

Chapter 9

Comparison of Health Impact of Air Pollution Between China and Other Countries

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Abstract Air pollution is the world's largest single environmental risk according to the World Health Organization (WHO), which caused around seven million deaths in 2012. Extensive epidemiological studies have been carried out worldwide to examine the health impacts of ambient air pollution, consistently demonstrating significant health impacts of ambient air pollution. Air pollution problem in China is especially serious; it has become the fourth biggest threat to the health of the Chinese people. In this review, we summarized existing literature, compared health impact of air pollution between China and other countries, and found substantial heterogeneity in the risk estimates of air pollution. The effect heterogeneities may be due to the differences in the characteristics of populations (e.g., the proportion of the elder population and people with preexisting diseases), exposure profile (e.g., air pollution concentrations and composition), and regional climate. Although the magnitude of relative risk estimates of air pollution is generally similar with that in other parts of the world, air pollution is one of China's most serious environmental health problems given the huge number of people exposed to high concentration levels of air pollution in China.

Keywords Particulate matter • Concentration-response functions • Effect heterogeneity

9.1 Introduction

Extensive epidemiological studies have been conducted worldwide to examine the health impacts of air pollution, and the evidence for the adverse effects of ambient air pollution on morbidity and mortality is convincing in both China [11, 40] and

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other countries ([21]; Isabelle [30, 36]) (details discussed in Chaps. 4 and 5). However, the concentration-response functions (CRFs), the percent change of a given health outcome per $\mu\text{g}/\text{m}^3$ increment in concentration of air pollution, may differ according to geographical regions. These effect heterogeneities may be due to the difference in the characteristics of populations, exposure profile (e.g., air pollution concentration, particulate matter size distribution and composition), and regional climate. Insights into these modifying factors might help to understand the health effects of air pollution and could lead to different air quality guidelines for different parts of the world.

China is a developing country and is in the process of fast industrialization and rapid economic development. With fast development over the past three decades, China had become the world's second largest economy in terms of gross domestic product (GDP) in 2010 and was the world's biggest energy consumer in 2009. Coal is the major source of energy; the amount of coal consumption in China constitutes about 50% of the world's total coal consumption in 2012. At the same time, the number of on-road civilian vehicles increased dramatically from 16.09 million in 2000 to 93.56 million in 2011 [12]. China's extensive industrial development, substantial coal-dependent energy consumption, and increasing number of vehicles have led to a rise in emissions of air pollutants. Consequently, air pollution is one of China's most serious environmental problems. Ambient air pollution has become the fourth biggest threat to the health of the Chinese people according to the Global Burden of Disease Study 2010 [46].

China has its unique characteristics when compared to the developed countries (e.g., the United States and the European countries). Being the largest producer and consumer of coal in the world, together with outdated technologies and poor maintenance of coal burning facilities, China is one of the few countries with the highest air pollution levels (both outdoor and indoor) in the world [41, 42]. In addition, sociodemographic characteristics of residents (e.g., age structure, disease pattern, and socioeconomic status) in China also differ from those of developed countries. Thus, comparing the health effects of air pollution between China and other countries may provide some insights into the potential modifiers of the adverse health effects of air pollution.

In this chapter, we focus on the short-term health effects of particulate matter (PM) and ozone (O_3), comparing the findings from China and other countries, and discussing the potential effect modifiers for the adverse health effect of air pollution.

9.2 Comparison of CRFs Between China and Other Countries

Case-crossover and time-series analysis are the two main methods to analyze the short-term health effects of air pollution. The case-crossover study design, however, has its own disadvantages: it does not account for over-dispersion and may

underestimate the variance of health estimates in each city, and it can induce larger estimates for heterogeneity than does time-series analysis [21]. On the other hand, there is a broad spectrum of model specification in the time-series analysis, including the methods used for smoothing (e.g., natural splines or penalized spline) and the number of degrees of freedom for smoothing, but effect estimates remained fairly stable [21]. Thus, we restricted our comparative analysis to those estimates from time-series studies in order to reduce the complexities introduced by differing analytic strategies.

Although numerous effect estimates have been generated from single-city studies, these results are hard to synthesize. Multi-city studies combining estimates from various locations using a common protocol may reduce potential bias (e.g., analytic bias, lag selection bias, and publication bias) and increase precision, thus can be used to evaluate the heterogeneity of the air pollution effect across cities. In addition, there are relatively few studies in China which have examined effects of air pollution on hospital admissions, and few studies have examined long-term effects of air pollution. Currently, the China Kadoorie Biobank (CKB) cohort study is under way and has not produced findings on air pollution and health yet. This large, well-established study should generate new findings for China. Thus, in this chapter, we mainly compare results from multi-city studies in the world, supplemented by meta-analysis results in the literature.

9.2.1 *Particulate Matter (PM)*

Airborne particulate matter (PM) is a complex mixture of solid and liquid particles of primary and secondary origin, which contain a wide range of inorganic and organic components. It is created by both natural and man-made sources and is found both outdoors and indoors. From the health perspectives, PM is typically defined by size, with the smaller particles having larger health impacts. PM with aerodynamic diameter less than 10 μm is called PM_{10} , and PM with aerodynamic diameter less than 2.5 μm is called $\text{PM}_{2.5}$.

Numerous large-scale multi-city epidemiological studies have been carried out to estimate the health effects of air pollution worldwide. Air pollution and health: A European and North American Approach (APHENA) was a Health Effects Institute (HEI)-sponsored project consisting of three components: (1) the Air Pollution and Health: A European Approach Phase 2 (APHEA-2) study involving 32 European cities; (2) the National Morbidity, Mortality, and Air Pollution Study (NMMAPS), conducted in 90 largest US cities; and (3) multi-city research on the health effects of air pollution in 12 Canadian cities [21]. Estudio de Salud y Contaminación del Aire en Latinoamérica (ESCALA) was another HEI-sponsored project that aimed to estimate the health effects of air pollution in 9 Latin American cities [30]. Simpson et al. conducted a multi-city study to estimate the short-term mortality effects of air pollution in 4 Australian cities (Melbourne, Sydney, Brisbane, and Perth), which account for about 50% of the Australian population [36]. The China Air Pollution

and Health Effect Study (CAPES) was the largest multi-city study conducted in China which included 16 Chinese cities covering about 69 million population [11]. These multi-city epidemiological studies generally applied two stages of analysis to estimate city-specific and national average associations of PM_{10} with daily mortality. In the first stage of the analysis, city-specific estimate was estimated followed by a common protocol for fitting the Poisson regression models to the air pollution and mortality time-series data in each city. In the second stage, hierarchical model or meta-analysis was used to pool city-specific estimates and obtain the national average estimates of the association of PM_{10} with mortality. Potential effect modification by sociodemographic characteristics and regional climate was also assessed across study regions.

The associations between PM_{10} and all-cause mortality were positive and statistically significant for the all-age group in Canada, Europe, the United States, Latin America, and China, but not in Australia. When pooling estimates from cities, the percentage excess risks per $10 \mu g/m^3$ increase of PM_{10} concentration for all-cause mortality were 0.84% (95% confidence interval: 0.30%, 1.40%), 0.26% (0.15%, 0.36%), 0.26% (0.15%, 0.37%), 0.77% (0.60%, 1.00%), 0.20% (−0.80%, 1.20%), and 0.35% (0.18%, 0.52%) for Canada, Europe, the United States, Latin American, Australia, and China, respectively. The effect estimates for Canada (0.84%) and Latin America (0.77%) were two or three times higher than those for Europe (0.26%), United States (0.26%), Australia (0.20%) and China (0.35%).

