International Journal of Basic and Applied Biology
p-ISSN: 2394-5820, e-ISSN: 2349-2539, Volume 3, Issue 4; October-December, 2016, pp. 238-241

http://www.krishisanskriti.org/Publication.html

Air Pollutants and their Effects on Plants and Human Health

Seema Talwar[1], Nupur Mondal[1] and Sujata Bhardwaj[2]

[1]Department of Botany, Shivaji College, University of Delhi
[2]Department of Biology, Bhaskaracharya College of Applied Sciences, University of Delhi
E-mail: [1]seematalwar2014@gmail.com, nupur.mondal2010@gmail.com

1. INTRODUCTION

Anthropogenic emission of harmful substances into the atmosphere is called air pollution. These pollutants modify the chemical composition of the natural atmosphere. Increase in the concentration of greenhouse gases is also responsible for air pollution. Besides anthropogenic emissions, geogenic and biogenic emissions also increases the risk of air pollution. Geogenic emission comes from the non-living world such as volcanic emissions, sea-salt emissions, and natural fires, whereas biogenic emissions release from the living world; such as volatile organic compound (VOC) emissions from forests and emission of methane from swamps. Human activity also influences geogenic and biogenic emissions. Use of nitrogen fertilizers (urea) in agriculture results in increased biogenic emissions. Therefore, we can conclude that pollution is any substance which is emitted into the air from an anthropogenic, biogenic, or geogenic source. These substances are not part of the natural atmosphere and their concentration is more than the normal. They cause short-term or long-term negative effects. Primary or secondary air pollutants consist of gas and particle contaminants. These pollutants are responsible for damaging our environment, as they have direct as well as indirect negative impact on the environment, human health, vegetation and our cultural heritage. It has been reported that more than 3,000 substances that are not part of the atmospheric composition, emitted in the atmosphere can be considered air pollutants [1]. These pollutants are usually emitted by industrial activities and by the transport sector and are transported in the air over long distances. The most famous kind of atmospheric pollution is the photochemical cloud, whose components are formed due to the complicated chemical reactions in atmosphere. The principal reactants are the hydrocarbons, nitrogen oxides, sulphur oxides, ozone and ultraviolet radiation. All air pollutants do not have the same capability for producing same toxic effects due to the difference in their physical and chemical properties [2].

Different air pollutants have different impact on organisms. Plants are the sole food producer on earth and hence are the food provider for all organisms directly or indirectly. Air pollutants have an adverse affect on the plants and their parts. They also affect the overall yield of the crop. When these plants are consumed by animals and human beings, they are indirectly affected by them. Moreover, air pollutants have a direct impact on human beings as well. The present study gives a review of how air pollutants are affecting plants and human beings and hence, why it is necessary to combat air pollution.

2. EFFECT ON PLANTS

Sulphur dioxide is a very common air pollutant, which is produced in large amount due to the combustion of coal and other fuels. It is also a major constituent of the acid rain. One of the byproducts of sulphur dioxide is sulphuric acid which is also a harmful chemical for the plants. The young leaves in angiosperms are most perceptive to sulphur dioxide. Leaves exposed to these chemicals begin to loose their colour, and white spots are formed. Some leaves develop red, brown or black spots. In the severe attack, leaves start to fall off, and ultimately reduce the crop yield. Being the strong reducing agent, sulphur dioxide in high concentration causes the swelling of thylakoids and thus interferes with electron transport chain. It also reduces the rate of photosynthesis and protein synthesis. Sulphur dioxide is also responsible for closing of stomata and thus reduces the rate of transpiration. It also affects the structural proteins in cell membrane and changes the membrane permeability [1].

Nitrogen dioxide affects leaves and seedlings. It is responsible for the formation of crystalloid structures in the stroma of chloroplast and causes the swelling of thylakoid membrane, which ultimately reduces the photosynthetic activity. In most of the angiosperms, leaves show the water-soaked intraveinal areas which later on become necrotic. Chlorosis and tip burn are also very common symptoms in case of angiosperms and in conifer needles respectively [1].

