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# EFFECTS OF OUTDOOR AIR POLLUTION ON RESPIRATORY HEALTH AND PREVENTIVE MEASURES

Respiratory Medicine

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## ABSTRACT

**INTRODUCTION:** In the words of the World Health Organization, air pollution is defined as contamination of the domestic or outdoor environment by chemical, physical, or biological pollutants that have an adverse effect on the natural components of the atmosphere. The consequence could be adverse health effects that could be sub-clinical or overt. Despite these, little is known about the health impact of urban air pollution at the cellular and sub-cellular levels among people residing in the Indian mega cities. Against this background, this literature review is being conducted in order to identify ways for reducing environmental pollution and the effects on the body. **METHODS:** An extensive search of all materials related to the topic was carried out in PubMed, Medline, World Health Organization website and Google Scholar search engines. Relevant documents, reports, research articles focusing on the air pollution and its impact on health published in the period 1992 to 2021 were included in the review. A total of 49 articles on our topic were included in this study. **RESULTS:** In this review article we discussed about the distribution and multiple determinants of respiratory diseases caused by the air pollutants, its sources and pathogenesis of the diseases. **CONCLUSION:** People today are constantly exposed to outdoor air pollution causing increased respiratory morbidity and mortality. The continuous health education on air pollution and personal protective measures at regular intervals is mandatory to improve respiratory health. The impact of adherence, change in behaviour and lifestyle modification helps in significantly reducing the exposure, symptoms and respiratory health among people.

## KEYWORDS

Air Pollution, Respiratory morbidity, Respiratory health, Pollutants.

### Introduction and epidemiology:

The World Health Organization defines air pollution as the detrimental effects of chemical, physical, or biological pollutants on natural components of the atmosphere. The most damaging gases in the atmosphere are particulate matter, carbon monoxide, and ozone. Indoor and outdoor air pollution causes fatal respiratory and other illnesses. (1) Chemical, particle, or biological pollutants polluting the atmosphere, causing human disease and death, damaging other living species such as plants, or limiting visual acuity. Pollution comes from mobile (automobiles), stationary (factories, power plants, refineries) and indoor sources (e.g., building materials, gas stoves, passive smoking, etc.)(2-4)

The effects of urban air pollution on the general public are well-known. Globally, pollution causes 5,000 fatalities per year from pneumonia, COPD, and other causes. (5) Global public health concerns about air pollution have grown. Pollutants include particulate and gaseous contaminants (CO,NO, SO2). The latest Global Burden of Disease report lists outdoor air pollution as a leading cause of death and lost disability-adjusted life years, accounting for almost 3 million premature deaths each year (6). Inhaling gasoline vapour or dust can severely damage the lungs and other organs. Because of their regular exposure to these toxic substances, traffic officers are especially susceptible (7). Early preventative and corrective measures help reduce long-term effects on pulmonary function (8).

The World Health Organization estimates that air pollution causes around 7 million premature deaths every year. This staggering number of deaths occurs because 91 percent of the world's population lives in locations with air quality indexes that exceed acceptable levels of pollution exposure. Increasing urbanisation and industrialisation, coal combustion for electricity generation, windblown mineral dust, agricultural stubble burning, waste burning, transportation vehicles, and diesel generators are all major sources of air pollution. The most common ways of measuring air pollution are ambient particle, indoor particle, and ambient ozone. In this context, this literature review aims to discover solutions to reduce environmental pollution and its consequences on the body.(10)

### Various pollutants and sources

#### Particulates

Particulates are solid or liquid materials in the air. Construction and demolition, powdered insecticides, bioaerosols and volcanic ash are also sources of particles. These particles can fly thousands of kilometres over oceans and land on distant continents. Biomass burning of wood, leaves, crops, and forests is a major source of particulates globally (11).

Particulates less than 10 m (PM10) or 2.5 m (PM2.5) are considered particularly toxic due to their ability to easily penetrate the lungs into the alveoli. Whether PM10, PM2.5, or PM2.5–10 are more harmful to human health is currently debated (Brunekreef and Forsberg, 2005).(12). Particulate toxicity varies with chemical makeup. Toxic metals like lead and mercury, poly aromatic hydrocarbons (PAHs), and persistent organic pollutants like dioxins are all of concern.

