# All-cause mortality risk associated with long-term exposure to ambient $PM_{2.5}$ in China: a cohort study

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## Summary

**Background** Evidence from cohort studies in North America and Europe indicates that long-term exposure to fine particulate matter ($PM_{2.5}$) is associated with an increased mortality risk. However, this association has rarely been quantified at higher ambient concentrations. We estimated the hazard ratio (HR) for all-cause mortality from long-term exposure to $PM_{2.5}$ in a well established Chinese cohort of older adults.

**Methods** The Chinese Longitudinal Healthy Longevity Survey (CLHLS) is a prospective cohort study of men and women aged 65 years and older enrolled in 2008 and followed up through 2014 for mortality events. We studied individuals for whom residential locations were available in 2008 for linkage to 1 km grids of $PM_{2.5}$ concentrations, derived from satellite remote sensing. Cox proportional hazards models were used to estimate the effect of long-term exposure to $PM_{2.5}$ on all-cause mortality, controlling for age, sex, smoking status, drinking status, physical activity, body-mass index, household income, marital status, and education. We then used our results to estimate premature mortality related to $PM_{2.5}$ exposure in the population aged 65 years and older in China in 2010.

**Findings** 13 344 individuals in the CLHLS cohort had data for all timepoints, yielding follow-up data for 49 440 person-years. In a 3-year window, these individuals were exposed to a median $PM_{2.5}$ concentration of 50·7 μg/m³ (range 6·7–113·3). The overall HR for a 10 μg/m³ increase in this value was 1·08 (95% CI 1·06–1·09). In stratified analyses, HRs were higher in rural than in urban locations, in southern versus northern regions, and with exposure to lower versus higher $PM_{2.5}$ concentrations. Based on the overall HR, we estimated that 1 765 820 people aged 65 years and older in China in 2010 had premature mortality related to $PM_{2.5}$ exposure.

**Interpretation** Long-term exposure to $PM_{2.5}$ is associated with an increased risk of all-cause mortality among adults aged 65 years and older in China, but the magnitude of the risk declines as the concentration of $PM_{2.5}$ increases.

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## Introduction

Previous studies[1–8] in North America and Europe have provided compelling evidence of an association between increased mortality risk and long-term exposure to fine particulate matter ($PM_{2.5}$) at reasonably low concentrations (<30 μg/m³). However, only one cohort study[9] has investigated an association between long-term mortality and exposure to high concentrations of $PM_{2.5}$, such as those found in low-income and middle-income countries (LMICs), and this study was only in men. Global burden estimates for outdoor air pollution are dominated by $PM_{2.5}$-related deaths in China and India.[10] However, estimates of the mortality burden attributable to ambient air pollution in LMICs have been based on extrapolations from studies in high-income countries with lower ambient concentrations of $PM_{2.5}$, as well as effect estimates for high concentrations derived from studies of second-hand and active smoking.[11] Therefore, there is an urgent need for estimates of mortality from cohort studies investigating long-term exposure to high $PM_{2.5}$ concentrations in LMICs to support policy making and health promotion in these countries.

The gap in epidemiological information about the effects of $PM_{2.5}$ on mortality in LMICs is partly due to the absence of widespread ambient air monitoring data. However, this gap is being filled in China, where nationwide monitoring of $PM_{2.5}$ in cities began in 2013. According to data from state air pollution control stations in more than 300 cities in China, annual mean $PM_{2.5}$ concentrations for 2016 ranged from 11 μg/m³ to 157 μg/m³.[12] These data, combined with satellite-based remote sensing, make it possible to estimate long-term ambient concentrations of $PM_{2.5}$ to support national-level studies. Of particular interest is the shape of the concentration–response function (CRF) at concentrations higher than those observed in previous studies (ie, ≥30 μg/m³).

