Satellite-based estimates of long-term exposure to fine particles and association with mortality in elderly Hong Kong residents

Wong, Chit Ming; Lai, Hak Kan; Tsang, Hilda; Thach, Thuan Quoc; Thomas, G Neil; Lam, Kin Bong Hubert; Chan, King Pan; Yang, Lin; Lau, Alexis K H; Ayres, Jon G; Lee, Siu Yin; Man Chan, Wai; Hedley, Anthony J; Lam, Tai Hing

DOI: 10.1289/ehp.1408264

License: Creative Commons: Attribution (CC BY)

Citation for published version (Harvard):
Wong, CM, Lai, HK, Tsang, H, Thach, TQ, Thomas, GN, Lam, KBH, Chan, KP, Yang, L, Lau, AKH, Ayres, JG, Lee, SY, Man Chan, W, Hedley, AJ & Lam, TH 2015, 'Satellite-based estimates of long-term exposure to fine particles and association with mortality in elderly Hong Kong residents', Environmental Health Perspectives, vol. 123, no. 11, pp. 1167-72. https://doi.org/10.1289/ehp.1408264

Link to publication on Research at Birmingham portal
Introduction

Among the World Health Organization criteria pollutants, particulate matter (PM) is often considered the most policy-relevant because it is emitted from burning of fossil fuels in road traffic, shipping, and power generation, and it affects almost all organ systems of the body (WHO 2005). In particular, evidence from animal studies, but not from North America and Europe. Findings typically focused on mortality, and are mostly outcomes through record linkage to the death registry until 31 December 2011. Socioeconomic factors, lifestyle characteristics, and morbidity status were collected by face-to-face interview during enrollment and follow-up visits by registered nurses (Lam et al. 2004). All participants provided informed consent. Ethics approval was obtained from the Ethics Committee of the Faculty of Medicine, The University of Hong Kong.

In Hong Kong there are no zip codes or postal codes. We geocoded addresses for all participants onto an area map with demarcation of areas of District Boards and Tertiary Planning Units (TPU) for which ecological level sociodemographic variables could be obtained from the 2001 Census (Census and Statistics Department, Government of the Hong Kong Special Administrative Region 2002).

Satellite optical depth (AOD) retrieved from remote sensing data of the two NASA Earth Observing System satellites is a measure of transparency for electromagnetic radiation as well as an indication of PM levels in the troposphere (NASA 2013). AOD data are originally retrieved in 10 × 10 km resolution, and they are used to calculate the long-term exposure at each address of the participants.

Background: A limited number of studies on long-term effects of particulate matter with aerodynamic diameter < 2.5 μm (PM2.5) on health suggest it can be an important cause of morbidity and mortality. In Asia where air quality is poor and deteriorating, local data on long-term effects of PM2.5 to support policy on air quality management are scarce.

Objectives: We assessed long-term effects of PM2.5 on the mortality in a single Asian city.

Methods: For 10–13 years, we followed up a cohort of 66,820 participants ≥ 65 years of age who were enrolled and interviewed in all 18 Elderly Health Centres of the Department of Health, Hong Kong, in 1998–2001. Their residential addresses were geocoded into x- and y-coordinates, and their proxy exposures to PM2.5 at their addresses in 1 × 1 km grids were estimated from the U.S. National Aeronautics and Space Administration (NASA) satellite data. We used Cox regression models to calculate hazard ratios (HRs) of mortality associated with PM2.5.

Results: Mortality HRs per 10-μg/m³ increase in PM2.5 were 1.14 (95% CI: 1.07, 1.22) for all natural causes, 1.22 (95% CI: 1.08, 1.39) for cardiovascular causes, 1.42 (95% CI: 1.16, 1.73) for ischemic heart disease, 1.24 (95% CI: 1.00, 1.53) for cerebrovascular disease, and 1.05 (95% CI: 0.90, 1.22) for respiratory causes.

