ehp ENVIRONMENTAL HEALTH PERSPECTIVES
http://www.ehponline.org

Satellite-Based Estimates of Long-Term Exposure to Fine Particles and Association with Mortality in Elderly Hong Kong Residents

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

http://dx.doi.org/10.1289/ehp.1408264

Received: 11 February 2014
Accepted: 22 April 2015
Advance Publication: 24 April 2015

This article will be available in its final, 508-conformant form 2–4 months after Advance Publication. If you need assistance accessing this article before then, please contact ehp508@niehs.nih.gov. Our staff will work with you to assess and meet your accessibility needs within 3 working days.

NIH National Institute of Environmental Health Sciences

Environ Health Perspect doi: 10.1289/ehp1408264
Advance Publication: Not Copyedited

Satellite-Based Estimates of Long-Term Exposure to Fine Particles and Association with Mortality in Elderly Hong Kong Residents

Chit Ming Wong,[1] Hak Kan Lai,[1] Hilda Tsang,[1] Thuan Quoc Thach,[1] G. Neil Thomas,[2] Kin Bong Hubert Lam,[3] King Pan Chan,[1] Lin Yang,[1,4] Alexis K.H. Lau,[5] Jon G. Ayres,[2] Siu Yin Lee,[6] Wai Man Chan,[6] Anthony J. Hedley,[1] and Tai Hing Lam[1]

[1]School of Public Health, The University of Hong Kong, Hong Kong; [2]School of Health and Population Sciences, University of Birmingham, Edgbaston, Birmingham, United Kingdom; [3]Nuffield Department of Population Health, University of Oxford, United Kingdom; [4]School of Nursing, The Hong Kong Polytechnic University, Hong Kong; [5]Division of Environment, Hong Kong University of Science and Technology, Hong Kong; [6]Department of Health, the Government of Hong Kong, Hong Kong

Address correspondence to Hak Kan Lai, School of Public Health, 5/F William MW Mong Block, Li Ka Shing Faculty of Medicine, The University of Hong Kong, Hong Kong, China. Telephone: (852)-2831-5057. Fax: (852)-2855 9528. E-mail: tsangh@hku.hk

Running title: Long-term $PM_{2.5}$ and mortality in Hong Kong

Acknowledgments: This work was supported by the Wellcome Trust [#094330/Z10/Z]. We thank the Hong Kong Government departments including the Department of Health (Elderly Health Services) for the cohort data, Census and Statistics Department for mortality data and Environmental Protection Department for air pollution data. We thank Hong Kong University of

Science and Technology for satellite information and Department of Geography of The University of Hong Kong for geocoding the addresses. We thank Dr Edward Hughes of Edward Hughes Consulting and Dr Richard Burnett of Canada Health, Ottawa, Canada for provision of the software and advice in running the spatial random effects survival model.

Completing financial interests: The authors declare they have no actual or potential competing financial interests.

Abstract

Background: A limited number of studies on long-term effects of particulate matter with aerodynamic diameter <2.5 μm ($PM_{2.5}$) 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 $PM_{2.5}$ to support policy on air quality management are scarce.

Objectives: We assessed long-term effects of $PM_{2.5}$ on the mortality in a single Asian city.

Methods: We followed up for 10–13 years a cohort of 66,820 participants aged ≥65 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 geo-coded into x- and y- coordinates and their proxy exposures to $PM_{2.5}$ at their addresses in 1×1 km grids were estimated from the US National Aeronautics and Space Administration (NASA) satellite data. We used Cox regression models to calculate hazard ratios (HRs) of mortality associated with $PM_{2.5}$.

Results: Mortality HRs per 10-$\mu g/m^3$ increase in $PM_{2.5}$ 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 $PM_{2.5}$ exposures in a single city. This approach can expand the capacity to conduct environmental accountability studies in areas with few measurements of fine particles.

Introduction

Among the World Health Organization criteria pollutants, particulate matter (PM) is often considered the most policy relevant one, emitted from burning of fossil fuels in road traffic, shipping and power generation, and affects almost all organ systems of the body (WHO 2005). In particular, evidence from animal studies, but not yet confirmed in humans, shows that particles with aerodynamic diameter less than 2.5 μm ($PM_{2.5}$), can enter the blood stream via the alveolar capillaries, with the potential to cause serious health problems (Anderson et al. 2012).

