AIR POLLUTION AND RESPIRATORY HEALTH: AN EVOLVING EPIDEMIC

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ABSTRACT

The spectrum of adverse effects of air pollution is wide and includes the consequences of acute and chronic exposure, which may lead to health care problems. Physicians are more likely to be concerned with the less common and more acute clinical consequences of acute pollution exposure, such as emergency hospital admission due to respiratory problems. Air pollution is the preamble of chemical, biological and particulate materials that can cause harm to human health and damage the natural environment. Air pollution can originate outdoors as well as indoors and become a major health concern. There have been significant advances in the knowledge regarding the effects of air pollutants on human health in the past few years. The general public especially patients with upper and lower respiratory symptoms are aware of the adverse respiratory effects due to air pollution. Long-term exposure to air pollutants increases the risk of respiratory illnesses such as allergies, asthma, chronic obstructive pulmonary disease, impaired lung function and lung cancer. The aim of this review is to highlight the adverse effects of air pollution due to dust storms on human health and provide the clinicians with appropriate context for monitoring and assessing the risk of air pollution.

KEY WORDS: Air pollution, Sandstorm, Dust storm, Respiratory problems.

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INTRODUCTION

Desert winds and storms aerosolize several billion tons of soil-derived dust each year.[1] Large dust storm events are capable of trans-continental, transoceanic and global dispersion.[2] The Sahara-Sahel region of Africa is the largest source of aerosolized soil dust on earth, contributing as much as one billion metric tons of dust per year to the global atmosphere. Research on the content and biological particles traveling between continents, with the dust, has only been recently initiated. These studies have provided valuable information because of concerns about adverse health effects of allergens carried in the dust and the possible transport of pathogens.[3-5]

Patho-physiology: Air pollutants are derived from a variety of sources, of which the combustion of fossil-fuel products is the principal source. Air pollutants are usually classified on the basis of their sources, which include chemical composition, size and mode of release into indoor or outdoor environments.[6] The primary pollutants are directly emitted into the atmosphere, whereas, the secondary pollutants are the result of chemical reactions with other pollutants or atmospheric gases.

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The diseases of the respiratory system induced by air pollutants are influenced by the type of dust, duration of exposure, concentration and size of airborne dusts in the breathing zone[7]. The deposition of inhaled particles and gases are influenced by the physical and chemical properties of the inhaled agent, and also by various host factors. The physical properties of importance, include particle size and density, shape and penetrability, surface area, electrostatic charge, and hygroscopicity. Among the more important chemical properties influencing the respiratory tract's response is the acidity or alkalinity of the inhaled agent. The air pollutant particles with an aerodynamic diameter in excess of 10µm are deposited on the mucous membrane in the nose and pharynx. Because of their momentum, they do not follow the air stream as it curves downward into the lungs and they impact on or near the tonsils and adenoids in the back of pharynx. Particles between 3-10µm in diameter can be deposited throughout the tracheobronchial tree, where they initiate reflex bronchial constriction and coughing. Particles between 0.1-3µm in diameter are mostly deposited within the alveoli. However, the particles smaller than 0.1µm remain in the air stream and are exhaled. [8]
The defensive mechanisms of the lungs are grouped in three categories (i) surface fluids, including mucous, surfactant, and other materials contained in the surface lining layer of the lung (ii) epithelial resources including cilia, goblet cells, mucous and serous glands with type I and II epithelial cells of the alveolar lining and alveolar macrophages and (iii) immunocytologic reserves of both cellular and protein nature, including the leukocytes of blood and tissue and various immunoglobulin proteins IgA, IgE, IgG and IgM. These defensive mechanisms do not work in isolation or as separate mechanisms but are closely interrelated and integrated in their defensive function or near the surface of the respiratory membrane.[9] The numerous mechanisms operate to prevent the foreign particles from reaching the alveoli, these include hairs in the nostrils, which strain out particles larger or equal to 10mm in diameter. The entire surface of the respiratory passages, both in the nose and in the lower passages, down as far as the terminal bronchioles, are lined with ciliated epithelia with about 200 cilia on each epithelial cell. These cilia are capable of moving the particles at a rate of 5-20 mm/min in the trachea and main bronchi. However, the clearance rate is slower in bronchioles, which is 0.5-1 mm/min. The cilia in the lower respiratory passages beat upward while those in the nose beat downward. This continual beating causes the coat of mucous to move slowly at a velocity of about 10 mm/min toward the pharynx, from where the mucous and its entrapped particles are either swallowed or coughed to the exterior.[10,11] Disruption in one of these systems can easily result in the respiratory health problems.

