International Journal of Basic and Applied Medical Sciences ISSN: 2277-2103
Online, International Journal Available at http://www.cibtech.org/jms.htm
2020 Vol. 10(1) January-June, pp. 33-37/Gaude
***Review Article** (Open Access)*

ATMOSPHERIC AIR POLLUTION AND ASTHMA

***Gajanan S Gaude**
Gajanan S. Gaude, Department of Respiratory Medicine,
KAHER's J. N. Medical College, Belgaum -590010
**Author for Correspondence: gsgaude922@gmail.com*

ABSTRACT
Air pollution is an important contributing factor in aggravating the stable state of bronchial asthma leading to an exacerbation and may contribute to reduction in the lung functions if the exposure continues. Exposure to air pollution early in life contributes to the development of asthma throughout childhood and adolescence, particularly after age 4 years. Traffic-related air pollution in particular is associated with the development of asthma in school children. Air pollution exposure is thought to potentially cause asthma in children by impacting the developing lung and immune system. We hereby summarize the effects of air pollution in bronchial asthma patients.

Keywords: *Air Pollution, Asthma, Traffic Air Pollution, Clinical Effects*

INTRODUCTION
Around two thirds of people with asthma tell us poor air quality makes their asthma worse, putting them at risk of an asthma attack. This is because pollution can quickly irritate the airways and trigger asthma symptoms. Some pollution particles are small enough to enter into the lungs. Air pollution is a possible risk factor for everyone with asthma, but some people are more at risk, and may be affected by pollution even on moderate or low pollution days. In order to study the effects of atmospheric pollution on health, the effects of a brief exposure to a high content of pollutants must be distinguished from the symptoms of a prolonged exposure to average or low levels of pollution. It is acknowledged that historical pollution peaks (SO2 and particles simultaneously more than 500mg/m^3) such as of London in the 1950s, provoked respiratory and cardiac disorders with an increase in mortality affecting sensitive populations. The effects of atmospheric pollution on health, at the level at which they are encountered, are more difficult to study[1]. Two types of approaches are possible: The first is experimental studies on animals or on healthy volunteers; they do not produce actual conditions of exposure and of the environment in human beings, but enable us to draw up a hypothesis on the mechanisms of action of pollutants and characterize the effect of a specific pollutant. The second approach is epidemiological studies: they are less precise than laboratory tests but have the advantage of taking place in the actual conditions of human exposure and can permit the evaluation of the consequences of a prolonged exposure on the general population.

Allergy symptoms, especially bronchial asthma, are increasingly becoming frequent in all industrialized countries. It has been also observed that the not only the prevalence but also the severity of asthma has greatly increased in the course of the last few years (Aubier, 2000). Thus, the prevalence of asthma, which was 3% in 1972 in French University population, went to 5.6% in 1982 and to 14% in 1992. Even if the management of asthma has clearly progressed since the last 20 years, due to the better knowledge of the physio-pathological mechanisms of illness and the arrival of the new drugs and its combinations in the market, it still remains important to understand the root factors of its progress. The main etiological factors for the development of allergic diseases and asthma in particular, are of two kinds: genetic and environmental, with important interactions existing between the two. Identification and modification of genes responsible for the immunopathology of asthma could not constitute a coherent explanation for the increase in asthma prevalence observed in the last 20 years. Environmental factors therefore appear, to be the most plausible explanation. Among these factors, airborne pollens, air pollution and changes in

lifestyle are in the forefront as possible culprits. Bronchial asthma is defined as bronchial obstruction which is reversible, either spontaneously, or under the effects of bronchodilator drugs. On the physio-pathological level, asthma is characterized by an inflammation of the bronchial mucous membrane. This inflammation is continuous, more or less significant, according to the degree of seriousness of illness, triggered by many inflammatory cells including eosinophils and macrophages. Many studies have characterized this bronchial inflammation and studied the factors that could induce and/or aggravate it (Anderson *et al.,* 2013). Many atmospheric pollutants could interact with airborne allergens, magnifying response by aeroallergens. This interaction could be linked to the fact that a pollutant could be a co-factor of allergic reaction or it can induce a bronchial inflammation that could add to the inflammation which is itself produced by inhalation of the allergen. It's not only the outdoor air pollution that contributes to the development and exacerbation of asthma, but also the indoor air pollution including biogas mass fuel, chulah burning as in India, which contributes to a great extent in increasing development of bronchial asthma.