Conclusions: Our methods in using NASA satellite data provide a readily accessible and affordable approach to estimation of a sufficient range of individual PM2.5 exposures in a single city. This approach can expand the capacity to conduct environmental accountability studies in areas with few measurements of fine particles.
but with a 99% cloud-free local environment and adjustment for local meteorological conditions, they can be refined into 1 × 1 km resolution, providing a stronger correlation with PM than the original resolution (Li et al. 2005). We used surface extinction coefficients (SEC) for measuring AOD within 1 km of ground level to predict PM$_{2.5}$ (Division of Environment, The Hong Kong University of Science and Technology 2012). We regressed the annual SEC on annual PM$_{2.5}$ of the four Hong Kong Environmental Protection Department monitors, which measured the pollutant in the period 2000–2011 (see Supplemental Material, Figure S1). For each year, annual PM$_{2.5}$ exposures at geographical locations of individual participants were estimated using the same regression equation, with the annual SEC as the explanatory variable.

Missing SEC data (15.7%), which were mainly due to cloud cover problems (usually occurred from February to May), were filled in by the predicted mean matching method in multiple imputation using the MI procedure in SAS 9.2 (SAS Institute Inc., Cary, NC, USA). Missing data for individual-level covariates were recovered if they were reported in later years; otherwise, the participants were excluded case-wise.

We calculated hazard ratios for deaths categorized according to the International Classification of Diseases, 10th Revision (ICD-10; WHO 2010): all deaths from natural causes (codes A00–R99); cardiovascular diseases (I00–99) with subcategories of ischemic heart disease (IHD; I20–25) and cerebrovascular disease (I60–69); respiratory diseases (J00–47, 80–99) with subcategories of pneumonia (J12–18) and chronic obstructive pulmonary disease (COPD; J40–44, 47); and external causes (S00–T99). Study participants were excluded from analyses if they died within 1 year of enrollment or died from a cause other than the one being modeled.

We categorized the participants into four quartiles (Q1–Q4) of PM$_{2.5}$ exposure and plotted the survival curve of mortality from all natural causes for each group using the Kaplan Meier method. We adopted Cox proportional hazard models for survival, with the time scale setting as duration from year of recruitment to the year of death for the causes being modeled or censored at the year of the follow-up in 2011. The independent variable was exposure to average PM$_{2.5}$ at the baseline. Model covariates included individual-level demographic, socioeconomic, and lifestyle factors obtained from interviews; TPU-level sociodemographic variables obtained from the 2001 Census; and district-level data including the proportion of smokers (> 15 years of age) from 1998 to 2011 (Census and Statistics Department, Government of the Hong Kong Special Administrative Region 2011). Individual-level variables in the final model were age (continuous), sex, body mass index (BMI; < 21.6, 21.6–26.3, > 26.3 kg/m$^2$), smoking (never, ex-smoker, current smoker), physical exercise (days per week), education (< primary, primary, ≥ secondary), and monthly expenses (< 128, 128–384, 384–768, > 768 US dollars).

Figure 1. Spatial distribution of geocoded addresses of participants and boundaries of the 18 districts (n = 60,221). Each district has one Elderly Health Centre to provide health service for persons ≥ 65 years of age who have enrolled voluntarily. Those enrolled in 1998–2001 were recruited to this study, and their residential addresses were geocoded into x- and y-coordinates, which fell into 1 × 1 km grids on the Hong Kong map.

Figure 2. Distribution of PM$_{2.5}$ estimated at geocoded addresses of participants (n = 59,591). The width of each bar (x-axis) represents a class interval for a range of PM$_{2.5}$ exposure proxy of individuals, and the height (y-axis) represents the frequency of addresses in that class interval.
and 409 from external causes (Table 2). Survival was highest among residents in the lowest exposure group (Q1), slightly lower for those in Q2 and Q3, but markedly lower for those in the highest quartile of exposure (see Supplemental Material, Figure S2).