APHENA studied the health effects of PM_{10} on cardiovascular mortality for people aged less than 75 and people aged 75 years and older separately, while other multi-city studies examined the effects of PM_{10} on cardiovascular mortality for all ages. The risk estimates of PM_{10} on cardiovascular mortality were positive and statistically significant in Canada (people ≥ 75 years), Europe (people ≥ 75 years), the United States (people ≥ 75 years), Latin America (all-age group), and China (all-age group).

Effects of PM_{10} on respiratory mortality were less consistent across regions. A $10 \mu g/m^3$ increase in PM_{10} was associated with respiratory mortality of −1.4% (−3.2%, 0.5%), 0.37% (0.03%, 0.73%), 0.15% (−0.28%, 0.59%), 1.39% (0.98%, 1.81%), and 0.56% (0.31%, 0.81%) in Canada, Europe, the United States, Latin America, and China, respectively. Those associations were statistically significant in Europe, Latin America, and China only.

In summary, there is indeed heterogeneity for mortality effects of PM_{10} . The relative risks estimated in Canadian cities were much larger than in other regions. The effect sizes in China were comparable with those in Europe and the United States (Fig. 9.1 and Table 9.1).

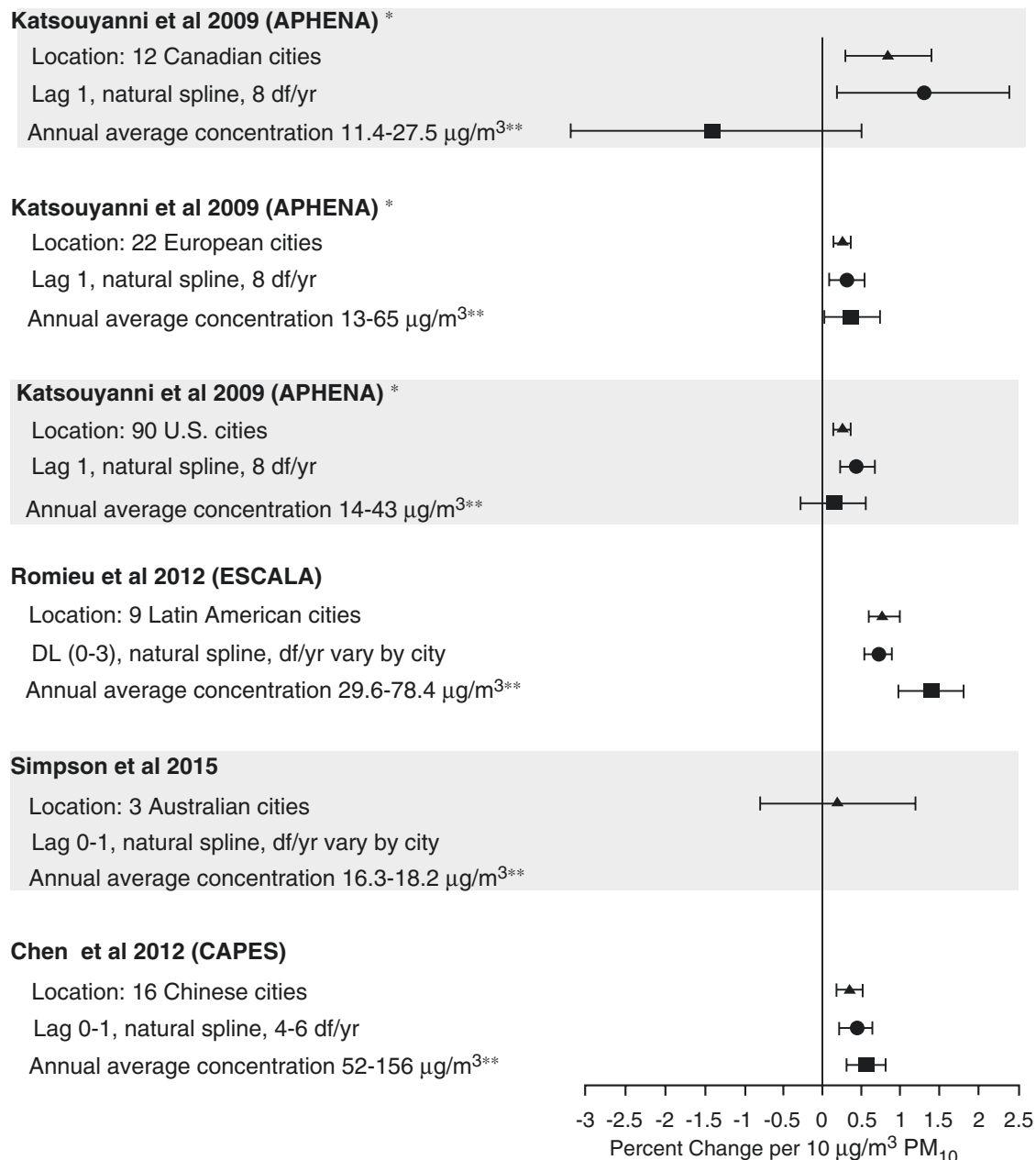


Fig. 9.1 Percentage excess risk (ER %) of all-natural, cardiovascular, and respiratory mortality per 10 µg/m³ increase in PM₁₀ in multi-city studies. Estimates with ▲ indicate all-natural mortality, estimates with ● represent cardiovascular mortality, and estimates with ■ denote respiratory mortality. *Estimates are for all ages, except estimates of cardiovascular mortality are for people ≥75 years. **Concentration ranges across cities (Source: Adapted from Romieu et al. [30]; p. 100)

9.2.2 Gaseous Pollutants

Multi-city studies of APHENA, ESCALA, and Simpson et al. [36] also examined the mortality effects of ozone (O₃). A four-city time-series study investigated the short-term effects of O₃ in the Pearl River Delta (PRD) of southern China [40].

Table 9.1 Mortality risk estimates for PM₁₀ exposure based on meta-analyses results

Country or region	Daily mortality	Reference	ER (%) per 10 µg/m ³	95% CI
Europe	All-cause	[2]	0.6%	0.4–0.8%
Europe	Cardiovascular	[2]	0.9%	0.5–1.3%
Europe	Respiratory	[2]	1.3%	0.5–2.09%
Asia	All-cause	[14]	0.49%	0.23–0.76%
China	All-cause	[34]	0.32%	0.28–0.35%
China	Cardiovascular	[34]	0.43%	0.37–0.49%
China	Respiratory	[34]	0.32%	0.23–0.40%

ER(%) percentage excess risk, CI confidence interval

Ozone was associated with all-natural mortality in each of the six regions, except for Australia and Latin America. The excess risks of ozone per 10 µg/m³ increase in all-natural mortality in Canada [0.52% (0.24%, 0.80%)] and China [0.81% (0.63%, 1.00%)] were greater compared to effects in Europe [0.13% (0.05%, 0.21%)] and the United States [0.18% (0.01%, 0.35%)]. For cardiovascular mortality, the risk estimates varied substantially. The effect estimates are positive and statistically significant for Canada (people ≥75 years), Latin America (all-age group), and China (all-age group). The association between ozone and respiratory mortality was generally nonsignificant, except Latin American, Australia, and China with percentage excess risks per 10 µg/m³ increase in ozone of 0.21% (0.10%, 0.31%), 1.10% (0.10%, 2.10%), and 1.33% (0.89%, 1.76%), respectively.

In summary, the magnitude of excess risks of ozone in China was generally larger than in other countries or regions for all-natural, cardiovascular, and respiratory mortality among the multi-city studies (Fig. 9.2 and Table 9.2). The underlying reasons should be explored in future studies.

