Relationship between environmental air pollution and allergic asthma

Vikas Deep Mishra[1,*], Nikhil Sinha[2]

[1]Asstistant Professor, Dept. of TB and Chest Diseases, [2]Associate Professor, Dept. of Internal Medicine, Heritage Institute of Medical Sciences, Bhadwar, Varanasi

***Corresponding Author:**
Email: vikasdmishra@gmail.com

Abstract
The ill-effects of environmental air pollution on respiratory and allergic diseases is increasing. The evidence is accumulating that the rise in the incidence of allergic diseases in the developing countries is largely due to the increase in air pollution as a result of rapid and ill-planned urbanization and industrialization. Major source of energy for today's world is biomass fuel and burning of biomass fuel produces high concentration of respirable particulate matter, toxic gases like nitrogen dioxide, sulphur dioxide and carbon monoxide and various other organic compounds. These form an important constituent of environmental pollution, capable of worsening respiratory and allergic diseases.

Keywords: Particulate matter, Nitrogen dioxide, Ozone.

Introduction

In recent times respiratory diseases and allergy are showing an increasing association with environmental (both indoor and outdoor) air pollution. The potential role of air pollution in increasing the prevalence of asthma and allergic diseases during the past few decades, has drawn the attention of researchers. Such an association was initially seen in the western world but, now it is being observed in few of the rapidly developing countries of the South East- Asia.[1] Nearly 50 percent of the world population depends on the fossil fuel for household energy, this leads to a mammoth 20 lakh tons of biomass fuel being burned everyday.[2,3] In rural India nearly 90 percent of combustion fuel is contributed by biomass fuel.[4] *Health Effect Institute* published a data in 2010 with reference to some major cities of India and China, showing that all natural causes of mortality increased by 0.6% on 10µg/m^3 increase in PM_{10}.[5] Previously low levels of allergy and asthma seen in developing countries, is now rising to match those of the western world. This is being reflected in experimental and epidemiological studies, implicating a harmful effect of traffic air pollution on the increasing trend of allergic diseases and asthma.

Outdoor pollutants

Sources for outdoor pollution are many. Burning of fossil fuel in factories and exhaust from motor vehicles engine all contribute to outdoor pollutants. Outdoor pollutants can be classified into *primary*[which are directly released into the environment e.g sulphur dioxide(SO_2), nitrogen monoxide(NO), nitrogen dioxide(NO_2) and particulate matter(PM)] and *secondary*[which are formed in the air by reactions of various primary pollutants and gases e.g. ozone(O_3)], and as *gaseous* and *particulate*, depending on their source, chemical composition, size and mode of release into the environment(6). PM are mainly produced by diesel powered motor vehicles but, can also be produced by various factories, mining areas, construction sites, power generation and biomass fuel. PM is a mixture of particles of varying size, shape and chemical composition, containing various oxides, polycyclic aromatic hydrocarbons, free radicals and transition metals. Diesel exhaust particles are produced by diesel powered motor vehicle, these have soot, ash particles, sulphates, silicates and metallic abrasion particles.

PM is sub-classified on the basis of particle size, which in turn is dependent on its source (natural or anthropogenic) or whether it is obtained from combustion or not. PM can be classified as follows-

a. Coarse PM: known as PM_{10} ; size 2.5 - 10µm
b. Fine PM: known as $PM_{2.5}$; size 0.1- 2.5 µm
c. Ultrafine PM: known as $PM_{0.1}$; size < 0.1µm

Because of their respirable size fine and ultrafine particles can be directly inhaled into the small airways and alveoli of the lungs and, can potentially interact with alveolar macrophages and epithelial cells. The ultrafine particles can adversely affect various other body organs as they can pass through the alveolar epithelial-endothelial layer and thus enter the blood stream.[7] It is also important to understand that mixture of various pollutants is more dangerous than individual components.

Indoor pollutants

Biomass fuel on combustion in poorly ventilated kitchens and/or poorly designed stoves leads to high concentration of respirable particulates (fine and ultrafine PM), gases *viz.* carbon monoxide, SO_2 and oxides of nitrogen and organic compounds like formaldehyde and benzene.[8,9] Few studies have shown strong association between combustion of indoor biomass fuel and increased incidence of bronchitis in

adult females and acute respiratory infections in children.[10,11] In 2000 Smith *et al.*[12] estimated that 5-6% of the national burden of disease in India is due to diseases caused by use of indoor biomass fuel.