Before adjusting for any covariates, the HR for all-cause mortality in association with a 10-μg/m³ increase in PM2.5 was 1.23 [95% confidence interval (CI): 1.16, 1.31]. After adjusting for individual-level covariates only, the HR was 1.13 (95% CI: 1.06, 1.21), whereas the HR from the fully adjusted model (including TPU and district-level covariates) was 1.14 (95% CI: 1.07, 1.22) (see Supplemental Material, Table S1). A natural spline model of the association between PM2.5 and all-cause mortality (fully adjusted model) confirmed that the association was linear (p-value comparing the fit of the spline model to a linear model = 0.8) (Figure 3).

A 10-μg/m³ increase in PM2.5 was associated with all cardiovascular disease (HR = 1.22; 95% CI: 1.08, 1.39) and with the subcategories IHD (HR = 1.42; 95% CI: 1.16, 1.73) and cerebrovascular disease (HR = 1.24; 95% CI: 1.00, 1.53) (Table 3). Associations with respiratory mortality and COPD were positive but not statistically significant (HR = 1.05; p = 0.030).

Table 1. Descriptive statistics of the participants by quartiles of PM2.5 concentration derived from the NASA satellite data (n = 59,591).

| Variable | Q1 | Q2 | Q3 | Q4 |
|----------|----|----|----|----|
| PM2.5 concentration (μg/m³, mean ± SD) | 32.6 ± 1.03 | 34.6 ± 0.43 | 36.2 ± 0.53 | 38.8 ± 1.34 |
| Participants (n) | 14,907 | 15,167 | 14,884 | 14,833 |
| Incidence rate for all deaths (per 100,000 person-years) | 447 | 461 | 462 | 495 |
| Age (years, mean ± SD) | 71.8 ± 5.4 | 71.9 ± 5.5 | 71.8 ± 5.5 | 72.2 ± 5.5 |
| Sex (%) | | | | |
| Male | 34.7 | 33.7 | 34.9 | 33.6 |
| Female | 65.3 | 66.4 | 65.1 | 66.4 |
| BMI quartile (%) | | | | |
| 2nd–3rd (21.6–26.3 kg/m²) | 50.0 | 50.6 | 51.0 | 51.6 |
| 1st (< 21.6 kg/m²) | 21.6 | 23.5 | 22.3 | 24.0 |
| 4th (> 26.3 kg/m²) | 28.4 | 25.9 | 26.7 | 24.4 |
| Smoking (%) | | | | |
| Never | 71.8 | 71.5 | 70.9 | 70.5 |
| Quit | 18.7 | 19.0 | 19.6 | 19.5 |
| Current | 9.5 | 9.5 | 9.5 | 10.0 |
| Exercise | | | | |
| Days per week (mean ± SD) | 5.6 ± 2.5 | 5.5 ± 2.6 | 5.4 ± 2.7 | 5.4 ± 2.7 |
| Education (%) | | | | |
| ≤ Secondary | 17.6 | 18.4 | 17.8 | 15.3 |
| Primary | 34.6 | 37.4 | 38.3 | 38.3 |
| < Primary | 47.8 | 44.2 | 43.9 | 46.4 |
| Expense per month (US$, %) | | | | |
| < 128 | 17.0 | 15.7 | 14.0 | 12.4 |
| 128–384 | 69.7 | 70.5 | 67.7 | 67.7 |
| ≥ 385 | 13.3 | 13.8 | 18.3 | 19.9 |
| TPU level (mean percent ± SD) | | | | |
| ≥ 65 years of age | 11.4 ± 4.2 | 11.5 ± 3.9 | 12.0 ± 3.9 | 13.6 ± 4.4 |
| > Secondary education | 13.7 ± 8.5 | 13.4 ± 8.1 | 13.3 ± 8.1 | 11.8 ± 7.0 |
| Income ≥ 1,923 US$/month | 63.3 ± 11.0 | 61.1 ± 10.5 | 58.9 ± 11.3 | 55.1 ± 12.0 |
| District level | | | | |
| Smoking rate (mean percent of smokers ± SD) | 11.5 ± 0.4 | 11.6 ± 0.4 | 11.6 ± 0.4 | 11.6 ± 0.3 |

TPU, Tertiary Planning Units.