Studies of long-term effects of $PM_{2.5}$ have typically focused on mortality, and are mostly from North America and Europe. Findings have been inconsistent, with American studies reporting associations with cardiovascular diseases (Crouse et al. 2012; Krewski et al. 2009), and European studies reporting associations with respiratory diseases (Beelen et al. 2013; Carey et al. 2013).

To our knowledge, there are seven published studies on long-term effects of air pollution on mortality in Asia (Cao et al. 2011; Dong et al. 2012; Katanoda et al. 2011; Nishiwaki et al. 2013; Ueda et al. 2012; Zhang et al. 2011; Zhou et al. 2014). Among them only one study assessed the effects of $PM_{2.5}$; but due to a lack of direct measurements $PM_{2.5}$was estimated from PM_{10} by means of a presumed ratio of $PM_{2.5}/PM_{10}$ (Katanoda et al. 2011).

Mitigation of air pollution is urgently needed in Asia where air quality has deteriorated quickly due to rapid industrialization and urbanization (Hedley et al. 2008). In the present study, we estimated $PM_{2.5}$ with a novel method using high resolution satellite data from the National Aeronautics and Space Administration (NASA) (Paciorek and Liu 2012), and assessed long-term

effects on mortality in a large elderly cohort in Hong Kong, the most economically developed city in China.

Methods

We included a total of 66,820 participants aged 65 or older (≥65) who enrolled in each Elderly Health Centre of the Department of Health in all 18 districts of Hong Kong in 1998–2001, and examined mortality outcomes through record linkage to the death registry until the 31st of December, 2011. Socio-economic factors, lifestyle characteristics, and morbidity status were collected by face-to-face interview during enrolment and follow-up visits by registered nurses (Lam et al. 2004). All the 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 geo-coded addresses for all participants onto an area map with demarcation of areas of District Boards, and Tertiary Planning Units (TPU) for which ecological level socio-demographic variables could be obtained from the 2001 Census (Census and Statistics Department 2002).

Aerosol optical depth (AOD) retrieved from remote sensing data of the two Earth Observing System satellites of NASA is a measure of transparency for electro-magnetic radiation as well as an indication of PM levels in the troposphere [National Aeronautics and Space Administration (NASA) 2012]. AOD data are originally retrieved in 10×10 km resolution, but with 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 one (Li et al. 2005). We used surface extinction coefficients (SEC) for measuring AOD within 1 km of ground

level to predict $PM_{2.5}$ [Hong Kong University of Science and Technology (HKUST) 2012]. We regressed annual SEC on annual $PM_{2.5}$ of the four Hong Kong Environmental Protection Department (EPD) 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 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. Missing data for individual-level covariates were recovered if they were reported in later years, or otherwise the participants were excluded case-wise.

We calculated hazard ratios for all deaths from natural causes [International Classification of Diseases version 10 (ICD10) (WHO 2010): 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]; external causes [S00–T99]. Study participants were excluded from analyses if they died within one year of enrollment, or died from a cause other than the one being modeled.

We categorized the participants into four quartiles namely Q1–Q4 of $PM_{2.5}$ exposure and plotted the survival curve of mortality from all natural causes for each group using 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 socio-demographic variables obtained from the 2001 Census; and district-level data including the proportion of smokers (over 15 years of age) from 1998 to 2011 (Census and Statistics Department 2011). Individual-level variables in the final model were age (continuous), sex, BMI (<21.6, 21.6–26.3, >26.3), smoking (never, ex-smoker, current smoker), physical exercise (days per week), education (< primary, primary, secondary and above), and monthly expenses (<128, 128–384, >384 US$). In addition we adjusted for the TPU-level proportion of the population ≥65 years of age, and the proportion with tertiary education, and the average monthly income in each TPU. Finally, we also adjusted for the proportion of smokers in each district.

From each model, the mortality hazard ratio (HR) associated with a 10 $\mu g/m^3$ (defined as a unit) increase in $PM_{2.5}$ concentration was estimated with adjustment for confounding variables. We plotted the relationship between $PM_{2.5}$ and all deaths from natural causes using the natural spline smoother in COXPH of R program version 3.0.1 (R Development Core Team, Vienna, Austria) with two degrees of freedom.