Ozone is one of the most harmful pollutants to plants which gets released into the atmosphere from the burning of fossil fuels. Tropospheric ozone is an oxidant that damages

agriculture and ecosystems [3]. It can be carried to the long distances. Ozone is produced when oxides of nitrogen and other volatile organic compounds react in the presence of sunlight and heat. Plants exposed to higher concentration of ozone show different colour of spots on their leaves depending upon the concentration and ultimately it dies. Common symptoms of plants exposed to ozone are yellowing, stippling (small darkly pigmented areas approximately 2-4 mm diameter) flecking (tiny light-tan irregular spots less than 1mm diameter), blotching in leaves, premature senescence and early maturity [1]. Ozone also inhibits male reproductive features such as pollen formation, pollination, pollen germination and pollen tube growth. Dicot plants (soybean, cotton, peanut) are more sensitive than monocots (sorghum, field corn and winter wheat) [4]. Ozone also affects the yield of the plant. It has been observed in Cotton plant where ozone has significantly reduced the yield [5, 6]). Ozone destroys rubisco, an enzyme crucial for photosynthesis. Ozone also inhibits oxidative phosphorylation and thus changes in membrane permeability of the membrane. The impact of ozone on plants increases with the increase in humidity and decreases with low temperature and drought [1].

3. EFFECTS ON HUMAN HEALTH

Sulphur dioxide is easily soluble in water and as a result this gas when inhaled is absorbed in upper respiratory tract. But when a small amount of sulphur dioxide gas reaches into the lung's airways it can cause asthma. Patients suffering with asthma, when exposed to low level to sulphur dioxide results in the constriction of airways which leads to difficulty in breathing [7]. In severe condition, it may also cause asthmatic attack. Nose and throat irritation, followed by broncho constriction and dyspnoea, especially in asthmatic individuals, are usually experienced after exposure to increased levels of sulphur dioxide [8].

The European ESCAPE studies have observed considerable associations between long-term exposure to NO_2 and lung malfunction in children, respiratory infections in early childhood and effects on adult lung function also. Nitrogen oxides increase the susceptibility to respiratory infections [9, 10]). Emphysema-like lesions have also been reported in mice when exposed to nitrogen dioxide [11]. Direct exposure of nitrogen dioxide on skin can cause irritations and burns.

Carbon monoxide, one of the pollutants of green house gases combines with haemoglobin and changes its conformation. Some processes in modern technology, such as iron smelting, still produce carbon monoxide as a byproduct [12]. Thus formed carboxy haemoglobin reduces its capacity to transport the oxygen to different body parts [13]. And thus affects the functioning of different organs, especially those organs which required more oxygen such as the brain and the heart. Blood clotting can obstruct the blood vessels, which may results in angina [14].

Heavy metals (lead, arsenic, mercury, nickel) reduce lung functioning [15, 16], and are responsible for various diseases such as asthma, emphysema, and lung cancer [17 ,18]. These metals can cause the kidney damage which decreases glomerular filtration rate (GFR). These metals also increase the threat of stone formation or nephrocalcinosis [19-21]; and renal cancer [22, 23]. Increased blood pressure and anaemia are also due to consequence of heavy metal pollution [24]. Heavy metals and dioxins affect the nervous system also. Neurotoxicity, with symptoms such as memory disturbances, sleep disorders, anger, fatigue, hand tremors, blurred vision, and slurred speech, have been observed after the exposure with arsenic, lead and mercury [25, 26]. Lead exposure may be detrimental to the dopamine system, glutamate system, and N-methyl-D-Aspartate (NMDA) receptor complex, which play a key role in memory functions [27, 28]. Besides this, lead also increases the risk for spontaneous abortion and reduced foetal growth. There are also evidences suggesting that parental lead exposure is also responsible for congenital malformations [29], and lesions of the developing nervous system, causing important impairment in newborn's motor and cognitive abilities [30]. Mercury is also responsible for certain cases of neurological cancer. Dioxins decrease nerve conduction velocity and impaired mental development of children [31, 32]. Dioxins were found to be transferred from the mother to the fetus via the placenta. They act as endocrine disruptors and affect growth and development of the central nervous system of the foetus [33]. Epidemiologic studies have also linked dioxin exposure to increased mortality caused by ischemic heart disease [34].