*All of the 60,221 participants included in the study, 630 had a PM2.5 estimate only at baseline. **PM2.5 concentrations (μg/m³): minimum, 26.4; 25th percentile, 33.8; 50th percentile, 35.3; 75th percentile, 37.2; maximum, 44.6. Thus, Q1: 25th percentile; Q2: 25th–50th percentile; Q3: 50th–75th percentile; Q4: > 75th percentile.

Table 2. Mortality outcomes after 10–13 years of follow up at end of study in 2011.

| ICD-10 codes | Mortality cause | No. of deaths | Percent |
|--------------|----------------|---------------|---------|
| A00–A99      | All natural causes | 16,006 | 97.5 |
| I00–99       | Cardiovascular | 4,656 | 28.4 |
| I20–I25      | IHD | 1,910 | 11.0 |
| I60–69       | Cerebrovascular | 1,621 | 9.9 |
| J00–47, 80–99 | Respiratory | 3,150 | 19.2 |
| J12–18       | Pneumonia | 2,057 | 12.5 |
| J40–44, 47   | COPD | 940 | 5.7 |
| S00–T99      | External causes | 409 | 2.5 |
| All included codes | All causes | 16,415 | |

Environmental Health Perspectives • VOLUME 123 • NUMBER 11 • November 2015 1169
In stratified analyses (Table 4), the association between a 10-μg/m³ increase in PM_{2.5} and mortality was closer to the null (or essentially null) in the ≥ 71 age group compared with the < 71 age group for all-mortality outcomes, with significant differences by age for cardiovascular mortality (HR = 1.15; 95% CI: 1.00, 1.33 vs. HR = 1.42; 95% CI: 1.10, 1.84; interaction p-value 0.04) and IHD (HR = 1.22; 95% CI: 0.96, 1.53 vs. HR = 2.20; 95% CI: 1.47, 3.29; interaction p-value 0.002). Differences were also pronounced for COPD (HR = 1.13; 95% CI: 0.81, 1.57 vs. HR = 2.20; 95% CI: 1.26, 3.86; interaction p-value 0.06). There was little evidence of consistent differences in associations between PM_{2.5} and any of the outcomes according to sex (Table 4) or education (see Supplemental Material, Table S2).

Stratifying for different periods of follow-up (Table 5), the HR for a 10-μg/m³ increase in PM_{2.5} and all natural cause mortality was highest for deaths 2–4 years after baseline (HR = 1.32; 95% CI: 1.11, 1.52) and lower for deaths within 5–8 years (HR = 1.12; 95% CI: 1.00, 1.25) and after ≥ 9 years (HR = 1.09; 95% CI: 0.99, 1.19). Mortality for cardiovascular disease and the subcategories IHD and cerebrovascular disease followed a similar pattern. In mortality for respiratory disease and the subcategories pneumonia and COPD, the HRs were markedly high in the first period and were much lower in the second and third periods.

Estimates from multilevel models that account for clustering at the TPU and district levels were similar to the main analysis (see Supplemental Material, Table S3), as were estimates from models that account for spatial autocorrelation (see Supplemental Material, Table S4).

Discussion

In Hong Kong, a highly dense subtropical city (2001 population of 6.7 million, land area of 1,104 km²), exposure to PM_{2.5} was significantly associated with mortality from natural and cardiovascular causes, and with mortality due to IHD and cerebrovascular disease specifically, in people ≥ 65 years of age. The findings with adjustment for potential confounding factors measured at individual and ecological levels are, in general, robust to different periods of exposure measurement and inclusion and exclusion criteria.

The results of our study from a satellite-based measure of PM_{2.5} provide new evidence on mortality from long-term effects of PM_{2.5}.

