Review Article on Pollutional Haze

Approaches to prevent the patients with chronic airway diseases from exacerbation in the haze weather

Jin Ren[1], Bo Li[1,2], Dan Yu[3], Jing Liu[1], Zhongsen Ma[1]

[1]Department of Respiratory Medicine, the Second Hospital, Jilin University, Changchun 130041, China; [2]Department of Occupational Disease Prevention, Jilin Provincial Occupational Disease Prevention and Treatment, Changchun 130000, China; [3]Department of Otolaryngology Head and Neck Surgery, the Second Hospital, Jilin University, Changchun 130041, China

Contributions: (I) Conception and design: J Ren; (II) Administrative support: Z Ma; (III) Provision of study materials or patients: D Yu; (IV) Collection and assembly of data: J Liu; (V) Data analysis and interpretation: B Li; (VI) Manuscript writing: All authors; (VII) Final approval of manuscript: All authors.

Correspondence to: Zhongsen Ma. Department of Respiratory Medicine, the Second Hospital, Jilin University, 218 Ziqiang Street, Changchun 130041, China. Email: mazhongsen2005@163.com.

Abstract: Haze weather is becoming one of the biggest problems in many big cities in China. It triggers both public anxiety and official concerns. Particulate matter (PM) plays the most important role in causing the adverse health effects. Chemical composition of PM2.5 includes primary particles and secondary particles. The toxicological mechanisms of PM2.5 to the human body include the oxidative stress, inflammation and carcinogenesis. Short or long-term exposure to PM (especially PM2.5) can cause a series of symptoms including respiratory symptoms such as cough, wheezing and dyspnea as well as other symptoms. There are positive associations between PM2.5 and mortality due to a number of causes. PM2.5 is considered to contribute to the onset of asthma, the exacerbation of chronic obstructive pulmonary disease (COPD) in haze weather. Some approaches including outdoor health care, indoor health care and preventive medications can prevent the patients with chronic airway diseases from exacerbations.

Keywords: Haze weather; PM2.5; toxicity; chronic airway diseases; preventive approach

Submitted May 29, 2014. Accepted for publication Oct 28, 2015.
doi: 10.3978/j.issn.2072-1439.2015.11.61
View this article at: http://dx.doi.org/10.3978/j.issn.2072-1439.2015.11.61

Introduction

In recent years, haze weather is becoming one of the biggest problems in many big cities in China. The haze triggers both public anxiety and official concerns. Haze is an atmospheric phenomenon that is characterized by visibility of less than 10 km due to complex material that is suspended in the air, such as dust, smoke and other fine particles. The haze not only can affect the air condition that obscuring the clarity of the sky, damage forests and crops, and contaminate lakes and rivers, but also can influence the human health. Among all these air pollutants in haze, particulate matter (PM) plays the most important role in causing the adverse health effects in human. According to the aerodynamic diameter of the particles, there are PM10, PM2.5–10, PM2.5, PM_1 and even the smaller ultrafine PM (UFPs) respectively. PM10 are the particles less than 10 μm in aerodynamic diameter; PM2.5, also known as fine particles are particles less than 2.5 μm in aerodynamic diameter; and PM10–2.5, also known as coarse particles are particles between 10 and 2.5 μm in aerodynamic diameter. diameter; the smaller ultrafine PM are less than 0.01 μm in aerodynamic diameter.

PM10 cannot be inhaled deep into the lungs, they will deposit primarily in the primary bronchi. PM2.5 and even smaller PM can penetrate into the lungs and embed themselves in the alveoli and the ultrafine PM even can be transmitted into the blood circulation. PM2.5 is also the carriers of some chemical elements and organisms. So it is generally accepted that PM2.5 is more harmful than PM10

for public health. The toxicological mechanisms of PM2.5 to the human body include the oxidative stress, inflammation, allergic reaction and carcinogenesis. These reactions in the body can lead to acute diseases such as acute upper airway infection, acute bronchitis, and also trigger the exacerbation of chronic airway diseases such as chronic obstructive pulmonary disease (COPD) and asthma. The long-term exposure to PM2.5 can induce the coronary heart disease, dysfunction of immune system, the formation of thrombosis and even carcinoma. For the patients suffered from chronic airway diseases, the haze weather is a potential risk factor for the exacerbation, and the methods to prevent themselves from the exacerbation in the bad weather are very helpful.