Actually these metals accumulate in cellular organelles and obstruct with their function. For example lead accumulation in mitochondria causes the inhibition of Ca^{+2} uptake, which reduces the transmembrane potential, oxidation of pyridine nucleotides, and a speedy discharge of accumulated Ca^{+2} [35]. Metals also bind to proteins [36] and reduce a large number of enzymes, including the mitochondrial ones [37]. Metals also bind to DNA, which affects the expression of genes. Nucleic acid binding proteins are also involved. Nickel enters the nucleus, interacts with chromatin and silences the expression of genes which suppresses the growth of tumour and thus induces the formation of cancer [38]. Some metals interfere with various voltage- and ligand-gated ionic channels exerting neurotoxic effects. For instance lead affects the N-methyl-D-aspartic acid (NMDA) receptor, subtypes of voltage- and calcium-gated potassium channels, cholinergic receptors and voltage-gated calcium channels [30, 39].

Dioxin also causes the adverse effects as they modify the metabolism by inducing a number of various metabolic enzymes (e.g. CYPs, glutathione-transferase, tyrosine kinase etc.), homeostasis, through hormone modulation (e.g. estrogens, androgens glucocorticoids, insulin, thyroid hormones) and their receptors, and growth and differentiation by interfering with growth factors (e.g. EGF, TGFa, TNFa) and their receptors [40].

4. CONCLUSION

Air pollution from road transport and industrial activities has become an unavoidable part of our life. Research depicts that there is a severe impact of air pollutants on plants and their various parts. Since plants are the only manufacturers of food on earth, consumption of affected plants indirectly affects other organisms also. Moreover, studies have revealed that exposure to high level of air pollution has negative effects on human health directly, such as cardio-pulmonary diseases, premature deaths due to diseases of the heart and lungs. These pollutants suppress body's immune system to fight against infections. In nut shell, living in an environment with high air pollution for longer duration can affect the person's physical and mental health.

REFERENCES

[1] Gheorghe, I.F., and Ion, B., "The Effects of Air Pollutants on Vegetation and the Role of Vegetation in Reducing Atmospheric Pollution", *The Impact of Air Pollution on Health, Economy, Environment and Agricultural Sources*, 2011, Dr. Mohamed Khallaf (Ed.), In Tech, DOI: 10.5772/17660.

[2] Aburto, V., H., B., Castillo, J.,A.,R., Meza,V.,M.,T., Corey, G. and Fernández, G., O., "Evaluation of health effects of pollution" Organization for Economic and Co-operation Development, Paris, 2009, pp. 1-67.

[3] Hogsett W.E., Weber J.E., Tingey D., Herstrom A., Lee E.H., and Laurence J.A., "Environmental Auditing: An Approach for Characterizing Tropospheric Ozone Risk to Forests", *Environmental Management*, 21, 1997, pp. 105-120.

[4] Heck, W. W., Cure, W. W., Rawlings, J. O., Zaragoza, L. J., Heagle, A. S., Heggestad, H E., Kohut, R. J., Kress, L. W. and Temple, P. J., "Assessing impacts of ozone on agricultural crops: II. crop yield functions and alternative exposure statistics" *Journal of the Air Pollution Control Association*, 34, 1984, pp. 810-817.

[5] Heagle, A. S., "Ozone and crop yield", *Annual Review of Phytopathology*, 27, 1989, pp. 397-423.

[6] Krupa, S.V., Grunhage, L., Jager H.-J., Nosal, M., W.J. Manning, W.J., Legge, A.H., and Hanewald, K., "Ambient ozone (O_3) and adverse crop response: A unified view of cause and effect", *Environmental Pollution*, 87, 1995, pp.119-126.

[7] Walters, S., Griffiths, R.K., Ayres, J.,G., "Temporal Association Between Hospital Admissions for Asthma in Birmingham and Ambient Levels of Sulphur Dioxide and Smoke", *Thorax* 49 (2) 1992, pp. 133-140.