We used a novel strategy to estimate exposure of individuals to PM_{2.5} concentrations based on SEC estimated from AOD data within 1 km of ground level captured by NASA satellites. With improved resolution of 1 × 1 km, SEC is correlated (r = 0.6) with the PM_{2.5} concentration measured at the monitors (see Supplemental Material, Figure S1). In one of the few studies using NASA satellite data in Canada, with 10 × 10 km resolution, similar correlations were obtained, and HRs for 10-μg/m³ increase in PM_{2.5} were 1.15 (95% CI: 1.13, 1.16) for all natural cause mortality and 1.31 (95% CI: 1.27, 1.35) for IHD (Crouse et al., 2012), consistent with our findings for the

![Figure 3. Concentration–response relationship between PM_{2.5} exposure and all natural cause mortality. The figure demonstrates the relative risk and confidence intervals of PM_{2.5} concentrations measured in μg/m³. Dashed lines represent 95% CI (p-value 0.772 for log likelihood Chi-square test for linear vs. natural spline model).](image-url)
Long-term PM$_{2.5}$ and mortality in Hong Kong

south China city of Hong Kong. These results demonstrate the feasibility of using satellite data to derive a valid proxy measure for individual long-term exposure to PM$_{2.5}$ in a typical Asian city with high population density in areas with complicated terrain. The resolution of 1 × 1 km would be adequate to define the exposure of older persons who are most likely retired and confined to within 0.5 km of their place of residence (Chau et al. 2002).

In Hong Kong, pollution levels are generally heterogeneous from a public health risk perspective; therefore, while our approach is applicable to an older population with limited mobility, it may be less so for very mobile populations.

The HR estimates of mortality from all natural, cardiovascular, and respiratory causes are consistent with those reported in a recent review (Hoek et al. 2013) and with the combined analysis of 22 European cohorts within the ESCAPE project (Beelen et al. 2014). In an extended analysis of the American Cancer Society study (Krewski et al. 2009), in the nationwide assessment the HR of mortality from all causes was lower than ours; in Los Angeles, California, the HR was similar to ours, but in New York City (New York) the HR of around unity was lower than ours. However, for IHD, the estimates were lower than ours. In two other studies published after the review (Hoek et al. 2013), the estimates for respiratory mortality were different and higher (Beelen et al. 2014; Carey et al. 2013; Katanoda et al. 2011). The heterogeneity in effect sizes for respiratory mortality among studies may be due to the different local polluting sources, particularly from traffic. Because the age range (25–85 years) in these studies is broad, the heterogeneity may also be due to the differences in susceptibility. In a previous study we observed that the relative reduction in respiratory mortality was greater in the 15–24 years group after restrictions on sulfur content of fuel in Hong Kong, suggesting heterogeneity of air pollution effects among age groups (Hedley et al. 2002).

In our stratified analysis, participants recruited at ≥ 71 years of age were potentially at lower risk from air pollution than those recruited at < 71 years, probably due to a healthy survivor effect. Particularly for older people, caution is needed in interpreting mortality effects in long study periods, which may vary because of changes in susceptibility of the survivors in different periods of follow-up. Our results show no sex differences. Indeed, the current evidence for sex differences in susceptibility is weak and inconsistent among studies. For example, the American Cancer Society study (Krewski et al. 2009) and the Netherlands study (Beelen et al. 2008) showed a higher risk in females, but the Harvard Six City study showed a lower risk in females for cardiovascular mortality associated with PM$_{2.5}$ (Pope et al. 2002).

Long-term PM$_{2.5}$ exposure was positively associated with all respiratory and COPD mortality, particularly with strong associations for the latter, during the first 2–4 years of follow-up (data not shown). In contrast, PM$_{2.5}$ exposure was not associated with pneumonia mortality. Although long-term exposure to air pollutants is likely to be a key determinant of a person’s susceptibility to viral and bacterial infections, other factors such as health service accessibility could be equally important (Neupane et al. 2010).

After the first coordinated project among four Asian cities in early 2000 (Wong et al. 2008), several studies of the short-term effects of air pollution in Asia have been reported (Balakrishnan et al. 2013; Bae and Park 2009; Chen et al. 2012; Mahiyuddin et al. 2013; Rajarathnam et al. 2011; Wong et al. 2008) showing that the effect estimates for PM$_{10}$ are, in general, comparable to those from North America and Europe (Samoli et al. 2008). Short-term effects are limited to health outcomes, which are responsive to short periods of exposure (Künzli et al. 2001).