We performed sensitivity analyses with exposures to average $PM_{2.5}$ yearly; inclusion of participants who died during the first year after enrollment; exclusion of participants who died in the first 1–3 years. We also performed separate analyses stratified by age (<71 or ≥71 years, based on the median age of 70), sex, and education (< primary, or primary or above). In addition,

we stratified according to the length of follow-up (2 to <5, 5 to <9, or ≥9 years after baseline). We also used models with random effects set at the intercepts to take account of possible intra-district correlations and with an extension to take account of spatial autocorrelations due to adjacency or distance-decay between TPUs (Burnett et al. 2001; Ma et al. 2003).

All statistical analyses were performed using Stata10.0 (STATA Corporation, College Station, TX, USA) and SAS 9.2 (SAS Institute, Inc., Cary, North Carolina).

Results

A total of 66,820 participants were recruited and 64,888 addresses geo-coded (97%); they were distributed over the 18 districts of Hong Kong (Figure 1), accounting for 6.5% to 17.2% of the ≥65 population. After the exclusion of missing data due to missing individual-level covariates (0.2%), problems in geo-coding (8.1%) or problems in satellite data (1.5%), a final sample of 60,221 participants (90.1%) were included for $PM_{2.5}$ estimation and analysis.

During the study period, $PM_{2.5}$ was in general increasing but there were ups and downs in some years. Such patterns were similar across geographic areas (data not shown). At the baseline, the estimated concentrations of $PM_{2.5}$ approximated to a Normal distribution (Figure 2), approximately 70% of the participants were 65–74 years of age, and 65% were female. Participants in the highest exposure category (Q4), tended to be older and were more likely to be smokers, to habitually exercise less, to be less likely to have secondary or higher education and more likely to have higher levels of personal expenditure, compared with those with lower exposure (Q1–Q3) (Table 1). TPU with higher concentration of $PM_{2.5}$ tended to be associated with lower household income (Spearman correlation: -0.155; p=0.030) (data not shown).

After 10–13 years follow-up before excluding the 204 deaths in the first year, there were 16,006 all deaths from natural causes 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-$\mu g/m^3$ increase in $PM_{2.5}$ was 1.23 (95% CI: 1.16, 1.31). After adjusting for individual-level covariates only, the HR was 1.13 (95% CI: 1.06, 1.21), while 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 $PM_{2.5}$ 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-$\mu g/m^3$ increase in $PM_{2.5}$ 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; 95% CI: 0.90, 1.22 and HR = 1.30; 95% CI: 0.98, 1.74; respectively).

In sensitivity analyses for all natural causes and for overall and subcategories of cardiovascular mortality (Table 3), the estimates and levels of statistical significance remained similar, except for cerebrovascular mortality where the estimates became non-significant when yearly average concentration were used as the exposure or when deaths within 1–3 years were excluded. The

comparisons between the main and sensitivity analyses for respiratory mortality showed similar levels of estimates and significance.

In stratified analyses (Table 4), the association between a 10-$\mu g/m^3$ 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 versus 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 versus 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 versus 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-$\mu g/m^3$ 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, 1.56), and lower for deaths 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 and subcategories IHD and cerebrovascular disease followed a similar pattern. In mortality for respiratory and 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 multi-level 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 km^2), 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 aged ≥65 years. 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/m^3 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 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 so 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 and with the combined analysis of 22 European cohorts within the ESCAPE project (Hoek et al. 2013). In an extended analysis of the American Cancer Society study (Krewski et al. 2009), in the nationwide assessment the HR of mortality from all-cause was lower than ours, but in Los Angeles the HR was similar to ours, while in New York city the HR of around unity was lower than ours. However for IHD the estimates were lower than ours. However, in two other studies published after the review, the estimates for respiratory mortality were different and higher (Beelen et al. 2013; 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. But as the age range (25 to 85 years) is broad in these studies 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 15–64 than the ≥65 age group after restrictions on sulphur 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 the age of ≥71 years are potentially at lower risk from air pollution than at <71, 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 and the Netherlands study 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; Beelen et al. 2008; Krewski et al. 2009).

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. In contrast, $PM_{2.5}$ exposure was not associated with pneumonia mortality. While 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 short-term effects of air pollution studies 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 (Künzliet 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 enrolment in the care centres, and were therefore likely to have included health conscious participants who were less susceptible than those of the general population. Second, as the participants were 65 or older on recruitment, the study could not assess health problems which 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 at four EPD monitors. A better assessment should be carried out by setting up and measuring with sufficient number of monitors around the whole area of Hong Kong.

