SCIENCE CHINA
Life Sciences

• NEWS AND VIEWS •

June 2015 Vol.58 No.6: 624–626
doi: 10.1007/s11427-015-4878-4

Health effects of fine particles ($PM_{2.5}$) in ambient air

HAN YiQun & ZHU Tong*

State Key Joint Laboratory for Environmental Simulation and Pollution Control, College of Environmental Sciences and Engineering, Center for Environment and Health, Peking University, Beijing 100871, China

Received April 18, 2015; accepted April 28, 2015; published online May 27, 2015

Citation: Han YQ, Zhu T. Health effects of fine particles ($PM_{2.5}$) in ambient air. Sci China Life Sci, 2015, 58: 624−626, doi: 10.1007/s11427-015-4878-4

Air pollutants have serious health effects on human beings. The 2010 Global Burden of Disease study reports that each year, ambient particulate matter ($PM_{2.5}$, particles with aerodynamic diameter < 2.5 micrometer) is causing 3.2 million premature deaths globally, and 1.2 million in China alone. Ambient PM pollution ranked fourth among all risk factors in China [1].

Although the association of $PM_{2.5}$ exposure with premature deaths from lung cancer, chronic obstructive pulmonary disease (COPD), ischemic heart disease (IHD) and cerebrovascular disease are well documented, there is growing evidence that $PM_{2.5}$ exposure in ambient air has health effects on the brain, reproduction, immune system, and metabolism. However, the underlying mechanism for such effects is complicated, with various hypothetical pathways proposed. It is generally believed that oxidative stress and pulmonary inflammation are direct effects of $PM_{2.5}$ particles, which in turn lead to systematic inflammation and endothelial dysfunction, automatic nervous system imbalance, and others [2]. Substantial evidence from toxicological and epidemiological studies has accumulated to reveal the mechanism of the $PM_{2.5}$ health effects, but this evidence is frequently inconsistent and at times even contradictory.

To address the complicated nature of $PM_{2.5}$ health effects, one needs to consider both those effects and PM characteristics simultaneously, e.g., time after exposure (lag effects), particle size and chemical composition, and susceptible populations. Here, we suggest that these variables compose four dimensions that are necessary to describe the $PM_{2.5}$ health effects and their underlying mechanism (Figure 1).

Time after exposure (Lag effect). Health effects of air pollution represent a continuous pathophysiological process, evolving from acute to chronic effects. The order of this process may be described as external exposure, internal exposure, change in subclinical biomarkers, change in function/clinical endpoints, disease, and death. Based on difference in research purpose and design, selected health endpoints and related observation time windows of the effects vary. For example, panel and controlled exposure studies focus on subclinical biomarkers and functional change, such as inflammatory cytokines in serum or altered heart rate variability (HRV). Therefore, the observed lag effect may be from hours to days. Time-series studies have examined acute effects using clinical visits, hospitalization rates, or mortality as endpoints, with time windows from days to weeks [3]. Cohort and cross-sectional studies usually aim at chronic effects after years or even decades-long air pollution exposure, using morbidity/mortality of chronic disease and life expectancy as health endpoints.

Researches on the health effects of air pollution in China have mainly been based on time-series studies. Cohort and panel studies are limited, and no prospective cohort study has been reported. In addition to strength of causality, cohort studies in China can provide specific exposure-response relationships at concentration levels observed in most Chinese cities (e.g., annual mean $PM_{2.5}$ concentrations 50–100 μg m^{-3}). This could furnish much greater accuracy in the estimation of premature death caused by air pollution. Panel studies would enhance understanding of the mechanisms underlying health effects, identification of spe-

*Corresponding author (email: tzhu@pku.edu.cn)

