Am J Respir Crit Care Med 162:981–988. https://doi.org/10.1164/ajrccm.162.3.9911115

Gold DR, Litonjua A, Schwartz J et al (2000) Ambient pollution and heart rate variability. Circulation 101:1267–1273

Gorman D, Drewry A, Huang YL, Sames C (2003) The clinical toxicology of carbon monoxide. Toxicology 187(1):25–38

Goudarzi G, Mohammadi MJ, Angali KA, Neisi AK, Babaei AA, Mohammadi B, Soleimani Z, Geravandi S (2012) Estimation of health effects attributed to NO2 exposure using AirQ model. Arch Hyg Sci 1:59–66

Graber JM, Macdonald SC, Kass DE et al (2007) Carbon monoxide: the case for environmental public health surveillance. Public Health Rep 122:138–144. https://doi.org/10.1177/003335490712200202

Hazari MS, Haykal-Coates N, Winsett DW et al (2011) TRPA1 and sympathetic activation contribute to increased risk of triggered cardiac arrhythmias in hypertensive rats exposed to diesel exhaust. Environ Health Perspect 119:951–957. https://doi.org/10.1289/ehp.1003200

He C, Song Y, Ichinose T et al (2018) Lipopolysaccharide levels adherent to PM2.5 play an important role in particulate matter induced-immunosuppressive effects in mouse splenocytes. J Appl Toxicol 38:471–479. https://doi.org/10.1002/jat.3554

Heinrich J, Hoelscher B, Frye C, Meyer I, Pitz M, Cyrys J, Wjst M, Neas L, Wichmann H-E (2002) Improved air quality in reunified Germany and decreases in respiratory symptoms. Epidemiology 13(4):394–401

Hemminki K, Pershagen G (1994) Cancer risk of air pollution: epidemiological evidence. Environ Health Perspect 102(Suppl 4):187–192. https://doi.org/10.1289/ehp.94102s4187

Henrotin J-B, Zeller M, Lorgis L et al (2010) Evidence of the role of short-term exposure to ozone on ischaemic cerebral and cardiac events: the Dijon Vascular Project (DIVA). Heart 96:1990–1996. https://doi.org/10.1136/hrt.2010.200337

Hong Y-C, Lee J-T, Kim H, Kwon H-J (2002) Air pollution: a new risk factor in ischemic stroke mortality. Stroke 33:2165–2169

Hou J, Sun H, Guo Y et al (2018) Associations between urinary monohydroxy polycyclic aromatic hydrocarbons metabolites and Framingham Risk Score in Chinese adults with low lung function. Ecotoxicol Environ Saf 147:1002–1009. https://doi.org/10.1016/j.ecoenv.2017.09.058

Ibeneme S, Egbosionu V, Ibeneme G et al (2016) Evidence of allergic reactions and cardiopulmonary impairments among traders operating from foodstuff warehouses. Biomed Res Int 2016:1–11. https://doi.org/10.1155/2016/1081258

Jacobs L, Emmerechts J, Hoylaerts MF et al (2011) Traffic air pollution and oxidized LDL. PLoS One 6:e16200. https://doi.org/10.1371/journal.pone.0016200

Ji J, Hedelin A, Malmlöf M et al (2017) Development of combining of human bronchial Mucosa models with XposeALI® for exposure of air pollution nanoparticles. PLoS One 12:e0170428. https://doi.org/10.1371/journal.pone.0170428

Kan H, Chen R, Tong S (2012) Ambient air pollution, climate change, and population health in China. Environ Int 42:10–19

Katsouyanni K, Touloumi G, Spix C, Schwartz J, Balducci F, Medina S, Rossi G, Wojtyniak B, Sunyer J, Bacharova L, Schouten JP, Ponka A, Anderson HR (1997) Short term effects of ambient sulphur dioxide and particulate matter on mortality in 12 European cities: results from time series data from the APHEA project. Air pollution and health: a European approach. BMJ 314(7095):1658–1663

Kaufman JD, Adar SD, Barr RG et al (2016) Association between air pollution and coronary artery calcification within six metropolitan areas in the USA (the multi-ethnic study of atherosclerosis and air pollution): a longitudinal cohort study. Lancet 388:696–704. https://doi.org/10.1016/S0140-6736(16)00378-0

Kelly FJ, Fussell JC (2015) Air pollution and public health: emerging hazards and improved understanding of risk. Environ Geochem Health 37:631–649. https://doi.org/10.1007/s10653-015-9720-1

Kelly FJ, Fussell JC (2017) Role of oxidative stress in cardiovascular disease outcomes following exposure to ambient air pollution. Free Radic Biol Med 110:345–367. https://doi.org/10.1016/j.freeradbiomed.2017.06.019

Kettunen J, Lanki T, Tiittanen P et al (2007) Associations of fine and ultrafine particulate air pollution with stroke mortality in an area of low air pollution levels. Stroke 38:918–922. https://doi.org/10.1161/01.STR.0000257999.49706.3b

Kim HH, Lee CS, Do YS et al (2016) Near-road exposure and impact of air pollution on allergic diseases in elementary school children: a cross-sectional study. Yonsei Med J 57:698. https://doi.org/10.3349/YMJ.2016.57.3.698

Kim H-J, Park SJ, Park CS et al (2018) Surface-modified polymer nanofiber membrane for high-efficiency microdust capturing. Chem Eng J 339:204–213. https://doi.org/10.1016/j.cej.2018.01.121

Kinney P (1999) The pulmonary effects of outdoor ozone and particle air pollution. Semin Respir Crit Care Med 20(06):601–607

Klimont Z, Smith SJ, Cofala J (2013) The last decade of global anthropogenic sulfur dioxide: 2000–2011 emissions. Environ Res Lett 8(1):014003

Koken PJM, Piver WT, Ye F, Elixhauser A, Olsen LM, Portier CJ (2003) Temperature, air pollution, and hospitalization for cardiovascular diseases among elderly people in Denver. Environ Health Perspect 111(10):1312–1317

Kumar P, Patton AP, Durant JL, Frey HC (2018) A review of factors impacting exposure to PM2.5, ultrafine particles and black carbon in Asian transport microenvironments. Atmos Environ 187:301–316. https://doi.org/10.1016/J.ATMOSENV.2018.05.046

Künzli N, Medina S, Kaiser R et al (2001) Assessment of deaths attributable to air pollution: should we use risk estimates based on time series or on cohort studies? Am J Epidemiol 153:1050–1055

Künzli N, Jerrett M, Mack WJ et al (2005) Ambient air pollution and atherosclerosis in Los Angeles. Environ Health Perspect 113:201–206. https://doi.org/10.1289/ehp.7523

Latza U, Gerdes S, Baur X (2009) Effects of nitrogen dioxide on human health: systematic review of experimental and epidemiological studies conducted between 2002 and 2006. Int J Hyg Environ Health 212:271–287. https://doi.org/10.1016/j.ijheh.2008.06.003

Li F, Liu Y, Lü J et al (2015) Ambient air pollution in China poses a multifaceted health threat to outdoor physical activity. J Epidemiol Community Health 69:201–204. https://doi.org/10.1136/jech-2014-203892

Li Z, Che W, Frey HC et al (2017) Characterization of PM2.5 exposure concentration in transport microenvironments using portable monitors. Environ Pollut 228:433–442. https://doi.org/10.1016/J.ENVPOL.2017.05.039

Liu W, Xu Y, Liu W et al (2018) Oxidative potential of ambient PM 2.5 in the coastal cities of the Bohai Sea, northern China: Seasonal variation and source apportionment. Environ Pollut 236:514–528. https://doi.org/10.1016/j.envpol.2018.01.116

Louwies T, Int Panis L, Provost E et al (2018) DNA hypomethylation in association with internal and external markers of traffic exposure in a panel of healthy adults. Air Qual Atmos Heal 11:673–681. https://doi.org/10.1007/s11869-018-0574-4

Luo M, Hou X, Gu Y et al (2018) Trans-boundary air pollution in a city under various atmospheric conditions. Sci Total Environ 618:132–141. https://doi.org/10.1016/j.scitotenv.2017.11.001

Luyts K, Van Den Broucke S, Hemmeryckx B et al (2018) Nanoparticles in the lungs of old mice: pulmonary inflammation and oxidative stress without procoagulant effects. Sci Total Environ 644:907–915. https://doi.org/10.1016/j.scitotenv.2018.06.301

Machado MN, Schmidt AC, Saldiva PHN et al (2014) Pulmonary functional and morphological damage after exposure to tripoli dust. Respir Physiol Neurobiol 196:17–24. https://doi.org/10.1016/j.resp.2014.02.007

Mahowald N, Albani S, Kok JF et al (2014) The size distribution of desert dust aerosols and its impact on the Earth system. Aeolian Res 15:53–71. https://doi.org/10.1016/J.AEOLIA.2013.09.002

Manigrasso M, Vitali M, Protano C, Avino P (2017) Temporal evolution of ultrafine particles and of alveolar deposited surface area from main indoor combustion and non-combustion sources in a model room. Sci Total Environ 598:1015–1026. https://doi.org/10.1016/j.scitotenv.2017.02.048

McCarthy MC, O'Brien TE, Charrier JG, Hafner HR (2009) Characterization of the chronic risk and hazard of hazardous air pollutants in the United States using ambient monitoring data. Environ Health Perspect 117:790–796. https://doi.org/10.1289/ehp.11861

Møller P, Danielsen PH, Karottki DG et al (2014) Oxidative stress and inflammation generated DNA damage by exposure to air pollution particles. Mutat Res Rev Mutat Res 762:133–166. https://doi.org/10.1016/j.mrrev.2014.09.001

Nemmar A, Hoet PHM, Vanquickenborne B et al (2002) Passage of inhaled particles into the blood circulation in humans. Circulation 105:411–414

Newby DE, Mannucci PM, Tell GS, Baccarelli AA, Brook RD, Donaldson K, Forastiere F, Franchini M, Franco OH, Graham I, Hoek G, Hoffmann B, Hoylaerts MF, Künzli N, Mills N, Pekkanen J, Peters A, Piepoli MF, Rajagopalan S, Storey RF (2015) Expert position paper on air pollution and cardiovascular disease. Eur Heart J 36(2):83–93

Nuvolone D, Petri D, Voller F (2018) The effects of ozone on human health. Environ Sci Pollut Res 25:8074–8088. https://doi.org/10.1007/s11356-017-9239-3

O'Neill MS, Veves A, Zanobetti A et al (2005) Diabetes enhances vulnerability to particulate air pollution-associated impairment in vascular reactivity and endothelial function. Circulation 111:2913–2920. https://doi.org/10.1161/CIRCULATIONAHA.104.517110

Olatunji AS, Kolawole TO, Oloruntola M, Günter C (2018) Evaluation of pollution of soils and particulate matter around metal recycling factories in Southwestern Nigeria. J Heal Pollut 8:20–30. https://doi.org/10.5696/2156-9614-8.17.20

Olopade CO, Frank E, Bartlett E et al (2017) Effect of a clean stove intervention on inflammatory biomarkers in pregnant women in Ibadan, Nigeria: a randomized controlled study. Environ Int 98:181–190. https://doi.org/10.1016/j.envint.2016.11.004

Omidi Y, Goudarzi G, Heidari AM, Daryanoosh SM (2016) Health impact assessment of short-term exposure to NO2 in Kermanshah, Iran using AirQ model. Environ Health Eng Manag 3(2):91–97

Patton AP, Perkins J, Zamore W et al (2014) Spatial and temporal differences in traffic-related air pollution in three urban neighborhoods near an interstate highway. Atmos Environ (1994) 99:309–321. https://doi.org/10.1016/j.atmosenv.2014.09.072

Penney D, Benignus V, Kephalopoulos S, Kotzias D, Kleinman M, Verrier A (2010) Carbon monoxide WHO guidelines for indoor air quality: selected pollutants

Peters A, Döring A, Wichmann H-E, Koenig W (1997) Increased plasma viscosity during an air pollution episode: a link to mortality? Lancet 349:1582–1587. https://doi.org/10.1016/S0140-6736(97)01211-7

Piantadosi CA (1999) Diagnosis and treatment of carbon monoxide poisoning. Respir Care Clin N Am 5:183–202

Pikhart H, Bobak M, Gorynski P, Wojtyniak B, Danova J, Celko MA, Kriz B, Briggs D, Elliott P (2001) Outdoor sulphur dioxide and respiratory symptoms in Czech and Polish school children: a small-area study (SAVIAH). Int Arch Occup Environ Health 74(8):574–578

Pissuwan D, Somkid K, Kongseng S et al (2016) Respiratory tract toxicity of titanium dioxide nanoparticles and multi-walled carbon nanotubes on mice after intranasal exposure. Micro Nano Lett 11:183–187. https://doi.org/10.1049/mnl.2015.0523

Pope CA, Dockery DW (2006) Health effects of fine particulate air pollution: lines that connect. J Air Waste Manage Assoc 56:709–742. https://doi.org/10.1080/10473289.2006.10464485

Pope CA III, Burnett RT, Thun MJ et al (2002) Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. JAMA 287:1132. https://doi.org/10.1001/jama.287.9.1132

Pope CAI, Dockery DW, Kanner RE et al (1999) Oxygen saturation, pulse rate, and particulate air pollution. Am J Respir Crit Care Med 159:365–372. https://doi.org/10.1164/ajrccm.159.2.9702103

Pope CA, Burnett RT, Thurston GD et al (2004a) Cardiovascular mortality and long-term exposure to particulate air pollution. Circulation 109:71–77. https://doi.org/10.1161/01.CIR.0000108927.80044.7F

Pope CA, Hansen ML, Long RW et al (2004b) Ambient particulate air pollution, heart rate variability, and blood markers of inflammation in a panel of elderly subjects. Environ Health Perspect 112:339–345. https://doi.org/10.1289/ehp.6588

Prospero JM (1999) Long-range transport of mineral dust in the global atmosphere: impact of African dust on the environment of the southeastern United States. Proc Natl Acad Sci USA 96:3396–3403. https://doi.org/10.1073/PNAS.96.7.3396

Przyklenk K, Kloner RA (1989) ‘Reperfusion injury’ by oxygen-derived free radicals? Effect of superoxide dismutase plus catalase, given at the time of reperfusion, on myocardial infarct size, contractile function, coronary microvasculature, and regional myocardial blood flow. Circ Res 64:86–96

Qin G, Wu M, Wang J, Xu Z, Xia J, Sang N (2016) Sulfur dioxide contributes to the cardiac and mitochondrial dysfunction in rats. Toxicol Sci 151(2):334–346

Rahman MM, Mazaheri M, Clifford S, Morawska L (2017) Estimate of main local sources to ambient ultrafine particle number concentrations in an urban area. Atmos Res 194:178–189. https://doi.org/10.1016/j.atmosres.2017.04.036

Rall DP (1974) Review of the health effects of sulfur oxides. Environ Health Perspect 8:97–121. https://doi.org/10.1289/ehp.74897

Raub J (1999) Carbon Monoxide, 2nd edn. World Health Organization, Geneva

Samoli E, Aga E, Touloumi G et al (2006) Short-term effects of nitrogen dioxide on mortality: an analysis within the APHEA project. Eur Respir J 27:1129–1138. https://doi.org/10.1183/09031936.06.00143905

Samoli E, Touloumi G, Schwartz J et al (2007) Short-term effects of carbon monoxide on mortality: an analysis within the APHEA project. Environ Health Perspect 115:1578–1583. https://doi.org/10.1289/ehp.10375

Schwartz J (2001) Is there harvesting in the association of airborne particles with daily deaths and hospital admissions? Epidemiology 12:55–61

Setiawan B, Kania N, Nugrahenny D et al (2014) Subchronic inhalation of particulate matter 10 coal dust induces atherosclerosis in the aorta of diabetic and nondiabetic rats. Biomarkers Genomic Med 6:67–73. https://doi.org/10.1016/j.bgm.2014.03.002