Conclusion

In an observation window of 10–13 years for a population based cohort of 65 or older, exposure to $PM_{2.5}$ estimated from NASA satellite data at 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 adverse health outcomes and $PM_{2.5}$ 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, Sen Roy S, Chatterjee A. 2013. A spatially disaggregated time-series analysis of the short-term effects of particulate matter exposure on mortality in Chennai, India. Air Quality, Atmosphere & 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 Perspect116:196–202.

Beelen R, Raaschou-Nielsen O, Stafoggia M, Andersen ZJ, Weinmayr G, Hoffmann B, et al. 2013. Effects of long-term exposure to air pollution on natural-cause mortality: an analysis of 22 European cohorts within the multicentre ESCAPE project. Lancet pii: S0140–6736(13)62158–3.

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

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.

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.

Census and Statistics Department. 2002. Hong Kong 2001 Population Census - Basic Tables for Tertiary Planning Units. Avialable: http://www.censtatd.gov.hk/major_projects/2001_population_census/basic_tables_for_tertiary_planning_units/index.jsp [accessed 26 March 2015].

Census and Statistics Department. 2011. Thematic household survey Report No. 48. Avaliable: 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.

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. Respiration 84:360–368.

Hedley AJ, McGhee SM, Barron B, Chau P, Chau J, Thach TQ,et al. 2008. Air pollution: costs and paths to a solution 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.

Hedley AJ, Wong CM, Thach TQ, Ma S, Lam TH, Anderson HR. 2002. Cardiorespiratory and all-cause mortality after restrictions on sulphur content of fuel in Hong Kong: an intervention study. Lancet 360:1646–1652.

HKUST (Hong Kong University of Science and Technology). 2013. Satellite Informatics System for Surface Particulate Matter Distribution. Avaliable: http://envf.ust.hk/itf-si/ [Accessed 26 March 2015].

Hoek G, Krishnan RM, Beelen R, Peters A, Ostro B, Brunekreef B, et al. 2013. Long-term airpollution exposure and cardio-respiratory mortality: a review. Environ Health 12:43.

Katanoda K, Sobue T, Satoh 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 Epidemiol21:132–143.

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 153:1050–1055.

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 140:5–114; discussion 115–136.

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.

Lam TH, Li ZB, Ho SY,Chan WM, Ho KS, Tham MK, et al. 2007. Smoking, quitting and mortality in an elderly cohort of 56,000 Hong Kong Chinese. Tob Control 16:182–189.

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 T Geosci Remote 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, Thach TQ, Wong CM. 2013. Short-term effects of daily air pollution on mortality. Atmospheric Environment65:69-79.

Miller KA, Siscovick DS, Sheppard L, Shepherd K, Sullivan JH, Anderson GL, et al. 2007. Long-term exposure to air pollution and incidence of cardiovascular events in women. N Engl J Med 356:447–458.

NASA (National Aeronautics and Space Administration). 2013. Earth Observing System. Available: http://eospso.nasa.gov/ [accessed 26 March 2015].

Neupane B, Jerrett M, Burnett RT, Marrie T, Arain A, Loeb M.2010. Long-term exposure to ambient air pollution and risk of hospitalization with community-acquired pneumonia in older adults. Am J Respir Crit Care Med 181:47–53.

Nishiwaki Y, Michikawa T, Takebayashi T, Nitta H, Iso H, Inoue M,et al. 2013. Long-term exposure to particulate matter in relation to mortality and incidence of cardiovascular disease: the JPHC Study. J Atheroscler Thromb 20:296–309.

Ou CQ, Hedley AJ, Chung RY, Thach TQ, Chau YK, Chan KP, et al. 2008. Socioeconomic disparities in air pollution-associated mortality. Environ Res 107:237–244.

Paciorek CJ, Liu Y. 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 167:5–83; discussion 85–91.

Pope CA 3rd, 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.

Puett RC, Schwartz J, Hart JE, Yanosky JD, Speizer FE, Suh H,et al. 2008. Chronic particulate exposure, mortality, and coronary heart disease in the nurses' health study. Am J Epidemiol 168:1161–1168.