Haze formation and chemical composition of PM2.5

Haze formation

Haze is an atmospheric phenomenon in which fine particles contribute to light extinction through scattering and absorption, which obscure the clarity of the sky (1). The sources of PM emissions are from both natural and man-made. Natural sources include wind-blown dust, sea salt, volcanic ash, pollens, fungal spores, soil particles, the products of forest fires and the oxidation of biogenic reactive gases. Man-made sources constitute fossil fuel combustion (especially in vehicles and power plants), industrial processes (producing metals, cement, lime and chemicals), construction work, quarrying and mining activities, cigarette smoking and wood stove burning. The road transport and the burning of fossil fuels in power stations and factories are main sources of PM in urban areas. Components of traffic-derived PM include engine emissions and wear, brake and tyre wear and dust from road surfaces (2). The largest single source of airborne PM from motor vehicles is derived from diesel exhaust. Because the number of new cars with diesel engines in industrialised countries increases rapidly, diesel exhaust particles (DEPs) account for most airborne particulate matter (up to 90%) in the world's largest cities (3,4).

Fine particles refer to PM2.5. Because fine particles are small and have low densities and high buoyancies, they survive for longer time in the atmosphere (5). The number concentration of fine particles per unit volume has a more direct impact on the formation of haze. Fine particles also act as carriers for heterogeneous chemical reactions (6).

Chemical composition of PM2.5

PM2.5 can be divided into primary particles and secondary particles. The primary particles are released directly from their source, primarily by combustion into the atmosphere. They are mainly derived from road transport, stationary combustion and industrial processes. Secondary particles are formed because of chemical reactions within the atmosphere. The chemical reactions produce substances of low volatility that consequently condense into solid or liquid phase, thereby becoming PM. Secondary particles include sulphur dioxide (SO_2) which are primarily derived from power generation and industrial combustion processes and nitrogen dioxide (NO_2) which are primarily derived from road transport and power generation. Carbonaceous particulates also contain secondary organic aerosol (SOA) formed from the oxidation of volatile organic compounds (VOCs). Metals carried by the fine particles such as iron (Fe), vanadium (V), nickel (Ni), chromium (Cr) and copper (Cu) are also the reasons for exerting adverse health effects. The chemical processes in the formation of secondary particles are relatively slow and their persistence in the atmosphere is prolonged (2).

Soluble secondary inorganic particles such as sulphates and nitrates are all acidic. They usually exist as pure aqueous or solid particles, or sometimes as a surface layer on other solid particles such as carbon or ash particles. One epidemiological study showed the sulphate particles from coal burning power stations increased the risk of mortality in Boston (7). Another study by Atkinson *et al.* reported a positive association between concentrations of secondary pollutants (particularly non-primary PM2.5, sulphate and nitrate) and increases in numbers of respiratory admissions in London (8).

Metals within the PM2.5 may most likely to exert adverse health effects. They are produced by metallurgical processes, from impurities in fuel additives and in non-exhaust emissions such as mechanical abrasion. Some metals are transition metals such as Fe, Ni, V, Cu and Cr, when they are inhaled and absorbed by the tissues, reactive oxygen species (ROS) can be produced and do harm to the tissues.

Elemental carbon (EC) and organic carbon (OC) are also very important elements of ambient PM2.5. In many areas, these ambient PM containing significant amounts (up to 80-90% of UFP mass) of EC and OC are derived from combustion processes (9,10). Some biological sources such as viruses, pollen grains, plant debris also contribute to the

production of carbonaceous aerosol. The carbonaceous aerosol contains SOA formed from the oxidation of biogenic and anthropogenic hydrocarbon emissions. More than 200 organic species have been identified, including alkanes, alkenes, aromatics, oxygenated compounds (including aldehydes, ketones and carboxylic acids), amino compounds, nitrates, polyaromatic hydrocarbons (PAH) and PAH derivatives (11). Since specific OC components may undergo chemical modifications and their ability to induce biological effects could be altered, identifying the potential toxicity of them is further complicated (12).