Dust associated microbes: Intercontinental dust events can facilitate long-distance dispersal (LDD) of dust-associated biological particles. It has been demonstrated that microbes survive in dust storms because many genera of bacteria (e.g. Bacillus) and most fungi can form spores, a dormant state that is resistant to desiccation, heat, radiation and nutrient-poor conditions. Many of the bacteria that are isolated from dust related aerosol samples are highly pigmented, suggesting that pigmentation also helps shield the microbes from ultra-violet radiation,[12] in addition to the protection afforded by clouds, fog, smoke and desert dust particles. After variable times and distances traveled, microbes, pollens and mineral dust particles eventually fall to the ground by gravity or precipitation.[13]
In a survey of airborne fungi in the eastern and western deserts of Egypt, 44 genera and 102 species (*Aspergillus was the dominant genus*) were recovered, with the areas of highest fungal concentrations and diversity corresponding to increases in vegetative cover and anthropogenic activity.[14] The most prevalent fungal genera in airborne dust samples collected from the atmosphere of Taif, Saudi Arabia (31 genera and 70 species) were *Aspergillus, Drechslera, Fusarium, Mucor, Penicillium, Phoma,* and *Stachybotrys*, and it was noted that genus prevalence was dependent on the nutrient medium used.[15] It has also

been reported that desert environments harbor diverse mycological communities, fungi such as *Coccidiodes immitis* and *Coccidiodes posadasii* are known to be restricted geographically.[16]

Abdul Hafez et al.,[15] and Kwaasi et al.,[17] reported that many genera of bacteria and fungi found in dust storm samples in Saudi Arabia were identified by using microscope method, such as *Bacillus, Pseudomonas, Staphylococcus* (bacterial genera), and *Alternaria, Aspergillus, Botrytis, Cladosporium, Mortierella, Mucor*, *Penicillium, Pythium Ulocladium, Verticillium* (fungal genera).

Moreover, desert dust research in Kuwait, which focused on "military personnel health protection," identified 147 bacteria, including representatives from 10 genera. Studies by chromatographic analysis of fatty acid methyl esters and 16S rRNA gene sequencing in settled dust identified *Arthrobacter, Bacillus, Cryptococcus, Flavimonas, Kurthia, Neisseria, Paenibacillus, Pseudomonas, Ralstonia, Staphylococcus* (as bacterial genera) while studies using 26S rRNA gene sequencing method identified *Alternaria, Cryptococcus, Mortierella*, *Penicillium. Phoma, Rhodotorula, Stemphylium* (as fungal genera).[18]

Dust associated bacteria: Studies on African dust identified bacterial isolates which were mostly Gram positive microbes and spore formers.[19] These are the types of micro-organisms that are most likely to survive trans-oceanic transport in a dust event. Bacillus & microbacterium [20,21] were the numerically dominant genera of bacteria isolated. Many dust-related studies use culture-based analyses to isolate micro-organisms. Culture methods are relatively easy and inexpensive. They also showed that microbes were viable even after being transported several thousand kilometers (e.g. the epidemic of meningitis, which spread from the African belt to Scandinavian countries including Sudan to Sweden) and are capable of causing an infection.[22]

Dust-associated fungi: The dust-associated studies on fungi show that, the composition of the aerosolized microbial communities vary dramatically during dust events and consist of a greater number of taxa than do the background samples. In several studies *Cladosporium* was the numerically dominant fungal germ detected during dust events, however this could be because Cladosporium is both ubiquitous and commonly found in aerosol samples.[18] *Aspergillus* species were also found associated with dust events. [19] Although this genus is also frequently observed in aerosol samples, the species are of interest due to their roles as allergens.