Seymour V (2016) The human-nature relationship and its impact on health: a critical review. Front Public Health 4:260. https://doi.org/10.3389/fpubh.2016.00260

Shi S, Zhu S, Lee ES et al (2016) Performance of wearable ionization air cleaners: Ozone emission and particle removal. Aerosol Sci Technol 50:211–221. https://doi.org/10.1080/02786826.2016.1139045

Silbajoris R, Osornio-Vargas AR, Simmons SO et al (2011) Ambient particulate matter induces interleukin-8 expression through an alternative NF-κB (nuclear factor-kappa B) mechanism in human airway epithelial cells. Environ Health Perspect 119:1379–1383. https://doi.org/10.1289/ehp.1103594

Silva AH, Locatelli C, Filho UP et al (2017) Visceral fat increase and signals of inflammation in adipose tissue after administration of titanium dioxide nanoparticles in mice. Toxicol Ind Health 33:147–158. https://doi.org/10.1177/0748233715613224

Simonetti G, Conte E, Perrino C, Canepari S (2018) Oxidative potential of size-segregated PM in an urban and an industrial area of Italy. Atmos Environ 187:292–300. https://doi.org/10.1016/j.atmosenv.2018.05.051

Smith KR (2002) Indoor air pollution in developing countries: recommendations for research. Indoor Air 12:198–207. https://doi.org/10.1034/j.1600-0668.2002.01137.x

Stafoggia M, Cesaroni G, Peters A et al (2014) Long-term exposure to ambient air pollution and incidence of cerebrovascular events: results from 11 European cohorts within the ESCAPE project. Environ Health Perspect 122:919–925. https://doi.org/10.1289/ehp.1307301

Suwa T, Hogg JC, Quinlan KB et al (2002) Particulate air pollution induces progression of atherosclerosis. J Am Coll Cardiol 39:935–942

Taillé C, El-Benna J, Lanone S, Boczkowski J, Motterlini R (2005) Mitochondrial respiratory chain and NAD(P)H oxidase are targets for the antiproliferative effect of carbon monoxide in human airway smooth muscle. J Biol Chem 280(27):25350–25360

Tham A, Lullo D, Dalton S et al (2017) Modeling vascular inflammation and atherogenicity after inhalation of ambient levels of ozone: exploratory lessons from transcriptomics. Inhal Toxicol 29:96–105. https://doi.org/10.1080/08958378.2017.1310333

Tunnicliffe WS, Hilton MF, Harrison RM, Ayres JG (2001) The effect of sulphur dioxide exposure on indices of heart rate variability in normal and asthmatic adults. Eur Respir J 17(4):604–608

Urch B, Silverman F, Corey P et al (2005) Acute blood pressure responses in healthy adults during controlled air pollution exposures. Environ Health Perspect 113:1052–1055. https://doi.org/10.1289/ehp.7785

Voidazan S, Moldovan H, Huțanu A et al (2018) Could fibrinogen and hsCRP be useful for assessing personal risk in workers exposed to a mixture of ultrafine particles and organic solvents? Rev Rom Med Lab 26:177–187. https://doi.org/10.2478/rrlm-2018-0011

Von Burg R (1999) Toxicology update. J Appl Toxicol 19(5):379–386

Vora R, Zareba W, Utell MJ et al (2014) Inhalation of ultrafine carbon particles alters heart rate and heart rate variability in people with type 2 diabetes. Part Fibre Toxicol 11:31. https://doi.org/10.1186/s12989-014-0031-y

Vreeland H, Weber R, Bergin M et al (2017) Oxidative potential of PM 2.5 during Atlanta rush hour: measurements of in-vehicle dithiothreitol (DTT) activity. Atmos Environ 165:169–178. https://doi.org/10.1016/j.atmosenv.2017.06.044

Weuve J, Kaufman JD, Szpiro AA et al (2016) Exposure to traffic-related air pollution in relation to progression in physical disability among older adults. Environ Health Perspect 124:1000–1008. https://doi.org/10.1289/ehp.1510089

WHO (2002) The World Health Report 2002: reducing risks, promoting healthy life. World Health Organization, Geneva

WHO (2012) Burden of disease from the joint effects of household and ambient air pollution for 2012. World Heal Organ

WHO (2016) Burden of disease from ambient air pollution for 2016

World Bank (2004) Reducing air pollution from urban transport

Wold LE, Simkhovich BZ, Kleinman MT et al (2006) In vivo and in vitro models to test the hypothesis of particle-induced effects on cardiac function and arrhythmias. Cardiovasc Toxicol 6:69–78

Wold LE, Ying Z, Hutchinson KR et al (2012) Cardiovascular remodeling in response to long-term exposure to fine particulate matter air pollution. Circ Heart Fail 5:452–461. https://doi.org/10.1161/CIRCHEARTFAILURE.112.966580

Yang B, Deng Q, Zhang W et al (2016) Exposure to polycyclic aromatic hydrocarbons, plasma cytokines and heart rate variability. Sci Rep 6:19272. https://doi.org/10.1038/srep19272

Yang L, Hou X-Y, Wei Y et al (2017) Biomarkers of the health outcomes associated with ambient particulate matter exposure. Sci Total Environ 579:1446–1459. https://doi.org/10.1016/j.scitotenv.2016.11.146

Yang B-Y, Guo Y, Morawska L et al (2019) Ambient PM1 air pollution and cardiovascular disease prevalence: insights from the 33 communities Chinese health study. Environ Int 123:310–317. https://doi.org/10.1016/J.ENVINT.2018.12.012

Yin Z, Xu H-J, Yao X-L et al (2017) Ambient fine particles (PM 2.5) attenuate collagen-induced platelet activation through interference of the PLCγ2/Akt/GSK3β signaling pathway. Environ Toxicol 32:530–540. https://doi.org/10.1002/tox.22257

Ying Z, Xu X, Bai Y et al (2014) Long-term exposure to concentrated ambient PM 2.5 increases mouse blood pressure through abnormal activation of the sympathetic nervous system: a role for hypothalamic inflammation. Environ Health Perspect 122:79–86. https://doi.org/10.1289/ehp.1307151

You S, Neoh KG, Tong YW et al (2017a) Variation of household electricity consumption and potential impact of outdoor PM2.5 concentration: a comparison between Singapore and Shanghai. Appl Energy 188:475–484. https://doi.org/10.1016/j.apenergy.2016.12.019

You S, Yao Z, Dai Y, Wang C-H (2017b) A comparison of PM exposure related to emission hotspots in a hot and humid urban environment: concentrations, compositions, respiratory deposition, and potential health risks. Sci Total Environ 599–600:464–473. https://doi.org/10.1016/j.scitotenv.2017.04.217

Zanobetti A, Canner MJ, Stone PH et al (2004) Ambient pollution and blood pressure in cardiac rehabilitation patients. Circulation 110:2184–2189. https://doi.org/10.1161/01.CIR.0000143831.33243.D8

Zhang Y, Li M, Bravo MA et al (2014) Air quality in Lanzhou, a Major Industrial City in China: characteristics of air pollution and review of existing evidence from air pollution and health studies. Water Air Soil Pollut 225:2187. https://doi.org/10.1007/s11270-014-2187-3

Zhang T, Gao B, Zhou Z, Chang Y (2016) The movement and deposition of PM2.5 in the upper respiratory tract for the patients with heart failure: an elementary CFD study. Biomed Eng Online 15:138. https://doi.org/10.1186/s12938-016-0281-z

Zhao Y, Wang S, Duan L, Lei Y, Cao P, Hao J (2008) Primary air pollutant emissions of coal-fired power plants in China: current status and future prediction. Atmos Environ 42(36):8442–8452

Chapter 9
Health Risk Assessment and Management of Air Pollutants

Atar Singh Pipal, Stuti Dubey, and Ajay Taneja

Abstract Air pollution is a vital global public health concern which is generally addressed by collective societal action, particularly to control emissions, i.e. primary air pollutants which are precursors in the formation of secondary air pollutants via different atmospheric chemical reactions. The massive increase in emission of air pollutants in the atmosphere is major cause of human health and environmental problems. According to WHO, it is revealed that particulate matter (PM) exposure is responsible for ~800,000 premature deaths alone each year as compared to other air pollutants. Therefore, more systematic studies for the measurement of various air pollutants are still required to examine the current scenario and their physicochemical characteristics especially focused on PM. This will aid in health risk assessment of air pollutants by using various tools and estimation methods. The present chapter describes the brief introduction of air pollutants and their emission source characteristics along with detailed systematic findings and outcomes of the different studies. In addition, the methods/equations and diverse tools used for risk assessment by scientific community and various researchers have been introduced at regional and global level. Also, the methods and approaches that can be employed for the management of air pollutants (indoor and outdoor) in Indian context have been described. Overall, the chapter gives an idea about the deterioration of air quality due to emission of various pollutants, their formation and management methods along with the concerned health issues. This will serve as an imperative document for the scientific community and policy makers to develop effective mitigation policies with respect to air quality improvement.

Keyword Air quality · Pollutants · Health risks · Sources and pollutant's management

---

A. S. Pipal (✉) · S. Dubey · A. Taneja
Department of Chemistry, Dr. B R Ambedkar University, Agra, India
e-mail: aspippal@gmail.com

P. Saxena, A. Srivastava (eds.), *Air Pollution and Environmental Health*,
Environmental Chemistry for a Sustainable World 20,
https://doi.org/10.1007/978-981-15-3481-2_9

9.1 Introduction

Air is the principal liable constituent of our environment that is prone to pollution (Agarwal 2009). Air pollution may be defined as an undesirable state of natural air being contaminated with harmful substances as a consequence of human activities and natural disasters (Manahan 1999). This state of the air is termed as air pollution. It may be also defined as *"an introduction of harmful substance and chemicals into the natural environment which can have undesirable effects on human life, climate and the ecosystem"*. Air pollution can be present in both indoor and outdoor environment and characterise in terms of physical, chemical and morphological properties (Taneja et al. 2008; Pipal et al. 2014; Pipal and Satsangi 2015; Saxena and Sonwani 2019). Apart from this, meteorological conditions, sources and spatial characteristics are the factors that play a vital role to contribute towards air pollution.

9.1.1 Indoor Air Pollution (IAP)

IAP also known as Household Air Pollution (HAP) is defined as pollution arising from pollutants released in homes or working places or any closed environment from the various sources (furniture, paints, pets, etc.) and other burning activities such as cooking, cigarette burning, consumption of fuels (biomass, dung, wood, leaves), dusting, burning of incense and candles etc. Indoor air pollution occurs when the building is not properly ventilated and the pollutants get accumulated and concentration becomes higher than outdoor air pollutants (Daly and Zannetti 2007).

9.1.2 Outdoor/Ambient Air Pollution

The pollution in which pollutants are released in outdoor atmosphere is known as outdoor/ambient air pollution. The most common source of ambient air pollution are man-made activities which include combustion of fossil fuels (coal, oil and gas) in industries, power stations, homes and road vehicles. In addition oxides of sulphur and nitrogen from volcanic eruption, biological decay, oceans, lightning strike, forest fires, Volatile Organic Compounds (VOCs) and pollen from plants, trees and PM from dust storm are known as natural sources (Saxena and Ghosh 2018, 2019).

9.2 Air Pollutants

The substances that are in excess amount in the air are known to be air pollutants. These substances in air are present in high enough concentration that could harm animals, humans, vegetation, and materials. They may exist in the form of solid, liquid, gas or PM in the atmosphere.

9.2.1 Classification of Air Pollutants

Air pollutants may be released directly in atmosphere and can be formed by the substances that already exist in environment. They may also be released from natural as well as anthropogenic sources. On the basis of formation, air pollutants may be categorised as primary and secondary air pollutants.

9.2.1.1 Primary Air Pollutants (Precursors)

Pollutants that are released directly into the atmosphere and can be formed on their own are known as primary pollutants. They can be emitted from anthropogenic, biological and geogenic sources. The main primary pollutants which affect the human being are:

- Carbon compounds like CO, CO_2, CH_4 and VOCs
- Nitrogen compounds (NO, NO_2 and NH_3)
- Sulphur compounds such as SO_2, H_2S
- Halogen compounds such as chloride (Cl^-), fluoride (F^-), bromide (Br^-)
- Solid or liquid form of PM and it is classified into many classes on the basis of aerodynamic diameter of the particles (Harrison 2000)

9.2.1.2 Secondary Air Pollutants

Secondary pollutants are substances that are formed in atmosphere from precursor gases via different chemical reactions as they are not emitted directly into the atmosphere. The main secondary pollutants which affect human beings are:

- NO_2 and HNO_3 formed from NO precursors via chemical reaction
- Photochemical oxidation of nitrogen oxides and VOCs leads to the formation of O_3
- Sulphuric acid droplets formed from SO_2, and nitric acid (HNO_3) droplets formed from NO_2

- Reactions of sulphuric acid (H_2SO_4) and nitric acid (HNO_3) droplets with NH_3 and water vapours leads to the formation of sulphates (SO_4^{2-}) and nitrates (NO_3^-) aerosols (i.e. NH_4SO_4 and NH_4NO_3)

9.3 Sources of Air Pollutants

Air pollutants present in the atmosphere can be released from both natural as well as anthropogenic sources that are discussed below.

9.3.1 Natural Sources

Natural sources of air pollutants include volcanic eruption which release dust, ash, and other gases, pollen grains from flowers, forest fires that are naturally caused by lightning. The emission of air pollutants from natural sources have short life period in atmosphere and also play important role in changing the composition of atmosphere (Falkowska 2015).

9.3.2 Anthropogenic (Human-Made) Sources

Man-made activities such as fuel and biomass burning, household combustion of coal; sewers and domestic drains emanating foul gases, agriculture equipment's, vehicle exhausts, factories, shipping, airplanes are known as anthropogenic sources. They produce many harmful primary pollutants such as carbon monoxide (CO), nitrogen dioxide (NO_2), nitrogen oxide (NOx), sulphur dioxide (SO_2), ammonia (NH_3), PM, VOCs along with secondary pollutants like SO_3, HNO_3, H_2SO_4, H_2O_2, O_3 and various organic components. The environmental air pollution is thus evil of all anthropogenic processes leading to the unfavourable alteration of the environment (Volkamer et al. 2006; Sonwani and Saxena 2016).

9.4 Effect of Air Pollutants on Human Health

The various particle and gaseous pollutants released from natural and anthropogenic sources results into different respiratory acute and severe diseases. When people are exposed to these pollutants, they would cause distinct adverse health effects. However, the effects of pollutants depend upon the concentration of pollutants as well as the exposure period. More the concentration and exposure time of pollutants, more will be the harmful effects on human health as well as climate change.

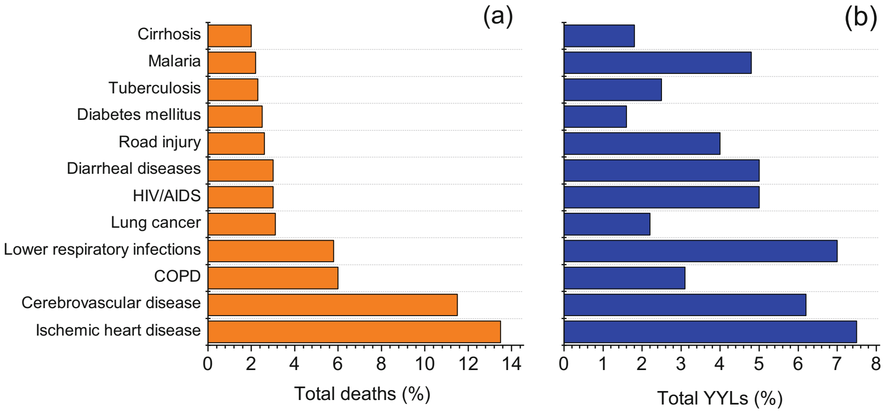


Fig. 9.1 (**a** and **b**) Leading causes of global deaths and premature deaths

In the year of 2012, 7 million deaths globally were caused due to both ambient and household air pollution in which ambient air pollution was responsible for 3.7 million deaths while household air pollution was attributable for 4.3 million deaths (WHO 2014a). In the year 2017, air pollution had taken away life of 12.4 lakh people in India. In Indian perspective, even some of the air pollutant levels were less than the permissible level but still they are causing health problems and subsequently, the loss of life expectancy would have been 1.7 years higher and declared as the leading risk factor for deaths (Saxena and Naik 2018). (https://economictimes.indiatimes.com/news/politics-and-nation/around-12-4-lakh-deaths-in-india-in-2017-attributable-to-air-pollution-study/articleshow/66972252.cms?from=mdr). Recently, the study conducted by Balakrishnan et al. (2019) indicated that 26.2% of global air pollution Disability Adjusted Life-years (DALYs) occurred in India in 2017.