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

Samoli E, Peng R, Ramsay T, Pipikou M, Touloumi G, Dominici F, et al. 2008. Acute effects of ambient particulate matter on mortality in Europe and North America: results from the APHENA study. Environ Health Perspect 116:1480-1486.

Ueda K, Nagasawa SY, Nitta H, Miura K, Ueshima H. 2012. Exposure to particulate matter and long-term risk of cardiovascular mortality in Japan: NIPPON DATA80. J Atheroscler Thromb 19:246–254.

WHO (World Health Organization). 2005. Health effects of transport-related air pollution. KrzyzanowskiM, Kuna-Dibbert B, Schneider J (Eds.). Copenhagen, WHO Regional Office for Europe. Available: http://www.euro.who.int/__data/assets/pdf_file/0006/74715/E86650.pdf [accessed 26 March 2015].

WHO (World Health Organization). 2010. International Statistical Classification of Diseases and Related Health Problems 10th Revision. WHO Regional Office for Europe. Available: http://apps.who.int/classifications/icd10/browse/2010/en [accessed 26 March 2015].

Wong CM, Vichit-Vadakan N, Kan H, Qian Z. 2008. Public Health and Air Pollution in Asia (PAPA): A multi-city study for short-term effects of air pollution on mortality. Environmental Health Perspectives116:1195-1202.

Zhang P, Dong G, Sun B, Zhang LW, Chen X, Ma NN, et al. 2011. Long-term exposure to ambient air pollution and mortality due to cardiovascular disease and cerebrovascular disease in Shenyang, China. PLoS One 6:e20827.

Zhou 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. 186:1-6.

Table 1. Descriptive statistics of the participants by four categories by quartiles of $PM_{2.5}$ concentration derived from the NASA satellite data (n=59,591[a]).

Variables	**$PM_{2.5}$ Category[b]**			
	Q1	**Q2**	**Q3**	**Q4**
$PM_{2.5}$ concentration (μg/m^3, mean±SD)	32.6±1.03	34.6±0.43	36.2±0.53	38.8±1.34
Subject [n]	14 907	15167	14 684	14 833
Incidence rate for all deaths (per 100,000 person-year)	447	461	462	495
Individual level:				
Age in year (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 quartiles: 2nd – 3rd [21.6-26.3] (%)	50.0	50.6	51.0	51.6
1st [<21.6]	21.6	23.5	22.3	24.0
4th [>26.3]	28.4	25.9	26.7	24.4
Smoking: Never (%)	71.8	71.5	70.9	70.5
Quitted	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 or above (%)	17.6	18.4	17.8	15.3
Primary	34.6	37.4	38.3	38.3
Below primary	47.8	44.2	43.9	46.4
Expense per month in 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[c]:				
Age ≥65 (mean%±SD)	11.4±4.2	11.5±3.8	12.0±3.9	13.6±4.4
Tertiary education (mean%±SD)	13.7±8.5	13.4±8.1	13.3±8.1	11.8±7.0
Income ≥ US$1,923/m (mean%±SD)	63.3±11.0	61.1±10.5	58.9±11.3	55.1±12.0
District level:				
Smoking rate (mean% of smokers±SD)	11.5±0.4	11.6±0.4	11.6±0.4	11.6±0.3

[a]The number was smaller than the total number of participants included in the study of 60,221 by 630 which were participants who did not have $PM_{2.5}$ estimate only at baseline. [b]$PM_{2.5}$ concentration (μg/m^3): Minimum 26.4, 1st quartile 33.8, 2nd quartile 35.3, 3rd quartile 37.2, maximum 44.6; Q1: <1st quartile; Q2: 1st–2nd quartile; Q3: 2nd–3rd quartile; Q4: >3rd quartile. [c]TPU: Tertiary Planning Units.

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

ICD10	Mortality causes	No. of deaths	%
A00-R99	All natural causes	16,006	97.5
I00-99	Cardiovascular	4,656	28.4
I20-I25	- Ischemic heart disease	1,810	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
	Total no. of deaths	16,415	

Table 3. Hazard ratio (95%CI) per 10 μg/m^3 increase of $PM_{2.5}$ in main analysis for average exposure at baseline period and sensitivity analyses for exposure to average $PM_{2.5}$ yearly and for different inclusion and exclusion criteria.