9.3 Modifying Factors for CRFs

Sources of heterogeneity in the risk estimates for PM and O₃ may include different exposure profiles, regional climate, and population characteristics.

9.3.1 Concentration Ranges of Air Pollutants

Cities of the developing countries currently have the highest air pollution concentration (Figs. 9.3 and 9.4), particularly those cities in Asia with extensive coal burning [29]. For example, the ranges of annual mean concentrations of PM₁₀ and O₃ in 74 Chinese cities were from 42 to 233 µg/m³ and 69–200 µg/m³, compared with the mean concentrations of 105 µg/m³ and 145 µg/m³ in those 74 Chinese cities in 2014, respectively [13], which is far exceeding the limits of both national air quality

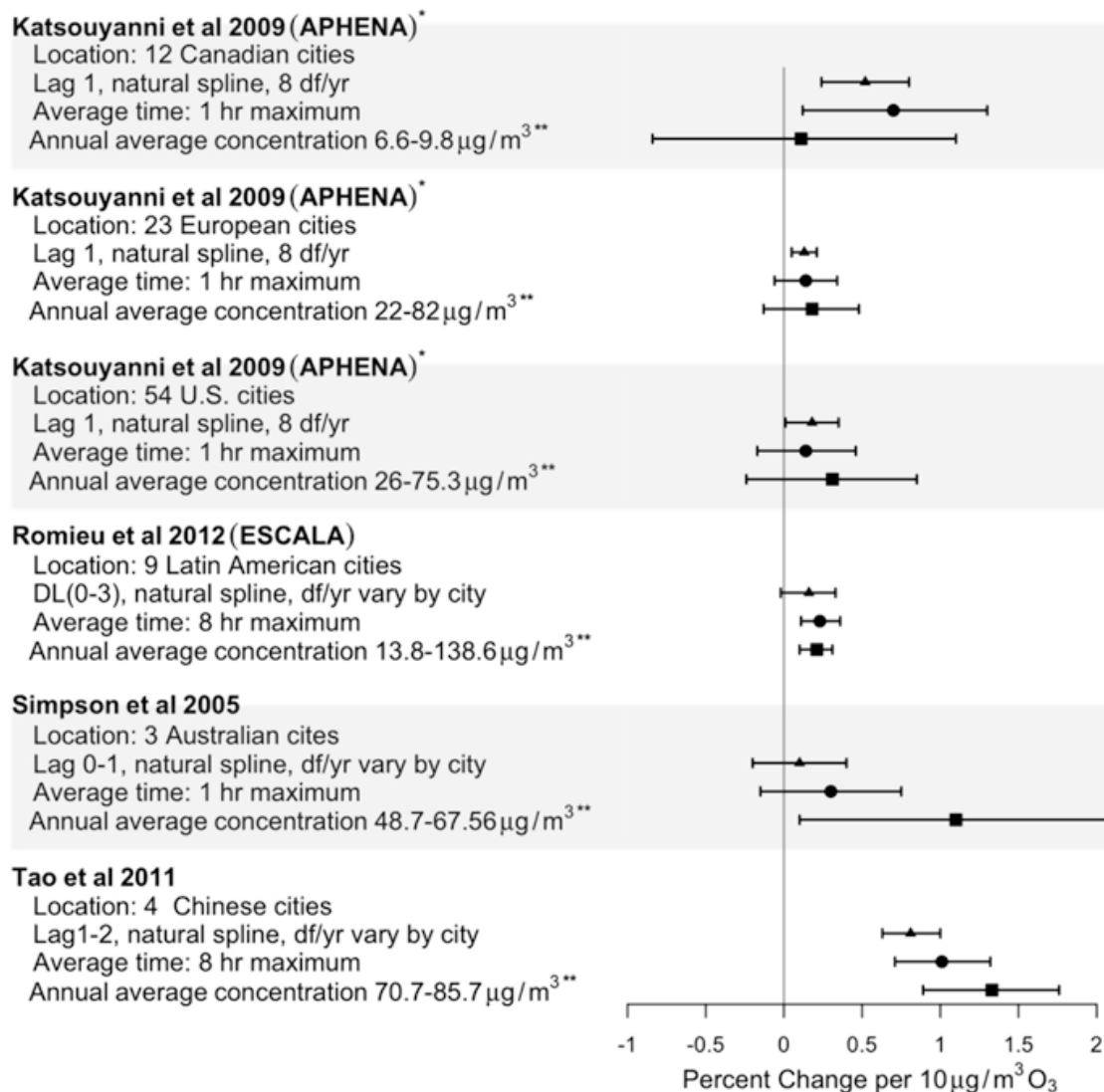


Fig. 9.2 Percentage excess risk (ER %) of all-natural, cardiovascular, and respiratory mortality per 10 µg/m³ increase in O₃ in multi-city studies. Estimates with ▲ indicate all-natural mortality, estimates with ● represent cardiovascular mortality, and estimates with ■ denote respiratory mortality. *Estimates are for all ages, except estimates of cardiovascular mortality are for people ≥75 years. **Concentration ranges across cities (Source: Adapted from Romieu et al. [30]; p. 101)

standards and the air quality guidelines recommended by the World Health Organization (WHO). Cities of the developed countries seem to have better air quality in terms of PM₁₀ and O₃ concentration compared to developing countries. For instance, the median concentration of PM₁₀ ranges from 14.0 µg/m³ to 43.7 µg/m³ and 13.1 µg/m³ to 65.0 µg/m³ in the United States and European cities, respectively. The median concentration of O₃ ranges from 26.0 µg/m³ to 75.3 µg/m³ and 37.5 µg/m³ to 82.2 µg/m³ in the United States and European cities, respectively [21].

The CRF for PM₁₀ with mortality was found to be linear in *NMMAPS* [17] and *APHEA-2* project [31, 32] at low air pollution concentrations levels with no threshold identified. The CRF outside the range of concentration in North America and

Table 9.2 Mortality risk estimates for ozone exposure based on meta-analyses results

Country or region	Daily mortality	Reference	ER (%) per 10 $\mu\text{g}/\text{m}^3$	95 % CI
Europe	All-cause	[2]	0.3%	0.1–0.4%
Europe	Cardiovascular	[2]	0.4%	0.3–0.5%
Europe	Respiratory	[2]	0%	–0.4–0.5%
The United States	All-cause	[4]	0.84%	0.48–1.20%
The United States	Cardiovascular	[4]	0.85%	–0.66–2.39%
The United States	Respiratory	[4]	0.65%	–1.84–3.21%
China	All-cause	[34]	0.48%	0.38–0.58%
China	Cardiovascular	[34]	0.45%	0.29–0.60%
China	Respiratory	[34]	0.73%	0.49–0.97%