Outdoor air pollution has been ranked among top 10 health risks in India as per assessments of global burden of disease (GBD). The study conducted by Institute for Health Metrics and Evaluation (IHME) found that outdoor $PM_{2.5}$ and O_3 lead to 0.695 million premature deaths and loss of 18.2 million healthy life years (IHME 2013). In order to assess health risk factor, it is found that outdoor air pollution ranked fifth in terms of mortality while overall health burden is on seventh rank. However, a million additional premature deaths in India have been caused from household air pollution due to solid fuel combustion.

Figure 9.1a and b shows the total deaths and Years of Life Lost (YLLs) (%) because of different diseases caused due to air pollutants. From Fig. 9.1a, it is inferred that highest proportion of deaths is because of ischemic heart disease followed by cardiovascular disease and lowest due to cirrhosis. In case of years of life lost (YLL) the same trend of deaths gained is seen (Fig. 9.1b). This statistical data on number of deaths and YLLs caused due to different diseases shown in above figures was obtained from Global Burden of Diseases (2010) report published by IHME (2013). According to the State of Global Air Report 2019, India is facing third leading risk factor for mortality accounting for 1.2 million deaths due to air

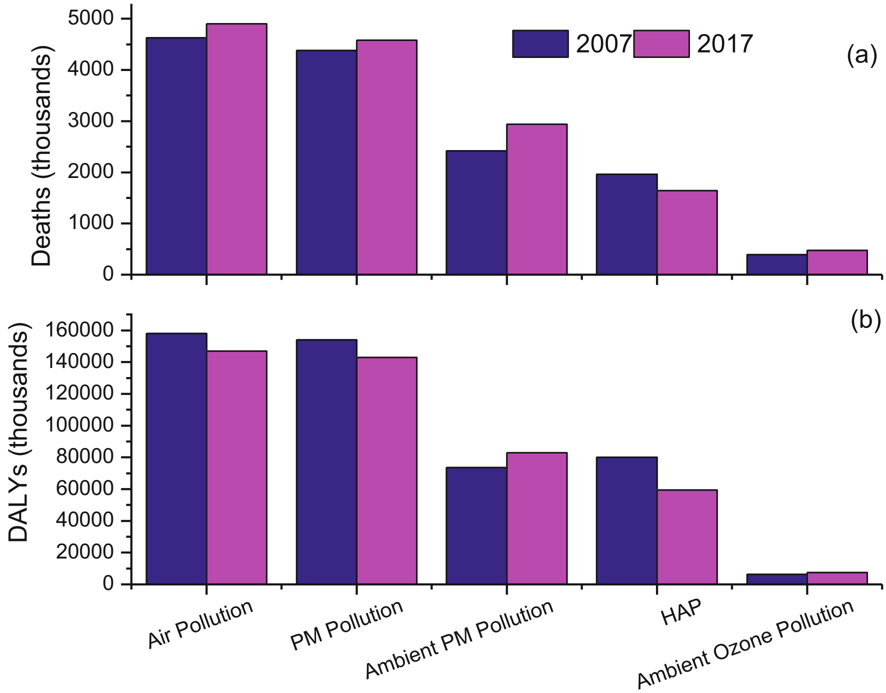


Fig. 9.2 (**a** and **b**) Number of deaths and DALYs due to different types of air pollution (all causes). *HAP* House hold air pollution

pollution. Further, the percentage distribution of various diseases indicates that 49% of deaths are attributed by COPD cases followed by lung cancer, diabetes, and ischemic heart diseases.

According to Global Burden Disease Study (2017) published by IHME (2018), the number of deaths attributed due to different types of air pollution was found to have increased except household air pollution which recorded a decrease of 320 thousands deaths (Fig. 9.2a) while the number of DALYs was found to be decreased in all type of pollution cases (i.e. air pollution, PM pollution, household air pollution), whereas in case of ambient PM pollution it has increased by 520 thousands (Fig. 9.2b).

The different types of air pollutants released from distinct sources and activities both in indoor and outdoor environment along with their health effects have been discussed in Table 9.1.

Table 9.1 Different pollutants, their sources and associated health effects

S. no.	Pollutants	Sources	Associated health effects
1.	Carbon monoxide (CO)	Fossil fuel combustion in vehicles, industries and power plants, fireplaces, cook tops, ovens, wood/coal stoves and tobacco smoke	Interference in the blood's ability to carry O_2, slows reflexes and causes drowsiness. It also leads to headache and stress on heart (Mofenson et al. 1984)
2.	Nitrogen dioxide (NO_2)	Same as carbon monoxide	It makes the body vulnerable to respiratory infections, lung diseases and may cause cancer (Chauhan et al. 1998)
3.	Sulphur dioxide (SO_2)	Same as carbon monoxide	Short-term exposure leads to breathing difficulties and obstructive airways. Long-term exposure causes chronic bronchitis, emphysema and respiratory illness (Chen et al. 2007)
4.	Volatile organic compounds (VOCs)	Glues, cleaning materials, fabric softeners, paints, moth repellents, deodorisers, pesticides	It affects eye, upper and lower respiratory irritation, nasal congestion, headache, rash, dyspnoea, epistaxis and may cause cancer (Berglund et al. 1992)
5.	Environmental tobacco smoke (ETS)	Cigarettes, cigars and pipes	It may cause diseases like acute respiratory symptoms, tuberculosis, asthma, etc. to non-smokers. Lung cancer, impaired breathing and cardiovascular disease in smokers (Yanbaeva et al. 2007)
6.	Particulate matter (PM)	Construction materials, unpaved roads, burning activities fields, smokestacks, cooking; combustion of candles, incense; heaters, and environmental tobacco smoke	PM causes a variety of respiratory problems and cardiovascular diseases. It also leads to cancer due to long-term exposure (Englert 2004)
7.	Heavy metals	Combustion processes, industrial activities, insecticides, manufacturing of steel, smelting, dust, combustion process, ETS	Kidney failure, abnormalities in skeleton system, different kinds of cancers such as liver, lung, nose, larynx and prostate, skin and lungs problem, anaemia, cardiovascular diseases, birth defects, asthma and chronic bronchitis, heart disorders (Mahurpawar 2015)
8.	Ozone (O_3)	O_3 formed from precursors released by road transport, power plants, industrial boilers, paint, dry cleaning, and other solvent uses	Respiratory and cardiovascular problems, premature mortality (Tager et al. 2005)
9.	Aerosols	Various natural and anthropogenic activities such as aerosol sprays, hair sprays, perfumes, solvents, glues, cleaning agents	It can cause asthma, skin irritation, breathing difficulty, cough, lung problems, headache, diarrhoea, cancer, effects infants and pregnant women (Poschl 2005)

9.5 Health Risk

Health risks are a measure of the chance that anyone has to experience health issues, due to exposure of toxic air pollutants (https://www3.epa.gov/airtoxics/3_90_024.html). In order to know health-related issue due to air pollutants, various tools and methods have been applied by different researchers at regional and global level.

9.5.1 *Estimation of Health Risk*

The health risk of air pollution in inhabitants is represented by the concentration–response function. It is typically based on estimates of Relative Risk (RR) and obtained from epidemiological studies. It outlines the possibility of adverse health outcome such as premature death, heart attack, asthma attack, emergency room visit, and hospital admission occurring in the population who is exposed to the higher level of air pollution as compared to the population that are at comparatively lower exposure level. It also defines the proportional increase in the evaluated health outcome related with an increase in the concentration of pollutants (μg/m^3 or ppb) (Katsouyanni 2003). However, RR also estimates the health risk only in a defined population and for a specific person, i.e. no individual health risk can be measured (McAuley and Hrudey 2006; Australian Department of Health 2012).

9.5.2 *Quantification of Health Impact*

Number of attributable deaths or cases of diseases, years of life lost (YLL), disability-adjusted life years (DALYs) and change in life expectancy occur due to total exposure to air pollution or change in exposure and are the terms that are used to describe air pollution health risk assessment (AP-HRA) (WHO Regional Office for Europe 2016). These matrices collect different types of health impact and can also be used to highlight the different aspects of the population with respect to health (Murray and Lopez 2013).

9.5.2.1 Number of Attributable Deaths or Cases of Diseases

It is calculated as the difference in number of deaths or cases of diseases between the incidence/rate at the exposure measured over a specific period at baseline exposure (WHO Regional Office for Europe 2016).

9.5.2.2 Years of Life Lost (YLL)

It is a measure of the years of life lost as a result of premature death. In general term, the calculated number of deaths attributable to changes in exposure to air pollution is multiplied by the standard life expectancy at the age at which death occurs (WHO 2014b).

9.5.2.3 Years Lost Due to Disability (YLD)

It measures the years of lost due to disability and estimated by multiplying the number of incident cases of a particular health outcome in a particular period. It is measured by the average duration of the case until remission or death (years) and a disability weight factor that reflects the severity of the disease on a scale from 0 (perfect health) to 1 (dead) (WHO 2014b).

9.5.2.4 Disability-Adjusted Life Years

One DALY is one lost year of healthy life. The sum of DALYs across a population—the burden of disease can be thought of as a measurement of the gap between actual health status and an ideal situation in which the entire population lives to an advanced age, free of disease and disability. Total DALYs for a particular disease or health condition in a population are calculated as the sum of YLL and YLD (Murray and Lopez 2013; WHO 2014b).

9.5.3 Health Risk Assessment (RA)

RA is the process used by various scientists, researchers and government agencies to estimate the increased risk of health problems in humans exposed to toxic substances. The RA approach outlined by the WHO in the Environmental Burden of Disease (EBD) series (Prüss-Üstün et al. 2003; Ostro 2004) includes the following steps:

1. Evaluation of the air exposure of the population through data obtained from air model or monitoring networks. A target concentration is also required to determine the attributable disease or the potential gains of a management plan.
2. Ample number of persons exposed to air pollutants.
3. Baseline data of incidence of the adverse health outcomes associated with air pollutants (like mortality rate).
4. Concentration–response functions (CRFs) related to the incidence of adverse health effects.

There are various steps and methods which are used by various researchers in their study to estimate the health risk assessment of different air pollutants especially heavy metals.

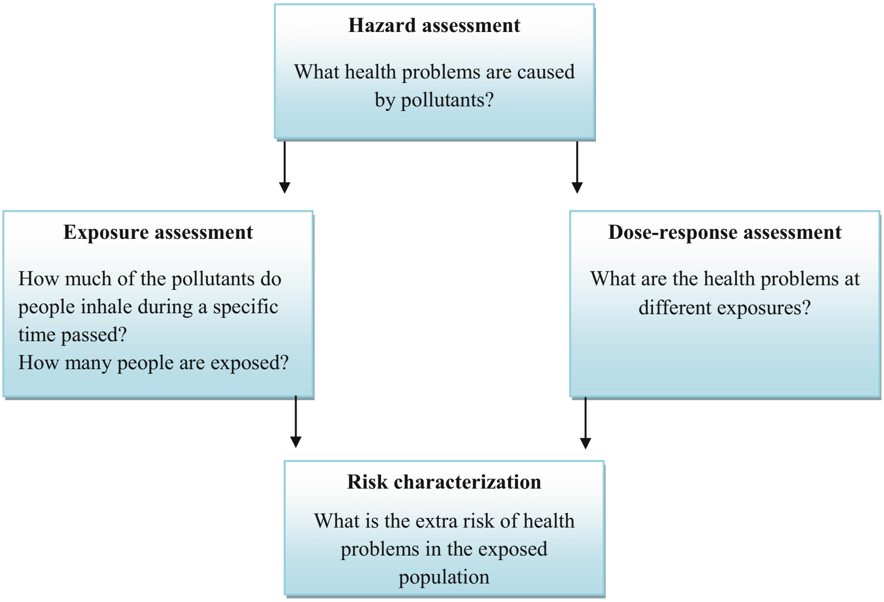


Fig. 9.3 Different steps given by USEPA for risk assessment

The entire process used for risk assessment is divided into four steps by US Environmental Protection Agency (USEPA) as depicted by the flow chart (Fig. 9.3).

The mechanism of risk assessment and various factors which are responsible for health issues due to air pollutants in different ways are mentioned in A Citizen's Guide (1991), Risk Assessment for Toxic Air Pollutants published as EPA 450/3-90-024. March 1991.

In the year 1992, USEPA produced different equations in order to calculate the health risk associated with PM-bound metal. It is known that the time of exposure to pollutants is a major factor to determine the health hazard due to that pollutant. Firstly, the average daily dose (A_{DD}) is calculated with the Eq. (9.1):

$$A_{DD} = C \times InhR \times ED/BW \times AT \tag{9.1}$$

where C = concentration ($\mu g/m^3$) of contaminants in air
InhR = average inhalation rate
ED = exposure duration;
USEPA has given standard value of all these terms
BW = body weight
AT = average exposure time

With the help of A_{DD} the non-carcinogenic risk of metals can be calculated by Eq. (9.2):

$$HQ = A_{DD}/Rfc \tag{9.2}$$

Rfc = Reference concentration of metals or other toxicity value.

When HQ value is found to be >1 the metal causes non-carcinogenic risk while HQ value <1, it does not cause any risk.

The carcinogenicity of four metals (Cr, Ni, Cd and Pb), i.e. Lifetime cancer risk (R_{ic}) can be calculated as:

$$R_{ic} = C \times ED \times UR/\text{Average life} \tag{9.3}$$

UR = unit risk.

Using the above formula, Kushwala et al. 2012; Li et al. 2015; Wang et al. 2015; Fang et al. 2013; Jan et al. 2018 and many others have calculated health risk of various metals associated with PM exposure.

Along with the above method the health risk analysis (both for adult and child) for receptor due to metal-bound PM can also be done by the following formulas provided by USEPA 2009.

The exposure concentration can be determined by using Eq. (9.4):

$$EC = \quad (CA \times ET \times EF \times ED)/AT \tag{9.4}$$

where: EC (μg/m^3) = exposure concentration
CA (μg/m^3) = contaminant concentration in air
ET = exposure time usually 24 h/day
EF (days/year) = exposure frequency (350 days/year)
ED (years) = exposure duration
AT = average time (period over which exposure is averaged)

Risk characterisation for a person who is exposed to air pollutants through inhalation pathway can be examined by calculating Hazard Quotient (HQ) (Eq. 9.5):

$$HQ = EC/(\text{Toxicity Value} \times 1000\ \mu g/mg) \tag{9.5}$$

while Eq. (9.6) helps to calculate Excess Lifetime Cancer Risk (ELCR) for non-carcinogenic and carcinogenic risk, respectively:

$$ELCR = IUR \times EC \tag{9.6}$$

Chronic Reference concentration (RfC) (mg/m^3) and IUR (μg/m^3) = Inhalation Unit Risk.