Pathogenesis: A better understanding of pathogenesis of asthmatic individuals who are susceptible to outdoor air pollution exposure should enable the design of effective preventive strategies. The theory that air pollution can cause exacerbations of pre-existing asthma is supported by several studies over the years (Friedman *et al.,* 2001), but recent evidence suggests that air pollution might cause new onset asthma as well (Nishimura *et al.,* 2013). A recent meta-analysis of cross-sectional studies that included different levels of pollution showed no effect of long-term exposure to pollution on asthma prevalence (Anderson *et al.,* 2013). Although most of the outdoor air pollution almost always occurs as a mixture, air quality is regulated by most jurisdictions in terms of its individual components. With increasing attention to traffic-related air pollution (TRAP) as the exposure variable of interest, a shift has focussed on individual components of the pollution mixture. Different types of pollutants can cause different level of damage to the airways. Specific pollutants can induce airway inflammation (eg, ozone, nitrogen dioxide, and PM <2·5 μm in diameter) and airway hyper-responsiveness (ozone and nitrogen dioxide) (Aris *et al.,* 1993; Poynter *et al.,* 2006). In addition, oxidative stress has been associated with pollutant exposures (ozone, nitrogen dioxide, and PM2·5) (Liu *et al.,* 2009). Therefore, exposure to these pollutants leads to exacerbations and possibly even the onset of asthma. The mechanisms by which pollutants induce these effects are not completely clear. Because the pollutants of interest, including TRAP, can cause oxidative stress, and the ability of antioxidant defences to handle the increased load of reactive oxygen species generated in the lungs after exposure is an important determinant of risk for subsequent adverse effects (Guarnieri *et al.,* 2014). There may be other pathways through which oxidising pollutants might also affect severity of asthma involving immune responses. TRAP, specifically ambient polycyclic aromatic hydrocarbons and diesel-exhaust particles, affect regulatory T cell (Treg) function through an epigenetic mechanism (Brunst *et al.,* 2013). Hypermetylation in Foxp3 associated with chronic exposure to polycyclic aromatic hydrocarbons or diesel-exhaust particles leads to suppression of Treg function and increased asthma severity as assessed by symptoms and lung function.

Even indoor air pollution also can be a significant problem for the asthmatic patients. It can come from indoor cooking or heating, open fireplaces, passive smoking, indoor plants and offgassing of building materials. This problem has been increasingly recognized and was termed 'sick building sydrome' (SBS) referring to health issues with subsequent loss of productivity and increased sick leave. Indoor air pollution leads to increased incidence of pneumonia, allergy, chronic obstructive pulmonary diseases (including asthma), lung cancer and others. In fact, the World Health Organization estimates that 4.3 million people worldwide die every year from indoor air pollution, mostly from indoor use of solid fuels (Bowatte *et al.,* 2015). Interventions include switching to alternative fuels such as electricity or solar power, using improved stoves and having adequate ventilation.

Pro-inflammatory Effects: Many atmospheric pollutants are capable of inducing an inflammation at different levels of the airways. The former is at the root of a bronchial hyper-reactivity of the airways. These pollutants are responsible for bronchial irritation, depending upon different individual mechanisms, which are often accompanied by cytotoxic effect on epithelial cells of airways. These effects have been studied experimentally in vitro, on animals or human beings at controlled exposures. The principal pollutants studied are sulphur dioxide, oxides of nitrogen and ozone. All these studies have shown that pollutants, generally with high concentration, could be the root cause of a transient bronchial inflammation. The clinical significance of this inflammation is, however, difficult to establish. Many studies have shown that asthmatic or allergic subjects were not more sensitive to the effects of ozone than healthy subjects, contrary to the results obtained with SO_2 or NO_2. In an identical population, some subjects might react to ozone exposure with a decrease of FEV_1 and other measures. In elderly population, exposure to ozone only entails little or no modification of the pulmonary functions (Linn *et al.,* 1988). The response to ozone would decrease with age, or at certain periods during the year. Moreover, it must be noted that in healthy subjects these effects were observed at higher concentration of ozone than the maximal concentrations that could have been measured in the atmosphere.

Numerous epidemiological studies concerning mortality and morbidity figures have been conducted since 1970s for studying the relation between atmospheric pollution and asthma. In ecological studies, the state of the health is estimated from aggregate data of hospitalizations, emergency visits, absenteeism etc. These studies are generally retrospective, and allow into account certain confounding factors, such as influenza epidemics, meteorological factors, and long-term variations. Prospective studies evaluates the state of health from individual data like focussing on healthy patients or asthmatic patients and its risk factors, or take into account numerous environmental factors linked to respiratory ailments and, finally, utilizing the appropriate techniques of analysis. The long term effects of continued exposure to pollution are determined from cross-sectional studies carried out in geographically different zones. Finally, one must understand that the relation between atmospheric pollution and asthma is complex. Multi-factor phenomena have an influence on pollution, as well as on health.