ER (%) percentage excess risk, CI confidence interval

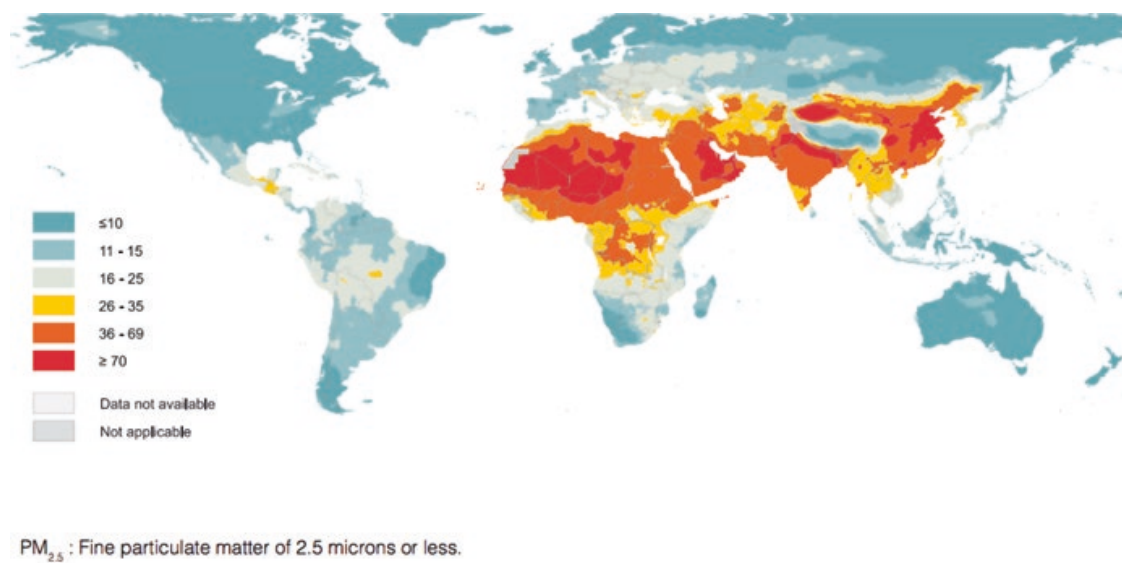


Fig. 9.3 Global map of modeled annual median concentration of PM_{2.5}, in $\mu\text{g}/\text{m}^3$ (Source: World Health Organization, website: <http://who.int/phe/publications/air-pollution-global-assessment/en/>)

Europe is less clear. It is likely that the CRF will begin to flatten at very high concentrations as studies suggested [3, 45] (Fig. 9.5); therefore, variation in air pollution levels could contribute to heterogeneity in CRF, particularly if the underlying curves are nonlinear.

9.3.2 The Concentration of Copollutants

The concentration of copollutants (e.g., nitrogen dioxide) is the highest in China (Fig. 9.4). It might be an effect modifier for the mortality effects of PM. For example, higher nitrogen dioxide (NO₂) concentrations were found to be associated with larger PM mortality effect estimates in single-city studies [16, 18]. A multi-city study [21]

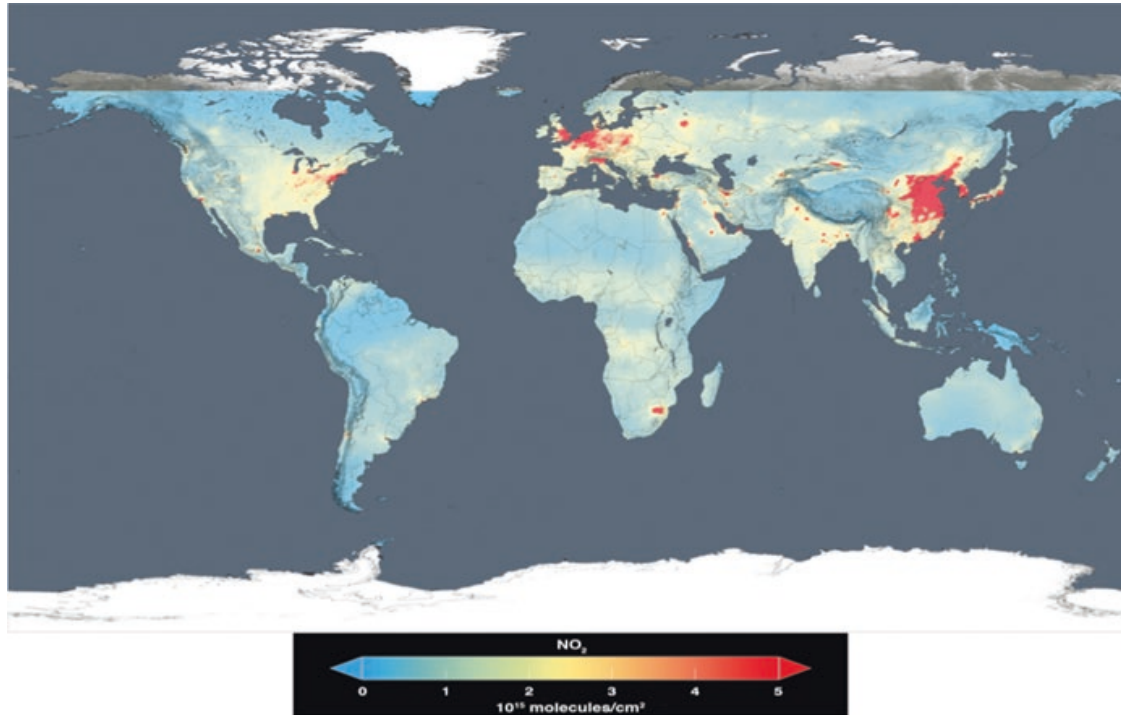


Fig. 9.4 Global map of the 2014 annual average concentration of nitrogen dioxide (Source: The National Aeronautics and Space Administration (NASA), website: <https://www.nasa.gov/press-release/new-nasa-satellite-maps-show-human-fingerprint-on-global-air-quality>)

also found that in cities with higher mean NO₂ levels and mean NO₂/PM₁₀ ratios, the risk estimates of mortality were higher [0.44% (cities with higher mean NO₂ levels) versus 0.17% (cities with lower mean NO₂ levels) and 0.42% (cities with higher mean NO₂/PM₁₀ ratios) versus 0.17% (cities with lower mean NO₂/PM₁₀ ratios) increase per 10 µg/m³ change in PM₁₀ for all-cause mortality in Europe, respectively, and 0.28% versus 0.01% and 0.27% versus 0.16% in the United States, respectively]. This may suggest an interaction between NO₂ and PM. The reason for this interaction may be that exposure to long-term NO₂ could deteriorate lung function and induce oxidative stress, which may increase the vulnerability to the short-term effects of PM [18]. NO₂ is an indicator of pollution originating from traffic. It may also indicate that PM originating from traffic is more toxic than those from other sources.

9.3.3 Chemical Composition and Emission Sources

As discussed in Chap. 2, PM is a complex mixture of different components including elemental carbon (EC), organic carbon (OC), sulfate (SO₄), nitrate (NO₃), and trace elements. Variation in estimated health effects could be driven by regional variation in the chemical composition of PM.