[8] Balmes, J.R., Fine, J.M., and Sheppard, D., "Symptomatic bronchoconstriction after short-term inhalation of sulfur dioxide", *The American Review of Respiratory Disease*, 136 (5), 1987, pp.1117-1121.

[9] Chauhan, A.J., Krishna, M.T., Frew, A.J., and Holgate, S.T., "Exposure to nitrogen dioxide (NO2) and respiratory disease risk", *Reviews on Environmental Health*, 13, 1998, pp.73-90.

[10] Vagaggini, B., Paggiaro, P.L., Giannini, D., Franco, A.D., Cianchetti, S., Carnevali, S., Taccola, M., Bacci, E., Bancalari, L., Dente, F.L. and Giuntini, C., "Effect of short-term NO_2 exposure on induced sputum in normal, asthmatic and COPD subjects", *European Respiratory Journal*, 1996, 9, pp. 1852-1857.

[11] Wegmann, M., Fehrenbach, A., Heimann, S., Fehrenbach, H., Renz, H., Garn, H., Herz, U., "NO2-induced airway inflammation is associated with progressive airflow limitation and development of emphysema-like lesions in C57bl/6 mice" *Experimental and Toxicologic Pathology*, 56, 2005, pp.341-350.

[12] Robert, U., A., and Edward, H., A., "Crossing the Energy Divide: Moving from Fossil Fuel Dependence to a Clean-Energy Future", 2009, Wharton School Publishing, 36.

[13] Badman, D.G., and Jaffe, E.R., "Blood and air pollution: state of knowledge and research needs", *Otolaryngoloy- Head and Neck Surgery*, 1996, pp. 114-205.

[14] Vermylen, J., Nemmar, A., Nemery, B., Hoylaerts, M.F., "Ambient air pollution and acute myocardial infarction", *Journal of Thrombosis And Haemostasis* 3, 2005, pp.1955-1961.

[15] Rastogi, S.K., Gupta, B.N., Husain, T., Chandra, H., Mathur, N., Pangtey, B.S., Chandra, S.V., Garg, N., "A cross-sectional study of pulmonary function among workers exposed to multimetals in the glass bangle industry", *American Journal* of *Industrial Medicine*, 20, 1991, pp. 391-399.

[16] Tager, I.B., Balmes, J., Lurmann, F., Ngo, L., Alcorn, S., Kunzli, N., "Chronic exposure to ambient ozone and lung function in young adults", *Epidemiology*, 16, 2005, pp. 751-759.

[17] Kuo, C.Y., Wong, R.H., Lin, J.Y., Lai, J.C., and Lee, H., "Accumulation of chromium and nickel metals in lung tumors from lung cancer patients in Taiwan", *Journal* of *Toxicology and Environmental Health A* 69, 2006, pp.1337-1344.

[18] Nawrot, T., Plusquin, M., Hogervorst, J., Roels, H.A., Celis, H., Thijs, L., Vangronsveld, J., Van Hecke, E., and Staessen, J.A., "Environmental exposure to cadmium and risk of cancer: a prospective population-based study" *The Lancet Oncology*, 7, 2006, pp. 119-126.

[19] Damek-Poprawa, M., and Sawicka-Kapusta, K., "Damage to the liver, kidney, and testis with reference to burden of heavy metals in yellow-necked mice from areas around steelworks and zinc smelters in Poland" *Toxicology* 186, 2003, pp.1-10.

[20] Jarup, L., "Hazards of heavy metal contamination", *British Medical Bulletin*, 68, 2003, pp.167-182.

[21] Loghman-Adham, M., "Renal effects of environmental and occupational lead exposure" *Environmental Health Perspectives*, 105, 1997, pp. 928-939.

[22] Boffetta, P., Merler, E., and Vainio, H., "Carcinogenicity of mercury and mercury compounds" The *Scandinavian Journal of Work, Environment* & *Health*, 19, 1993, pp. 1-7.

[23] Vamvakas, S., Bittner, D., Koster, U., "Enhanced expression of the protooncogenes c-myc and c-fos in normal and malignant renal growth", *Toxicology Letter*, 67, 1993, pp.161-172.