## Research in context

### Evidence before this study

We searched PubMed, Web of Science, and Embase up to June 30, 2018, without language restrictions. We used the search terms "fine particulate matter", "$PM_{2.5}$", "mortality", "long-term", and "chronic". Searches were supplemented by hand-searching the reference lists of any identified systematic reviews. Previous studies have reported that long-term exposure to $PM_{2.5}$ increases risk of all-cause mortality, with most evidence coming from cohort studies in North America and Europe. However, little evidence exists of the association between long-term exposure to $PM_{2.5}$ and mortality at the high concentrations of $PM_{2.5}$ found in low-income and middle-income countries (LMICs), which can reach 100 μg/m³. Only one cohort study identified an association between long-term mortality and $PM_{2.5}$ in China, and this study was in men only. Global burden estimates for outdoor air pollution are dominated by $PM_{2.5}$-related deaths in China and India. However, estimates of the mortality burden attributable to ambient air pollution in LMICs have relied on extrapolations from studies with lower ambient $PM_{2.5}$ concentrations, combined with high-concentration effect estimates derived from studies of second-hand and active smoking, which introduces uncertainty in the estimation of the mortality burden.

### Added value of this study

To our knowledge, this study is the first to show that long-term exposure to $PM_{2.5}$ is associated with an increased risk of all-cause mortality over a broad range of $PM_{2.5}$ concentrations (7–113 μg/m³) in a community-based, nationwide, prospective cohort study of men and women aged 65 years and older in China. We also addressed the question of the shape of the concentration–response function at concentrations higher than those observed in most previous studies.

### Implications of all the available evidence

Our data provide confirmatory evidence that mortality risk is non-linear, with stronger effects occurring at lower concentrations of $PM_{2.5}$. These findings support the integrated exposure–response concept, which assumes that effects level off at higher $PM_{2.5}$ concentrations. Our data might inform future studies that investigate concentration–response relationships and estimate disease burden from exposure to $PM_{2.5}$ in LMICs such as China.

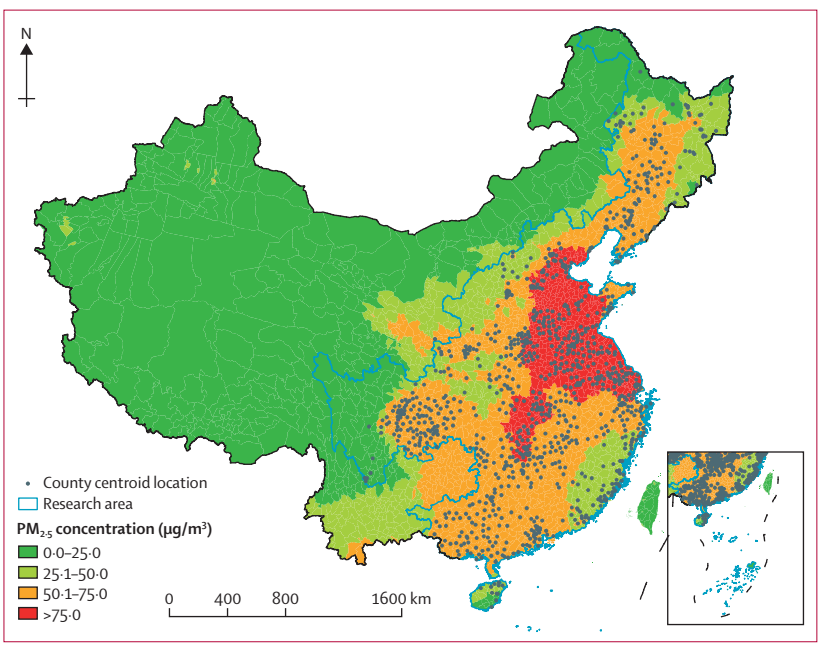


*Figure 1:* **Map of $PM_{2.5}$ concentration in China during the study period (2008–14)**
Islands in the South China Sea are shown in the box.

See Online for appendix

Here, we investigate the associations between long-term exposure to $PM_{2.5}$ and all-cause mortality in China using a cohort of individuals aged 65 years and older from the Chinese Longitudinal Healthy Longevity Survey (CLHLS). Our objectives were to estimate the hazard ratio (HR) for all-cause mortality from long-term $PM_{2.5}$ exposure; investigate evidence of non-linearity in the CRF; compare estimated mortality risk across age, sex, smoking status, and drinking status subgroups; and estimate the burden of $PM_{2.5}$-related mortality in the population aged 65 years and older in China in 2010.