Dust-associated viruses: A study conducted by Griffin et al,[21] in the Caribbean atmosphere, mentioned virus-like particles[15] as being associated with transoceanic African desert dust. In his report, a total bacterial count was almost identical to the total viral count. It may be that the higher UV radiation and dry air associated with long-distance transport in dust events is unfavorable for survival of viral particles.

Dust-associated pollen: Pollens have been shown to be dispensed by dust storm events, epidemiologic studies investigating the role of allergen-pollutant combinations as triggers of asthmatic attacks have revealed that ozone, NO_2, and aeroallergens were independently or interactively related with asthma symptoms and changes in peak expiratory flow rate. It has been shown that ozone and fungal spores were co-factors associated with increased asthma symptoms and inhaler use.[22,23]

Respiratory symptoms and lung functions: The air pollution has been associated with numerous adverse respiratory outcomes. There are acute as well as chronic effects causing cough, bronchitis, respiratory illness and exacerbations of asthma.[24,25] Moreover, strongest evidences are available for adverse longterm effect of air pollution on lung function.[26] The exact mechanisms by which air pollutants cause adverse health effects are complex and not properly understood. A number of possibilities have been suggested. One hypothesized general pathway includes pollution induced lung damage including oxidative lung damage and inflammation leading to decline in lung function, respiratory distress, and cardiovascular disease related to hypoxia. [27] Evidence of pollution-related inflammation has been observed.[28] It has also been suggested that the autonomic nervous system may play an important role in the pathophysiologic pathways between particulate exposure and cardio pulmonary disease. Seaton, et. al,[29] hypothesized that fine particulate air pollution may provoke alveolar inflammation, resulting in the release of cytokines and increased blood coagulatibility. Autonomic nervous system-activated changes in blood viscosity and heart rate variability may increase the likelihood of cardiac death.[30] In general, it is speculated that interactions between inflammation, abnormal haemostatic function, and altered cardiac rhythm may play an important role in the pathogenesis of cardiopulmonary disease related to air pollution.

Children's health and air pollution: There is a strong evidence regarding susceptibility of children to health problems as a result of exposure to polluted air.[31,32] In general, children suffer from higher exposure to air pollutants than adults, because children spend more time outdoors than adults, their lungs are not well formed with immature immune system, higher physical activity, elevated metabolic rate and the resultant increase in minute ventilation.[33] Exposure to high particle conditions, such as dust storm may increase the risk of lung and vascular damage in children, because total particle deposition increases in proportion to minute ventilation and the deposition fraction nearly increases many times from the rest. Moreover, the developing lungs in children are particularly vulnerable to the adverse consequences of particle inhalation. The accumulated evidence indicates that children's health is adversely affected by a combination of air pollution and their susceptibility characteristics. The air pollutants cause wide range of acute and chronic effects, either as a single risk factor or, more often, in combination with other external agents. Air pollutants also interact with other environmental exposures, such as allergens, viruses and diet resulting in an overall adverse impact on children's health.

Hospitalizations: Many studies have demonstrated that the emergency department visits of patients with asthma, chronic obstructive pulmonary disease and other respiratory ailments tend to increase during episodes of particulate air pollution.[34,35] Moreover, the relationship between increased air pollution and hospitalization for cardiovascular diseases has also been demonstrated.[36]

CONCLUSIONS

Dust storm events occur frequently in the Kingdom of Saudi Arabia, especially in the Riyadh region. These storms have direct impact on the adult and children's health. Massive dust events create an atmospheric bridge over land and sea and aid in long distant dispersal. This is of paramount importance, because of concerns about the adverse health effects of allergens carried in the dust and the possible transport of pathogens. The air pollution caused by dust storm has an ill effect on health, especially on the respiratory system of adults and children leading to increased mortality. Further clinical studies are needed to investigate the effects of dust storms on the health of individuals in Saudi Arabia and other dust storms prone regions.