#### Ozone (O3)

The ozone molecule has three oxygen atoms (O3) instead of two (O2). Ozone is important in the stratosphere because it blocks harmful UV light from the sun, yet it is deadly to humans. Ozone is formed indirectly by atmospheric reactions involving volatile organic compounds, nitrogen oxides, and sunshine; this indirect ozone generation is most efficient during warm weather. Ozone, like particles, may travel thousands of km. Opacity is 1–2 weeks in the summer and 1–2 months in the winter.(13)

Ozone irritates the lungs at high doses, especially in the summer. Ozone pollution causes asthma and other lung problems. Severe asthmatics' symptoms worsen even when ambient ozone levels are below the EPA's standards. High ozone levels exacerbate existing heart conditions like angina.

#### Carbon monoxide (CO)

Incomplete combustion produces CO. It comes from automotive, heating, coal-fired power plants, and biomass combustion. Carbon monoxide is created in greater proportions by inefficient combustion, colder weather, and higher elevations. Carbon monoxide has a half-life of 1–2 months in the atmosphere and can travel thousands of kilometres (14).

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**Sulfur oxides (SO2, So3), hydrogen sulfide (H2S), acid gases (HF, HCl)**

Car emissions, oil/gas fields, and refineries all produce sulphur dioxide (Godish, 2003). The degradation of oil or dead plants produces hydrogen sulphide. In addition to sulphur dioxide, mercaptans and other sulfur-containing compounds are created by industries such as papermaking and rayon production. Many industries and waste combustion sources emit acid gases like HCl and HF.

**Nitrogen oxides (NO2 and others)**

Nitrogen oxides are emitted via industrial/vehicular combustion and fertiliser oxidation (Godish, 2003). Nitrogen oxides are created in the atmosphere when reactive oxygen molecules react with nitrogen.

**Lead (Pb) and other metals**

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**Volatile organics (VOC's), solvents, pesticides and methane (CH4)**

This category includes compounds that easily volatilize (evaporate) into the air. A large number of companies and various industries produce volatile emissions from coniferous woods.

**Bioaerosols: molds, bacteria, pollen and others**

Bioaerosols are microscopic particles created by living organisms. The list includes pollen, seeds, bacteria (including endotoxins), moulds (fungi), algae (protozoa), flour (latex), pollen and seeds. Some bioaerosols are harmful due to their infectiousness, allergies, or poisons. Mold spores can span thousands of kilometres of ocean to land on neighbouring continents (16).

**Radiation**

Radioactive chemicals can reach the atmosphere through natural decay, nuclear power plants, weapons testing, and industrial, research, and medical uses of radioisotopes.

**Standards for air pollutant concentrations**

Several governments and organisations have set regulations for contaminant concentrations in outdoor air. The daily air quality index (AQI) is used in many nations throughout the world. Both India and the US use the same AQI formula. AQI values of 0-5 are regarded 'Good', 51–100 'Satisfactory', 101–200 'Moderately Polluted', 201–300 'Poor', 301–400 'Very Poor', and 401–500 'Severe'.

Priority air pollutants regularly exceed these limitations, especially in developing countries' major cities. For example, in 2004, the EPA calculated that 18% of the world's urban regions (cities with populations over 100,000) had ambient air containing over 100 ug/m3 PM10, which is double the US EPA standard (17).