In recent years, China has been undergoing a stage of transition from mainly economic development to include issues in the environment, for which tighter air quality standards are needed (Chen et al. 2011). With air movements over China, air pollution from the highly polluted northern cities could affect the southern cities, and joint efforts among cities are needed to combat the problems of air pollution. Reliable estimates of health effects of air pollution from epidemiologic studies are urgently needed to provide important scientific evidence for environmental accountability as well as for health impact assessment of new air quality objectives. Our study can fill an important gap in missing long-term effect estimates for Asia and the impact on life expectancy and value of life years, which could be gained due to reduction in the pollutant as a result of government intervention (Hedley et al. 2002). These estimates can form the basis of essential public health information, including communication of the risks of air pollution and supporting the benefit–cost ratios of achieving clean air.

There are some limitations of our study. First, the participants were self-selected for enrollment in the care centers; thus our study was likely to have included health-conscious participants who were less susceptible than those of the general population. Second, because the participants were ≥ 65 years of age on recruitment, the study could not assess health problems that affect younger people. Third, occupational exposures and those experienced before the baseline were not measured, which might have led to bias in estimation of the health effects. Last but not least, the data we used for verifying the estimation model for PM$_{2.5}$ were directly measured by four Hong Kong Environmental Protection Department monitors. A better assessment could be carried out by setting up and measuring the whole area of Hong Kong using a sufficient number of monitors.

### Conclusion

In an observation window of 10–13 years for a population-based cohort of ≥ 65 years of age, exposure to PM$_{2.5}$ estimated from NASA satellite data at the area of residence was associated with mortality for all natural and cardiovascular causes. The effect estimates corroborate the existing evidence for a causal relationship between PM$_{2.5}$ and adverse health outcomes, and support formulation and implementation of policies for the mitigation of the pollutant and its disease burden.

### References

Anderson JO, Thundiyil JG, Stolbach A. 2012. Clearing the air: a review of the effects of particulate matter air pollution on human health. J Med Toxicol 8:166–175.

Bae HJ, Park J. 2009. Health benefits of improving air quality in the rapidly aging Korean society. Sci Total Environ 407:5971–5977.

Balakrishnan K, Ganguli B, Ghosh S, Sambandam S, Roy SS, Chatterjee A. 2013. A spatially disaggregated time-series analysis of the short-term effects of particulate matter exposure on mortality in Chennai, India. Air Qual Atmos Health 6:111–121.

Beelen R, Hoek G, van den Brandt PA, Goldbohm RA, Fischer P, Schouten LJ, et al. 2008. Long-term effects of traffic-related air pollution on mortality in a Dutch cohort (NLCS-AIR study). Environ Health Perspect 116:196–202; doi:10.1289/ehp.10767.

Beelen R, Raaschou-Nielsen O, Stafoggia M, Andersen ZJ, Weinmayr G, Hoffmann B, et al. 2014. Environmental Health Perspectives • VOLUME 123 | NUMBER 11 | November 2015 1171

| Table 5. Hazard ratio (95% CI) per 10-μg/m$^3$ increase of PM$_{2.5}$ in stratified analyses by period of follow-up. |
| Cause of death | 2–4 years | 5–8 years | ≥ 9 years |
|-----------------|-----------|-----------|----------|
| All natural causes | 1.32 (1.11, 1.58)** | 1.12 (1.00, 1.25) | 1.09 (0.98, 1.19) |
| Cardiovascular | 1.81 (1.32, 2.50)** | 1.16 (0.92, 1.45) | 1.11 (0.93, 1.32) |
| IHD | 2.36 (1.42, 3.93)** | 1.06 (0.73, 1.54) | 1.43 (1.03, 1.90)** |
| Carevascular | 1.64 (0.94, 2.67) | 1.39 (0.96, 2.00) | 1.07 (0.79, 1.43) |
| Respiratory | 1.72 (1.08, 2.73)* | 1.07 (0.63, 1.71) | 0.93 (0.76, 1.14) |
| Pneumonia | 1.42 (0.72, 2.73) | 1.03 (0.72, 1.47) | 0.86 (0.67, 1.10) |
| COPD | 2.30 (1.15, 4.63)* | 1.16 (0.71, 1.91) | 1.12 (0.76, 1.67) |
| External cause | 0.97 (0.44, 2.17) | 0.80 (0.38, 1.66) | 1.30 (0.63, 2.46) |