Short Term Effects: There has been some relation between atmospheric pollution and readmissions to emergency departments due to asthma exacerbations. The first study showed that the correlation between exposure to PM10 (particles of size equal to 10u) and asthma severity in people more than 65 years (Neukirch *et al.,* 1999). Pollution levels observed in the region of Paris in the course of this period were linked to problems of health in spite of relatively low pollution peaks. Thus, it's not only pollution peaks which were implicated, but also pollution levels commonly observed in the area. For example, an increase in the level of fine suspected particles upon 26, 74 and 111 mg/m^3, on a daily average, was accompanied by increases of: 2%, 4% and 5% of non – accidental mortality; 2%, 5% and 10% of the daily number of hospitalizations at state hospitals for respiratory and cardiovascular causes; and 4%, 18% and 30% of the daily number of SOS home visits for asthma (Neukirch *et al.,* 1999). Some of the studies have shown the short-term effects of pollution on respiratory health. These studies were analysed according to suitable techniques for this type of data, taking into account some factors which are weather-dependent at the time of the study (Segala *et al.,* 1998). The objective was to study the daily relation between the acid-particulate pollution of winter and the morbidity linked to asthma. The results of these studies indicated that there was harmful effect of acid-particulate pollution in asthmatic subjects, after taking into account the temperature and the relative humidity. At the same time, the incidence and duration of the symptoms slightly but significantly increased with pollution, in spite of the low levels of black fumes and SO_2.

Long Term Effects: The long-term effect of a continued exposure to atmospheric pollution was studied by comparing different geographic zones. Forastiere *et al.,* (1994) compared the prevalence of bronchial hyper-reactivity in a polluted industrial pollution and that of non- polluted rural zone, in a representative sample of children from 7 to 14 years. They concluded that the facts of living in a polluted area, even if

pollution levels are lower than standard levels, could increase bronchial hyper-reactivity, independently of asthma, atopy and level of pulmonary functions. In Germany, the reunification allowed for the comparison of populations, which were genetically close, but had very different life styles and environmental exposures. Mutius *et al.,* (1995) studied children from 9 to 11 years, in Munich, where industrial pollution was weak, but the automobile traffic was dense, and at Leipzig, in former East Germany where industrial pollution was high. They concluded that sensitivity to aeroallergens was much more frequent in Munich. This could explain the differences between the former West Germany and the former East Germany regarding the prevalence of asthma and hay fever. On the other hand, in the former East Germany, the prevalence of bronchitis was observed to be higher than in the West, and a significant association was found between the levels of SO_2 and suspended particles, and the symptoms of the upper airway were observed (Forastiere *et al.,* 1994).

Concurrent exposure to aeroallergens and air pollutants can enhance airway inflammation and lead to asthma exacerbations. A cross-sectional study using population-level data showed that high exposure to fungal and pollen allergens can interact with PM2.5 or coarse particulate matter measuring less than 10 μm in diameter to increase the risk of hospitalizations for asthma. In another study (Stevens *et al.,* 2019), co-exposure to diesel exhaust particles (DEPs) and dust mite allergen increased TH2 cytokine levels and airway responsiveness in a murine model, and high exposure to DEPs was associated with asthma in atopic children but not in non-atopic children. Small clinical trials (Brandt *et al.,* 2015; Carlsten *et al.,* 2016) in adults have demonstrated enhanced allergen-induced airway inflammation in participants exposed to DEPs, ozone, and NO_2. Whether by modifying outdoor activity during the high pollen season or reducing indoor allergen levels attenuates pollutant effects on asthma requires further research.

CONCLUSION

In conclusion, the effects of atmospheric pollution, including indoor air pollution in patients with bronchial asthma are still unclear. Current evidence suggests that chronic stress and an unhealthy diet or low levels of antioxidant vitamins interact with air pollution to cause or worsen asthma. Well-designed randomized clinical trials should help determine the extent to which dietary interventions or vitamin supplementation mitigate the harmful effects of air pollution on asthma. Because of the complex etiology of asthma, multifactorial trials examining several interventions (*i.e.,* weight loss, stress reduction, and a healthy diet) might be best suited to identify the most effective modifiers of pollutant-related effects.