Cause of death	Main analysis[a] - baseline Exposure (n=59,362)	Yearly exposure (n=59,421)	Including deaths within 1 year - baseline exposure (n=59,566)	Excluding deaths within 3 years - baseline exposure (n=57,405)
All natural causes	1.14 (1.07, 1.22)***	1.11 (1.03, 1.20)**	1.14 (1.07, 1.22)***	1.15 (1.08, 1.24)***
Cardiovascular	1.22 (1.08, 1.39)**	1.15 (1.00, 1.33)	1.23 (1.08, 1.39)**	1.19 (1.04, 1.36)*
- IHD	1.42 (1.16, 1.73)***	1.40 (1.12, 1.76)**	1.43 (1.17, 1.74)***	1.40 (1.13, 1.73)**
- Cerebrovascular	1.24 (1.00, 1.53)*	1.15 (0.91, 1.46)	1.24 (1.01, 1.53)*	1.18 (0.94, 1.47)
Respiratory	1.05 (0.90, 1.22)	1.06 (0.89, 1.26)	1.02 (0.87, 1.18)	1.01 (0.86, 1.19)
- Pneumonia	0.94 (0.77, 1.14)	1.02 (0.82, 1.26)	0.92 (0.76, 1.11)	0.91 (0.75, 1.11)
- COPD	1.30 (0.98, 1.74)	1.26 (0.92, 1.73)	1.32 (1.00, 1.74)	1.28 (0.95, 1.73)
External causes	1.04 (0.69, 1.58)	1.09 (0.69, 1.75)	1.03 (0.69, 1.56)	1.10 (0.71, 1.72)

*p<0.05,**p<0.01,***p<0.001

[a]Death within one year excluded.

Table 4. Hazard ratio (95%CI) per 10 μg/m^3 increase of $PM_{2.5}$ in stratified analyses by age or by sex with exposure at baseline (deaths within the first year excluded).

Cause of death	Age<71	Age≥71	Interaction[a]	Male	Female	Interaction[a]
All natural causes	1.23 (1.08, 1.40)**	1.11 (1.03, 1.19)**	0.07	1.17 (1.06, 1.29)**	1.13 (1.03, 1.23)**	0.7
Cardiovascular	1.42 (1.10, 1.84)**	1.15 (1.00, 1.33)	0.04	1.28 (1.05, 1.57)*	1.19 (1.01, 1.40)*	0.5
- IHD	2.20 (1.47, 3.29)***	1.22 (0.96, 1.53)	0.002	1.39 (1.02, 1.90)*	1.44 (1.10, 1.89)**	0.8
- Cerebrovascular	1.21 (0.80, 1.84)	1.24 (0.97, 1.59)	0.8	1.32 (0.93, 1.87)	1.21 (0.92, 1.58)	0.9
Respiratory	1.37 (0.96, 1.95)	0.98 (0.83, 1.16)	0.1	1.11 (0.90, 1.38)	0.97 (0.78, 1.22)	0.5
- Pneumonia	1.03 (0.63, 1.69)	0.91 (0.74, 1.13)	0.6	1.00 (0.75, 1.32)	0.88 (0.68, 1.15)	0.7
- COPD	2.20 (1.26, 3.86)	1.13 (0.81, 1.57)	0.06	1.21 (0.85, 1.72)	1.53 (0.93, 2.52)	0.6
External cause	0.84 (0.41, 1.73)	1.14 (0.68, 1.90)	0.8	1.03 (0.60, 1.77)	1.07 (0.57, 1.99)	0.9

*p<0.05,**p<0.01,***p<0.001. [a]P-value for the interaction term in the model for the combined data set.

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, 1.56)**	1.12 (1.00, 1.25)	1.09 (0.99, 1.19)
Cardiovascular	1.81 (1.32, 2.50)***	1.16 (0.92, 1.45)	1.11 (0.93, 1.32)
- Ischemic heart disease	2.36 (1.42, 3.93)**	1.06 (0.73, 1.54)	1.43 (1.09, 1.89)*
- Cerebrovascular	1.64 (0.94, 2.87)	1.39 (0.96, 2.00)	1.07 (0.79, 1.43)
Respiratory	1.72 (1.09, 2.73)*	1.07 (0.81, 1.41)	0.93 (0.76, 1.14)
- Pneumonia	1.42 (0.72, 2.79)	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 (0.69, 2.46)

*p<0.05,**p<0.01,***p<0.001.