Among various epidemiological studies in which these equations have been used for the estimation of health risk of metals associated with PM include Hieu and Lee (2010), Taner et al. (2013), and Rohra et al. (2018). These researchers have estimated health risk of different metals in various pathways and their findings with respect to health risk caused due to chemical constituent associated with PM.

Apart from estimation process, health risk is also accessed by various tools that have been devised by various organisations. The details of some of the tools which are used for health risk assessment from ambient air pollution are provided in Table 9.2 (WHO Regional office 2016).

Table 9.2 Different tools developed by various organisations for health risk assessment caused due to air pollutants

Tools	Developing institution	Geographical scope	Health endpoint addressed
Air Counts	Abt Associates	Global (42 cities, additional 3000 under development)	Mortality
AirQ2.2 (update under development	World Health Organization	Population specified by size, mortality and morbidity characteristics	Mortality and morbidity
Aphekom	French Institute of Public Health Surveillance	Global (current version focuses on Europe)	Mortality and morbidity
Economic Valuation of Air Pollution (EVA)	Aarhus University	Northern hemisphere, continental (e.g. Europe), local areas	Mortality and morbidity
EcoSense	University of Stuttgart	Europe	Mortality and morbidity
Environmental Benefits Mapping and Analysis Program—Community Edition (BenMAP-CE)	USEPA	Continental USA and China predefined	Mortality and morbidity
EBD Assessment tool for ambient air pollution	World Health Organization	Global	Mortality and morbidity
GMAPS	World Bank	Global	Mortality and morbidity
IOMLIFET	Institute of Occupational Medicine	Places where background mortality data and measured and predicted pollutant concentrations level	Mortality and morbidity
Rapid Co-benefits Calculator	US Environmental Protection Agency, Stockholm Environment Institute	Underdevelopment for all countries globally	Mortality
SIM-Air	Urban Emissions	Asia, Africa, Latin America	Mortality
TM5-FASST	European Commission Joint Research Centre	Global	Mortality and morbidity
ICRP	International Commission on Radiological Protection	Global	Morbidity

USEPA, EBD

The health risk of air pollutants accessed by estimation process (using values or equations and software methods) are not as accurate as the function of epidemiological studies. As these methods are based on the values given by various organisations from various places having different environmental conditions they are not perfect but these results are useful to researcher for further assessment of risk concerned with exposure of toxic air pollutants. Moreover, the factors which are used for health estimation help the government bodies to set regulatory standards to reduce people's exposures from toxic air pollutants and assessment of health issues.

There are various standards set out by national and international bodies for ambient air that provide the concentration limit of exposure according to time (annual and 24 h) of toxic pollutants. Environmental protection agency (EPA) has assigned a specific air quality index (AQI) category and range to make it easier for people to understand quickly whether air pollution is reaching unhealthy levels in their communities (Table 9.3). For example, the mass concentration of fine particle ($PM_{2.5}$) in the range of 36–66 μg m^{-3} is unhealthy for sensitive groups, while 66–150 μg m^{-3} means that conditions may be unhealthy for everyone. The overall average concentrations of PM ($PM_{2.5}$: 98 μg m^{-3} and PM_{10}: 148 μg m^{-3}) in the study conducted by Pipal et al. (2019) was found to be much higher than its standards and AQI values over atmosphere in Pune. This may cause respiratory problems and health issue to people who have been continuously affected by these pollutants along with climatic problems. This may be due to less application of emission control measures, which are responsible for respiratory problems and human health. The CPCB and other government monitoring programmes also reported that concentration of PM in Indian cities is higher than the common guidelines. However, no such standards exist till date for indoor air. Such standards should be framed for indoor air quality as people spend majority of their time in indoor and exposed to various toxic pollutants which lead to acute and severe diseases.

In this connection, some relevant research work done at national and international level so far which focused on risk assessment and epidemiology are discussed and examined; their major findings and outcomes have been discussed in Table 9.4. Exposure to toxic pollutants results in increased health risk and leads to different acute and severe diseases. However, concentration and exposure time are also important factors for health risk. On the basis of assessment methods and tools,

Table 9.3 Air quality index and recommendations

AQI level	AQI values	$PM_{2.5}$	PM_{10}
Good	0–50	0–15	0–50
Moderate	51–100	16–35	51–155
Unhealthy for sensitive groups	101–150	36–65	155–254
Unhealthy for sensitive groups	151–200	66–150	255–354
Very unhealthy	201–300	>150	>354

Air quality index: a guide to air quality and health (Anderson et al. 2012)

Table 9.4 List of epidemiological studies that relate pollutants with health risk

References	Year	Region	Findings
American Industrial Hygiene Association (AIHA)	1968	US	Exposure to formaldehyde is toxic which results in strong irritating to eyes, skin, nose, respiratory tract or mucous membrane
Pandey et al.	2005	India	Exposure to NO_2 and Suspended Particulate Matter (SPM) is more harmful as compared to SO_2. The average health risk possessed by NO_2 and SPM was found to be 22.11 and 16.13 times more than that of SO_2 for all age categories
Brook et al.	2010	US	Cardiovascular effects of PM exposure have been linked to oxidative stress (OS), pulmonary and systemic inflammation, endothelial cell dysfunction, atherosclerosis and altered cardiac autonomic function (ACAF)
WHO	2011	–	Respiratory system and lungs function can be affected due to exposure of SO_2, causing various problems of coughing, mucus secretion, aggravation of asthma and chronic bronchitis that "make people more prone to infections of the respiratory tract". Also reported that SO_2 is the main cause of irritations of the eyes. Long-term exposure to NO_2 leads to proportional increase in the symptoms of bronchitis in asthmatic children. Ozone initiates asthmatic problems, reduces lung function and leads to lung diseases
Yeh et al.	2011	Taiwan	Sewer gas releases hazardous pollutants, i.e. benzene and trichloromethane. Its cancer risks reached the ranges of $2.77–3.98 \times 10^{-3}$ and $29.74–42.7 \times 10^{-3}$, respectively, for workers without protective equipment as the cancer risk assessment
Wichmann and Voyi	2012	South Africa	Exposure to PM is strongly associated with respiratory, cardiovascular and cerebrovascular risks
Sancini et al.	2014	Italy	Systematic adverse effects are related with repeated instillations of $PM_{2.5}$. Susceptible population which include elder people and people who have unrecognised coronary artery or structural heart disease are more prone to $PM_{2.5}$
Maji et al.	2017	India	The number of morbidity cases (except COPD) have increased by 30% from 1992–2002 to 2003–2013 and PM_{10} resulted in the excess number of mortality and morbidity as compared to gaseous pollutants

(continued)

Table 9.4 (continued)

References	Year	Region	Findings
Chen et al.	2017	China	Home environment alone contributes 96% of the total average health risk. The carcinogenic health risks were found to be in the bedrooms (6.8 × 10^4 for male, 7.4 × 10^4 for female) and living rooms (4.4 × 10^4 for male, 4.0 × 10^4 for female) with long exposure time. These all values were higher than the acceptable risk threshold
Khaniabadi et al.	2019	Iran	With increase in each 10 μg/m^3 concentration of PM_{10} the risk of mortality for all causes and cardiovascular diseases increase by 0.74% and 0.80%, respectively while the risk of hospitalisations for respiratory disease and cardiovascular diseases increased by 0.80% and 0.90%. In case of SO, increases in each of 10 μg m, the risk for cardiovascular mortality increased by 1.2%. while the case of hospitalisations for COPD and acute myocardial infarction increased by 0.44% and 1.0%, respectively
Satsangi et al.	2014	India	In silico study suggested that Ni actively formed coordination complex with histone proteins by maintaining strong hydrogen bonding interactions with Aspartic acid and Glutathione. The structural consequence of Ni in nucleosomal proteins and assessment epigenetic changes ultimately causing lung and nasal cancer

various national and international studies have been conducted which prove the relation of health risk due to exposure of pollutants.

9.6 Approaches to Improve Ambient Air Quality

In order to improve ambient air quality different measures can be taken. As is well known, plants are able to purify the air but reckless cutting of plants has led to the rise in the problem of ecological imbalance and also hinder in the process of purification of air. Along with this, source control is also an important step that helps in improving ambient air quality. Some of the common steps to control or manage pollutant concentration in the atmosphere can be taken by the citizens of a country who can help a lot in controlling air pollution which results to improve air quality.

9.6.1 Source Control

Source control is one of the most effective ways to improve ambient air quality. Reduction in the number of source that causes air pollution can effectively reduce the pollutants level in the atmosphere. The different sources that cause ambient air pollution in the Indian context (Guttikunda et al. 2014) and their control have been discussed below.

9.6.1.1 Transportation

It plays a vital role in urbanisation and industrialisation in which road transportation is one of the major growing transports in developing country and also plays a vital role in Indian economy. The fossil fuels combustion in vehicles release a lot of pollutants leading to the deterioration of air quality. Of the total, transport sector contributes 14% of the air pollution in India (https://en.wikipedia.org/wiki/Air_pollution_in_India) which is an abundant number. So, vehicles that are run by Compressed Natural Gas (CNG), biodiesel, solar technology and electricity must be used instead of those vehicles in which combustion of fossil fuels occur, specially diesel. However, in recent years, the use of CNG in vehicles has increased. Still, other steps must also be taken by the government in order to reduce the contribution of pollution due to transportation. Current effective policies related to transportation are needed to be implemented with strictness and if needed new policies must be formed in order to tackle air pollution.

Figure 9.4 shows that the largest contributor to air pollution is dust and construction followed by diesel generators and lowest is from domestic cooking (https://en.wikipedia.org/wiki/Air_pollution_in_India).

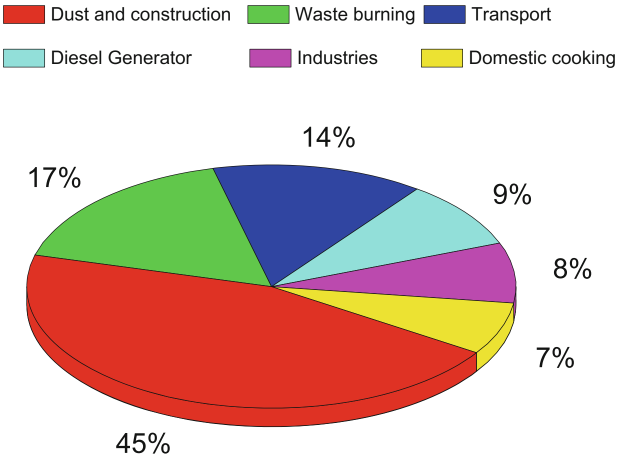


Fig. 9.4 Different sources contribute to air pollution

9.6.1.2 Waste Burning

Open waste burning contributes 17% of the air pollution in India. Waste collected by Municipal Corporation is usually burnt in open spaces which lead to emission of toxic pollutants such as SOx, NOx, PM, VOCs and other pollutants. There is no fixed procedure for management of these wastes and also landfill facilities are not maintained in a proper manner which is the main cause for burning of waste especially in small and medium cities. There is an urgent requirement for waste management programme that helps to tackle the problem related to air pollution.

9.6.1.3 Industries

Industries contribute 8% of air pollution in India. With increase in modernisation large number of industries has been set up which produce large number of pollutants along with goods. The percentage contribution of air pollution from industries in India alarms that immediate and effective measures to be taken for controlling the toxic pollutants emission from industries. Smoke from chimneys comes out without any purification and mixes in clean air leading to deterioration of ambient air quality day by day. For that air purification technique such as precipitators must be employed on the ejection point of smoke from chimneys in industries so that clean air may come out. An environment monitoring device known as Aurassure has been employed for the monitoring of different air quality parameters from different locations and it also helps industries to know the level of their pollutants emission.

9.6.1.4 Diesel Generator

The supply of electricity in India is also a big deal and due to improper supply of electricity people use diesel generators in homes and other working places that lead to emission of different pollutants in the air. Diesel generators contribute about 9% of air pollution in India which can be stopped by improving the electricity supply in India especially in small cities. However, the patented Chakr Innovation is a very important intervention that converts diesel soot from generator into purified carbon-based pigment after removing heavy metals and carcinogens from soot. The purified carbon-based pigment can be further used as ink and paints for various purposes.

9.6.1.5 Dust and Construction

Dust and construction activities are the largest contributor of air pollution which shares 45% of the air pollution in India. With increase in modernisation, massive construction work leads to large number of pollutants. The contribution of dust and construction towards air pollution in India is in such an alarming state that effective measures are needed to be taken for the control of pollutants from these types of activities (Sonwani and Kulshrestha 2018, 2019). According to a report of CPCB, particles released from wear and tear of tyres and brakes and dusts composed of particles from road's material, pavement and street furniture is a major concern for many cities in India. Unpaved roads lead to increase in dust loading (CPCB 2010). Intervention should also be taken in order to control dust as it contributes to 30–40% of course particulate matter (PM_{10}) pollution in most Indian cities (CPCB 2010; Guttikunda and Jawahar 2012).

9.6.1.6 Coal: Fired Power Plant

It is known that coal is usually used for the production of electricity in India. According to World Bank collection of development indicators, in the year 2014 coal shared 75.08% of the total source used for total electricity production in India. The amount of pollutants released from the combustion of coal used for electricity generation can be understood. According to a study, Indian coal that is used for electricity production comprises low level of sulphur as compared to other coal and leads to production of ash in large amount which finally may act as a contributor of PM emission (Pant and Harrison 2012). The production of electricity from renewable source and hydroelectricity should be focused rather than electricity production from coal to achieve better outcomes.

9.6.2 Other Measures

Use of public transportation instead of private vehicles can help in reducing air pollutant especially PM concentration which is emitted from combustion of fossil fuel and wearing and tearing of tyres.

9.6.3 Purification of Air

There are various air cleaning tools available that can be employed to purify the polluted air. China has recently established world's biggest (100-m-high) air purification tower in Xian, Shaanxi province that helps to tackle problem of smog. This is a good initiative to improve air quality. Such steps should also be taken in large scale by Indian government bodies and other agencies towards the most polluted areas and heavy traffic junctions. We know that Indian government has already started a start-up named "Kurin Systems" to develop air purifiers that can be installed in Delhi in order to improve the air quality. If it will be an effective device to improve the air quality of Delhi, then government can approach other cities also.

9.6.4 Implication of Policies

The harmful effects of air pollution have been confirmed by various studies and concern that there are several policies which have been assigned by the government to attain the goal of healthy air. These policies are required to be implemented in the right way to achieve the required goal.

9.7 Approaches to Improve Air Quality in Indoor Environment

The problem of air pollution is not only concerned with ambient air but it is a major topic of concern for indoor air pollution. People spend majority of their time indoor so it cannot be taken lightly. A number of studies have been conducted in indoor environment which shows that the concentration level of pollutants are enough to lead to ill health effects.

The following are three basic strategies used for improving indoor air quality:

1. Source control
2. Improved ventilation
3. Air cleaners

9.7.1 Source Control

The elimination of sources that cause air pollution is the best way to deal with the problem of poor indoor air quality. Sources that release asbestos can be sealed or enclosed; combustion process can be controlled; gas stoves can be maintained so that complete combustion can take place which lead to decrease in the amount of emissions. Source control is the most effective solution for indoor air quality problems in home.

9.7.1.1 Domestic Cooking

Domestic cooking is the main source of indoor air pollution and contributes to 7% of the air pollution of India. In 2011, 35% of urban and 89% of rural household depend on non-LPG fuels according to census of India (Census-India 2012).

The combustion of biomass leads to emission of pollutants which cause various acute and severe diseases. Most of the women are not aware about the harmfulness of biomass fuel smoke and some are aware but still they cannot use LPG for cooking purpose due to economic condition. In this connection, Indian government has started a scheme called Ujjwala Yojana on May 1, 2016 at Ballia which support free cooking LPG connection for the families who are living below the poverty line. Use of clean fuel will improve women's health who has to cook on unclean fuel. (http://yojana.gov.in/June%20Yojana%20Final.pdf).