REFERENCES

Anderson HR, Favarato G, Atkinson RW (2013). Long-term exposure to air pollution and the incidence of asthma: meta-analysis of cohort studies. *Air Quality and Atmosphere and Health,* **6** 47-56.

Aris RM, Christian D, Hearne PQ, Kerr K, Finkbeiner WE, Balmes JR (1993). Ozone-induced airway infl ammation in human subjects as determined by airway lavage and biopsy. *American Review in Respiratory Disease,* **148** 1363–72.

Aubier M (2000). Relation between asthma and atmospheric pollution. *Le Concouurs Medical,* **122**(34) 2419-2423.

Bowatte G, Lodge C, Lowe AJ, Erbas B, Perret J, Abramson MJ, Matheson M, Dharmage SC (2015). The influence of childhood traffic-related air pollution exposure on asthma, allergy and sensitization: a systematic review and a meta-analysis of birth cohort studies. *Allergy*, **70**(3) 245-256.

Brandt EB., Biagini Myers, JM, Acciani TH, Ryan PH., Sivaprasad U, Ruff B, *et al.* (2015). Exposure to allergen and diesel exhaust particles potentiates secondary allergen-specific memory responses, promoting asthma susceptibility. *Journal of Allergy and Clinical Immunology,* **136** 295–303.

Brunst KJ, Leung Y-K, Ryan PH, *et al.* (2013). Forkhead box protein 3 (FOXP3) hypermethylation is associated with diesel exhaust exposure and risk for childhood asthma. *Journal of Allergy and Clinical Immunology,* **131**(2)592-594.

Carlsten C, Blomberg A, Pui M, Sandstrom T, Wong SW, Alexis N, *et al.* (2016). Diesel exhaust augments allergen-induced lower airway inflammation in allergic individuals: a controlled human exposure study. *Thorax,* **71** 35–44.

Forastiere F, Corbo GM, Pistelli R, Michelozzi P, Agabiti N, Brancoto G, Ciappi G, Perruci CA (1994). Bronchial hyperresponsiveness in children living in areas with different air pollution levels. *Archives of Environmental Health,* **49** 111-118.

Friedman MS, Powell KE, Hutwagner L, Graham LM, Teague WG (2001). Impact of changes in transportation and commuting behaviors during the 1996 Summer Olympic Games in Atlanta on air quality and childhood asthma. *JAMA,* **285** 897–905.

Guarnieri M, Balmes JR (2014). Outdoor air pollution and asthma. *Lancet,* **383** 1581–1592.

Linn W, Avol E, Shamoo D (1988). Repeated laboratory ozone exposure of volunteer Los Angeles residents: Apparent seasonal variation in response. *Toxicology and Industrial Health,* **4** 505-520.

Liu L, Poon R, Chen L, *et al.* (2009). Acute eff ects of air pollution on pulmonary function, airway infl ammation, and oxidative stress in asthmatic children. *Environmental Health Perspective,* **117** 668–74.

Neukirch F, Segala CI, Le Moullec Y, Korobaeff M, Aubier M (1999). Short term effects of low level winter pollution on respiratory health of asthmatic adult. *Archives in Environment and Health,* **53**(5) 32-328.

Nishimura KK, Galanter JM, Roth LA, *et al.* (2013). Early-life air pollution and asthma risk in minority children. The GALA II and SAGE II studies. *American Journal of Respiratory Critical Care Medicine,* **188** 309–18.

Poynter ME, Persinger RL, Irvin CG, *et al.* (2006). Nitrogen dioxide enhances allergic airway inflammation and hyper responsiveness in the mouse. *American Journal of Physiology-Lung Cell Molecular Physiology,* **290** L144–52.

Segala C, Fauroux B, Just J, Pascual L, Grimfeld A, Neukirch F (1998). Short term effects of winter air pollution on respiratory health of asthmatic children in Paris. *European Respiratory Journal,* **11** 677-685.

Stevens EL, Rosser F, Forno E, Peden D, Celedon JC (2019). Can the effects of outdoor air pollution on asthma be mitigated? *Journal of Allergy and Clinical Immunology,* **143**(1) 2016-9.

Von Mutius E, Sherill DL, Fritzsch C, Martinez FD, Lebowitz MD (1995). Air pollution and upper respiratory symptoms in children from East Germany. *European Respiratory Journal,* **8** 723-728.