It is suggested that, the environmental protection agencies must involve the print and electronic media to provide appropriate information about the exact date and time of the dust storm to the public, especially in the dust storm regions. These agencies should also publicize the need for preventive measures, such as wearing of appropriate face masks and eye goggles during the dust storm events.

REFERENCES

1. Moulin, C, Lambert CE, Dulac F, Dayan U. Control of atmospheric export of dust from North Africa by the North Atlantic Oscillation, Nature 1997; 387:691-694.
2. Qian WL, Shi QS. Variations of the dust storm in China and its climatic control. J Climate 2002;15:1216-1229.
3. Hsiao-Man H, Carol Y. Rao, Hsiao-HsienYueh-Hsiu Chiu, Chi-Ming Liu, Chao HJ. Characteristics and determinants of ambient fungal spores in Hualien, Taiwan. Atmos Environ. 2005; 39:5839-5850.
4. Wu PC, Tsai JC, Li FC. Lung S-C., Increased levels of ambient fungal spores in Taiwan are associated with dust events from China. Atmos Environ 2004;38:4879-4886.

5. Shinn EA, GW Smith JM. Prospero, P Betzer ML, Hayes V. Garrison and RT Barber. 2000. African dust and the demise of Caribbean coral reefs. Geophysical Research Letters 27:3029-3032.
6. Bernstenin JA, Alexis N, Barnes C, Berstein IL. Health effects of air pollution. J Allergy Clin Immunol, 2004;114(5):1116-1123.
7. Mengesha YA, Bekele A. Relative chronic effects of occupational dusts on respiratory indices and health of workers in three Ethiopian factories. Am J Ind Med 1998;34:373-380.
8. Sheppard D, Hughson WG, Shellito J. Occupational lung diseases. In: Occupational Medicine 1990, edited by Joseph La Dou, Appleton and Lange, USA, pp. 221-236.
9. Green GMJ. Burns Amberson lecture. In defense of lung. Am Rev Rep Dis 1990;102:691-703.
10. Murray JF. The normal lung. Second edition. W.B. Saunders Philadelphia, 1986; pp 314- 322.
11. Xiao GG, Wang M, Lio N, Loo JA, Nel AE. Use of proteomics to demonstrate a hierarchical oxidative stress response to diesel exhausts particles in a macrophage cell line. J Biol Chem 2003; 278:50781-90.
12. Kellogg CA. Griffin DW, Garrison VH, Peak KK. Characterization of aerosolized bacteria and fungi from desert dust events in Mali, West Africa. Aerobiologia 2004;20:99-110.
13. Lighthart B. The ecology of bacteria in the alfresco atmosphere. FEMS Microbial Ecol 1997; 23:263-274.
14. Ismail MA, Abdel-Hafez SII, Moharram . Aeromycobiota of western desert of Egypt. Afr J Sci Technol 2002; 3:1-9.
15. Abdel –Hafez SII, Shoreit AAM. Mycotoxins producing fungi and mycoflora of air-dust from Taif, Saudi Arabia. Mycopathologia 1985; 92:65-71.
16. Hector RF, Laniado-Laborin R. Coccidioidomycosis a fungal disease of the Americas. PLoS Med 2005; 2:(1)e2.
17. Kwaasi AA, Parhar RS, Al-Mohanna FA, Harfi HA, X Collison KS, Al-Sedairy ST. Aeroallergens and viable microbes in sandstorm dust. Potential triggers of allergic and non-allergic respiratory ailments. Allergy 1998;53:255-265.
18. Lyles MB, Fredrickson HL, Bednar AJ, Fannin HB, Sobecki TM. The chemical, biological, and mechanical characterization of airborne micro-particulates from Kuwait. Abstr. 8th Ann. Force Health Protect. Conf., session 2586, Louisville, KY. 2005.