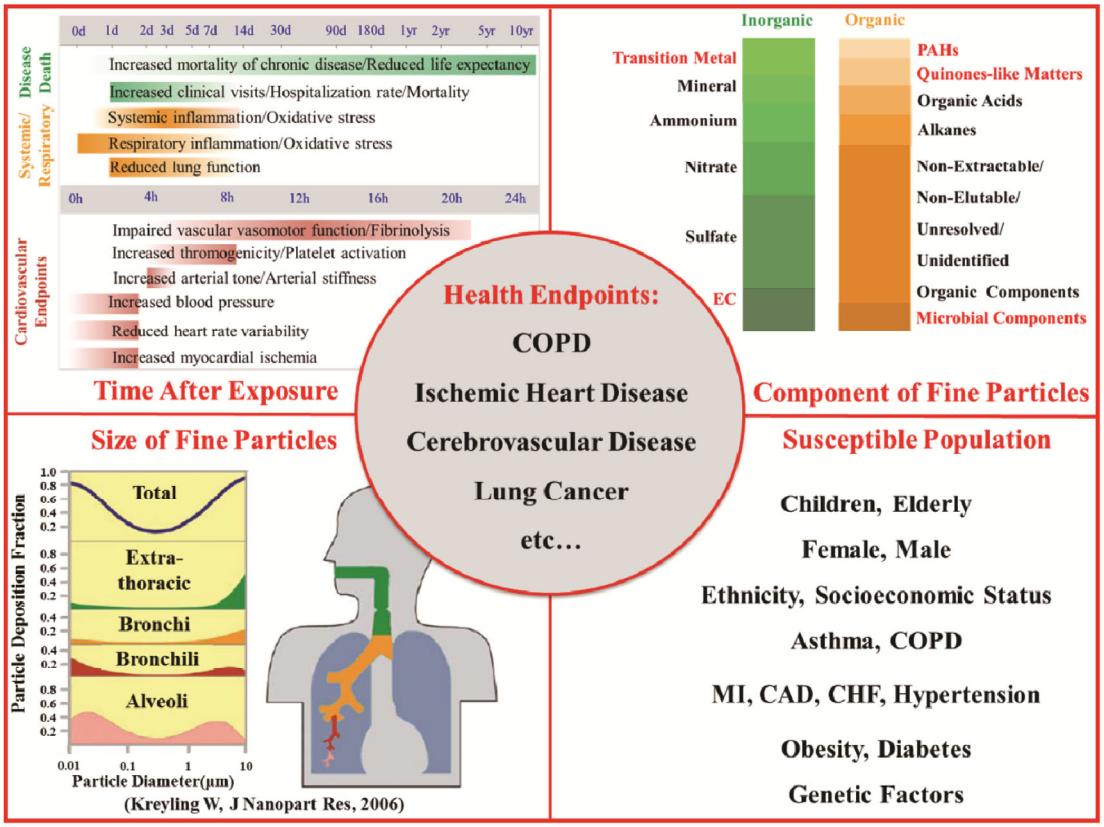

Figure 1 Four dimensions required to describe health effects of fine particles on human subjects, i.e., time after exposure, size and composition of fine particles, and susceptible populations.

cific air components of $PM_{2.5}$ that generate those effects, and identify representative biomarkers in the early phase of the effects.

Size effect of fine particles. Size distribution is an important characterization of particles in ambient air. Particles of varying aerodynamic size indicate differences not only in their compositions and sources but also related health effects.

According to the aerodynamic behavior of size-segregated particles in the human respiratory tract, a predictive mathematical model, the multiple path particle dosimetry model, was developed by the International Commission on Radiological Protection to estimate the deposition of inhaled particles of various size in the head/nasopharyngeal, tracheobronchial, and pulmonary/alveolar regions of the respiratory tract [4]. Results show that for particles with diameters of 20–100 nm, deposition efficiency in the deep pulmonary section increases with decreasing particle size. That is, smaller particles are more likely to be deposited in that section, contact epithelial cells and macrophages, causing subsequent inflammation.

Toxicological studies have also provided evidence that particles of smaller size were cleared slower than larger ones, and are thereby retained longer within the lung after deposition. Particles of size less than 100 nm may have the ability to cross the air-blood barrier in the alveolar and enter the cardiovascular system. In addition, such particles have a larger active surface area, which tends to absorb more toxic chemicals. Some time-series studies support the hypothesis that smaller particles have greater health effects, but results are inconsistent. Number concentration and specific composition of size-segregated particles in ambient air should also be considered. Moreover, population-based studies focused on the effect of size-segregated particles on comprehensive biomarkers are still lacking.