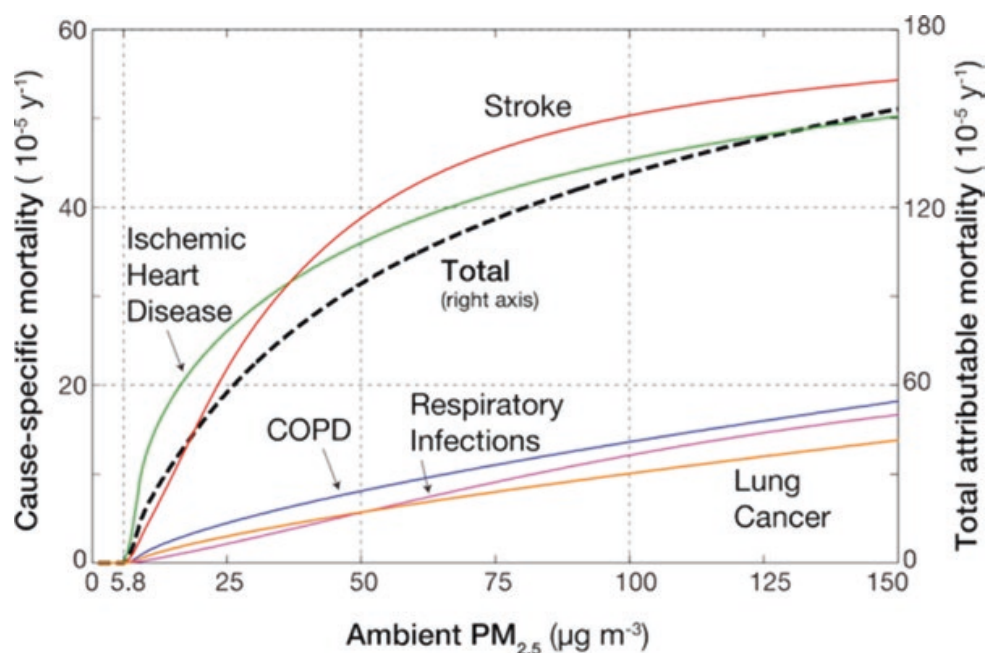


Fig. 9.5 Global concentration-mortality relationships for ambient $\text{PM}_{2.5}$ for five individual endpoints (solid lines, left axis) and total of five causes (dashed line, right axis) based on integrated exposure response curves developed for the Global Burden of Disease (GBD) studies [9]. Vertical axes indicate per-capita mortality rates attributable to $\text{PM}_{2.5}$ for a hypothetical global population uniformly exposed to a given level of $\text{PM}_{2.5}$. Plotted data illustrate the relative contribution of individual disease endpoints to total mortality for a typical population exposed at a given concentration by incorporating concentration-response curves and global disease incidence data (Source: Apte et al. [3])

Numerous studies have reported adverse health effects of each PM chemical constituents. Levy et al. conducted a multi-city time-series analysis to estimate the association between $\text{PM}_{2.5}$ constituents and hospital admissions in a population of 12 million US Medicare enrollees [23]. They found that EC was associated with an elevated risk of cardiovascular hospital admission. EC and NO_3 were also linked with emergency type 2 diabetes hospitalizations in Hong Kong elderly population [38]. NO_3 , sodium ion, chloride ion, magnesium, and nickel were associated with cardiovascular hospitalizations, while sodium ion, aluminum, and magnesium were found to be associated with respiratory hospitalizations [27]. Chemical components of PM were also associated with mortality [25, 51] and low birthweight [5].

Several recent multi-city studies have examined whether variability in proportions of PM constituents can explain the effect heterogeneity. Bell et al. [6] examined associations between elderly hospital admissions and 52 chemical components of $\text{PM}_{2.5}$ for 187 counties in the United States and found that variation in EC, nickel, and vanadium could explain the variations in $\text{PM}_{2.5}$ mass effect estimates for both cardiovascular and respiratory hospitalizations [6]. The association between PM_{10} and mortality was also modified when the PM was high in nickel and vanadium in the *NMMAPS* project [24].

Different chemical constituents of PM are generated by various pollutant sources. A few epidemiological studies have used source apportionment analysis (e.g., posi-

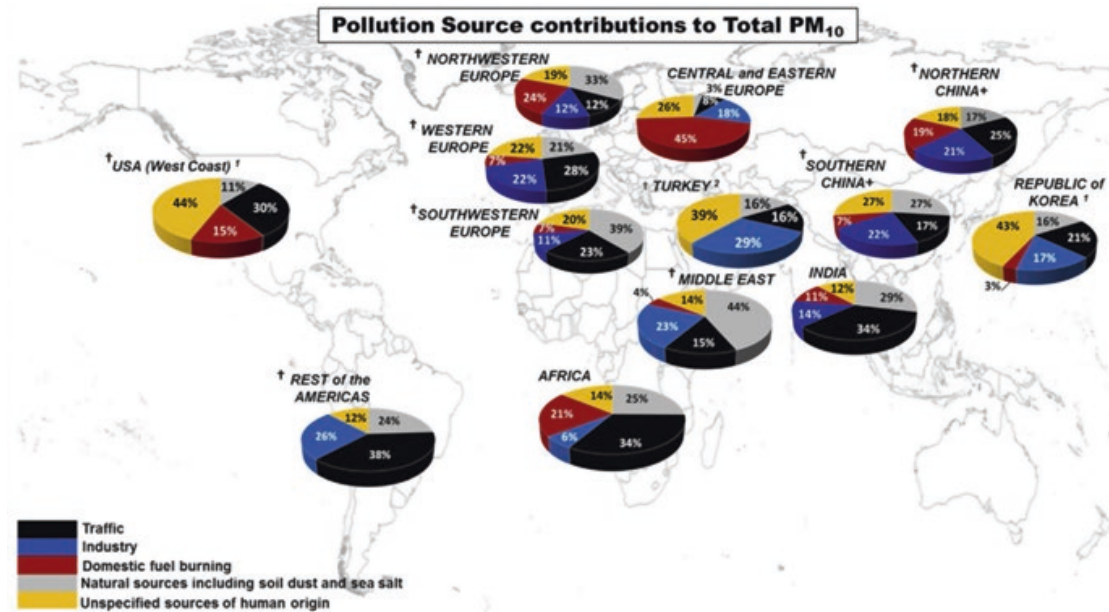


Fig. 9.6 Population-weighted averages for relative source contributions to total PM₁₀ in urban cities. †regions in which domestic fuel burning have not been assessed. †Based only on one study. ‡Based only on one study including traffic (Source: Karagulian et al. [19])

tive matrix factorization) to explore the health effects of PM sources and found some PM sources were more harmful than others. For example, Stanek et al. [37] conducted a literature review summarizing findings from 29 studies in the United States [37]. The authors found that PM from motor vehicle emission was associated with cardiovascular mortality, while PM from coal combustion was associated with total mortality. However, limited number of studies was conducted in China to estimate source-specific health risks of PM. Pun et al. [28] estimated PM₁₀ sources contributed from 19 chemical components by positive matrix factorization in Hong Kong and found that respiratory hospitalizations were associated with vehicle exhaust, regional combustion, and secondary nitrate [28].

Figure 9.6 shows population-weighted averages for relative source contributions to total PM₁₀ in urban sites utilizing the global source apportionment database at the website of the World Health Organization [44]. This database included a total of 529 source apportionment records in cities of 51 countries for a total of 560 million people. This global review [19] found that the contributions by source category varied substantially across regions. For example, traffic was the main contributor to urban ambient PM₁₀ in Africa and India (34%), the United States (30%), Western Europe (28%), and Northern China (28%); industry was an important contributor in Turkey (29%), Western Europe (22%), and China (21–22%); domestic fuel burning was the main contributor in Central and Eastern Europe (45%), Northwestern Europe (24%), Africa (21%), and the Northern China (19%). Contributions by source category in China are comparable with other regions.