19. Prospero, J. M., Blades E, Mathison G, Naidu R. Interhemispheric transport of viable fungi and bacteria from Africa to the Caribbean with soil dust. Aerobiologia 2005; 21:1-19.
20. Griffin DW, Kellogg CA, Garrison VH, Lisle JT, Borden TC, Shinn EA. Atmospheric microbiology in the northern Caribbean during African dust events. Aerobiologia 2003; 19:143-157.
21. Griffin DW., Garrison VH., Herman JR., Shinn EA. African desert dust in the Caribbean atmosphere: microbiology and public health. Aerobiologia 2001;17:203-213.
22. Papke RT, Ward DM. The importance of physical isolation to microbial diversification. FEMS Microbiol Ecol 2004; 48:293-303.
23. Delfino RJ, Zeiger RS, Seltzer JM, Street DH, Matteucci RM, Anderson PR, et al. The effect of outdoor fungal spore concentrations on daily asthma severity. Environ Health Perspect 1997; 105:622-35.
24. Boezen M, Schouten J, Rijcken B, Vonk J, Gerritsen J, Van Der Zee S, et al., Peak expiratory flow variability, bronchial responsiveness, and susceptibility to ambient air pollution in adults. Am J Respir Crit Care Med. 1998; 158: 1848-1854.
25. Braun-Fahlander C, Ackermann-Liebrich U. Schwartz J, Gnehm HP, Rutishauser M, Wanner HU. Air pollution and respiratory symptoms in preschool children. Am Rev Respir Dis 1992; 145:42-47.
26. Götschi T, Heinrich J, Sunyer J, Künzli N .Long-term effects of ambient air pollution on lung function: a review.Epidemiology. 2008 Sep;19(5):690-701.
27. Tan WC, Qui D, Liam BL, Ng TP, Lee SH, Van Eeden S. The human bone marrow response to acute air pollution caused by forest fires. Am J Respi Crit Care Med 2000;161(4 pt 1):1213-1217.
28. Salvi S. Blomberg A, Rudell B, Kelly F, Sandstrom T, Holgate ST. 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 1999; 159:702-709.
29. Seaton A, MacNee W, Donaldson K, Godden D. Particulate air pollution and acute health effects. Lancet 1995; 345:176-178.
30. Schwartz J, Air pollution and children's health, Pediatrics 2004; 113: 1037-1043.
31. Moshammer H, Bartonova A, Hanke W, van den Hazel P, Kopke JG and Krämer U. Air pollution: a threat to the health of our children, Acta Paediatr 2006; 95; 453: 93-105.
32. Bennett WD and Zeman KL, Deposition of fine particles in children spontaneously breathing at rest, Inhal Toxicol 1998; 10: 831-842.
33. Chalupa C, Morrow PE, Oberdorster G, Utell MJ and Frampton MW. Ultrafine particle deposition in subjects with asthma, Environ Health Perspect 2004; 112 (8): 879-882.
34. Anderson HR, Spix C, Medina S. Schouten JP, Castellsague J, Rossi G, et al. Air pollution and daily admission for chronic obstructive pulmonary disease in 6 European cities: results from the APHEA project. Eur Respire J 1997; 10:1064-1071.
35. Atkinson RW, Anderson HR, Strachan DP, Bland JM, Bremner SA, Ponce de Leon A. Short-term associations between outdoor air pollution and visits to accident and emergency departments in London for respiratory complaints. Eur Respir J 1999;13:257-265.
36. Burnett RT, Smith-Doiron M, Stieb D, Cakmak S, Brook JR. Effects of particulate and gaseous air pollution on cardio respiratory hospitalizations. Arch Environ Health. 1999; 54:130-139.