Toxicology of PM2.5

PM2.5 exhibits a special structure and has very large surface areas which can carry and absorb some toxic and harmful materials. Airborne PM2.5 is linked to adverse health effects of the respiratory system. PM2.5 can penetrate into the respiratory airways and deposited in the bronchioles and the alveoli.

The most important pathogenic mechanism of PM2.5 to the respiratory system is oxidative stress. The components of PM2.5 especially the transition metals can generate ROS by a variety of reactions and induce lung injury. These redox-active metals can contain unpaired electrons in their d-orbital, and are capable of generating free radical species via redox cycling mechanisms with biological reductants. Despite transition metals, there are also non-redox active metals such as aluminium (Al), zinc (Zn) and Pb are contained in the particulate air pollution, these metals can influence the toxic effects of transition metals, either exacerbating or lessening the production of free radicals. A study reported that personal exposure of healthy human subjects to water-soluble V and Cr, but not Fe, Ni, Cu or Pt, in PM2.5 were associated with significant increases in oxidative stress and DNA damage as measured in blood (13). Cakmak *et al.* used generalized linear mixed models; daily changes in ambient PM2.5-associated metals were compared to daily changes in physiologic measures in 59 healthy subjects who spent 5-day near a steel plant and 5-day on a college campus. The results showed that interquartile increases in calcium, cadmium, lead, strontium, tin, V and Zn were associated with statistically significant increases in heart rate of 1-3 beats per minute, increases of 1-3 mmHg in blood pressure and/or lung function decreases of up to 4% for total lung capacity. Metals contained in PM2.5 were found to be associated with acute changes in cardiovascular and respiratory physiology (14).

Studies showed that in healthy volunteers who inhaled concentrated ambient particles (CAP) there were neutrophilic inflammation in the lungs, and increased blood fibrinogen levels associated with Fe/Se/sulphate and Cu/Zn/V factors respectively (15,16).

The instillation studies on humans using PM filter extracts sampled in Utah Valley when the steel mill was working, induced pulmonary injury and inflammation, and particle analysis indicated that a higher metal content (Fe, Cu, Ni, Pd and Zn among others) in the extracts from the active periods of the steel mill might contribute to the higher biological activity (17).

In addition, other air pollutants such as ozone play a role in synergistic redox effects and contribute to oxidative stress in the respiratory system. Studies showed that bronchiolar epithelium is highly susceptible to injury and oxidative stress induced by acute exposure to ozone (18). PM2.5 can also stimulate and interact with the target cells such as lung epithelial cells and pulmonary macrophages to generate ROS.

ROS which produced by airborne PM are implicated in the activation of mitogen-activated protein kinase (MAPK) family members and activation of transcription factors such as NF-κB and AP-1. These signaling pathways contribute to processes of inflammation, apoptosis, proliferation, transformation and differentiation (19). In addition, studies showed that stable free radicals in the PM especially hydroxyl radicals play an important role in increasing ROS production (20,21).

ROS can cause damage to cellular macromolecules such as nucleic acids, lipids and proteins. In a chronic inflammatory process, persistent ROS production can induce considerable tissue damage. Thus, people suffering from chronic pulmonary inflammation may be susceptible for adverse health effects of PM inhalation (22). Several *in vitro* studies have demonstrated that PM induces DNA damage in the form of strand breaks and 8-hydroxy-2'-deoxyguanosine (8-OHdG) formation in lung epithelial cells (23,24).

The EC and OC also can elicit pulmonary injuries. Take diesel for example, many studies showed exposures to diesel exhaust in animal models caused respiratory effects including acute inflammatory responses, allergic processes and enhanced susceptibility to pulmonary infections (4,25).

Large numbers of studies showed that ambient PM can generate ROS in pulmonary cells such as epithelium and macrophages and this is considered the most important mechanism for carcinogenesis (26).