**Respiratory diseases caused by the pollutants**

**Effect On Lung Function**

P Pal and colleagues investigated the impact of air pollution on traffic cops' lung function tests in Pondicherry, India. This study used the case-control method, using traffic cops as cases and general police officers of similar age and BMI as controls. Smokers and non-smokers were sub-grouped because prolonged smoking has been demonstrated to affect PFT. The difference between cases and controls was 0.05, so there was a significant decline in vital capacity, FEV1, FEF-25, and PIF among nonsmokers. VC, FEV1, PEF, MVV, FEV-25, and PIF all decreased in smokers. These modifications show that car pollution limits lung expansion, as well as obstructs and constrictes airways. (18)

V Paul and colleagues conducted a cross-sectional study of traffic cops at several crossroads in Mangalore. The study included 100 traffic cops and 100 non-teaching employees of Father Muller Medical College as controls. Years of exposure were used in traffic crew subgroup analysis. The exposed group had lower FVC, FEV1, and PEFR, and a higher FEV1/FVC ratio than the control group. (19)

U P Santos and associates examined the impact of air pollution on adult respiratory function. The study included 101 nonsmoking volunteers, most of them were taxi drivers, traffic controllers, and forest rangers. Participants in this cohort study were asked to take a spirometer test and a 24-hour personal PM 2.5 test over four weeks. A regression model that adjusts for age and characteristics like waist circumference, job time, daily labour hours, diabetes or hypertension, and smoking history. They produced a 12.2% decrease in FVC and a -9.1% fall in FEV1, as well as a 14.9% increase in FEF 25-75 percent /FVC without affecting FEV1/FVC. (20)

**Asthma**

Most studies on pollution's health consequences have focused on respiratory difficulties. Children are often the subjects of studies that relate greater rates of asthma and other respiratory disorders to higher levels of priority pollutants such particulates, ozone, sulphur and nitrogen oxides, and Co2.

Adult asthma is becoming more severe in high-income countries, including Denmark. The economic impact connected with hospitalisation, drugs, and missed work years owing to illness and mortality is enormous. The burden is expected to grow as the number of older people with asthma increases. (21)

High levels of numerous common pollutants (PM10 or PM2.5, O3, CO, SO2, NO2, benzene) are linked to increased asthma symptoms, consultations, and/or hospitalizations. small concentrations Asthma in children can be worse by PM10, CO, O3, NO2, and SO2 levels below the US EPA's.

**Chronic Obstructive Pulmonary Disease (COPD)**

Chronic Obstructive Pulmonary Disease (COPD) causes irreversible constriction of the airways. COPD is caused by contaminants that promote inflammation and an immune response. Chronic bronchitis is the inflammatory response in bigger airways. It causes emphysema in the tiny air cells at the end of the lungs' tiniest passages. Although most COPD patients are smokers, air pollution plays a role in the development of COPD and the origin and development of acute exacerbations.

Song, Q. A meta-analysis found that outdoor air pollution increases the incidence and prevalence of COPD. The same study's authors claim that a 10 g/m3 rise in PM10 in outdoor air might cause severe COPD exacerbations and even mortality. [22]

Low to moderate outdoor air pollution can exacerbate geriatric respiratory issues. A 1980–1995 study of Tokyo residents aged 65+ indicated increased rates of asthma and bronchitis with increasing outdoor PM10 concentrations (23).

**Lung Cancer**

Lung cancer is the biggest cancer killer in both men and women in the United States. While this is true, additional variables such as air pollution increase the risk of lung cancer. Particulate particles and ozone may affect lung cancer mortality. Four out of five studies in Europe and the US indicated an increased risk of lung cancer from greater outdoor levels of PM10/PM2.5, vehicle traffic, and NO2, while the fifth study reported a non-significant elevated risk (24). A analysis of 24 research indicated that subjects residing near garbage incinerators had considerably increased rates of cancer (lung, larnyx, and non-Hodkins lymphoma). Studies on asphalt and coke oven workers have established a relationship between PAHs such benzo(a)pyrene and lung cancer.