*p < 0.05, **p < 0.01, ***p < 0.001.
Effects of long-term exposure to air pollution on natural-cause mortality: an analysis of 22 European cohorts within the multicentre ESCAPE project. Lancet 383:785–795.

Burnett R, Ma R, Jerrett M, Goldberg MS, Catmaks M, Pope CA III, et al. 2001. The spatial association between community air pollution and mortality: a new method of analyzing correlated geographic cohort data. Environ Health Perspect 109:suppl 3:375–380.

Cao J, Yang C, Li J, Chen R, Chen B, Gu D, et al. 2011. Association between long-term exposure to outdoor air pollution and mortality in China: a cohort study. J Hazard Mater 186:1594–1600.

Carey IM, Atkinson RW, Kent AJ, van Staa T, Cook DG, Anderson HR. 2013. Mortality associations with long-term exposure to outdoor air pollution in a national English cohort. Am J Respir Crit Care Med 187:1226–1233.

Census and Statistics Department, Government of the Hong Kong Special Administrative Region. 2002. Hong Kong 2001 Population Census: Basic Tables for Tertiary Planning Units. Available: http://www.statistics.gov.hk/pub/B11200242001XXXXB0400.pdf [accessed 26 March 2015].

Census and Statistics Department, Government of the Hong Kong Special Administrative Region. 2011. Thematic Household Survey. Report No. 48. Available: http://smokefree.hk/UserFiles/resources/Statistics/Thematic_Household_Survey_No48.pdf [accessed 26 March 2015].

Chau CK, Tu EY, Chan DW, Burnett J. 2002. Estimating the total exposure to air pollutants for different population age groups in Hong Kong. Environ Int 27:617–630.

Chen B, Kan H, Chen R, Jiang S, Hong C. 2011. Air pollution and health studies in China—policy implications. J Air Waste Manag Assoc 61:1292–1299.

Chen R, Kan H, Chen B, Huang W, Bai Z, Song G, et al. 2012. Association of particulate air pollution with daily mortality: the China Air Pollution and Health Effects Study. Am J Epidemiol 175:1173–1181.

Crouse DL, Peters PA, van Donkelaar A, Goldberg MS, Villeneuve PJ, Brion O, et al. 2012. Risk of nonaccidental and cardiovascular mortality in relation to long-term exposure to low concentrations of fine particulate matter: a Canadian national-level cohort study. Environ Health Perspect 120:708–714; doi:10.1289/ehp.1104049.

Dong GH, Zhang P, Sun B, Zhang L, Chen X, Ma N, et al. 2012. Long-term exposure to ambient air pollution and respiratory disease mortality in Shenyang, China: a 12-year population-based retrospective cohort study. Respiraion 84:360–368.

Hedley AJ, McGhee SM, Barron B, Chau P, Chau J, Thach TQ, et al. 2008. Air pollution: costs and paths to solutions in Hong Kong—understanding the connections among visibility, air pollution, and health costs in pursuit of accountability, environmental justice, and health protection. J Toxicol Environ Health A 71:544–554.

Hoek G, Krishnan RM, Beelen R, Peters A, Ostro B, Brunekeef B, et al. 2013. Long-term air pollution exposure and cardio-respiratory mortality: a review. Environ Health 12:43; doi:10.1186/1476-074X-12-43.