Epidemiological studies

Symptoms and mortality related to ambient air pollution

Short or long-term exposure to PM (especially PM2.5 or UFPs) can cause a series of symptoms including respiratory symptoms such as cough, wheezing and dyspnea as well as other symptoms like headache, dizziness, chest pain, mental and physical slowing (27,28). Long *et al.* studied the adverse health effects of PM on the population of Winnipeg, Canada and found that 42% of the population reported that symptoms (cough, wheezing, chest tightness, shortness of breath) developed or became worse due to the air pollution episode and 20% reported that they had breathing trouble (29). Ho *et al.* reported that during the South Asian haze crisis in 2013, the five most common physical symptoms include mouth or throat discomfort (68.8%), nose discomfort (64.1%), eye discomfort (60.7%), headache (50.3%) and breathing difficulty (40.3%) (30).

There are positive associations between PM2.5 and mortality due to a number of causes. A study based on meta-analysis of 110 peer-reviewed time series studies indexed in medical databases to May 2011 reported that based upon 23 estimates for all-cause mortality, a 10 μg/m^3 increment in PM2.5 was associated with an increase in the risk of death. Associations for respiratory causes of death were larger than for cardiovascular causes. Positive associations with mortality for most other causes including ischaemic heart disease and stroke were also observed (31). Another study investigated the short-term effects of PM2.5 on deaths from diabetes, cardiac and cerebrovascular causes, lower respiratory tract infections (LRTI) and COPD in 10 European Mediterranean metropolitan areas participating in the MED-PARTICLES project during 2001-2010. The results showed positive associations between PM2.5 and mortality due to diabetes, cardiac causes, COPD, and to a lesser degree to cerebrovascular causes (32).

Relationship between chronic airway diseases and PM2.5

Chronic airway diseases include asthma, COPD. PM2.5 is considered to contribute to the onset of asthma, the exacerbation of COPD in haze weather. Epidemiological and clinical studies showed that people exposed to combined air pollutants, such as ozone and cigarette smoke or PM showed increased pulmonary oxidative stress and inflammation associated with an increase in pulmonary diseases and mortality (33).

Several studies investigated the relationship between PM2.5 and hospital admissions for COPD. Dadvand *et al.* found that exposure to ambient PM2.5 with NO_2 increases systemic inflammation in COPD patients, especially in former smokers in Barcelona, Spain, in 2004-2006 (34). Halonen *et al.* reported a 5.3% increase in the risk of COPD admission per 10 μg/m^3 increase in the level of PM2.5 (35). Dominici *et al.* reported a 1.61% (95% CI, 0.56-2.66%) increase in hospitalization for COPD per 10 μg/m^3 increase in PM2.5 level (36). A case-crossover study in Taipei by Tsai *et al.* reported a 6.87% and 1.72% increase in hospitalization for COPD per 10 μg/m^3 increments in the 3 day moving average concentrations of PM2.5 for warm days and cool days, respectively (37).

There are also a large number of studies showed the associations between PM2.5 and asthma morbidity.

A nationwide study in the United States reported that positive ozone–asthma associations were detected across all spatial settings, while positive PM2.5/asthma associations were detected only in central cities of a metropolitan statistical area (MSA) and in outer ring suburbs. They found two significant positive location-specific associations between PM2.5 and asthma risk: the higher the PM2.5 concentration, the greater the asthma risk in central cities of an MSA (OR: 1.050, 95% CI, 1.010-1.092) and in outer ring suburban counties (OR: 1.041, 95% CI, 1.002-1.082) (38). Evans *et al.* reported that interquartile range increases in ultrafine particles and carbon monoxide concentrations in the previous 7 days were associated with increases in the relative odds of a pediatric asthma visit, with the largest increases observed for 4-day mean ultrafine particles and 7-day mean carbon monoxide (39). A pilot observational study from the central valley of California by Vempilly *et al.* found wheezing and dyspnea in asthmatics were worsened with increasing ambient PM2.5. Increasing PM2.5 decreased FEV_1% predicted (–0.51, –0.79 to –0.23) and FEF 25-75% predicted (–0.66, –1.07 to –0.24) in subjects with asthma (all $P<0.01$). The reductions in FEV 25-75% and FEV_1 per 10 μg/m^3 increase in ambient PM2.5 were 6% and 5% respectively. Increasing ambient PM2.5 worsened airway function in asthma (40).