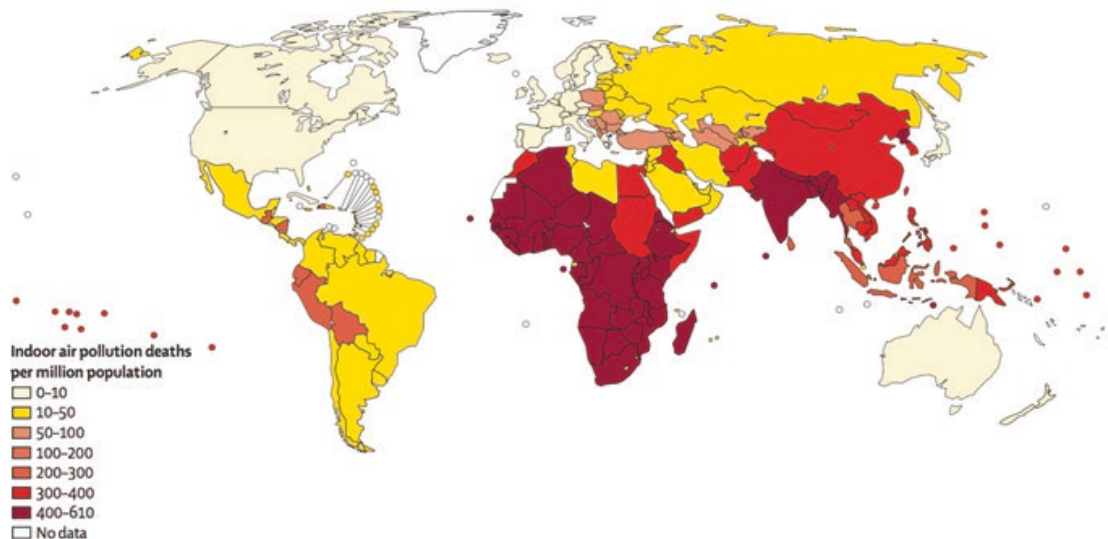


Fig. 9.7 WHO map of household air pollution and mortality (Source: World Health Organization (WHO), website: <http://www.who.int/heli/risks/indoorair/en/iapmap.pdf>)

9.3.4 Indoor Air Pollution

High ambient air pollution concentration does not necessarily result in high personal exposure. Human exposure is determined by the concentration of air pollution in the environments and by the amount of time people spend there. The majority of epidemiological studies utilized outdoor concentrations of air pollutants measured in fixed monitoring stations as a surrogate for personal exposure. Although outdoor air pollutants may penetrate indoors, the concentration of indoor and outdoor air pollutants may differ substantially due to outdoor-to-indoor migration and source of indoor pollutants. Humans spend much more time indoors than outdoors. Indoor air pollution would contribute substantially to personal exposure, especially in countries in which cooking and heating depend on solid fuels (e.g., coal and biomass) with open or poorly ventilated stoves, such as in Africa, India, and China (Fig. 9.7). During cooking or heating, on average, the levels of PM are about 10–20 times higher than the levels in international health guidelines [43]. Thus, simply utilizing outdoor air pollution concentration as a surrogate for personal exposure may result in bias of personal exposure assessment [35]. This bias may vary among countries, which could be one of the reasons to explain effects heterogeneity among countries.

9.3.5 Responsiveness of the Population

Individuals respond differently when exposed to air pollution. Health effects might be exacerbated in susceptible subgroups when exposed to the same levels of pollutants as the average population. This susceptibility to air pollution has been linked to

personal characteristics, such as inherent personal characteristics (e.g., age, socioeconomic status, and chronic diseases) and aspects of lifestyle that may enhance the adverse health effects of air pollutants (e.g., cigarette smoking and exercise).

Population with lower **socioeconomic status** tends to have higher risks of both mortality and morbidity when exposed to the same levels of air pollution. For example, higher susceptibility is found in persons with the least educated, lower income, lower occupational status, and unemployment [8, 50]. The multi-city study of the *APHENA* project [21] found cities with higher percentage of unemployment were associated with a greater effect of PM_{10} on all-cause mortality (0.57% versus 0.27% and 0.23% versus 0.11% increase per $10 \mu g/m^3$ change of PM_{10} in Europe and the United States, respectively). Higher mortality risk estimates were also linked to low educational status in the multi-city study of the *CAPEs* study in China [11].

Higher risk estimates were also found in individuals already affected by **preexisting cardiopulmonary disease** [26, 39, 49] as well as **diabetes** [48]. For example, the risk estimates of PM_{10} for people with respiratory diseases have almost doubled for those with cardiovascular diseases [47]. People with diabetes have 0.7% higher risks (2.0% versus 1.3%) per $10 \mu g/m^3$ increase in PM_{10} for cardiovascular hospital admissions than people who do not have diabetes [48].

Age is another effect modifier for the health effects of PM. Stratified analyses have consistently reported the increased risk of hospital admissions [15] and mortality [10, 33] among **older adults** compared with younger population when exposed to air pollutants. Multi-city study of the *APHENA* project [21] also found cities with higher percentage of older people (≥ 75 years) were associated with a greater effect of PM_{10} on all-cause mortality (0.32% versus 0.25% and 0.24% versus 0.03% increase in mortality per $10 \mu g/m^3$ change in PM_{10} for all-cause mortality in Europe and the United States, respectively). Both the meta-analysis of 144 effect estimates from 39 time-series studies and *NMMAPS* of 95 US cities showed that elders had much higher ozone-associated mortality risk than younger groups [4]. A literature review summarized 23 related studies and concluded that age is the most consistent effect modifier of the association between short-term exposure to PM and mortality and hospitalization with risk estimates to be 0.30% (0.11%, 0.49%) higher for older than for younger population [7]. The multi-city study of the *CAPEs* study also reported that older people (≥ 65 years) tended to be more vulnerable than younger population (5–64 years) in China [11].

Figure 9.8 shows the age distribution by countries. The proportion of the older population (≥ 65 years) is higher in developed countries or regions (e.g., European areas, Australia, and the United States). China has the lowest proportion of older among the four countries or regions (China, European areas, Australia, and the United States) in 2011. However, China's population is growing old at a faster rate than almost all other countries. It is expected that more than a quarter of China's population will be over 65 in 2050, which is almost 500 million people [1] (Fig. 9.9). With population aging in China, the effect sizes of air pollution are expected to increase.

Fig. 9.8 Age distribution by country and area in 2011 (Source: World Bank, World Development Indicators, website: <https://www.indexmundi.com/facts/visualizations/age-distribution/#country=au:cn:x:c:us>)