9.7.2 Ventilation Improvements

The concentration of pollutant can be diluted by ventilating (opening door and windows and using exhaust fans) in homes. However, it is not always helpful as the pollutants from outdoor air seep in indoor and increase the level of pollutants.

Air quality of indoor environment can be effectively improved by introducing outdoor air as it helps in dispersion of indoor pollutants concentration. There are several ways that aid in introduction of good air in indoor spaces; such as:

- Natural ventilation through doors and windows.
- Mechanical means, such as Heating, Ventilation and Air Conditioning (HVAC) system uptake outdoor air which helps to promote good indoor air quality.
- Infiltration is the process which involves the circulation of outdoor air from opening in house, joints and cracks in walls, ceilings and floors; and also doors and windows.

9.7.3 Air Cleaners

Air cleaners with different technologies and sizes are available in the market which help to reduce the indoor pollutants to a large extent. These air cleaners are available at the cost that can be afforded by low income families also. The effectiveness of these air cleaners depends on the amount of air drawn from cleaning or filtering element and efficiency to collect pollutants from indoor air (https://www.epa.gov/indoor-air-quality-iaq/improving-indoor-air-quality). In this regard; one of the campaign studies conducted at Agra by using two air purifiers found that they are able to reduce 12–73% of PM from indoor chamber.

9.8 Conclusion

The problem of air pollution has increased with great pace from the times of urbanisation, industrialisation and modernisation. From the various reports and literature it is concluded that air pollution leads to large number of diseases and deaths worldwide. Pollutants are not only harmful for human health but they also affect our environment and other living organism. Therefore, the pollutants released in indoor and outdoor environment from various sources and activities are needed to be controlled. Urbanisation and modernisation are indicators of the development of a country but it seems to be a sin when they lead to ill health and deaths. As the awareness about harmfulness regarding air pollution has increased, the management of air pollutants has been introduced. There are large number of pollution control tools which are available these days that are widely used to purify the air but these will not be fruitful until we stop damaging our environment. Source control is the best solution to tackle the problem of air pollution. The study conducted recently in China by Huang et al. (2018) has confirmed that reduction in air pollution can also reduce the cases of mortality and YLL. Overall, our findings revealed that the ambient and indoor air pollutants concentration levels are above the prescribed standard values in many cities and associated with increased risk of various health issues. Therefore, the effective policies to reduce emissions sources are obviously preferable and evidences support the effectiveness of individual actions to reduce exposure levels and health risks of air pollutants.

References

Agarwal SK (2009) Air pollution. APH Publishing Corporation, New Delhi

AIHA (1968) Community air quality guides, aldehydes. Am Ind Hyg Assoc J 29:404–512

Anderson JO, Thundiyil JG, Stolbach AJ (2012) Clearing the air: a review of the effects of particulate matter air pollution on human health. J Med Toxicol 8(2):166–175

Australian Department of Health (2012) Environmental health risk assessment. Guidelines for assessing human health risks from environmental hazards [online]. Canberra, Australian Government Department of Health

Balakrishnan K, Dey S, Gupta T, Dhaliwal RS, Brauer M, Cohen AJ, Sabde Y (2019) The impact of air pollution on deaths, disease burden, and life expectancy across the states of India: the Global Burden of Disease Study 2017. Lancet Planet Health 3(1):26–39

Berglund B, Brunekreef B, Knöppe H, Lindvall T, Maroni M, Mølhave L, Skov P (1992) Effects of indoor air pollution on human health. Indoor Air 2(1):2–25

Brook RD, Rajagopalan S, Pope CA III, Brook JR, Bhatnagar A, Diez-Roux AV, Peters A (2010) Particulate matter air pollution and cardiovascular disease: an update to the scientific statement from the American Heart Association. Circulation 121(21):2331–2378

Census-India (2012) Census of India, 2011. The Government of India, New Delhi, India

Chauhan AJ, Krishna MT, Frew AJ, Holgate ST (1998) Exposure to nitrogen dioxide (NO_2) and respiratory disease risk. Rev Environ Health 13(1–2):73–90

Chen TM, Kuschner WG, Gokhale J, Shofer S (2007) Outdoor air pollution: nitrogen dioxide, sulfur dioxide, and carbon monoxide health effects. Am J Med Sci 333(4):249–256

Chen X, Li F, Liu C, Yang J, Zhang J, Peng C (2017) Monitoring, human health risk assessment and optimized management for typical pollutants in indoor air from random families of university staff, Wuhan city, China. Sustainability 9(7):1115–1127

CPCB (2010) Air quality monitoring, emission inventory and source apportionment study for Indian cities. Central Pollution Control Board, the Government of India, New Delhi, India

Daly A, Zannetti P (2007) An introduction to air pollution–definitions, classifications, and history. In: Zannetti P, Al-Ajmi D, Al-Rashied S (eds) Ambient air pollution. The Arab School for Science and Technology and The Environ Comp Institute, Freemont, CA, pp 1–14

Englert N (2004) Fine particles and human health—a review of epidemiological studies. Toxicol Lett 149(1–3):235–242

Falkowska L (2015) Environmental characteristics of gaseous pollutants and related adverse health effects. In: Pastuszka JS (ed) Synergic influence of gaseous particulate, and biological pollutants on human health. CRC Press, Boca Raton, pp 3–38

Fang W, Yang Y, Xu Z (2013) PM10 and PM2.5 and health risk assessment for heavy metals in a typical factory for cathode ray tube television recycling. Environ Sci Technol 47:12469–12476

Guttikunda SK, Jawahar P (2012) Application of SIM-air modeling tools to assess air quality in Indian cities. Atmos Environ 62:551–561

Guttikunda SK, Goel R, Pant P (2014) Nature of air pollution, emission sources, and management in the Indian cities. Atmos Environ 95:501–510

Harrison RM (ed) (2000) Pollution causes, effects and control, IV edn. Royal society of chemistry, Paston Pre Press, Beccles Suffolk

HEI (Health Effects Institute) (2019) State of Global Air 2019. A special report on global exposure to air pollution and its disease burden. Health Effects Institute, Boston, MA

Hieu NT, Lee BK (2010) Characteristics of particulate matter and metals in the ambient air from a residential area in the largest industrial city in Korea. Atmos Res 98:526–537

Huang J, Pan X, Guo X, Li G (2018) Health impact of China's air pollution prevention and control action plan: an analysis of national air quality monitoring and mortality data. Lancet Planet Health 2(7):313–323

IHME (2013) The global burden of disease 2010: generating evidence and guiding policy. Institute for Health Metrics and Evaluation, Seattle, USA

IHME (2018) Findings from the global burden of disease study 2017. Institute for Health Metrics and Evaluation, Seattle, WA

Jan R, Roy R, Yadav S, Satsangi PG (2018) Chemical fractionation and health risk assessment of particulate matter-bound metals in Pune, India. Environ Geochem Health 40:255–270

Katsouyanni K (2003) Ambient air pollution and health. Br Med Bull 68(1):143–156

Khaniabadi YO, Sicard P, Takdastan A, Hopke PK, Taiwo AM, Khaniabadi FO, Daryanoosh M (2019) Mortality and morbidity due to ambient air pollution in Iran. Clin Epidemiol Global Health 7(2):222–227. https://doi.org/10.1016/j.cegh.2018.06.006

Kushwala R, Srivastava A, Jain V (2012) Human exposure to particulate matter and their risk assessment over Delhi, India. Natl Acad Sci Lett 35(6):497–504

Li T, Wang Y, Li WJ, Chen JM, Wang T, Wang WX (2015) Concentrations and solubility of trace elements in fine particles at a mountain site, southern China: regional sources and cloud processing. Atmos Chem Phys 15:8987–9002

Mahurpawar M (2015) Effects of heavy metals on human health. Int J Res-Granthaalayah 1(7)

Maji KJ, Dikshit AK, Chaudhary R (2017) Human health risk assessment due to air pollution in the megacity Mumbai in India. Asian J Atmos Environ 11(2):61–70

Manahan SE (1999) Environmental chemistry, VII edn. CRC Press, Lewis Publishers

McAuley C, Hrudey SE (2006) Towards meaningful stakeholder comprehension of sour gas facility risk assessments. J Environ Eng Sci 5(1):1–11

Mofenson HC, Caraccio TR, Brody GM (1984) Carbon monoxide poisoning. Am J Emerg Med 2 (3):254–261

Murray CJ, Lopez AD (2013) Measuring the global burden of disease. N Engl J Med 369 (5):448–457

Ostro B (2004) Outdoor air pollution: assessing the environmental burden of disease at national and local levels. World Health Organization, Geneva. http://www.who.int/quantifying_ehimpacts/publications/ebd5/en/

Pandey JS, Kumar R, Devotta S (2005) Health risks of NO_2, SPM and SO_2 in Delhi (India). Atmos Environ 39(36):6868–6874

Pant P, Harrison RM (2012) Critical review of receptor modelling for particulate matter: a case study of India. Atmos Environ 49:1–12

Pipal AS, Satsangi PG (2015) Study of carbonaceous species, morphology and sources of fine (PM2.5) and coarse (PM10) particles along with their climatic nature in India. Atmos Res 154:103–115

Pipal AS, Tiwari S, Satsangi PG, Taneja A, Bisth DS, Srivastava AK, Srivastava MK (2014) Sources and characteristics of carbonaceous aerosols at Agra “World heritage site” and Delhi “capital city of India”. Environ Sci Pollut Res 21:8678–8691

Pipal AS, Singh S, Satsangi GP (2019) Study on bulk to single particle analysis of atmospheric aerosols at urban region. Urban Clim 27:243–258

Poschl U (2005) Atmospheric aerosols: composition, transformation, climate and health effects. Review on atmospheric chemistry. Angew Chem Int Ed 44:7520–7540

Prüss-Üstün A, Mathers C, Corvalán C, Woodward A (2003) Introduction and methods: assessing the environmental burden of disease at national and local levels. Environmental burden of disease series No. 1, World Health Organization Protection of the Human Environment Geneva 2003

Rohra H, Tiwari R, Khandelwal N, Taneja A (2018) Mass distribution and health risk assessment of size segregated particulate in varied indoor microenvironments of Agra, India – a case study. Urban Clim 24:139–152

Sancini G, Farina F, Battaglia C, Cifola I, Mangano E, Mantecca P, Palestini P (2014) Health risk assessment for air pollutants: alterations in lung and cardiac gene expression in mice exposed to Milano winter fine particulate matter ($PM_{2.5}$). PLoS One 9(10):109685

Satsangi PG, Yadav S, Pipal AS, Kumbhar N (2014) Characteristics of trace metals in fine ($PM_{2.5}$) and inhalable (PM_{10}) particles and its health risk assessment along with in-silico approach in indoor environment of India. Atmos Environ 92:384–393

Saxena P, Ghosh C (2018) Sustainable way to mitigate ozone pollution by reducing biogenic Vocs through landscape management programme. IJETT 56(2):87–91

Saxena P, Ghosh C (2019) Establishing correlation between abiotic stress and isoprene emission of selected plant species. In: Emerging issues in ecology and environmental science. Springer, Cham, pp 43–65

Saxena P, Naik V (eds) (2018) Air pollution: sources, impacts and controls. CABI

Saxena P, Sonwani S (2019) Primary criteria air pollutants: environmental health effects. In: Criteria air pollutants and their impact on environmental health. Springer, Singapore, pp 49–82

Sonwani S, Kulshrestha U (2018) Morphology, elemental composition and source identification of airborne particles in Delhi, India. J Indian Geophys Union 22(6):607–620

Sonwani S, Kulshrestha UC (2019) PM 10 carbonaceous aerosols and their real-time wet scavenging during monsoon and non-monsoon seasons at Delhi, India. J Atmos Chem 76:171–200

Sonwani S, Saxena P (2016) Identifying the sources of primary air pollutants and their impact on environmental health: a review. IJETR 6(2):111–130

Tager IB, Balmes J, Lurmann F, Ngo L, Alcorn S, Künzli N (2005) Chronic exposure to ambient ozone and lung function in young adults. Epidemiology:751–759

Taneja A, Saini R, Masih A (2008) Indoor air quality of houses located in the urban environment of Agra, India. Ann N Y Acad Sci 1140:228–245

Taner S, Pekey B, Pekey H (2013) Fine particulate matter in the indoor air of barbeque restaurants: elemental compositions, sources and health risks. Sci Total Environ 454–455:79–87

U.S. Environmental Protection Agency (1992) Guidance for data usability in risk assessment, OSWER 9285.7-09

USEPA (2009) Risk assessment guidance for superfund: volume I – Human Health Evaluation Manual (Part F, Supplemental Guidance for Inhalation Risk Assessment). Washington, DC

Volkamer R, Jimenez JL, San Martini F, Dzepina K, Zhang Q, Salcedo D, Molina MJ (2006) Secondary organic aerosol formation from anthropogenic air pollution: rapid and higher than expected. Geophys Res Lett 33(17):L17811

Wang Y, Hu L, Lu G (2015) Health risk assessments based on existing data of arsenic, chromium, lead, and zinc in China’s air. Hum Ecol Risk Assess 21:560–573

WHO (2014a) WHO news release. 7 million premature deaths annually linked to air pollution. Geneva. http://www.who.int/mediacentre/news/releases/2014/air-pollution/en/

WHO (2014b) Health statistics and information systems. Metrics: disability-adjusted life year (DALY) [online]. World Health Organization, Geneva. http://www.who.int/healthinfo/global_burden_disease/metrics_daly/en/

WHO (2016) Health risk assessment of air pollution – general principles. Copenhagen: WHO Regional Office for Europe, Geneva

Wichmann J, Voyi K (2012) Ambient air pollution exposure and respiratory, cardiovascular and cerebrovascular mortality in Cape Town, South Africa: 2001–2006. Int J Environ Res Public Health 9(11):3978–4016

World Health Organisation (WHO) (2011) Air quality and health. Fact sheet no. 313. http://www.who.int/mediacentre/factsheets/fs313/en/index.html

Yanbaeva DG, Dentener MA, Creutzberg EC, Wesseling G, Wouters EF (2007) Systemic effects of smoking. Chest 131(5):1557–1566

Yeh SH, Lai CH, Lin CH, Chen MJ, Hsu HT, Lin GX, Lin TT, Huang YW (2011) Estimating cancer risk increment from air pollutant exposure for sewer workers working in an industrial city. Aerosol Air Qual Res 11:120–127

Chapter 10
Air Quality Management Practices: A Sustainable Perspective

Vibhav Mishra and Aditya Ranjan

Abstract The quality of air and its management should always be a matter of debate either in developed or developing society. Rapid industrialization, self-centered approach, and dire need to earn more without giving heed to community needs lead to catastrophic results. During the late eighteenth and nineteenth centuries, the pace of industrialization was tremendous. The two world wars and then reinstitution of humanity again shifted the burden on nature. Only after 1972, when it was realized that something has gone wrong and left behind, the attempts to regenerate the nature started. The concept of sustainable development was introduced and commitment towards the same was called upon. After Stockholm, the commitment was incorporated in the Constitution of India, 1950 and to adhere the international obligations, Water Act, 1974; Air Act, 1981; Environment Protection Act, 1986 were enacted. The catastrophe of Bhopal in 1984 compelled the Supreme Court of India to step in for air quality management and do the needful till the time Government thinks to legislate. A new rule of Absolute Liability (M.C. Mehta v. Union of India (*Oleum Gas Leak Case*), AIR 1987 SC 1086) was propounded by changing the age-old Strict Liability (Rylands v. Fletcher, [1868] UKHL 1, (1868) LR 3 HL 330). Since then, both the Government and the Courts have gone together to see, check, and curb the air pollution. Nevertheless, sparse funding elevates the problem exponentially. Commitment is the sine qua non for problem redressal, which predominantly seems absent. However, the judicial hunch of having public participation in the form of Public Interest Litigation has been the horse of the carriage. The introduction of the BS-VI norms for the vehicular emission, compulsory introduction of electric vehicles, removal of diesel locomotives, imposing penalty on stubble burning, making LPG cylinders available to all for alleviation of wood fuel, and compensatory afforestation are the primary check points of sustainable practices leading to air

---

V. Mishra (✉)
Supreme Court of India, New Delhi, India
e-mail: vibhavmishra@outlook.in

A. Ranjan
Faculty of Law, University of Delhi, New Delhi, India

P. Saxena, A. Srivastava (eds.), *Air Pollution and Environmental Health*,
Environmental Chemistry for a Sustainable World 20,
https://doi.org/10.1007/978-981-15-3481-2_10

quality management. Thus the present chapter focuses on the developments and implementations in air quality management and the possible sustainable measures for the same.