Prevention from exacerbation of chronic airway diseases in haze weather

Environmental epidemiology research has proved that short or long-term exposure to ambient PM2.5 was associated with increased mortality and morbidity, reduction in life expectancy and additional respiratory diseases. Therefore,

how to prevent the patients who suffer from COPD and asthma from exacerbation in haze weather should be an important issue.

Outdoor health care

The patients should stay indoors as much as possible during the haze weather to avoid the direct exposure to ambient air pollutants. If they must go out, there are some advices they should take. Avoid going out in the morning and evening when there are traffic peaks, because the gas and diesel exhausts from vehicles can contribute to the increasing the concentrations of PM2.5 in this two periods of time. Road travel should be minimized to the absolutely necessary. Choose taking cars, taxies or buses instead of walking and driving a bicycle. If the patients must drive cars or sit in the cars or taxies, keep all the windows closed and turn on the air-conditioner and keep it to the "recirculate inside air" mode.

Some patients wear masks when they go out in haze weather, but not all the masks are appropriate for protecting PM2.5. Wearing the wrong mask will lead to a false sense of security and encourage those using them to be out in the haze for extended periods. Using surgical masks is ineffective as it will not prevent the particles of 10 microns and less from entering our lungs. We need to wear appropriate mask such as N95 mask which can filter out 95% of particulate larger than 0.3 microns. N95 mask is a tighter fit than surgical masks, so it is not recommended to use it for extended periods in the patients who suffer from chronic airway diseases because it can make them feel hot and stuffy and deteriorate the shortness of breath.

Indoor health care

Even when you stay indoors, it is not mean that you are totally safe from haze, you should do more to reduce the possibilities of exposure to ambient air pollutants. All the people who live in the house should wash off dust and dirt as soon as they step indoors. While inside home, keep all doors and windows shut; turn on the air-conditioners (fans should be kept off as much as possible) and if your budget allows, consider fitting air-conditioners with high efficiency particulate (HEPA) filters. Brown *et al.* found that use of a minimum efficiency removal value (MERV) 12 or higher performing air filter in home ventilation systems can effectively reduce indoor levels of common asthma and allergy triggers including cat dander particles, fine PM (PM2.5) and respiratory virus (41). Air cleaners or purifiers can also be used to improve indoor air quality. Using a humidifier to dampen down the particulates is another feasible choice, and the additional moisture can also help reduce respiratory irritation. When cleaning the house, wet mop house and indoor areas instead of sweeping with a broom. Reduce the possibility of cooking with wood, gas or charcoal. Cooking with an electric stove, oven or microwave is recommended. If the patients smoke, quit smoking immediately. The patients should also avoid passive smoking in the room. Some botanists recommend placing some green plants such as scindapsus aureus, dracaena sanderiana, carota L in the room. The bigger blades can absorb more pollutants and make the air fresher. Do not place flowers in the room if the patient has asthma. Health experts recommend drinking two litres a day when the haze is bad. Drinking some green tea is also a good choice. The diet should be nutritious and balanced. Choose digestible food rich in vitamins and protein, eat fresh vegetables and fruits. Physical exercise in the room is not recommended.

Preventive medications

For asthma patients, antiallergic drugs such as astemizole and loratadine could be used to avoid allergic reactions caused by airborne allergens. Bronchodilators such as symbicort turbuhaler or seretide should be inhaled regularly. Take the SABA on hand when going outdoors. For COPD patients, oxygen therapy could be used at home. If the patients have wheezing and dyspnea, the bronchodilators should be used. Antibiotics could be used when there is evidence of infections in the lungs. If the patients have other chronic diseases like coronary heart disease, diabetes, appropriate medications should be taken to avoid deterioration of the diseases.

In addition, the patients could take some medications which can increase the immunologic function. The multivitamins medication could be taken. Vitamin C is an antioxidant which can reduce the oxidative stress reaction.

Conclusions

Haze weather is harmful for human health. The PM2.5 plays an important role in the adverse health effects. The patients with chronic airway diseases including asthma and COPD are prone to deteriorate in haze weather. Some approaches in daily life and preventive medications could reduce the possibilities of exacerbation of the diseases.

Acknowledgements

None.

Footnote

Conflicts of Interest: The authors have no conflicts of interest to declare.