Based on data from the International Agency for Research on Cancer, the IARC classified outdoor air pollution and related PM as a class I human carcinogen in October 2013. (IARC). [25]A meta-analysis by Chen G et al found that exposure to nitrogen oxides, sulphur dioxide, and PM2.5 from vehicle emissions increases the risk of lung cancer. [26]

**Pathogenesis and effects studies**

Particulate matter is a complex mixture of carbonaceous core, organic molecules, acids, and microscopic metal particles. Particles smaller than 100 nanometers are referred to as ultra-fine particles (UFP). Particulate matter typically accumulates in the lungs, airways, and centriacinar regions following inhalation, contributing to disorders including asthma and COPD. COPD, asthma, lung fibrosis, and cancer are connected to the formation of reactive oxygen species (ROS), oxidative stress, adaptive immunity, and innate immunity by UFP-mediated cell damage. (27,28)

Jalava and colleagues cultured airborne particles from six European cities in vitro for 24 hours using mouse macrophages. PM0.2-2.5

values of 300 pg/mL to 150 g/mL dramatically reduced alveolar macrophage viability. TNF expression in alveolar macrophages increased with particle concentration. [29]

**ROS and oxidative stress:**

Toxins, tachykinins, and neurokinins are released when reactive oxygen species (ROS) interact with airway epithelial cells. Thus, choline-esterase and brain endopeptidase levels would be reduced, whereas beta adrenergic receptor function would be impaired in the airway walls. According to study, asthmatic ROS levels are associated to airway responsiveness. (30–32) The anti-oxidant pathway is also downregulated in COPD exacerbation. This leads to severe lung injury and acute respiratory distress syndrome. Oxidative stress-induced TGF-synthesis in lung epithelial cells, as well as the production of cytokines and growth factors, are thought to be important in the development of lung fibrosis.

**Innate and Adaptive Immunity:**

The inflammasome modulates inflammation by activating specific cytokines to lessen inflammation in humans. Innate immunity is mainly reliant on these two. In experiments on mouse cells, OVA induced hyperresponsiveness and inflammation in the airways, which intensified when the animals were exposed to TiO2 particles. As previously noted, continuous exposure to 2.5 micron or smaller particulate matter reduces the human nasal epithelium's barrier function, as indicated by reduced translation of TJ proteins and increased production of pro-inflammatory cytokines.

In innate immunity, as in adaptive immunity, IL-13 production is the major effector for Th2 phenotypic modification. Like in adaptive immunity, alveolar macrophages produce IL-13 to regulate inflammatory responses towards the Th2 phenotype.

In innate immunity, alveolar macrophages regulate inflammatory responses towards the Th2 phenotype by producing IL-13 (33). Lung fibrosis and goblet cell hyperplasia have been linked to long-term diesel exhaust particle exposure, according to the American Lung Association.(34,35)

**Prevention And Protection From Air Pollution:**
**Targets For Intervention:**

Air pollution is a major cause of respiratory diseases. 88 Exposure to PM10 at 20 g/m3 resulted in economic expenses related with respiratory disorders of 0.194% of China's GDP (0.175% at 50 g/m3). Thus, successful actions to limit air pollution exposure can have significant economic benefits (36).

**Exposure Mitigation strategies**

Personal protection, behavioural change, and pharmaceutical intervention can all help reduce workplace exposure hazards. Avoiding pollution and exercising near highways and large arterial roads are two examples of actions that may help reduce traffic-related UFP and other environmental components. Avoiding air pollution by using a GPS warning system to help motorists find alternate routes. Staying indoors, reducing exercise and activity: The other two techniques to minimise pollution and death are non-linear dose-response function curves for exercise mediated health benefits and particulate matter caused mortality. (37)

**Addressing environmental health in all sectors**

The Royal College of Physicians of London(38) and The Lancet (39) have advocated various organizational-level approaches. Transportation plays a vital role in reducing traffic-related air pollution, which has serious health implications. Chronic pharyngitis, sputum output, chronic rhinitis, and throat pain were all associated with exposure to traffic-related air pollution. (40)

Governments cannot achieve environmental health alone. Research institutions should be actively involved in developing pollution-reducing technologies. Individual corporations must strictly follow all rules and regulations. Finally, schools should teach students how to reduce pollution.

**Fuel And Vehicle Upgrading**

Use of low or ultra-low sulphur diesel has banned lead-based anti-knock addictive. In 2013, the fifth National Vehicle Gasoline Standard set sulphur (10 ppm), manganese (2 mg/L), and alkenes (25 %) higher limits. Hybrid public cars using LNG and electricity have gained popularity since 2008, reducing pollutant emissions by 15–35 percent. Upgrading industrial tech Modernization of coal production and combustion facilities is required. Factories should be modernised and relocated. Relocating a cement mill (a major source of industrial air pollution) from downtown helps improve local air quality.