## Methods

### Study population

For this cohort study, individuals were selected from the fifth wave of the CLHLS in 2008 (at baseline) because of the inability to obtain residential address information before 2008. The CLHLS study is a prospective, longitudinal, population-based study, and the study design has been described in detail elsewhere.[13] We included individuals aged 65 years and older who had available residential addresses. Recruitment was done with the goal of having roughly similar numbers of men and women at each age from 65 years onwards.

For the baseline survey, we randomly selected half of the counties or cities in 22 of 31 provinces in China; this area had a total population of 985 million in 2008, representing roughly 85% of the Chinese population (figure 1). Nine provinces were not investigated because of their low population densities in 2008. Population densities in China according to the 2010 census[14] are shown in the appendix. Participants were followed up in 2011 and in 2014 to assess vital status. Follow-up continued until the date of death (ascertained from family members) or the end of the study (Dec 1, 2014).

All participants or their legal representatives signed written informed consent forms to participate in the baseline and follow-up surveys. Informed consent was obtained from study participants before completing the study questionnaire. The study was approved by the

Biomedical Ethics Committee of Peking University, Beijing, China (IRB00001052-13074).

### Exposure

Estimates of ground-level concentrations of $PM_{2.5}$ were obtained from the Atmospheric Composition Analysis Group.[15] Ground-level $PM_{2.5}$ concentrations were estimated by combining aerosol optical depth retrievals from the National Aeronautics and Space Administration's Moderate Resolution Imaging Spectroradiometer, Multi-angle Imaging SpectroRadiometer, and Sea-viewing Wide field-of-view Sensor satellite instruments; vertical profiles derived from the GEOS-Chem chemical transport model; and calibration to ground-based observations of $PM_{2.5}$ using geographically weighted regression, as described in detail elsewhere.[15–17] The resultant $PM_{2.5}$ concentration estimates were highly consistent ($R^2$=0·81) with out-of-sample cross-validated $PM_{2.5}$ concentrations from monitors.[16] We used full-composition annual $PM_{2.5}$ estimates from 1998 to 2014, covering all of China at a 1 km×1 km spatial resolution. This dataset is the longest and highest resolution exposure dataset available in China. We assessed the reliability of this dataset by comparing it with another exposure dataset in China,[18] and found them to be highly correlated ($R^2$=0·79). Relevant information related to our exposure data can be found in the appendix.

Residential locations for each participant during follow-up were obtained from the CLHLS database using face-to-face household surveys in 2008, and latitudes and longitudes were assigned with R version 3.3.1. The accuracy of the locations reached to the rooftops of residences. By linking residential locations to the nearest $PM_{2.5}$ grids, we could match 1 km $PM_{2.5}$ grid cells to each participant. We considered multiple windows of exposure, including average exposure in the 1–10 years before death or end of follow-up. The pairwise correlations for the ten time periods ranged from 0·96 to 0·99. Exposure to $PM_{2.5}$ was averaged for the 3 years before death or the end of the study (for participants who did not die) because this time window had the strongest association with mortality (appendix).

### Exposure–response relationship

We used Cox proportional hazards models to quantify the association between $PM_{2.5}$ exposure and all-cause mortality. Person-years (calculated as person-days divided by 365) of follow-up were calculated from study enrolment to the end of follow-up (Dec 1, 2014) or date of death. HRs and 95% CIs were calculated for a 10 μg/m³ increase in $PM_{2.5}$ exposure.

In the main analysis (model 1), estimates were adjusted for age (strata variable by 1 year), sex, smoking status, drinking status, physical activity, body-mass index, household income, marital status, and education. Education was categorised dichotomously in the model and coded as yes if the participant reported at least 1 year of any formal education in response to the question "How many years of formal education have you received?" Marital status was categorised as currently married if the participant was currently married and not married if the participant was divorced, widowed, or had never been married. Physical activity was defined as a yes or no answer to the question "Do you often engage in physical activities, including walking, playing ball, running, or Qigong?"