References

1. Watson JG. Visibility: science and regulation. J Air Waste Manag Assoc 2002;52:628-713.
2. Kelly FJ, Fussell JC. Size, source and chemical composition as determinants of toxicity attributable to ambient particulate matter. Atmospheric Environment 2012;60:504-26.
3. Shah SD, Cocker DR 3rd, Miller JW, et al. Emission rates of particulate matter and elemental and organic carbon from in-use diesel engines. Environ Sci Technol 2004;38:2544-50.
4. Riedl M, Diaz-Sanchez D. Biology of diesel exhaust effects on respiratory function. J Allergy Clin Immunol 2005;115:221-8; quiz 229.
5. Haglera GSW, Bergina MH, Salmonc LG, et al. Source areas and chemical composition of fine particulate matter in the Pearl River Delta region of China. Atmospheric environment 2006;40:3802-15.
6. Pathak RK, Wu WS, Wang T. Summertime PM2.5 ionic species in four major cities of China: Nitrate formation in an ammonia-deficient atmosphere. Atmospheric Chemistry and Physics 2009;9:1711-22.
7. Maynard D, Coull BA, Gryparis A, et al. Mortality risk associated with short-term exposure to traffic particles and sulfates. Environ Health Perspect 2007;115:751-5.
8. Atkinson RW, Fuller GW, Anderson HR, et al. Urban ambient particle metrics and health: a time-series analysis. Epidemiology 2010;21:501-11.
9. Heintzenberg J. Fine particles in the global troposphere. Tellus(1989) 41B:149-60.
10. Chow JC, Watson JG. Survey of measurement and composition of ultrafine particles. Aerosol Air Qual Res 2007;7:121-73.
11. Seinfeld J, Pandis S. Atmospheric Chemistry and Physics: from Air Pollution to Climate Change. NewYork: Wiley-Interscience, 1998.
12. Vione D, Barra S, De Gennaro G, et al. Polycyclic aromatic hydrocarbons in the atmosphere: monitoring, sources, sinks and fate. II: Sinks and fate. Ann Chim 2004;94:257-68.
13. Sørensen M, Daneshvar B, Hansen M, et al. Personal PM2.5 exposure and markers of oxidative stress in blood. Environ Health Perspect 2003;111:161-6.
14. Cakmak S, Dales R, Kauri LM, et al. Metal composition of fine particulate air pollution and acute changes in cardiorespiratory physiology. Environ Pollut 2014;189:208-14.
15. Ghio AJ, Kim C, Devlin RB. Concentrated ambient air particles induce mild pulmonary inflammation in healthy human volunteers. Am J Respir Crit Care Med 2000;162:981-8.
16. Huang YC, Ghio AJ, Stonehuerner J, et al. The role of soluble components in ambient fine particles-induced changes in human lungs and blood. Inhal Toxicol 2003;15:327-42.
17. Ghio AJ. Biological effects of Utah Valley ambient air particles in humans: a review. J Aerosol Med 2004;17:157-64.
18. Evans MD, Dizdaroglu M, Cooke MS. Oxidative DNA damage and disease: induction, repair and significance. Mutat Res 2004;567:1-61.
19. Terzano C, Di Stefano F, Conti V, et al. Air pollution ultrafine particles: toxicity beyond the lung. Eur Rev Med Pharmacol Sci 2010;14:809-21.
20. Shi T, Knaapen AM, Begerow J, et al. Temporal variation of hydroxyl radical generation and 8-hydroxy-2'-deoxyguanosine formation by coarse and fine particulate matter. Occup Environ Med 2003;60:315-21.
21. Valavanidis A, Fiotakis K, Vlachogianni T. The role of stable free radicals, metals and pahs of airborne particulate matter in mechanisms of oxidative stress and carcinogenicity. In: Zereini F, Wiseman CLS, editors. Urban airborne particulate matter. Berlin: Springer Berlin Heidelberg, 2011:411-26.
22. Taniyama Y, Griendling KK. Reactive oxygen species in the vasculature: molecular and cellular mechanisms. Hypertension 2003;42:1075-81.
23. Storr SJ, Woolston CM, Zhang Y, et al. Redox environment, free radical, and oxidative DNA damage. Antioxid Redox Signal 2013;18:2399-408.
24. Valavanidis A, Vlachogianni T, Fiotakis C. 8-hydroxy-2' -deoxyguanosine (8-OHdG): A critical biomarker of oxidative stress and carcinogenesis. J Environ Sci Health C Environ Carcinog Ecotoxicol Rev 2009;27:120-39.
25. Gilmour MI, Jaakkola MS, London SJ, et al. How exposure to environmental tobacco smoke, outdoor air pollutants,