[24] Huang, Y.C., and Ghio, A.J., "Vascular effects of ambient pollutant particles and metals" *Current Vascular Pharmacology*, 4, 2006, pp. 199-203.

[25] Ewan, K.B., and Pamphlett, R., "Increased inorganic mercury in spinal motor neurons following chelating agents" *Neurotoxicology*, 17, 1996, pp. 343-349.

[26] Ratnaike, R.N., 2003. Acute and chronic arsenic toxicity, *Journal* of *Postgraduate Medicine*, 79, pp.391-396.

[27] Lasley, S.M., Gilbert, M.E., "Glutamatergic components underlying lead induced impairments in hippocampal synaptic plasticity" *Neurotoxicology*, 21, 2000, pp.1057-1067.

[28] Lasley, S.M., Green, M.C., and Gilbert, M.E., "Rat hippocampal NMDA receptor binding as a function of chronic lead exposure level" *Neurotoxicology* and *Teratology* 23, 2001, pp.185-189.

[29] Bellinger, D.C., "Teratogen update: lead and pregnancy", *Birth Defects Research* Part A: *Clinical* and *Molecular Teratology*, 73, 2005, pp. 409-420.

[30] Garza, A., Vega, R., and Soto, E., "Cellular mechanisms of lead neurotoxicity" *Medical Science Monitor*, 12, 2006, RA57

[31] Thomke, F., Jung, D., Besser, R., Roder, R., Konietzko, J., Hopf, H.C., "Increased risk of sensory neuropathy in workers with chloracne after exposure to 2,3,7,8-polychlorinated dioxins and furans", *Acta Neurologica Scandinavica*, 100, 1999, pp.1-5.

[32] Walkowiak, J., Wiener, J.A., Fastabend, A., Heinzow, B., Kramer, U., Schmidt, E., Steingruber, H.J., Wundram, S., Winneke, G., "Environmental exposure to polychlorinated biphenyls and quality of the home environment: effects on psychodevelopment in early childhood" *The Lancet* 358, 2001, pp.1602-1607.

[33] Wang, S.L., Lin, C.Y., Guo, Y.L., Lin, L.Y., Chou, W.L., Chang, L.W., "Infant exposure to polychlorinated dibenzo-p-dioxins, dibenzofurans and biphenyls (PCDD/Fs, PCBs) correlations between prenatal and postnatal exposure" *Chemosphere* 54, 2004, pp.1459-1473.

[34] Dalton, T.P., Kerzee, J.K., Wang, B., Miller, M., Dieter, M.Z., Lorenz, J.N., Shertzer, H.G., Nerbert, D.W., and Puga, A., "Dioxin exposure is an environmental risk factor for ischemic heart disease" *Cardiovascular Toxicology*, 1, 2001, pp. 285-298.

[35] Chavez, E., Jay, D., and Bravo, C., "The mechanism of lead-induced mitochondrial Ca2þ efflux", *Journal of Bioenergetics and Biomembrane* 19, 1987, pp 285-295.

[36] Goering, P.L., "Lead-protein interactions as a basis for lead toxicity" *Neurotoxicology*, 14, 1993, pp. 45-60.

[37] Rossi, E., Taketani, S., Garcia-Webb, P., Lead and the terminal mitochondrial enzymes of haem biosynthesis, *Biomedical Chromatography* 7, 1993, pp.1-6.

[38] Costa, M., Yan, Y., Zhao, D., and Salnikow, K., "Molecular mechanisms of nickel carcinogenesis: gene silencing by nickel delivery to the nucleus and gene activation/inactivation by nickel-induced cell signaling", *Journal of Environmental Monitoring*, 5, 2003, pp. 222-223.

[39] Toscano, C.D., Guilarte, T.R., "Lead neurotoxicity: from exposure to molecular effects", *Brain Research Reviews* 49, 2005, pp. 529-554.

[40] Birnbaum, L.S., "The mechanism of dioxin toxicity: relationship to risk assessment" *Environmental Health Perspectives* 102 (Suppl. 9), 1994, pp.157-167.