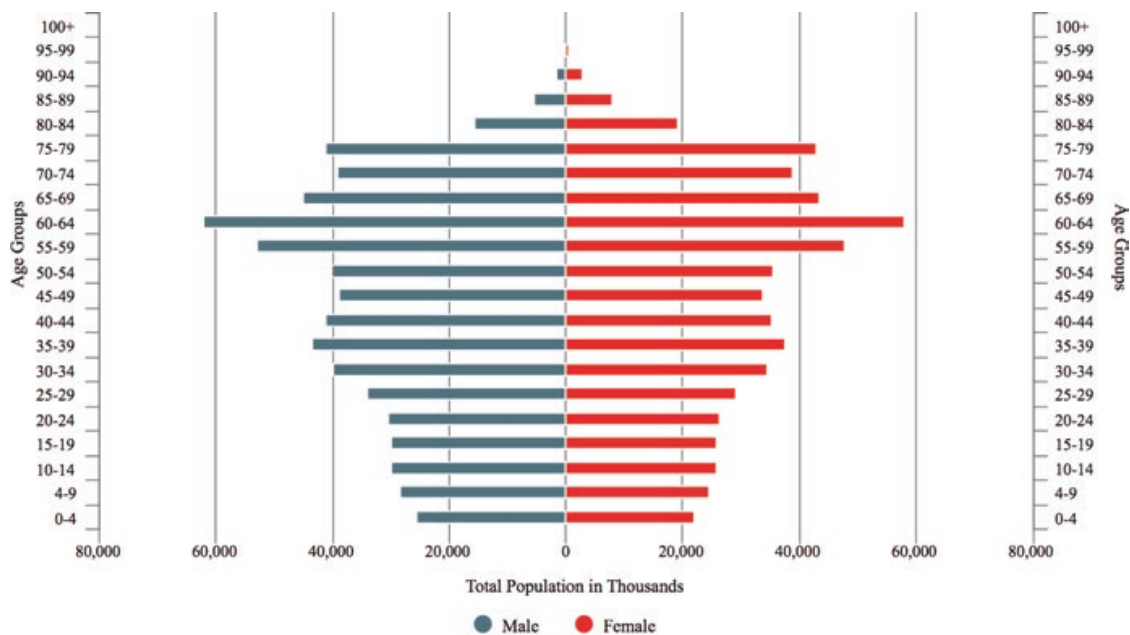
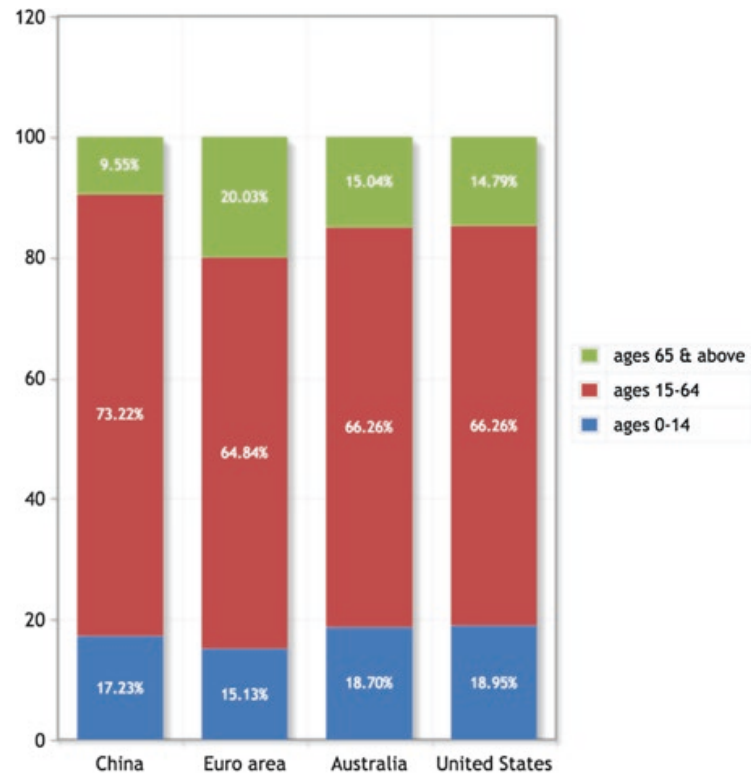


Fig. 9.9 Population projections for China in 2050 (Source: United Nations World Population Prospects, website: <http://chinapower.csis.org/aging-problem/>)

9.3.6 Regional Climate

Many multi-city studies have consistently shown that the effect estimates of air pollution were higher in warmer cities. Katsouyanni et al. [20] reported that the short-term mortality effect of PM₁₀ per 10 µg/m³ increase was 0.29% in cities with relatively cold climate, whereas it was 0.82% in warm climate in the APHEA-2 projects with a total population of more than 43 million [20]. Kioumourtzoglou et al. [22] assessed whether community-level variables modify the association between long-term PM_{2.5} exposures and mortality in a population of more than 35 million Medicare enrollees from 207 US cities. They found that temperature was the most consistent effect modifications [22]. Larger estimates of the effects of particles on mortality were found in warmer cities (e.g., 0.8% versus 0.3% increase in mortality per 10 µg/m³ change in PM₁₀). One possible explanation is that people tend to spend more time outdoors in warmer cities and keep their windows open so that the concentrations of air pollution measured at the outdoor fixed monitoring stations may better represent the average population exposure.

9.4 Summary

Health risk estimates of air pollution are heterogeneities across region. Differences in responsiveness of the population, exposure profiles such as air pollution concentration and composition, and regional climate may be contributable to this effect heterogeneity. Although the relative risk estimates of air pollution are generally similar between China and elsewhere, air pollution is one of China's most serious environmental health problems given the large number of people exposed to high concentration levels of air pollution in China.

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References

1. Center for Strategic & International Studies (CSIS). Does china have an aging problem? Available: <http://chinapower.csis.org/aging-problem/> (n.d.). Accessed 12 June 2017.
2. Anderson HR, Atkinson RW, Peacock JL, Marston L, Konstantinou K, World Health Organization. Meta-analysis of time-series studies and panel studies of particulate matter (PM) and ozone (O3): report of a WHO task group. WHO Regional Office for Europe: Copenhagen; 2004.
3. Apte JS, Marshall JD, Cohen AJ, Brauer M. Addressing global mortality from ambient PM_{2.5}. *Environ Sci Technol*. 2015;49(13):8057–66.

4. Bell ML, Dominici F, Samet JM. A meta-analysis of time-series studies of ozone and mortality with comparison to the national morbidity, mortality, and air pollution study. *Epidemiology*. 2005;16(4):436–45.
5. Bell ML, Belanger K, Ebisu K, Gent JF, Lee HJ, Koutrakis P, et al. Prenatal exposure to fine particulate matter and birth weight: variations by particulate constituents and sources. *Epidemiology*. 2010;21(6):884–91.
6. Bell ML. Assessment of the health impacts of particulate matter characteristics. *Health Eff Ins*. 2012;161:5–38.
7. Bell ML, Zanobetti A, Dominici F. Evidence on vulnerability and susceptibility to health risks associated with short-term exposure to particulate matter: a systematic review and meta-analysis. *Am J Epidemiol*. 2013;178(6):865–76.
8. Bell ML, Zanobetti A, Dominici F. Who is more affected by ozone pollution? A systematic review and meta-analysis. *Am J Epidemiol*. 2014;180(1):15–28.
9. Burnett R, Pope C III, Ezzati M, Olives C, Lim S, Mehta S, et al. An integrated risk function for estimating the global burden of disease attributable to ambient fine particulate matter exposure. *Environ Health Perspect*. 2014;122:397–403.
10. Cakmak S, Dales RE, Vidal CB. Air pollution and mortality in Chile: susceptibility among the elderly. *Environ Health Perspect*. 2007:524–7.
11. Chen R, Kan H, Chen B, Huang W, Bai Z, Song G, et al. Association of particulate air pollution with daily mortality: the china air pollution and health effects study. *Am J Epidemiol*. 2012;175(11):1173–81.
12. Chen Z, Wang JN, Ma GX, Zhang YS. China tackles the health effects of air pollution. *Lancet*. 2013;382(9909):1959–60.
13. Fu L, Wan W, Zhang WH. China air 2015: air pollution prevention and control progress in Chinese cities. Manila: Clean Air Asia; 2015.
14. HEI International Oversight Committee. Health effects of outdoor air pollution in developing countries of Asia: a literature review. Boston: Health Eff Inst; 2004.
15. D'Ippoliti D, Forastiere F, Ancona C, Agabiti N, Fusco D, Michelozzi P, et al. Air pollution and myocardial infarction in Rome: a case-crossover analysis. *Epidemiology*. 2003;14(5):528–35.
16. Deguen S, Petit C, Delbarre A, Kihal W, Padilla C, Benmarhnia T, et al. Neighbourhood characteristics and long-term air pollution levels modify the association between the short-term nitrogen dioxide concentrations and all-cause mortality in paris. *PLoS One*. 2015;10(7):e0131463.
17. Dominici F, Daniels M, Zeger SL, Samet JM. Air pollution and mortality: estimating regional and national dose-response relationships. *J Am Stat Assoc*. 2002;97(457):100–11.
18. Faustini A, Stafoggia M, Renzi M, Cesaroni G, Alessandrini E, Davoli M, et al. Does chronic exposure to high levels of nitrogen dioxide exacerbate the short-term effects of airborne particles? *Occup Environ Med*. 2016;73(11):772–8.
19. Karagulian F, Belis CA, Dora CFC, Prüss-Ustün AM, Bonjour S, Adair-Rohani H, et al. Contributions to cities' ambient particulate matter (PM): a systematic review of local source contributions at global level. *Atmos Environ*. 2015;120:475–83.
20. Katsouyanni K, Touloumi G, Samoli E, Gryparis A, Le Tertre A, Monopolis Y, et al. Confounding and effect modification in the short-term effects of ambient particles on total mortality: results from 29 European cities within the APHEA2 project. *Epidemiology*. 2001;12(5):521–31.
21. Katsouyanni K, Samet JM, Anderson H, Atkinson R, Le Tertre A, Medina S, et al. Air pollution and health: a European and North American approach (APHENA). *Health Eff Inst*. 2009;142:5–90.
22. Kioumourtzoglou MA, Schwartz J, James P, Dominici F, Zanobetti A. PM_{2.5} and mortality in 207 U.S. cities: modification by temperature and city characteristics. *Epidemiology*. 2016;27(2):221–7.
23. Levy JI, Diez D, Dou Y, Barr CD, Dominici F. A meta-analysis and multisite time-series analysis of the differential toxicity of major fine particulate matter constituents. *Am J Epidemiol*. 2012;175(11):1091–9.