Figure Legends

Figure 1. Spatial distribution of geo-coded addresses of participants with boundaries of the 18 districts (n=60,221). In each district there is one Elderly Health Centre to provide health service for persons 65 years of age or older who have enrolled voluntarily. Those enrolled in 1998–2001 were recruited to this study and their residential addresses were geo-coded into x- and y-coordinates which fell into 1x1 km grids in the Hong Kong map.

Figure 2. Distribution of $PM_{2.5}$ estimated at geo-coded addresses of participants (n=59,591). The width of each vertical rectangle in the x-axis represents a class interval for a range of $PM_{2.5}$ exposure proxy of individuals and the height in the y-axis represents the frequency of addresses in that class interval.

Figure 3. Concentration-response relationship between $PM_{2.5}$ exposure and all natural cause mortality. The figure demonstrates the relative risk (fully adjusted model) of all natural cause mortality in relation to long-term exposure to $PM_{2.5}$. The tick marks on the x-axis represent the position of $PM_{2.5}$ concentrations measured in $\mu g/m^3$. Dashed lines represent 95%CI (*p*-value=0.772 for log likelihood Chi-square test for linear vs nature spline model).

Figure 1.

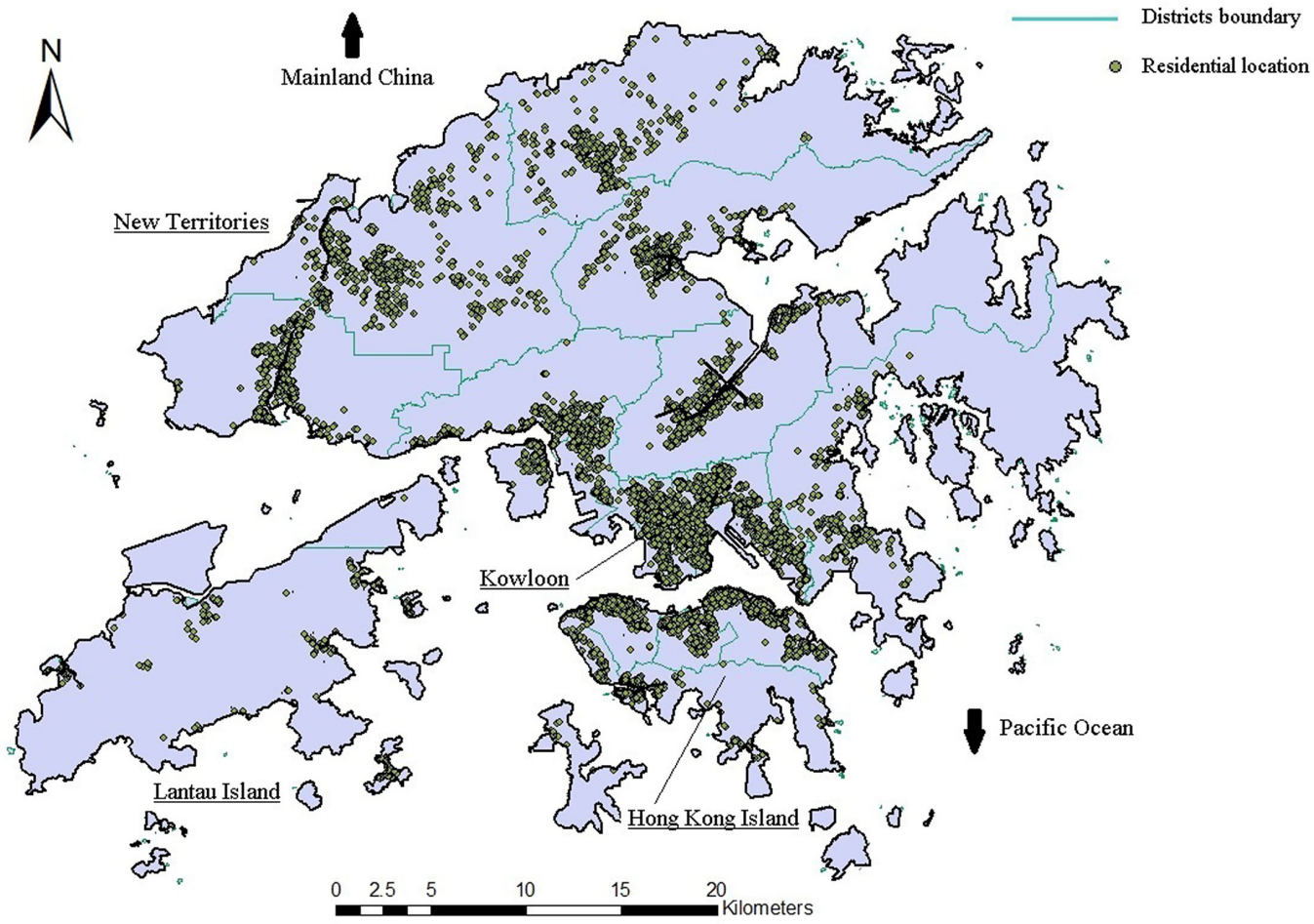
N
Mainland China
Districts boundary
Residential location
New Territories
Kowloon
Pacific Ocean
Lantau Island
Hong Kong Island
0 2.5 5 10 15 20
Kilometers