Effects of Oxides of Nitrogen

NO_2 is present in emissions from c

ar exhaust, power plants and burning of biomass fuel. Upon exposure to NO_2 both acute and chronic changes in lung function are seen. It also causes neutrophilic inflammation and proinflammatory cytokines production. An increase in allergen response is seen in atopic asthmatics if they had past exposure to NO_2.[13] Belanger *et. al*[14] concluded that an increase of 20 ppb in concentration of NO_2, increased both the chances of and the number of days of wheezing and chest tightness in children.

Effects of PM

In experimental models, exposure to PM has been shown to produce airway hyper-responsiveness, oxidative stress and airway remodeling.[15,16] Nightingale *et.al.* showed that, on controlled exposure of healthy volunteers, to reconstituted 200 $\mu g/m^3$ of diesel exhaust particles (which has high concentration of PM) caused neutrophilic activation and neutrophilic inflammation.[17] Poorly controlled asthma and reduction in lung function has been seen in patients who have history of long term exposure to PM.[18,19] Increase in utilization of health care use due to asthma exacerbation, as a result of both acute and chronic exposure of $PM_{2.5}$ or PM_{10} has been shown in many studies.[20,21]

Effect of Ozone

Ozone (O_3) is formed as a result of interaction between oxides of nitrogen and hydrocarbons emitted from traffic and industrial sources. The reaction is catalyzed by photochemical reactions. In animal studies, a single acute exposure of O_3 induced an airway neutrophilia and p38-MAP kinase dependent smooth muscle contractility leading to airway hyper-responsiveness[22] whereas, long term exposure leads to emphysematous changes.[23] Human airway epithelial cells on exposure to approximately 100 ppb of O_3 release proinflammatory cytokines *viz.* GM-CSF and sICAM-1. This was more in cells from asthmatic individuals than non asthmatics.[24] Exposure to high levels of O_3 (0.4 ppm for 2 hours) caused a reduction in FEV_1 and an increase in bronchial hyper-responsiveness, again, this change was more in asthmatics than non-asthmatic individuals, although the symptoms were similar.[25] A positive association between levels of O_3 in air and hospital admission of elderly patients for exacerbation of asthma/COPD and emergency visits by children for asthma exacerbation was pointed out by Halonen *et.al.*[26]

Conclusion

Sufficient evidences have now gathered to show the negative impact of air pollution on asthma and other allergic diseases. Ill-effects of air pollution affects children and elderly to a greater extent. Although, further large-scale and more inclusive studies are needed but, still the currently-available data would serve as an important evidence-based footing in establishing the relationship between the air pollution and the allergic diseases. While the epidemiological evidences alone carries some degree of unreliability in defining the environmental etiology, assembled experimental proofs has provided affirmation supporting their causative roles. The importance of local and within a city differences and the various constituents of air pollution should continue to be investigated. More knowledge is needed about the specific pollutants or mixture of pollutants responsible for the adverse health effects.

References

1. Chung KF, Zhang J, Zhong N. Outdoor air pollution and respiratory health in Asia. Respirology 2011;16:1023-6. [PubMed]
2. Barnes DF, Openshaw K, Smith KR, van der Plas R. 1994. WhatMakes People Cook with Improved Biomass Stove? A Comparative International Review of Stove Programs. Washington, DC: The World Bank.
3. Reddy AKN, Williams RH, Johansson TB. 1996. Energy after Rio: Prospects and Challenges. New York: United Nations Challenges.
4. TEDDY: Tata Energy Data Directory Yearbook 1998-1999. New Delhi: Tata Energy Research Institute.
5. HEI International Scientific Oversight Committee. 2010. Outdoor Air Pollution and Health in the Developing Countries of Asia: A Comprehensive Review. Special Report 18. Health Effects Institute, Boston, MA.
6. Qingling Z, Zhiming Q, Kian FC, Shan-Ku H. Link between environmental air pollution and allergic asthma: East meets West. *J Thorac Dis.* 2015 Jan;7(1):14-22
7. Nemmar A, Hoet PH, Vanquickenborne B, et.al. Passage of inhaled particles into the blood circulation in humans. Circulation 2002;105:411-4.[Pub Med]
8. Albalak R, Bruce N, McCraken J, Smith KR, Gallardo T. 2001. Indoor respirable particulate matter concentration from an open fire, Improved cook stove and LPG/open fire combination in a rural Guatemalan community. Environ Sci Technol 35:2650-55.
9. Smith KR. 1993. Fuel combustion, air pollution and health: the situation in developing countries. Annu Rev Energy Environ 18:529-66.
10. Pandey MR, Boleji JS, Smith KR, Wafula EM. 1989. Indoor air pollution in developing countries and acute respiratory infections in children. Lancet 1:427-29.
11. Smith KR 2000. The National burden of disease in India from indoor air pollution. Proc Natl Acad Sci USA 97:13286-93.
12. Smith KR, Mehta S. 2000. The burden of disease from indoor air pollution in developing countries. Comparision of estimates. Presented at the USAID/WHO Global Technical Consultation on the Health Impacts of Indoor Air Pollution and Household Energy in Developing Countries, 3-4 May, Washington, DC.
13. Strand V, Svartengren M, Rak S, et al. Repeated exposure