24. Lippmann M, Ito K, Hwang JS, Maciejczyk P, Chen LC. Cardiovascular effects of nickel in ambient air. *Environ Health Perspect.* 2006;114(11):1662–9.
25. Ostro B, Feng WY, Broadwin R, Malig B, Green R, Lipsett M. The impact of components of fine particulate matter on cardiovascular mortality in susceptible subpopulations. *Occup Environ Med.* 2008;65(11):750–6.
26. Park SK, O'Neill MS, Vokonas PS, Sparrow D, Schwartz J. Effects of air pollution on heart rate variability: the VA normative aging study. *Environ Health Perspect.* 2005;113(3):304–9.
27. Pun VC, Yu ITS, Qiu H, Ho KF, Sun Z, Louie PK, et al. Short-term associations of cause-specific emergency hospitalizations and particulate matter chemical components in Hong Kong. *Am J Epidemiol.* 2014;179(9):1086–95.
28. Pun VC, Tian L, Yu IT, Kioumourtzoglou MA, Qiu H. Differential distributed lag patterns of source-specific particulate matter on respiratory emergency hospitalizations. *Environ Sci Technol.* 2015;49(6):3830–8.
29. Romieu I, Samet JM, Smith KR, Bruce N. Outdoor air pollution and acute respiratory infections among children in developing countries. *J Occup Environ Med.* 2002;44(7):640–9.
30. Romieu I, Gouveia N, Cifuentes LA, de Leon AP, Junger W, Vera J, et al. Multicity study of air pollution and mortality in Latin America (the ESCALA study). *Health Eff Inst.* 2012;171:5–86.
31. Samoli E, Touloumi G, Zanobetti A, Le Tertre A, Schindler C, Atkinson R, et al. Investigating the dose-response relation between air pollution and total mortality in the APHEA-2 multicity project. *J Occup Environ Med.* 2003;60(12):977–82.
32. Samoli E, Analitis A, Touloumi G, Schwartz J, Anderson HR, Sunyer J, et al. Estimating the exposure-response relationships between particulate matter and mortality within the APHEA multicity project. *Environ Health Perspect.* 2005;113(1):88–95.
33. Sandström T, Frew A, Svartengren M, Viegi G. The need for a focus on air pollution research in the elderly. *Eur Respir J.* 2003;21(40 suppl):92s–5s.
34. Shang Y, Sun Z, Cao J, Wang X, Zhong L, Bi X, et al. Systematic review of Chinese studies of short-term exposure to air pollution and daily mortality. *Environ Int.* 2013;54(2013):100–11.
35. Shi S, Chen C, Zhao B. Modifications of exposure to ambient particulate matter: tackling bias in using ambient concentration as surrogate with particle infiltration factor and ambient exposure factor. *Environ Pollut.* 2017;220(2017):337–47.
36. Simpson R, Williams G, Petroeschevsky A, Best T, Morgan G, Denison L, et al. The short-term effects of air pollution on daily mortality in four Australian cities. *Aust N Z J Public Health.* 2005;29(3):205–12.
37. Stanek LW, Sacks JD, Dutton SJ, Dubois JJB. Attributing health effects to apportioned components and sources of particulate matter: an evaluation of collective results. *Atmos Environ.* 2011;45(32):5655–63.
38. Sun S, Qiu H, Ho KF, Tian L. Chemical components of respirable particulate matter associated with emergency hospital admissions for type 2 diabetes mellitus in Hong Kong. *Environ Int.* 2016;97(2016):93–9.
39. Sunyer J, Schwartz J, Tobías A, Macfarlane D, Garcia J, Antó JM. Patients with chronic obstructive pulmonary disease are at increased risk of death associated with urban particle air pollution: a case-crossover analysis. *Am J Epidemiol.* 2000;151(1):50–6.
40. Tao Y, Huang W, Huang X, Zhong L, Lu SE, Li Y, et al. Estimated acute effects of ambient ozone and nitrogen dioxide on mortality in the Pearl River Delta of southern China. *Environ Health Perspect.* 2012;120(3):393–8.
41. van Donkelaar A, Martin RV, Brauer M, Kahn R, Levy R, Verduzco C, et al. Global estimates of ambient fine particulate matter concentrations from satellite-based aerosol optical depth: development and application. *Environ Health Perspect.* 2010;118(6):847–55.
42. WHO. Guidelines for air quality. Geneva: World Health Organization; 2000.
43. WHO. Air quality guidelines: global update 2005: particulate matter, ozone, nitrogen dioxide, and sulfur dioxide. Geneva: World Health Organization; 2006.
44. WHO. Database on source apportionment studies for particulate matter in the air (PM₁₀ and PM_{2.5}). Available: http://www.who.int/quantifying_ehimpacts/global/source_apport/. n.d. Accessed 6 June 2017.

45. Xie W, Li G, Zhao D, Xie X, Wei Z, Wang W, et al. Relationship between fine particulate air pollution and ischaemic heart disease morbidity and mortality. *Heart*. 2015;101(4):257–63.
46. Yang G, Wang Y, Zeng Y, Gao GF, Liang X, Zhou M, et al. Rapid health transition in China, 1990–2010: findings from the Global Burden of Disease Study 2010. *Lancet*. 2013;381(9882):1987–2015.
47. Zanobetti A, Schwartz J, Gold D. Are there sensitive subgroups for the effects of airborne particles? *Environ Health Perspect*. 2000;108(9):841–5.
48. Zanobetti A, Schwartz J. Cardiovascular damage by airborne particles: are diabetics more susceptible? *Epidemiology*. 2002;13(5):588–92.
49. Zanobetti A, Schwartz J. The effect of particulate air pollution on emergency admissions for myocardial infarction: a multicity case-crossover analysis. *Environ Health Perspect*. 2005;113(8):978–82.
50. Zeka A, Zanobetti A, Schwartz J. Individual-level modifiers of the effects of particulate matter on daily mortality. *Am J Epidemiol*. 2006;163(9):849–59.
51. Zhou J, Ito K, Lall R, Lippmann M, Thurston G. Time-series analysis of mortality effects of fine particulate matter components in Detroit and Seattle. *Environ Health Perspect*. 2011;119(4):461–6.