Institute for the Environment, Hong Kong University of Science and Technology. 2013. Satellite Informatics System for Surface Particulate Matter Distribution. Available: http://envfl.ust.hk/ith-si/ [accessed 26 March 2015].

Katanoda K, Sobue T, Sato H, Tajima K, Suzuki T, Nakatsuka H, et al. 2011. An association between long-term exposure to ambient air pollution and mortality from lung cancer and respiratory diseases in Japan. J Epidemiol 21:132–140.

Krewski D, Jerrett M, Burnett RT, Ma R, Hughes E, Shi Y, et al. 2009. Extended follow-up and spatial analysis of the American Cancer Society study linking particulate air pollution and mortality. Res Rep Health Eff Inst 140:1–14.

Künzli N, Medina S, Kaiser R, Quénel P, Horak F Jr, Studnicka M. 2001. Assessment of deaths attributable to air pollution: should we use risk estimates based on time series or on cohort studies? Am J Epidemiol 152:1050–1055.

Kwok M, Xu X, Kan H, Wang L, Kuang X, Lan Q, et al. 2014. Associations between long-term exposure to ambient air pollution and respiratory disease mortality in Shenyang, China: a 12-year population-based retrospective cohort study. Environ Health Perspect 116:1195–1202; doi:10.1289/ehp.11257.

Lam TH, Li ZB, Ho SY, Chan WM, Ho KS, Li MP, et al. 2004. Smoking and depressive symptoms in Chinese elderly in Hong Kong. Acta Psychiatr Scand 110:195–200.

Li CC, Lau AKH, Mao JT, Chu DA. 2005. Retrieval, validation, and application of the 1-km aerosol optical depth from MODIS measurements over Hong Kong. IEEE Trans Geosci Remote Sens 43:2650–2658.

Ma R, Krewski D, Burnett RT. 2003. Random effects Cox models: a Poisson modelling approach. Biometrika 90:157–169.

Mahiyuddin WRW, Sahani M, Aripin R, Latif MT, Ismail A, et al. 2013. Long-term exposure to air pollution and risk of hospitalization with community-acquired pneumonia in older adults. Am J Respir Crit Care Med 185:47–53.

Natarajan S, Deo S, Jha P, Sheth J, Garg S, et al. 2013. Part 2. Time-series analysis of the American Cancer Society study linking particulate air pollution and mortality. Res Rep Health Eff Inst 140:1–14.

Nikouline VM, Konovalov AN, Gerasimov EA, Loseva ON, et al. 2005. Relative contributions of mortality for different diseases in Japan. J Epidemiol 21:132–140.

Paciorek CJ, Liu Y, HEI Health Review Committee. 2012. Assessment and statistical modeling of the relationship between remotely sensed aerosol optical depth and PM2.5 in the eastern United States. Res Rep Health Eff Inst 187:5–83.

Pope CA III, Burnett RT, Thun MJ, Calle EE, Krewski D, Ito K, et al. 2002. Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. JAMA 287:1132–1141.

R Core Team. 2013. R: A Language and Environment for Statistical Computing. Vienna, Austria:R Foundation for Statistical Computing. Available: http://www.R-project.org [accessed 22 February 2014].

Rajarathnam U, Sehgal M, Nair S, Patnayak RC, Chhabra SK, Kimani, et al. 2011. Part 2. Time-series study on air pollution and mortality in Delhi. Res Rep Health Eff Inst 157:47–74.

Sariola V, Rautava M, Hynynen K, Kivi H, Luostarinen T, Pasanen M, et al. 2005. Long-term exposure to air pollution and risk of mortality in the Finnish male population. J Epidemiol Community Health 59:172–176; doi:10.1136/jech.2003.017576.

Shen Y, et al. 2009. Extended follow-up and spatial analysis of the American Cancer Society study linking particulate air pollution and mortality. Res Rep Health Eff Inst 140:1–14.

Tang M, Liu Y, Wang L, Kuang X, Xu X, Kan H. 2014. Particulate air pollution and mortality in a cohort of Chinese men. Environ Pollut 188:1–6.

Wong et al.