to an ambient level of NO2 enhancesasthmatic response t o a nonsymptomatic allergen dose. Eur Respir J 1998;12: 6-12.[PubMed]
14. Belanger K, Gent JF, Triche EW, et al. Association of ind oor nitrogen dioxide exposure withrespiratory symptoms i n children with asthma. Am J Respir Crit Care Med 2006; 173:297-303. [PMC free article] [PubMed]
15. Stanek LW, Brown JS, Stanek J, et al. Air pollution toxic ology--a brief review of the role of thescience in shaping t he current understanding of air pollution health risks. Tox icol Sci 2011;120Suppl 1:S8-27. [PubMed]
16. Gowers AM, Cullinan P, Ayres JG, et al. Does outdoor ai r pollution induce new cases of asthma?Biological plausi bility and evidence; a review. Respirology 2012;17:887-9 8. [PubMed]
17. Nightingale JA, Maggs R, Cullinan P, et al. Airway infla mmation after controlled exposure to diesel exhaust particulates. Am J Respir Crit Care Med 2000;16 2:161-6. [PubMed]
18. Liu L, Poon R, Chen L, et al. Acute effects of air pollutio n on pulmonary function, airwayinflammation, and oxidat ive stress in asthmatic children. Environ Health Perspect 2009;117:668-74. [PMC free article] [PubMed]
19. Jacquemin B, Kauffmann F, Pin I, et al. Air pollution and asthma control in the Epidemiological study on the Genetics and Environment of Asthma. J Epid emiol Community Health 2012;66:796- 802. [PMC free article] [PubMed]
20. Malig BJ, Green S, Basu R, et al. Coarse particles and re spiratory emergency department visits inCalifornia. Am J Epidemiol 2013;178:58-69. [PubMed]
21. Silverman RA, Ito K. Age-related association of fine part icles and ozone with severe acute asthma inNew York Cit y. J Allergy Clin Immunol 2010;125:367-73. [PubMed]
22. Li F, Zhang M, Hussain F, et al. Inhibition of p38 MAPK -dependent bronchial contraction afterozone by corticoste roids. Eur Respir J 2011;37:933-42. [PMC free article] [P ubMed]
23. Triantaphyllopoulos K, Hussain F, Pinart M, et al. A mo del of chronic inflammation and pulmonary emphysema after multiple ozone exposures in mice. Am J Physiol Lung Cell MolPhysiol 2011;300:L691-700. [PM C free article] [PubMed]
24. Bayram H, Sapsford RJ, Abdelaziz MM, et al. Effect of o zone and nitrogen dioxide on the release of proinflammatory mediators from bronchial epithelial cells of nonatopic nonasthmatic subjects and atopic asthmatic patients in vitro. J Allergy Clin Immunol 2001; 107:287-94. [PubMed]
25. Kreit JW, Gross KB, Moore TB, et al. Ozone-induced ch anges in pulmonary function and bronchial responsiveness in asthmatics. J Appl Physiol (1985) 1989 ;66:217-22. [PubMed]
26. Halonen JI, Lanki T, Yli-Tuomi T, et al. Urban air polluti on, and asthma and COPD hospitalemergency room visits . Thorax 2008;63:635-41. [PubMed]