We did several sensitivity analyses (models 2–10), in which we added various indicator variables to model 1, including setting (urban *vs* rural; model 2), region (eastern *vs* southern *vs* central *vs* northern *vs* northwestern *vs* southwestern *vs* northeastern; model 3), region and setting (model 4), hypertension status (model 5), cognitive disorder status (model 6), and hypertension status and cognitive disorder status (model 7). In model 8, we controlled only for age and sex. In models 9 and 10, we assessed the potential nonlinear effects of $PM_{2.5}$ exposure on all-cause mortality by fitting penalised splines with 2 or 3 degrees of freedom, respectively, for $PM_{2.5}$ (appendix). We calculated Akaike information criterion estimates and log-likelihood ratios, which indicate the effect of adjustment on the model's degree of fit, for models 1, 9, and 10 (appendix).

| | Statistics |
|---|---|
| Total person-years | 49 440 |
| Age (years) | 89 (79–97) |
| Body-mass index | 20 (18–22) |
| Education (years) | 0 (0–3) |
| Household income (yuan) | 10 000 (4000–25 000) |
| Sex | |
| Women | 7687 (58%) |
| Men | 5657 (42%) |
| Marital status | |
| Married | 4242 (32%) |
| Not married | 9102 (68%) |
| Physical activity | |
| Yes | 8135 (61%) |
| No | 5209 (39%) |
| Smoking status | |
| Never | 8864 (66%) |
| Former | 2113 (16%) |
| Smoker | 2359 (18%) |
| Missing | 8 (<1%) |
| Drinking status | |
| Never | 9110 (68%) |
| Former | 1864 (14%) |
| Drinker | 2361 (18%) |
| Missing | 9 (<1%) |
| Hypertension | 7745 (58%) |
| Cognitive disorder | 5970 (45%) |

Data are n, median (IQR), or n (%).

***Table 1:*** **Descriptive characteristics of study population at baseline in 2008 (n=13 344)**

For the **GEOS-Chem chemical transport model** see http://acmg.seas.harvard.edu/geos/

| | Adjusted covariant | HR (95% CI) |
|---|---|---|
| Model 1 | Age, sex, smoking status, drinking status, physical activity, BMI, household income, marital status, and education | 1·08 (1·06–1·09) |
| Model 2 | Age, sex, smoking status, drinking status, physical activity, BMI, household income, marital status, education, and urban or rural residence | 1·06 (1·04–1·08) |
| Model 3 | Age, sex, smoking status, drinking status, physical activity, BMI, household income, marital status, education, and region | 1·1 (1·07–1·12) |
| Model 4 | Age, sex, smoking status, drinking status, physical activity, BMI, household income, marital status, education, urban or rural residence, and region | 1·08 (1·05–1·10) |
| Model 5 | Age, sex, smoking status, drinking status, physical activity, BMI, household income, marital status, education, and hypertension | 1·08 (1·06–1·09) |
| Model 6 | Age, sex, smoking status, drinking status, physical activity, BMI, household income, marital status, education, and presence of cognitive disorders | 1·07 (1·05–1·09) |
| Model 7 | Age, sex, smoking status, drinking status, physical activity, BMI, household income, marital status, education, hypertension, and presence of cognitive disorders | 1·07 (1·05–1·09) |
| Model 8 | Age and sex | 1·07 ( 1·05–1·08 ) |

HR=hazard ratio. BMI=body-mass index.

***Table 2:*** **HRs and 95% CIs for association between all-cause mortality and a 10 μg/m³ increase in 3-year average $PM_{2·5}$ concentration for eight models**