**Legislation to ban smoking**

In spite of tobacco ban advertising, the sponsorship, and promotion of tobacco products, the smoking rate remains steadily high. (41) this contributes to air pollution significantly worldwide.

**Healthy cities**

Sustainable development prioritises healthy cities. Cleaning up the air is important because it protects citizens' rights and welfare. It is also important because it improves access to health care facilities and improves the quality of care.

**Establishment of early warning systems**

During haze days, patients at risk should stay indoors. Weather forecasts may help reduce exposure. A national real-time display of PM2.5 concentrations is required.

Satellite monitoring has enabled real-time monitoring of air pollution, particularly PM, and dynamic monitoring over extended periods (>4 years) can help elucidate time trends of exacerbations. Compared to ground-based air quality monitoring, satellite monitoring provides estimates of pollutant exposure in locations (e.g. rural areas) where stationary monitoring data is lacking.

**Personal protection:**

When used effectively, portable air purifiers can reduce indoor PM 2.5 by 50% to 60%. (42) PAC has been shown to reduce the risk of respiratory issues. Compared to PAC, which was computed during wildfires and tested in a modelling analysis, high-efficiency domestic air filtration systems are less effective. Auto air filtration and cooling can protect against PM and UFP exposure. High-efficiency cabin filters reduce PM 2.5 levels in the cabin better than standard cabin air filters (43). Air-purifying respirators cover the nose and mouth, lowering particulate matter inhaled by the user. (44) . Closing windows and using cabin air filters lowered PM by 35%, reducing oxidative stress.

According to their investigation, Shakya et al found mixed findings in terms of filtration efficacy and particle size (all smaller than 1m). These size-dependent effects may be linked to the fact that submicron-sized particles are more likely to deposit by diffusion. Diesel exhaust particulates are so tiny that they can enter the alveolar portion of the lung and then enter the circulatory system. [45]

Bin Han et al. conducted a double-blind cross-over randomised trial on 39 healthy college students. For two hours, these volunteers were exposed to busy roadways while wearing either a powered air-purifying respirator (PAPR) or a N95 mask. The students were blinded by the mask (fine particle filter or volatile organic filter or sham filter or N95 mask). These masks were given to students at random and worn for a week. Using the sham filter raised post-exposure FENO by 2.3 ppb. The combined PM 2.5 and VOC PAPR filters showed no increase. The key components involved for airway inflammation are particulate particles or inorganic gases produced by TRAP. (46)

A study by Shakya et al found that a cotton mask's performance was unexpected, due to the mask's inability to completely seal around the face. Another study compared the ability of three masks to filter PM2.5 particles: bandana, biker, and surgical. They found that surgical masks filtered PM2.5 better than other varieties. But their performance was erratic(45). D Patel et al In another study, AmielAdoretal found that using a face mask helped patients with chronic allergic rhinitis who also had intermittent sickness. The study supports the idea that face masks can reduce atopic allergy symptoms. The proposed procedures involve physical filtration of face masks and the intake of humidified or heated air to alter the physiological response to allergens. The use of allergen-specific masks may be seen as a prophylactic measure in high-risk settings.(47)

**Air filters and monitors**

Indoor air pollution can be reduced with air filters. Using effective air filters in dormitories for 48 hours reduced PM2.5 levels by 57% (from 962 to 413/m3), which was associated with a 68% reduction in serum interleukin-1, a 69% reduction in serum soluble CD40 ligand, a 32% reduction in serum myeloperoxidase, and a 175% reduction in serum monocyte chemoattractant protein-1 in healthy college students. (48)

**Dietary recommendations**

By reducing oxidative stress, antioxidant-rich diets (leafy vegetables and fruits) may help reduce the effects of pollution. High doses of vitamin C and E antioxidants reduced nasal inflammation and partially restored antioxidant levels in asthmatic patients exposed to high amounts of ozone. Compared to filtered air, N-acetylcysteine supplementation reduced airway hyper-responsiveness by 42% in patients with diesel exhaust hyper-responsiveness. (49,50). Consumption of antioxidants and vitamins C should be encouraged everyday as part of healthy life.