Component effect of fine particles. Toxicological studies have proven that certain compositions of particles have a stronger toxicity than others, e.g., transition metal, carbonaceous components, polycyclic aromatic hydrocarbons, and quinones-like matters. Microbial components in particles have also been identified as potential risk factors in human health. Although some evidence from epidemiological studies supports the hypothesis that particle health effects are attributable to certain components, there have been no consistent conclusions. The mass concentration of PM (e.g., $PM_{2.5}$) remains the most robust indicator of health impacts.

The inconsistency in the population-based research may be attributed to the following reasons. First, despite the spatial uniformity of $PM_{2.5}$ concentration in the subject region,

certain components may have inhomogeneous spatial distributions. For instance, transition metal and carbonaceous components vary because of local source emissions, so using single-site monitoring data in a population-based study could lead to exposure misclassification. Second, PM health effects in ambient air are not simply additive effects of the toxicological results of various components, but more likely a combination of additive, synergistic, and antagonistic effects. Third, evaluation of the health effects of certain particulate compositions in ambient air requires long-term, multi-species measurements, which are difficult to apply in an epidemiological study.

Susceptible populations. Environmental protection criteria are in principle established based on integrated health risk assessment of the total population. However, epidemiological studies have suggested that certain subgroup of the population, such as children, older, asthmatics, those with COPD or IHD, are more susceptible than others to the health effects of air pollution [5]. This susceptibility may be related to differences in physiological structure, immune level, exposure pattern, and underlying disease condition. For example, the World Health Organization indicates that children are potentially more susceptible to PM than adults because of their greater activity levels, time spent outdoors, and minute volume per unit body weight, all of which can increase the PM dose per lung surface area and have subsequent adverse effects on their developing lungs. In comparison, the susceptibility of diabetic individuals to air pollution remains unclear and has attracted increased interest, which might be partially attributed to the association of PM with diabetes prevalence and various markers in plausible biological pathways. More population-based studies are needed to reveal the susceptibility of population subgroups to evaluate the health burden of ambient air pollution more accurately, and to implement specific control and prevention measures for those susceptible to such pollution.

The four dimensions of the variables summarized above will not only aid understanding of the complicated nature of $PM_{2.5}$ health effects and identify the main $PM_{2.5}$ size fraction and chemical composition that cause the effects, but also provide comprehensive evidence in a broad perspective for air pollution control and public health prevention strategies.

1 Lim SS, Vos T, Flaxman AD, Danaei G, Shibuya K, Adair-Rohani H, Amann M, Anderson HR, Andrews KG, Aryee M, et al. A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions, 1990-2010: a systematic analysis for the Global Burden of Disease Study 2010. Lancet, 2012, 380: 2224–2260
2 Brook RD, Rajagopalan S, Pope CA 3rd, Brook JR, Bhatnagar A, Diez-Roux AV, Holguin F, Hong Y, Luepker RV, Mittleman MA, et al. Particulate matter air pollution and cardiovascular disease: An update to the scientific statement from the American Heart Association. Circulation, 2010, 121: 2331–2378
3 Donaldson K, Duffin R, Langrish JP, Miller MR, Mills NL, Poland CA, Raftis J, Shah A, Shaw CA, Newby DE. Nanoparticles and the cardiovascular system: a critical review. Nanomedicine (Lond), 2013, 8: 403–423
4 Kreyling, WG, Semmler-Behnke, M, Moller, W. Health implications of nanoparticles. J Nanopart Res, 2006, 8, 543–562
5 Sacks JD, Stanek LW, Luben TJ, Johns DO, Buckley BJ, Brown JS, Ross M. Particulate matter-induced health effects: who is susceptible? Environ Health Perspect, 2011, 119: 446–454