Figure 2.

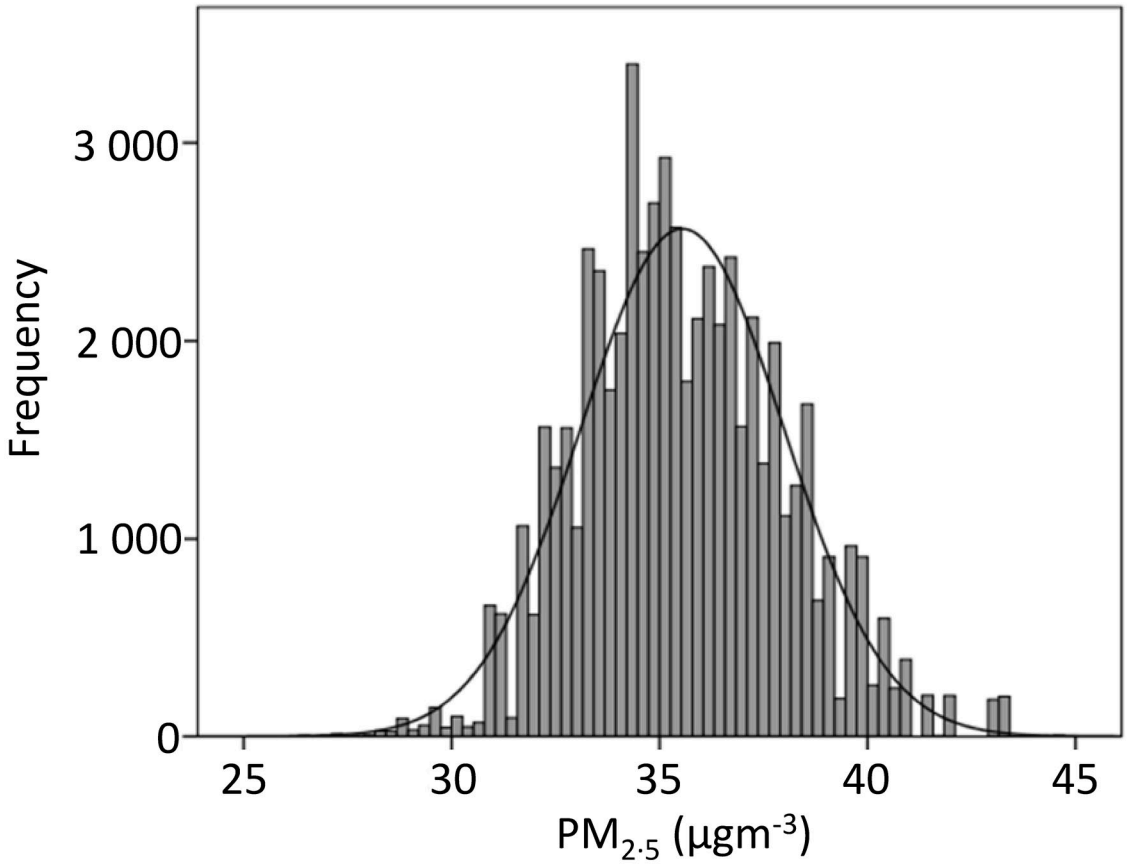

Figure 3.