**Reduced smoke exposure and improved ventilation**

Asthma in children is linked to second-hand smoke from indoor cigarette smoking. Policies preventing smoking in closed public locations could reduce environmental tobacco smoke exposure. Smoking rates remain high despite access to cessation clinics and drugs.(51)

## REFERENCES:


1. Lin H-H, Ezzati M, Murray M. Tobacco smoke, indoor air pollution and tuberculosis: a systematic review and meta-analysis. PLoS Med. 2007;4(1):e20.
2. American Thoracic Society. What constitutes an adverse health effect of air pollution? Official statement of the American Thoracic Society: This official statement of the American thoracic society was adopted by the ATS board of directors, July1999. Am J Respir Crit Care Med. 2000;161(2 Pt 1):665–73.
3. Ghio AJ, Carraway MS, Madden MC: Composition of air pollution particles and oxidative stress in cells, tissues, and living systems. *J Toxicol Environ Health B Crit Rev* 15:1–21, 2012.
4. Health effects of outdoor air pollution. Committee of the Environ- mental and Occupational Health Assembly of the American Tho- racic Society. *Am J Respir Crit Care Med* 153:3–50, 1996.
5. Cheng M F, Tsai S S, Wu T N, Chen P S, Yang C Y. Air pollution and hospital admissions for pneumonia in a tropical city: Kaohsiung, Taiwan. J Toxicol Environ Health A 2007; 70: 2021–2026
6. George D Thurston, Howard Kipen, Isabella Annesi-Maesano, et al. A joint ERS/ATS policy statement: what constitutes an adverse health effect of air pollution? An analytical framework. European research journal 2017 Issue 49.
7. Narendra Kumar Maddur[1], Surendar Reddy K[1], Sowmya J[2],Respiratory effects of air pollutants among non-smoking traffic policemen: An analytical cross sectional study. IAIM, 2016; 3(5): 8-13
8. Pravati Pal, John Robert., Dutta T K Pal G K. Pulmonary Function Test in Traffic Personnel in Pondicherry, Indian J Physiol Pharmacol 2010; 54 (4) : 329–336
9. India State-Level Disease Burden Initiative Air Pollution Collaborators, The impact of air pollution on deaths, disease burden, and life expectancy across the states of India: the Global Burden of Disease Study 2017 The lancet VOLUME 3, ISSUE 1, PE26-E39, JANUARY 01, 2019
10. Comroe JH Jr, Nadel JA: Current concepts: screening tests of pulmo- nary function. *N Engl J Med* 282:1249–1253, 1970.
11. Curtis L, Rea W, Smith-Willis P, Fenyves E, Pan Y. Adverse health effects of outdoor air pollutants. Environment international. 2006 Aug 1;32(6):815-30.
12. Brunekreef B. Air pollution and life-expectancy, is there a relation? Occup Environ Med 1997;54:781–4.
13. Godish T. Air quality. Fourth edition. Boca Raton, Florida: Lewis Books; 2003.
14. Akimoto H. Global air quality and pollution. Science 2003;302(5651):1716–9.
15. Hu SW, Shy CM. Health effects of waste incineration: a review of epidemiologic studies. J Air Waste Manage Assoc 2001;51:1100–9.
16. Shinn EA, Griffin DW, Seba DB. Atmospheric transport of mold spores in clouds of desert dust. Arch Environ Health 2003;58:498–504.
17. Cohen AJ, Ross Alexander H, Ostro B, Pandey KD, Kryzanowski M, Kunzai N, et al. The global burden of disease due to outdoor air pollution. J Toxicol Environ Health A 2005;68:1–7.
18. Dutta T, Pal G. Pulmonary function test in traffic police personnel in Pondicherry. Indian J Physiol Pharmacol. 2010;54(4):329–36.