and increased pollen burdens influences the incidence of asthma. Environ Health Perspect 2006;114:627-33.
26. Van Berlo D, Hullmann M, Schins RPF. Toxicology of Ambient Particulate Matter. In: Luch A, editor. Experientia Supplementum Molecular, Clinical and Environmental Toxicology. Heidelberg: Springer Basel AG, 2012:165-218.
27. Auchincloss AH, Diez Roux AV, Dvonch JT, et al. Associations between recent exposure to ambient fine particulate matter and blood pressure in the Multi-ethnic Study of Atherosclerosis (MESA). Environ Health Perspect 2008;116:486-91.
28. Wellenius GA, Boyle LD, Wilker EH, et al. Ambient fine particulate matter alters cerebral hemodynamics in the elderly. Stroke 2013;44:1532-6.
29. Long W, Tate RB, Neuman M, et al. Respiratory symptoms in a susceptible population due to burning of agricultural residue. Chest 1998;113:351-7.
30. Ho RC, Zhang MW, Ho CS, et al. Impact of 2013 south Asian haze crisis: study of physical and psychological symptoms and perceived dangerousness of pollution level. BMC Psychiatry. 2014 Mar 19;14:81.
31. Atkinson RW, Kang S, Anderson HR, et al. Epidemiological time series studies of PM2.5 and daily mortality and hospital admissions: a systematic review and meta-analysis. Thorax 2014;69:660-5.
32. Samoli E, Stafoggia M, Rodopoulou S, et al. Which specific causes of death are associated with short term exposure to fine and coarse particles in Southern Europe? Results from the MED-PARTICLES project. Environ Int 2014;67:54-61.
33. Sunil VR, Patel-Vayas K, Shen J, et al. Classical and alternative macrophage activation in the lung following ozone-induced oxidative stress. Toxicol Appl Pharmacol 2012;263:195-202.
34. Dadvand P, Nieuwenhuijsen MJ, Agustí À, et al. Air pollution and biomarkers of systemic inflammation and tissue repair in COPD patients. Eur Respir J 2014;44:603-13.
35. Halonen JI, Lanki T, Yli-Tuomi T, et al. Urban air pollution, and asthma and COPD hospital emergency room visits. Thorax 2008;63:635-41.
36. Dominici F, Peng RD, Bell ML, et al. Fine particulate air pollution and hospital admission for cardiovascular and respiratory diseases. JAMA 2006;295:1127-34.
37. Tsai SS, Chang CC, Yang CY. Fine particulate air pollution and hospital admissions for chronic obstructive pulmonary disease: a case-crossover study in Taipei. Int J Environ Res Public Health 2013;10:6015-26.
38. Li T, Lin G. Examining the role of location-specific associations between ambient air pollutants and adult asthma in the United States. Health Place 2014;25:26-33.
39. Evans KA, Halterman JS, Hopke PK, et al. Increased ultrafine particles and carbon monoxide concentrations are associated with asthma exacerbation among urban children. Environ Res 2014;129:11-9.
40. Vempilly J, Abejie B, Diep V, et al. The synergetic effect of ambient PM2.5 exposure and rhinovirus infection in airway dysfunction in asthma: a pilot observational study from the Central Valley of California. Exp Lung Res 2013;39:434-40.
41. Brown KW, Minegishi T, Allen JG, et al. Reducing patients' exposures to asthma and allergy triggers in their homes: an evaluation of effectiveness of grades of forced air ventilation filters. J Asthma 2014;51:585-94.

Cite this article as: Ren J, Li B, Yu D, Liu J, Ma Z. Approaches to prevent the patients with chronic airway diseases from exacerbation in the haze weather. J Thorac Dis 2016;8(1):E1-E7. doi: 10.3978/j.issn.2072-1439.2015.11.61