Keywords Challenges in legal sphere · Absolute liability · Suggestive remedy

10.1 Introduction

Unrecognized benefits of maintaining biological diversity are those services we receive when ecosystem functions normally.[1] These ecosystem functions include energy fixation, chemical cycling (oxygen production by rainforests), soil generation and maintenance, groundwater recharge, water purification, and flood protection. The list is not exhaustive. These services are provided to us at no cost and when we destroy the inability of ecosystem to function naturally, we not only lose these free services but also too often have to pay to replace them.[2] The judicial system only works if someone is allowed to have the last word and if that last word, once spoken, is loyally accepted.[3] Also, the law is the last interpretation of the law given by the last judge. A delicate balance has to be maintained between the interest of society and liberty of an individual[4] and a legitimate expectation must always yield to the larger public interest.[5] The concept of sustainable development has an ancient origin, but it was much more emphasized during the "Earth Summit" of 1992 in Rio de Janeiro. However, the same has not been followed up and adhered to *in-verbatim* and the consequence is apparent by mere peeping outside the windows. Amidst huge hue and cry, a significant amount of literature is passably available over ailing effect of air pollution and urge for sustainable development, but the same is sparingly for modus operandi and a small iota of it goes to implementation, especially when understood in Indian perspective. It has been clear, among all rationals, that for a hypothesis or assertion to be absolute, it must be proved thrice. However, the hypothesis or assertion made above for the Indian perspective needs no proof, for it shall raise a conundrum challenging the dogmatic premise of *"All's well"* and plausible efforts being put to address the ground reality which can be substantiated by the World Economic Forum's Report[6] placing 6 Indian cities amongst the World's top 10 air polluted cities. This chapter aims to harmonize the selected

---

[1] Dr. A.K. Sikri, J. [Union of India v. Zavaray S. Poonawala, (2015) 7 SCC 347, *para* 15].

[2] Ibid.

[3] Lord Diplock, (1972) 1 All ER 801, 874.

[4] Adarsh Kumar Goel, J. [Ramdev Food Products Pvt. Lts. V. State of Gujarat (2015) 6 SCC 439, *para* 19].

[5] Rohinton Fali Nariman, J. [P. Suseela v. University Grants Commission, (2015) 8 SCC 129, *para* 21].

[6] *Available at*: https://www.weforum.org/agenda/2018/05/these-are-the-worlds-most-polluted-cities (Last visited on February 2, 2019).

judicial hunch and policy relating to sustainable management practice regarding air pollution in India as against the policies proven successful in the nations, which could be adopted in India for an efficient result (Saxena and Sonwani 2019a, b).

10.2 The Development of the Concept of Sustainable Development

Air cannot be separated from the environment. Being its inseparable component, the concept relating to the environment in general are *suo-motto* applicable for the air quality, unless the context otherwise so requires or there is an express exception. The management is the application or execution of a draft policy. Therefore, the relevance of highlighting execution latches or suggestions to overcome the execution latches are cryptic in the absence of discussion over the guiding principles.

There are many conventions wherein the international community came up to deal with the concept of Sustainable Development. Sustainable, in essence, means—a thing which has the ability to sustain or be used in present while ensuring its availability in future (Saxena and Naik 2018). Now, the essential question which comes forth is whether this notion of sustainability *per-se* is about the development itself or about the resources which are being used for the development or the surroundings which are witnessing the development, *i.e.*, a third-party reference. Sustainable Development neutrally deals with both economic as well as ecological sustainability. However, the inseparable neutral approach for the same could be conceived as the intertwined double-helical DNA strand, where the major groove is the environment and the minor groove is economics. The judicial hunch and interpretation are the nitrogenous bases connecting and holding the intertwined two.

The idea of the benefit of future generations and present generations together has found an international support in the "*Maltese Proposal*" at the UNGA, 1967 although, this was not titled as the "Sustainable Development." It was only the *Cocoyoc Declaration* of 1974 which used the term "Sustainable Development" for the first time. This declaration was a document which was published after the conference of experts of various countries in Cocoyoc. These experts came together for discussion on the issues relating to "the use of resources, environment and the development strategies." All in all, this Cocoyoc declaration was a result of discussions during the Stockholm Conference of 1972; however, officially the term "Sustainable Development" was used in World Conservation Strategy of 1980 which was prepared by The World Conservation Union with UNEP (United Nations Environment Programme), WWF (Worldwide Fund for Nature). To note essentially, the prominent conventions relating to air which enunciated and emphasized on the concept of Sustainable Development are hereinunder.

10.2.1 The United Nations Conference on the Human Environment, 1972

The United Nations Conference on the Human Environment, popularly known as Stockholm Conference, was held in 1972 in Sweden. The outcome of this conference was a document which is known as the "Stockholm Declaration." This declaration consists of 26 principles. This Declaration dealt with the safeguarding of natural resources for present and future generations through careful planning and management under Principle 2. Further, Principle 3 of the Declaration stated that the capacity of the earth to produce vital renewable resources must be maintained and, wherever practicable, restored (Saxena and Sonwani 2020). Principle 11 enunciates the responsibility of States to prepare policies in such a way that they will not affect the development potential of the developing countries at present and in future as well and all States should come to some agreement to deal with the economic consequences based on the application of environmental measures. Principle 13 calls for the management of development planning of the States in a coordinated manner for the benefit of human environment according to their population. Principle 22 of the declaration again iterates an obligation of states to cooperate and develop some international law regarding liability and compensation for the victims of pollution and environmental damage irrespective of their jurisdictions. Notably, the most important provision in the aspect of Sustainable Development was Principle 5 of the Stockholm Declaration which provided that the exploitation of non-renewable natural resources should be such that these can be preserved against future exhaustion and the benefit of exploitation of these should be shared by all mankind. Therefore, with these principles laid down in Stockholm Declaration, it is evident that this Conference was the first formal attempt which took Sustainable Development as an important factor for the long run of human development and Human Environment (Sonwani and Saxena 2016).

10.2.2 World Commission on Environment and Development Report of 1987

The Commission on Environment and Development, popularly known as The Brundtland Commission, in its report titled as "Our Common Future" (1987) brings the term Sustainable Development in the common use. This commission was appointed by UN General Assembly in 1983. This commission has defined the term sustainable development as, "*a development that meets the needs of the present without compromising the ability of the future generations to meet their own needs.*" The report has explained this concept in two ways-: *firstly*, it was focused on the needs of indigents and it also made it clear that a priority should be given to the issues of such persons. *Secondly*, it was focused on the imposition of some limitations over technology and organizations by taking into account the ability of environment to meet the needs of present and future generations. This report has majorly emphasized the relationship between economics and environmental sustainable development as overlapping circles and stated that this economic sustainability and ecological sustainability should be the basis of every national strategy (Saxena and Sonwani 2019a, b). Along with these efforts, this Brundtland Commission Report has developed the World Ethics of Sustainability as well. It being so, the Brundtland Commission Report of 1987 was the first report which had a focus on the Sustainable Development as a term of common use and the first of its kind because it established a relationship between economics and Sustainable Development.

10.2.3 The United Nations Conference on Environment and Development (UNCED), 1992

The UNCED, 1992 popularly known as the Rio Conferrence was concluded with the Rio Declaration, affirming 27 Principles which focused on the behavior of nations toward more environmentally sustainable patterns of development. This included Agenda 21 as a separate voluntary action plan. Principle 1 of the Declaration stated that human beings are the main concern for the sustainable development, and that they deserve a healthy and productive life in harmony with the nature. Principle 2 of the Declaration has imposed a duty on the States for preparing their policies in such a manner; where, their sovereign rights of exploiting the environmental resources should not cause any damage to the environment in their areas of their jurisdictions, as well as the areas beyond their jurisdictions. Principle 3 of the Declaration was directly focused on Sustainable Development where it stated that the right of development should equitably meet the environmental needs of the present and the future generations simultaneously. Principle 4 stated that the environmental protection should be an integral part of the development and it should not be considered in isolation. Principle 5 stated that the people and States should cooperate in a manner through which poverty can be extinguished and the standards of living can be raised for the majority of the people. Principle 8 stated that states should reduce the unsustainable patterns of production and consumption to achieve sustainable development and it should promote the policies based on demography. Principle 12 stated that the States should cooperate to promote an international economic system which should be open to all and which would lead to the growth of economy and the sustainable development in all countries for addressing the problem of environmental degradation. Further, this principle emphasized on building up an international consensus to deal with the environmental measures for the reduction of the transboundary environmental problems. Principle 21 stated that the ideas, creativity, and courage of youths should be used to increase the global partnership in order to achieve sustainable development and for ensuring a better future for all. Principle 24 stated that the States should respect the international laws which provide for the environmental protection and it should be taken care of during the times of the armed conflicts and other than these, the States should cooperate in further development of the environment-related international laws. Along with these principles this declaration has emphasized on some other concepts which later were recognized as integral parts of the concept of Sustainable Development such as Precautionary Principle under Principle 15 and Environmental Impact Assessment (EIA) under Principle 17. Agenda 21 was adopted as a voluntary action plan at this Conference itself. It was a dynamic program for the twenty-first century which provided a docket for action at local, regional, and global level. In a manner, it is the most comprehensive, most far reaching and most effective program for international actions ever sanctioned by the international community. Agenda 21 can be said to be focused on the cooperation of the States in order to achieve the goal of sustainable development. It was divided into four sections dealing with Social and Economic Dimensions, Conservation and Management of Resources for Development, Strengthening the Role of the Major Groups, and lastly the means of implementation, respectively. Eventually, it played a major role in the implementation of the concept of Sustainable Development by establishing a commission known as The United Nations Commission on Sustainable Development under its Chap. 38. The Rio Conference further dealt with the issues of Climate Change in the form of Convention on Climate Change which imposes a requisite duty on the states to take steps for reduction of the emission of gases which are creating the condition of Global Warming. The other convention which was a part of the UNCED was The Convention on Biological Diversity which, however, falls beyond the scope of the present work. After going through the provisions of the Rio Declaration on Environment and Development, it can plausibly be concluded that this declaration has emphasized on the global development of the concept of "Sustainable Development" specifically.

10.2.4 World Summit on Sustainable Development (WSSD), 2002

The Summit was held in Johannesburg, to take a stock of the implementation of the Agenda 21. However, it was unable to fulfill the expectations of supplying the fruitful results with regard to the implementation of Agenda 21. Nevertheless, it got some achievements regarding the protection of fair and equitable sharing of benefit within the framework of the UNCBD. This summit got popularity as it played a vital role in giving the priority to poverty eradication and removal of unsustainable patterns of consumption in order to achieve the sustainable development at the global level. Another achievement of this summit was that it led to an agreement among States to halve the proportion of people without access to sanitation and safe drinking water and to restore fish stock levels by 2015, reduce the loss of biological diversity by 2010, sound management of chemicals injurious to human health and environment, all with a view to minimize risk by 2020. Further this Summit played a substantive role in bringing the idea for an advanced, cleaner, more efficient, affordable and cost-effective technology. Therefore, though the summit did not achieve a lot in practicality, it propounded a newer outlook, blueprint for further negotiations, and signalling pathways for better environment.

To conclude, it can be said that the concept of Sustainable Development was not developed in a day. The concept was developed by recurring substantive delebrations, in a systematic pattern, for over a span of about 4 decades through various international conferences, declaration. In recurring delebrations, various other fundamental concepts from domestic domain were incorporated as sub-set of sustainable development and it was taken as an important factor by various committees appointed by the United Nations on various occasions. The Concept of Sustainable Development has some important principles like Inter-Generational Equity, Use and Conservation of Natural Resources, Environmental Protection, The Precautionary Principles, The Polluter Pays Principle, Obligation to Assist and Cooperate, Eradication of Poverty, Financial Assistance to the Developing Countries under its sphere and these principles were evolved through some express provisions which were later on interpreted as the part of Sustainable Development.

10.3 Toward Sustainable Air Practices

In the United States of America, the Clean Air Act, 1963 (majorly amended in 1970) provides the legal framework for improving public health and public welfare by regulating the Air Quality through five identified goals.[7] These goals prescribe for the reduction of six major pollutants (including particulate matter and lead), limiting the hazardous air pollutants, protection and improvement of visibility in wilderness areas and national parks, reduction in emission of the substances which causes acid deposition, and, to curb the use of chemicals that have the potential to deplete the stratospheric ozone layer.[8] The legislation has empowered the Environment Protection Agency (EPA) to comprehensively establish National Ambient Air Quality Standards (NAAQS). The legislation also establishes a federal/state regulatory framework where the states are required to develop a State Implementation Plan (SIP) to set up the Ambient Air Quality Standards within its borders. Further, the said legislation has also established a more stringent system by establishing the New Source Performance Standards (NSPS) and National Emission Standards for Hazardous Air Pollutants (NESHAP) to regulate air toxics. Similarly, the United Kingdom has also done a great overhauling of their legal framework after the Great London Smog of 1952. The Parliament had enacted the Clean Air Act, 1956 which was sponsored by the Ministry of Housing and Local Government. The above Act

---

[7] *See* Air Quality Management in United States; *available at*: http://dels.nas.edu/resources/static-assets/materials-based-on-reports/reports-in-brief/aqm_final.pdf (Last Visited on August 16, 2019).

[8] Ibid.

was repealed by The Clean Air Act, 1993. Further, the UK has appointed an Air Quality Expert Group in 2001 which suggested various reforms. The United Kingdom has initiated for European Union Emissions Trading System (EU-ETS) in European Union which was first of its kind in the world to regulate the Greenhouse Gas Emissions. Similary, various nation-States, under the obligation of various international conventions, conferences and agreements have enacted the domestic legislations for the sustainable practices towards environment, including Air.

10.3.1 Indian Experience

Ancient Indian methods for the protection and preservation of envireomnet by maintaining sustainability has been promising. Nevertheless, it is flawed to equate the Ancient and the Modern efforts for achieving the sustainable air practices. The standards of morality in a society change with the passage of time.[9] A particular activity, which was treated as immoral a few decades ago, may not be so now. Societal norms are dynamic.[10] Though the ancient Indian practices and literature are rich enough to adopt and enact the measures for preserving and protecting the environment, especially the air,[11] the same do not find a vast recognition amidst global hunch for scientific validation. In legal parlance, the precautionary principles notionally premised on the Environmental Impact Assessment are the soul and essence of the sustainable practices relating to air pollution, whereas the Polluter Pays principle and Absolute Liability are the compensatory reliefs after the wrong has been inflicted upon the environment.

A basic shift in the approach to Environmental Protection occurred initially between 1972 and 1982. Earlier, the concept was based on the *"assimilative capacity rule"* as revealed from Principle 6 of the Stockholm Declaration of the United Nations Conference on Human Environment, 1972. The said principle *assumed* that science could provide policy-makers with information and means necessary to avoid encroaching upon the capacity of the environment to assimilate impacts; and, *presumed* that relevant technical expertise would be available when environmental harm was predicted and there would be sufficient time to act in order to avoid such harm. But in the 11th principle of the United Nations General Assembly Resolution on the World Charter for Nature, 1982, emphasis shifted to

---

[9] Dr. A.K. Sikri, J. in Indian Hotel and Restaurant Association v. State of Maharashtra, (2019) 3 SCC 429, para 79.