19. Paul V, Mascarenhas DG, Khilar S, Fernandes G. Impact of Air Pollution on the Lung Function of Traffic Policemen in Mangalore.
20. Santos UP, Garcia MLSB, Braga ALF, Pereira LAA, Lin CA, de André PA, et al. Association between traffic air pollution and reduced forced vital capacity: a study using personal monitors for outdoor workers. PLoS One. 2016;11(10):e0163225.
21. Andersen ZJ, Bønnelykke K, Hvidberg M, Jensen SS, Ketzel M, Loft S, Sørensen M, Tjønneland A, Overvad K, Raaschou-Nielsen O. Long-term exposure to air pollution and asthma hospitalisations in older adults: a cohort study. Thorax. 2012 Jan 1;67(1):6-11.
22. Song Q, Christiani DC, XiaorongWang Ren J. The global contribution of outdoor air pollution to the incidence, prevalence, mortality and hospital admission for chronic obstructive pulmonary disease: a systematic review and meta-analysis. International journal of environmental research and public health. 2014; 11:11822–11832. [PubMed: 25405599]
23. Ye F, Piver WT, Ando M, Portier CJ. Effects of temperature and air pollutants on cardiovascular and respiratory diseases for males and females older than 65 years of age in Toyko, July and August 1980–1995. Environ Health Perspect 2001;109:355–9.
24. Vineis P, Foraster F, Hoek G, Lippsett M. Outdoor air pollution and lung cancer: recent epidemiological evidence. Int J Cancer 2004;111:647–52.
25. Bang KM. Chronic obstructive pulmonary disease in nonsmokers by occupation and exposure: a brief review. Curr Opin Pulm Med. 2015; 21:149–154. [PubMed: 25590955]
26. Chen G, Wan X, Yang G, Zou X. Traffic-related air pollution and lung cancer: A meta-analysis. Thorac Cancer. 2015; 6:307–318. [PubMed: 26273377]
27. Surawski NC, Miljevic B, Ayoko GA, Elbagir S, Stevanovic S, Fairfull-Smith KE, et al. Physicochemical Characterization of Particulate Emissions from a Compression Ignition Engine: The Influence of Biodiesel Feedstock. Environ Sci Technol. 2011 Dec 15;45(24):10337–43.
28. Nel A, Xia T, Mädler L, Li N. Toxic Potential of Materials at the Nanolevel. Science. 2006 Feb 3;311(5761):622–7.
29. Jalava PI, Salonen RO, Pennanen AS, Sillanpää M, Hälinen AI, Happo MS, Hillamo R, Brunekreef B, Katsouyanni K, Sunyer J, Hirvonen MRInhal Toxicol. 2007 Mar; 19(3):213-25.
30. Nadeem A, Masood A, Siddiqui N. Review: Oxidant—antioxidant imbalance in asthma: scientific evidence, epidemiological data and possible therapeutic options. Ther Adv Respir Dis. 2008 Aug 1;2(4):215–35.
31. Tredaniel J, Boffetta P, Saracci R, Hirsch A. Exposure to environmental tobacco smoke and adult non-neoplastic respiratory diseases. European Respiratory Journal. 1994 Jan 1;7(1):173–85.
32. Calhoun WJ, Reed HE, Moest DR, Stevens CA. Enhanced Superoxide Production by Alveolar Macrophages and Air-Space Cells, Airway Inflammation, and Alveolar Macrophage Density Changes after Segmental Antigen Bronchoprovocation in Allergic Subjects. Am Rev Respir Dis. 1992 Feb 1;145(2_pt_1):317–25.
33. Kang C-M, Jang A-S, Ahn M-H, et al. Interleukin-25 and Interleukin-13 Production by Alveolar Macrophages in Response to Particles. Am J Respir Cell Mol Biol. 2005 Sep 1;33(3):290–6.
34. Kim B-G, Lee P-H, Lee S-H, et al. Long-Term Effects of Diesel Exhaust Particles on Airway Inflammation and Remodeling in a Mouse Model. Allergy Asthma Immunol Res. 2015 Nov 16;8(3):246–56.