[10] ibid.

[11] Rigveda, (Mandala 6, Hymn 48); Rigveda (Mandala 10, Hymn 168); Rigveda (Mandala 10, Hymn 186); Yajurveda 5:43; Yajurveda 6:33; Renugadevi R. "Environmental ethics in the Hindu Vedas and Puranas in India", African Journal of History and Culture (AJHC) Vol. 4(1), pp. 1–3, January 2012; Sarmah Rajib "Environmental awareness in the Vedic literature: An Assessment", International Journal of Sanskrit Research 2015; 1(4): 05–08; Arthashastra, Book-II, Chapter-I, II, IV.

the "*precautionary principle*," and this was reiterated in the Rio Conference of 1992 in its Principle 15. The inadequacies of science are the real basis that has led to the precautionary principle of 1982. It is based on the theory that it is better to err on the side of caution and prevent environmental harm which may indeed become irreversible.[12]

The environmental awareness started spreading in early 1970s; since then a number of specific and comprehensive environmental statutes have been enacted to deal with issues of environmental governance, pollution control, and sustainable development in India. The year 1972 can be marked as special for its immense importance at global as well as at the Indian level for the environmental awareness as the Stockholm Conference on Human Environment was organized in that year. The then Prime Minister of India took part in this conference and was committed toward having a national framework for the protection of environment. In order to fulfill those commitments, the Indian Parliament enacted several national legislations. However, the most significant step taken by the parliament was the introduction of 42nd Amendment to the Constitution of India, as this amendment created a suggestive state responsibility under Part IV for the "Protection and improvement of forests and wildlife"[13] which was certainly focused on a clean and better environment, though it has never been achieved like other directive principles of state policy. Further, it incorporated a new part in the Constitution of India in form of Part IV-A; which created certain fundamental duties on the part of the citizens, wherein one related to the environment was "to protect and improve the natural environment."[14] Therefore, this constitutional amendment has tried to secure a better environment for the citizens and later on the Supreme Court of India, through various judgments, has interpreted these provisions in the light of the "Right to get pollution free water and air,"[15] "protection of ecology and environmental pollution"[16] under the interpretaive ambit of the Article 21 of the Constitution[17] and incorporated the sustainable development principle. Further, major legislative changes were brought in through the Air (Prevention and Control of Pollution) Act, 1981 (Air Act henceforth). Some important features of the Air Act are discussed as under:

---

[12] *A.P. Pollution Control Board* v. *Prof. M.V. Nayudu*, (1999) 2 SCC 718.*see also:* Surendra Malik and Sumeet Malik, *Supreme Court Quinquennial Digest 1996–2000* 1880 (Eastern Book Company, Lucknow, Volume 2, 2006).

[13] The Constitution of India, Article 48A.

[14] The Constitution of India, Article 51A (g).

[15] *See Subhas Kumar* v. *State of Bihar*, AIR 1991 SC 420.

[16] *See Rural Litigation and Entitlement Kendra* v. *State of U.P.,* (1985) 2 SCC 431; *Indian Council for Enviro-Legal Action* v. *Union of India,* (1996) 3 SCC 212; *See also Vellore Citizen's Welfare Forum* v. *Union of India,* (1996) 5 SCC 647.

[17] Art. 21 "No person shall be deprived of his life or personal liberty except according to a procedure established by law." The Constitution of India, 1950.

10.3.2 *Introduction of* “Command and Control”

The Air Act followed the same model as the Water (Prevention and Control of Pollution) Act, 1974 (“Water Act”) and, in fact it enhanced the powers of the existing boards to include the air quality management. The Air Act envisaged and introduced a legal framework which is often referred to as the “command and control regime.”[18] This essentially refers to three things: (a) the establishment of a regulatory and monitoring agency, like the pollution control boards; (b) the setting up of standards regarding permissible levels of pollution; and (c) the enforcement of these standards through sanctions.

The Act vests the regulatory authority on the state boards and empowers them to establish and enforce the standards for emission of air pollutants by the industrial units into the atmospheric air.[19] The Central Board performs the same function for union territories and coordinates activities between state boards. Apart from this, the Pollution Control Boards are empowered in such a way that they can lay down different standards for different industrial plants for compliance, for example, if an industry is functioning in the “air pollution area” it has to follow the strict majors while on the other hand an industry operating in the same manner in a clean area and it is not directly affecting that area, can be permitted on different norms.

10.3.3 Justification for Air Act, 1981

The “statement of reasons and objects” of Air Act states that the Act was enacted in order to implement the decisions taken at the United Nations Conference on the Human Environment, Stockholm in 1972. This was enacted in furtherance of the obligation owed by the participating states, who undertook to take appropriate steps for the preservation of air quality and control of air pollution. The Act authorises the Central and State Pollution Control Boards constituted under the Water Act, to include air pollution control. The State Boards have the power to lay down, in consultation with the Central Board, standards for emission of air pollutants into the atmosphere by industrial plants, automobiles, and any other source.[20] Further, the State Governments, in consultation with the State Boards, have the power to declare “air pollution control areas.”[21] Section 21 of the Act contains the environmental clearance powers of the State Boards. The section, inter alia, prohibits

---

[18] *See* for instance, Sanjay Upadhyay and Videh Upadhyay, *Handbook on Environmental Law (Volume II)- Water Laws, Air Laws and the Environmental* 9–10 (Lexis Nexis, New Delhi, 2002); and Michael Watson, “Environmental Offences: the Reality of Environmental Crime” 7 *Envtl. L. Rev.* 190–200 (2005).

[19] The Air (Prevention and Control of Pollution) Act, 1981; s. 17 (1) (g).

[20] The Air (Prevention and Control of Pollution) Act, 1981 (Act 14 of 1981), s. 17(g).

[21] The Air (Prevention and Control of Pollution) Act, 1981 (Act 14 of 1981), s. 19.

industries operating within designated air pollution control areas must obtain "previous consent" from state board.

10.3.4 Amendments to the Air Act in the 1980s

After the enactment of the Environment Protection Act, the Air Act was also amended in 1987. The said amendment, however, introduced significant changes in the Act, strengthening the implementation provisions. Following significant changes were introduced through the amendment:

1. *Increased Penalties:* Penalties under the Acts were made more stringent.
2. *More power to Board to issue Directions:* The Boards were given the power to issue administrative direction which could extend to even closing the defaulting industrial plant or stoppage of supply of water, electricity or any other services to such a unit.[22]
3. *Power to apply to Courts for restraining persons from causing pollution:* Whenever it is apprehended that any person or industrial unit is likely to cause air and water polluting emissions beyond the standard lay down, Boards have been given the power to approach to the Courts for restraining the person who is likely to cause such pollution from so causing.[23]
4. *Enabling private prosecution:* Most importantly, the "citizen's initiative/complaint provision" was introduced in both the Acts, like in the Environment Protection Act, empowering individuals with the power to initiate privately criminal prosecution of erring persons, companies, and corporations, after giving a notice regarding the alleged offence with an intention to make a complaint to the board concerned.[24]

10.3.5 Central and State Pollution Control Boards

The Central Pollution Control Boards (CPCB) advises the Central Government, coordinates activities of State Pollution Control Boards (SPCBs), provides them with technical assistance and guidance, organizes training of personnel for pollution control boards, collects and complies technical and statistical data, lays down water and air quality standards, and executes nation-wide pollution control programs. It may step into the shoes of the SPCBs when the Central Government directs it to do so.[25] Further, the CPCB performs the functions of the SPCBs in the Union

---

[22] Air (Prevention and Control of Pollution) Act, 1981 (Act 14 of 1981), s. 31A.

[23] Air (Prevention and Control of Pollution) Act, 1981 (Act 14 of 1981), s. 22A.

[24] Air (Prevention and Control of Pollution) Act, 1981 (Act 14 of 1981) s. 43(1)(b).

[25] The Air (Prevention and Control of Pollution) Act, 1981 (Act 14 of 1981), s. 16.

Territories.[26] The SPCBs carry out programs identical to the CPCB within the territory of the concerned state. They plan comprehensive programs, advice state governments, collect and disseminate information, collaborate with the CPCB in organizing training programs, collect samples for the purpose of analysis, and so on. The most important aspect of their functions, however, is that they lay down standards for water and air quality. Further, the SPCBs manage the "consent provisions" under the Water and the Air Act. The permission by SPCB concerned is necessary for establishing the new industry or plant, etc.[27] Further, while the Central government can give directions to the CPCB, the latter can in turn issue directions to the SPCBs. The directions issued by the State Government will bind both CPCB and the SPCBs.[28]

To further integrate the existing organizational setup, the Environmental Action Programme, 1993 was formulated under the aegis of the Ministry of Environment and Forest with the sole aim and objective for integrating the command-chain model amongst various stakeholders in the government and keep a proactive vigil over the existing infrastructure. This was further strengthened by the National Environment Policy, 2006, the first unified aim and objective for the overall check over the environmental aspect and promotion of the sustainable development. The CPCB and SPCBs have to function under this command-chain model and frame the guidelines and policies in the light of above programs. Thus, the pollution control board, apart from other functions, perform the following main functions: (1) Consent Mechanism; (2) Laying down standards of emissions and discharge of effluent; and (3) Environment Quality Monitoring.

10.3.6 Powers of "Direct Enforcement": Power to Issue Directions

After the enactment of the EPA, the amendments to the Water and Air Act conferred more potent and meaningful powers to the pollution control Boards.[29]

The relevant provision for the same is Section 31A of the Air Act, which provides as under:

> [Notwithstanding anything contained in any other law, but subject to the provisions of this Act, and to any directions that the Central Government may give in this behalf, a Board may, in the exercise of its powers and performance of its functions under this Act, issue any direction in writing to any person, officer or authority, and such person, officer or authority shall be bound to comply with such directions.

---

[26] Air (Prevention and Control of Pollution) Act, 1981 (Act 14 of 1981), s. 6.

[27] The Air (Prevention and Control of Pollution) Act, 1981 (Act 14 of 1981), s. 17.

[28] Air (Prevention and Control of Pollution) Act, 1981 (Act 14 of 1981), s. 18.

[29] P. Leelakrishnan, *Environmental Law in India* 163 (Lexis Nexis, Gurgaon, 3rdedn., 2009).

> Explanation- For the avoidance of doubts, it is hereby declared that the power to issue directions under this section includes the power to direct-
>
> (a) the closure, prohibition or regulation of any industry, operation or process; or
> (b) the stoppage or regulation of supply of electricity, water or any other service.]

Therefore, a Board may, in exercise of its powers and functions, issue any direction in writing to any person, officer, or authority and such person, officer, or authority shall be bound to comply with such direction. This means that the SPCBs and CPCB can issue directions to any industry to stop functioning.

The SPCBs are empowered to take sample of effluents. They are empowered to enter any place for performing the functions of the Board and determine whether any order or direction is complied with. However, a plain reading of Section 25 and Section 26 of the Air Act makes it clear that this power is only for the purpose of procuring evidence for prosecution.[30] Similar powers are vested on the Central Government under Section 10 of the Environment Protection Act, 1986.

It may be noted that while pollution Control Boards are entrusted with powers which may extend to ordering the closure of industries, they do not have any penal powers, for instance, to impose fines. The establishment of the National Green Tribunal vide the NGT Act, 2010, has paved way for speeder justice. The tribunal, under S.14, has the jurisdiction over all Civil Cases where a substantial question relating to environment has arisen and under S.15, it has the power to order relief, compensation, and restitution. The power of the Tribunal under S.15 of the Act was largely under challenge before the Supreme Court of India in *State of Meghalaya v. All Dimasa Students Union* [(2019) 8 SCC177].

10.3.7 Mechanism for Enforcement: Importance of Criminal Sanctions

As is evident from the above discussion, environment statutes in India primarily function by laying down standards of permitted levels of emissions and employing the environmental clearance procedure, in order to preserve natural resources and regulate their use.

However, the relevant question to put forth is, as to what powers do the pollution control boards have in case the standards laid by them or the "previous consent" procedures are violated? The Air Act contains provisions for ensuring that the Boards have the power to restrain persons from violating these standards by either giving directions itself or applying to a competent court for restraining persons or industrial units who are likely to cause such pollution. However, these are the emergency provisions. The cardinal question which challenges the real-time efficacy and usual monitoring is the immediate power to act. Further, what if these directions

---

[30] Ibid.

and orders are also not complied with? What powers do the pollution control boards have to enforce these directions and orders, and the agency by which such powers of the pollution control boards be exercised, namely by the PCBs or by the courts?

The Supreme Court of India in *Indian Council for Enviro-Legal Action* v. *Union of India*[31] observed and held:

> If the mere enactment of laws relating to the protection of environment was to ensure a clean and pollution free environment, then India would, perhaps, be the least polluted country in the world. But this is not so. There are stated to be over 200 Central and State statutes which have at least some concern with environmental protection, either directly or indirectly. The plethora of such enactments has, unfortunately, not resulted in preventing environmental degradation which, on the contrary, has increased over the years.[32]

10.4 Judicial Attempts to Ensure Sustainable Air Practices

The precautionary principle was recommended by the UNEP Governing Council (1989). It is interesting to note that while the inadequacies of science have led to the precautionary principle, which in turn, has led to the special principle of burden of proof in environmental cases where burden as to the absence of injurious effect of actions proposed is placed on those who want to change the *status quo*. This is often termed as a reversal of the burden of proof, because otherwise in environmental cases, those opposing the change would be compelled to shoulder the evidentiary burden, a procedure which is not fair. Therefore, it is necessary that the party attempting to preserve the *status quo* by maintaining less polluted state should not carry the burden of proof and the party, who wants to alter it, must bear this burden. If the environmental risks being run by regulatory inactions are in some way uncertain but non-negligible, then regulatory action is justified.[33]

However, the recognition of the above thoughts, though recognized internationally, were sluggish to be recognized in the Indian judicial setup. While the world was making a shift from "*assimilative capacity principle*" to "*precautionary principle*" in the early 1980s, the Indian courts were dogmatic in their approach as seen in *M.C. Mehta* v. *Union of India*[34] where the Supreme Court observed:

> We cannot possibly adopt a policy of not having any chemical or other hazardous industry merely because they pose hazard or risk to the community. Industries, even if hazardous, have to be set up since they are essential for the economic development and advancement of well-being of the people. We can only hope to reduce the element of hazard or risk to the community by taking all necessary measures for locating such industries in a manner which would pose the least risk or danger to the community and maximizing safety requirements in such industries.

---

[31] 1996 (5) SCC 281.

[32] *Id.* at 293.

[33] ibid.

[34] AIR 1987 SC 1091.

Nevertheless, a decade later, the above transition started seeping in the judicial thoughts and the era of late 1990s emerged as a phase of environmental renaissance. This era saw exponential surge in the awareness regarding Air Quality Management and the need for the adoption of Sustainable Practices thereon. The Supreme Court observed that the courts now cannot afford to lightly deal with the cases involving the pollution of air and water. The parliamentary concern in the matter is adequately reflected in strengthening of the measures prescribed by the statutes and the courts have got no justification for ignoring the seriousness of subject.[35] During the phase the ambient air quality standards were recognized and the industries were ordered to shut when found defying the above.[36] The directions to the Government of India were issued to frame the standard of automobile emission and the same was directed to be complied within a timeframe.[37] Similarly, the concern was also expressed by the Supreme Court regarding delay in the adoption of the natural gas as industrial fuel by the Agra-based industries in the *Taj Trapezium case.*[38] New standards were laid down for the Brick Kilns and many were shut in the NCT of Delhi owing to hazardous emission.[39] The judiciary remains the guardian of the constitution even without the sword or the purse.[40] Hot-mix plants used in the construction industries and by the government either for the use of repairing roads resurfacing the runways at the IGI Airport (Delhi) were under strict scrutiny of the Supreme Court and the permission was given to carry out the uses of the hot-mix plants only after the applicant airport authority assured the Supreme Court that it would only use the technology which meets the permissible emission limits for the particulate matter prescribed by the EP rules for its hot-mix plants.[41]

Apart from the industrial air pollution, the vehicular pollution also plays a dangerous role in polluting the air. The National Capital Territory of Delhi is most affected by the health hazards created by the automobiles. For that reason, the Supreme Court and the National Green Tribunal has time and again passed innumerable orders wherein restrictions were imposed on the automobiles. In *MC Mehta* v. *Union of India,*[42] the Supreme Court reprimanded the Central Government for not complying the orders issued by it previously. Before making this order, the Supreme Court collected plethora of literature about the ill effects of the air pollution on human health. After this order, there was visible change in the type of fuel used by the public transport vehicles in Delhi. However, the very purpose of this order was not achieved as there is an uncontrolled growth of the number of the private vehicles

---

[35] *U.P. Pollution Control Board* v. *Mohan Meakins Ltd.*, (2000) 3 SCC 745.

[36] *Pollution Control Board* v. *Mahabir Coke Industry*, (2000) 9 SCC 344.

[37] *M.C. Mehta* v. *Union of India (Matter regarding emission standard for vehicles*), (1999) 6 SCC 12.

[38] *M.C. Mehta* v. *Union of India (Taj Trapezium)*, (1999) 6 SCC 611.

[39] *M.C. Mehta* v. *Union of India (Matter regarding Brick Kilns)*, (1998) 9 SCC 149.

[40] Dr. A.K. Sikri, J. in Shanti Bhushan v. Supreme Court of India, (2018) 8 SCC 396, para 36.

[41] *M.C. Mehta* v. *Union of India (Taj Trapezium)*, (1999) 7 SCC 522.

[42] (2002) 2 SCC 356, AIR 2002 SC 1696.

in the National Capital Territory of Delhi. On the other hand, in order to balance the conflicting interest of the convenience to the vehicle owners and aim to protect the trees, the petrol filling stations located to a site adjacent to, but not forming the part of the ridge area was though ordered to function, but it could not retain any alternative site given to it at another place.[43] The Supreme Court in *M.C. Mehta v. Union of India* [(2018) 18 SCC 745] ordered that the diesel vehicles above 4000cc for the present Prime Minister and former Prime Ministers of India, as per security standards, are not available under the environmental norms. Therefore, registration of said vehicles is permitted only subject to the payment of Environmental Compensation Charge (ECC) @ 30% of the cost of the vehicle to the Environment Pollution control Authority. Similarly, the usage of such vehicles was permitted for the Delhi Police on payment of the same. However, the garbage vehicles and water tankers above 2000cc were exempted to pay the ECC. For the private vehicles, all the companies/ automobile manufacturers who intend to sell the vehicle above 2000cc were required to deposit 0.1% of the ex-showroom price of the vehicle towards the Environment Protection Charge. However, the CPCB was given the liberty to enhance this charge, but not retrospectively. The thoughts for the larger protection of ambient air quality were developed and saw their recognition in the form of "*air pollution control areas*."[44]

The Supreme Court has opined that the *ex-post facto* Environmental Clearance (EC) or the retrospective EC is completely alien to the Environmental Jurisprudence.[45] Where on one part the recent trends suggest that the Court is adopting stringent measures against various polluters, the same seems to be stalled when it comes to the Governments. Considering tremendous rise in the air pollution in the National Capital Region, the use of the Furnace Oil and pet coke was prohibited; however, the State Governments of the adjoining States, viz, the State of Uttar Pradesh, Haryana, and Rajasthan were given liberty to issue such notifications,[46] which indicates that issuance of such notification does not attract a mandatory compliance on behalf of the State Government of the above said three states, which are also the major contributors to the pollution in the NCT of Delhi, depending upon the pattern and wind direction, were left on option. In addition to the above, directions were issued to curb on the use of SO_2, NOx, and SOx for 35 industries, which were again openended and optional for remaining States.[47] The National Auto Fuel Policy, 2003 was held to be effective by the Supreme Court and the OM was held not to be *carte-blanche* for the automobile industries to continue to manufacture BS-III vehicles when BS-VI vehicles are to be in the market soon and

---

[43] *M.C. Mehta* v. *Union of India (Regd. Pusa Service Station)*, (1998) 5 SCC 611.

[44] *Orissa State (Prevention and Control of Pollution) Board* v. *Orient Paper Mills,* (2003) 10 SCC 421.

[45] *Common Cause* v. *Union of India*, (2017) 9 SCC 499.

[46] *M.C. Mehta* v. *Union of India* (2017) 14 SCC 488.

[47] Ibid.

BS-IV vehicles are already in the market.[48] Recently, the National Green Tribunal, the apex body to deal with the Environmental Matters other than the Supreme Court, where appeal is conditional on leave, imposed a fine of Rs.500 Crore on Volkswagen, the prominent car producer for the usage of the "*Cheat Devices*" in its diesel cars which cheated the test devices over emissions. However, Volkswagen has preferred an appeal before the Supreme Court and the Court has provided the manufacturer the interim-relief vide order dated 07-05-2019 in the SLP Jurisdiction till the matter is sub-judice, on the submission of the car manufacturer that "on road" tests which have no legal basis or validity under Indian law, and thus cannot be made a basis for imposition of penalty or any adverse finding against Volkswagen Group Companies. Terming the reliance upon the "on road" test by NGT as completely arbitrary and unjustified, Volkswagen pleaded that NGT erred in concluding that the Central Motor Vehicles (11th Amendment) Rules, 2016 ("CMVR Amendment Rules"), which prescribe real-world driving cycle emission measurements from April 2023 can be applied retrospectively for a single manufacturer, i.e., Volkswagen Group Companies. In the same, the Volkswagen was ordered to submit the interim compensation of Rs.100 Crore as on date.

Even before this order, the Courts in India have made a shift from their earlier approach of supporting the industries over the environmental pollution to the closure of the industries. In *Syed Shah Asghar Hussaini* v. *A.P. Pollution Control Board and Ors.,*[49] Andhra Pradesh High Court held that:

> . . . .. in the event of any violation of the terms and conditions of the license and/or provisions of any Act, Rule or any directions issued by the Board is found, the Board shall immediately issue a closure notice to the erring industry and for the said purpose the first respondent is also entitled to initiate steps against the respondents in terms of the provisions of Section 31-A of the Air (Prevention and Control of Pollution) Act, 1981 and 33 of the Water (Prevention and Control of Pollution) Act, 1974, it will also be open to the State Government to proceed against the respondents under Section 5 of the Environment (Protection) Act, 1986. However, any party, shall be at liberty to file any other or further application in the matter if any cause of action therefore arises. The writ petition is disposed of with the aforesaid observations.

The approach taken by the High Court of Andhra Pradesh in the above quoted case was followed by the other courts as well. Still the question remained with the activeness of the Pollution Control Boards/State Governments, which seems very irresponsible most of the times. The practices which are being followed by the departments/authorities are not proactive as on most of the occasions they rise after some damage or when the situation goes out of their hands.

The Courts have been vigil on the Builders as well. Supreme Court in *Goel Ganga Developers (India) (P) Ltd. v. Union of India* [(2018) 18 SCC 257] rejected the contention that the damages for the breach of environmental norms and air pollution should be assessed on the basis of 'carbon footprint', as the courts cannot introduce a new concept of assessing and levying damages unless expert evidence in

---

[48] *M.C. Mehta* v. *Union of India* (2017) 14 SCC 243.

[49] ILR (2001) 2 AP 503, (504, 505) (DB).

this behalf is led or there are some well-established principles. In *Hanuman Laxman Aroskar v. Union of India* [(2019) 15 SCC 401] the Supreme Court postulated that the Environmental Clearance (EC) as under Environmental Impact Assessment Notification, 2006 is mandatory and that the EC is prerequisite for all the development projects. The process of EC constitutes 4 stages viz., (1) Screening; (2) Scoping; (3) Public Consultation; and (4) Appraisal. All these four stages are mandatory and that missing or misleading information provided by any applicant significantly impedes the functioning of the authority and that the defective application should be rejected immediately. Laxity and sheer apathy to the rule of law gives leaseholders a field day, being the primary beneficiary, with the State being left with some crumbs in the form of Royalty. For the State to have sustainable and equitable development, the implementation machinery needs strict vigilance. Unless the two marry, we will continue to be mute witnesses to the plunder of our natural resources and left wondering how to retrieve an irreversible situation.[50] Despite all, the judiciary also does not seem to be tough enough with the environmental offenders. Though the matter pertains to the Coastal Management, the approach of the Court could be inferred from the ratio. Despite the option of demolition of the project, the Court imposed the cost of Rs.1 Crore (10 Million INR) on the builder, with the direction of strict adherence to the norms in future and avoidance of contradictions with the authorities.[51] The doctrine of binding precedent is of utmost importance in the administration of our judicial system.[52] It promotes certainty and consistency in judicial decisions.[53] Judicial Consistency ensures the unanimity of approach and hamonises the variability. It further promotes the confidence of people in the administration and therefore, there is this need for this consistency and the enunciated legal principles.[54]

The Odd-Even Scheme introduced by the Government and the National Green Tribunal to curb vehicular emission in Delhi seem to have worked a bit. The same was when challenged before the NGT and the Supreme Court, the SC was of the view that there shall be no exemption to any person, officer, and individual from the ambit and scope of the odd and even scheme. They will be applied with equal rigor to all cases and all vehicles except those driven by women or two-wheelers.[55] However, it feels disheartening that the SC turned down the direction of the National Green Tribunal whereby the NGT ordered that all vehicles plying in the Airport area must be on CNG and if not, be converted to alternate fuel of CNG within 6 months to curb vehicular emissions and check air pollution.[56]

---

[50] Madan B. Lokur, J. Goa Foundation v. Sesa Sterlite Ltd. (2018) 4 SCC 218, para 2.

[51] Secretary, Kerala State Coastal Management Authority v. DLF Universal Ltd., (2018) 2 SCC 203.

[52] N. Santosh Hegde, J. (2002) 4 SCC 234, para 22.

[53] ibid.

[54] ibid.

[55] Government (NCT of Delhi) v. Vardhaman Kaushik, (2018) 10 SCC 633.

[56] Narang International Hotels Private Ltd. v. Society for protection of Culture, Heritage, Environment, Traditions and Promotion of National Awareness & Ors. (2018) 9 SCC 499.

Nevertheless, the judicial approach for maintaining the sustainable air practices found its rendezvous in *T.N. Godavarman Thirumalpad v. Union of India & Ors*.[57] The case is still pending and regular hearings are taking place. The Supreme Court has decised to keep the case open for continuous monitoring and in this, several orders relating to afforestation have been passed from time to time. In another plausible and debatable attempt, the Supreme Court in *Arjun Gopal v. Union of India* [(2019) 13 SCC 499] imposed the restrictions over the burning of fire-crackers during the most celebrated festival in India, Diwali, and on all other festive occasions in the National Capital Region of Delhi. Directions to the same effect were also issued to the Central Government for the applicability pan India. Compensatory Afforestation Funds were created by the Supreme Court for compensatory afforestation needs where the permission was granted for diverting forest area for non-forest purpose. Further, Compensatory Afforestation Fund Management and Planning Authority (CAMPA) was created as National Advisory Council under the chairmanship of the Union Minister of Environment and Forests for monitoring, technical assistance, and evaluation of compensatory afforestation activities.[58] However, despite the continuous monitoring of the SC over the Compensatory Afforestation Regime, the funds are to be allocated to the States by the Government of India. The pathetic and dwindling approach for compensatory afforestation in the eyes of the Government of India could be seen from the following table.[59]

S. no.	Annual year	Fund allocation
1.	2012–2013	Rs. 1029 Crore
2.	2013–2014	Rs. 1008 Crore
3.	2014–2015	Rs. 2057 Crore
4.	2015–2016	Rs. 2213 Crore
5.	2016–2017	Rs. 1827 Crore
6.	2017–2018	Rs. 465 Crore

Nevertheless, the total area of the land diverted from forest to non-forest usage is 310,063 ha and the total area of land earmarked for the compensatory afforestation is 385,434 ha, which excludes the land earmarked for the Compensatory afforestation plantation and other land (Non-CA) earmarked for plantation. Though the efforts seem plausible, the real question would be as if the scheme would be successful without there being sufficient funds for the same.

---

[57] (2008) 16 SCC 337.

[58] T.N. Godavarman Thirumalpad v. Union of India & Ors. [Order dated 10-07-2009].

[59] http://egreenwatch.nic.in/Portal.aspx [accessed on 24.08.2019 at 22:21 Hrs].

10.5 Conclusion

The Constitution is inherent in the concepts where words are transformed into concrete consequences.[60] The authorities in power should constantly remind themselves that they are the constitutional functionaries having the responsibility to ensure that the fundamental purpose of the administration is the welfare of the people in an ethical manner.[61] With the development of advanced techniques and increasing economical stakes, it is difficult to achieve a utopian sustainable development; however, the limits of the same can be stretched to the horizon, where the interpretation of law, the conflicting interests and other self-goals tend to harmonize for the creation of a better world, which is not only safe and serene for the present, but provides adequate environment for the coming generations. The judicial arm of the state being laden with the duty of being the final arbiter under constitution and protector of the constitutional ethos cannot usurp the power which it does not have.[62] Law cannot always be found fault with for the lack of its stringent implementation by the authorities concerned. Therefore, it is the solemn responsibility of all concerned to enforce the law as well as directions laid down by the Supreme Court from time to time in order to infuse the culture of purity[63] in vivid domains. The rule of law is the bedrock of democracy and would hardly require any reiteration. However firmly entrenched, the principle may be, it gets tested in the myriad of situation that confronts the courts from time to time.[64] The natural resources bestowed upon us by the nature are not the personal property of any individual or group of individuals. These resources are the trust of the future generation, for which we are the trustees, and so should be the utilization of the trust be regulated. Freedom of a person ends where the freedom of another person begins. In atmosphere, air is an omnipresent variable resource. The focus of the law as well as the enforcement agency should be straight jacketed to one rule: “No one should be left to breathe for what he is not responsible.”. This cardinal rule is the solution of the nuisance cum menace. Sustainable development is not a goal; it is the hard need of time for saving the planet and the existing life including flora and fauna. Moreover, it is very challenging to give sustainable solutions for air quality management particularly in India, where central as well as state government are working at their pace in a more healthier way but problem always arises from behavior of common people who does not have sustainable approach toward clean air and above all whole environment.

---

[60] Dipak Misra, CJ in State (NCT of Delhi) v. Union of India, (2018) 8 SCC 501, para 161.

[61] ibid., para 240.

[62] Dipak Misra, CJ in Public Interest Foundation v. Union of India (2019) 3 SCC 224, para 106.

[63] *Id*, para 107.

[64] Ranjan Gogoi, CJ in Alok Kumar Verma v. Union of India, (2019) 3 SCC 1, para 1.

References

Saxena P, Naik V (2018) Air pollution: sources, impacts and controls. CABI

Saxena P, Sonwani S (2019a) Primary criteria air pollutants: environmental health effects. In: Criteria air pollutants and their impact on environmental health. Springer, Singapore, pp 49–82

Saxena P, Sonwani S (2019b) Policy regulations and future recommendations. In: Criteria air pollutants and their impact on environmental health. Springer, Singapore, pp 127–157

Saxena P, Sonwani S (2020) Criteria air pollutants and their impact on environmental health. Springer, Singapore

Sonwani S, Saxena P (2016) Identifying the sources of primary air pollutants and their impact on environmental health: a review. IJETR 6(2):111–130