35. Hamilton RF, Holian A, Morandi MT. A Comparison of Asbestos and Urban Particulate Matter in the in Vitro Modification of Human Alveolar Macrophage Antigen-Presenting Cell Function. Experimental Lung Research. 2004 Jan 1;30(2):147–62.
36. Sun Z, An X, Tao Y, Hou Q. Assessment of population exposure to PM10 for respiratory disease in Lanzhou (China) and its health-related economic costs based on GIS. *BMC Public Health* 2013; **13:** 891.
37. Pasqua LA, Damasceno MV, Cruz R, Matsuda M, Garcia Martins M, Lima-Silva AE, et al. Exercising in Air Pollution: The Cleanest versus Dirtiest Cities Challenge. International Journal of Environmental Research and Public Health. 2018 Jul; 15(7): 1502.
38. Royal College of Physicians of London. Every breath we take: the lifelong impact of air pollution. Report of a working party. London: RCP, 2016.
39. Watts N, Adger WN, Agnolucci P, et al. Health and climate change: policy responses to protect public health. *Lancet* 2015; **386:** 1861–914.
40. Zhou W, Yuan D, Ye S, et al. Health eff ects of occupational exposures to vehicle emissions in Shanghai. Int J Occup Environ Health 2001; 7: 23–30.
41. Shan J. Smoking rate holds steady despite reduction eff orts. Dec 29, 2015. http://usa.chinadaily.com.cn/epaper/2015-12/29/ content_22855113.htm (accessed Sept 23, 2016).
42. Maestas MM, Brook RD, Ziemba RA, Li F, Crane RC, Klaver ZM, et al. Reduction of personal PM2.5 exposure via indoor air filtration systems in Detroit: an intervention study. J Expo Sci Environ Epidemiol. 2019 Jul;29(4):484–90.
43. Lee ES, Zhu Y. Application of a High-Efficiency Cabin Air Filter for Simultaneous Mitigation of Ultrafine Particle and Carbon Dioxide Exposures Inside Passenger Vehicles. Environ Sci Technol. 2014 Feb 18;48(4):2328–35.
44. Yu N, Shu S, Lin Y, She J, Ip HSS, Qiu X, et al. High efficiency cabin air filter in vehicles reduces drivers' roadway particulate matter exposures and associated lipid peroxidation. PLOS ONE. 2017 Nov 27;12(11):e0188498.
45. Shakya KM, Noyes A, Kallin R, et al. Evaluating the efficacy of cloth facemasks in reducing particulate matter exposure. J Expo Sci Environ Epidemiol 2017;27:352–7.
46. Morishita M, Wang L, Speth K, Zhou N, Bard RL, Li F, et al. Acute Blood Pressure and Cardiovascular Effects of Near-Roadway Exposures With and Without N95 Respirators. American Journal of Hypertension. 2019 Oct 16;32(11):1054–65.
47. Patel D, Shibata T, Wilson J, Maidin A. Challenges in evaluating PM concentration levels, commuting exposure, and mask efficacy in reducing PM exposure in growing, urban communities in a developing country. Science of The Total Environment. 2016 Feb 1;543:416–24.
48. Chen R, Zhao A, Chen H, et al. Cardiopulmonary benefits of reducing indoor particulates of outdoor origin: a randomized, double-blind cross-over trial of air purifi ers. J Am Coll Cardiol 2015; 65: 2279–87.
49. Sienra-Monqe JJ, Ramirez-Aquilar M, Moreno-Macias H, et al. Antioxidant supplementation and nasal infl ammatory responses among young asthmatics exposed to high levels of ozone. Clin Exp Immunol 2004; 138: 317–22.
50. Carlsten C, MacNutt MJ, Zhang Z, et al. Anti-oxidant N-acetylcysteine diminishes diesel exhaust-induced increased airway responsiveness in person with airway hyper-reactivity. Toxicol Sci 2014; 139: 479–87.
51. Dong GH, Cao Y, Ding HL, et al. Eff ects of environmental tobacco smoke on respiratory health of boys and girls from kindergarten: results from 15 districts of northern China. Indoor Air 2007; 17: 475–83.