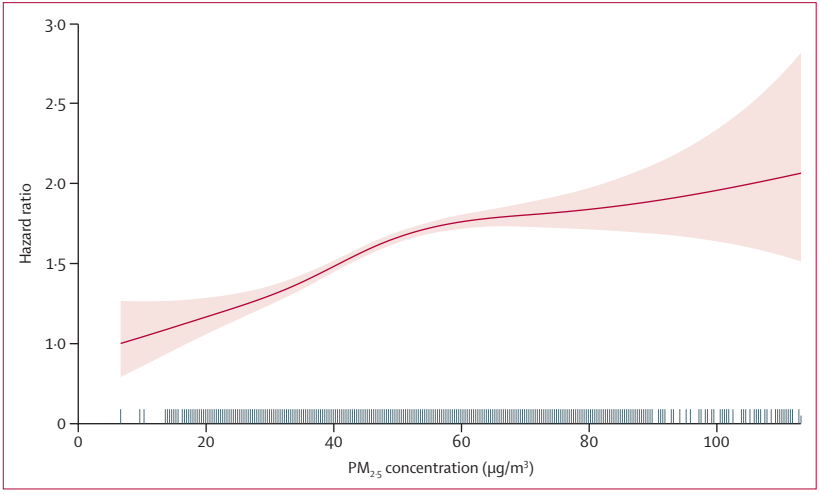


***Figure 2:*** **Curve association between all-cause mortality and a 10 μg/m³ increase in 3-year average $PM_{2·5}$ concentration**

Shading indicates 95% CIs. The reference $PM_{2·5}$ concentration was 6·7 μg/m³ (curve results from model 10).

We also did analyses stratified by age (65–74 years *vs* 75–84 years *vs* ≥85 years), sex, smoking status, drinking status, region (north *vs* south), setting (urban *vs* rural), and $PM_{2·5}$ concentration (<60·9 μg/m³ *vs* ≥60·9 μg/m³). We estimated 60·9 μg/m³ as the inflection point of the curve based on the maximum value of the second derivative on the curve of $PM_{2·5}$ and all-cause mortality. The maximum value of the second derivative represents the point of the curve where the slope of the tangent of the curve decreases suddenly. Analyses were done with R version 3.3.1.

### Estimation of all-cause mortality

We used the HR estimate from model 10 to calculate the burden of $PM_{2·5}$-related mortality in each county in China, using a method adapted from the Global Burden of Disease Study (GBD) 2010.[19,20] We calculated, for each county ($i$), the premature mortality ($M_i$) in the population aged 65 years and older that is attributable to ambient $PM_{2·5}$ as follows:

$$M_i = P_i \times I_i \times \left( \frac{HR(C_i) - 1}{HR(C_i)} \right)$$

where $P_i$ is the population aged 65 years and older in each county, $I_i$ is the average annual mortality rate in the population aged 65 years and older in each county (representing the hypothetical mortality rate that would remain if $PM_{2·5}$ concentrations were reduced to the theoretical minimum risk concentration throughout that county), $C_i$ is the annual average $PM_{2·5}$ concentration in each county, and $HR\ (C_i)$ is the HR at concentration $C_i$ for the population aged 65 years and older (estimated with model 10). All data sources used in estimation of the burden of disease are shown in the appendix.

### Role of the funding source

The funder of the study had no role in the study design, data collection, data analysis, data interpretation, or writing of the report. The corresponding author had full access to all data in the study and had the final responsibility for the decision to submit for publication.

## Results

In the study, 13 344 individuals had complete data for all timepoints (2008, 2011, and 2014), yielding follow-up data for 49 440 person-years. The mean duration of follow-up was 3·7 years (SD 2·1). Baseline characteristics of participants included in this analysis (table 1) were similar to the 3215 individuals who were lost to follow-up (appendix).

The median age at enrolment was 79 years (IQR 71–86) in 5134 participants who survived through follow-up, and 93 years (87–100) in 8210 participants who died. Total person-years during the 6-year follow-up were 29 737 for the surviving respondents and 19 702 for the respondents who had died. The median of the average 3-year $PM_{2·5}$ exposure was 50·7 μg/m³ (range 6·7– 113·3; appendix). $PM_{2·5}$ concentration statistics for several different regions and urban versus rural settings in China are shown in the appendix. We found that $PM_{2·5}$ concentrations varied substantially across China, and that these differences were largely unchanged during our study period (appendix).

Table 2 shows the HRs and 95% CIs for the association between all-cause mortality and a 10 μg/m³ increase in average 3-year $PM_{2·5}$ exposure according to the different models. In model 1, the HR was 1·08 (95% CI 1·06–1·09). Similar results were obtained for models 2–8. The HR (reference concentration 6·7 μg/m³ as the lowest concentration that participants were exposed to) for the association between all-cause mortality and $PM_{2·5}$ exposure was non-linear, with a steeper slope at concentrations below 60·9 μg/m³ (figure 2).

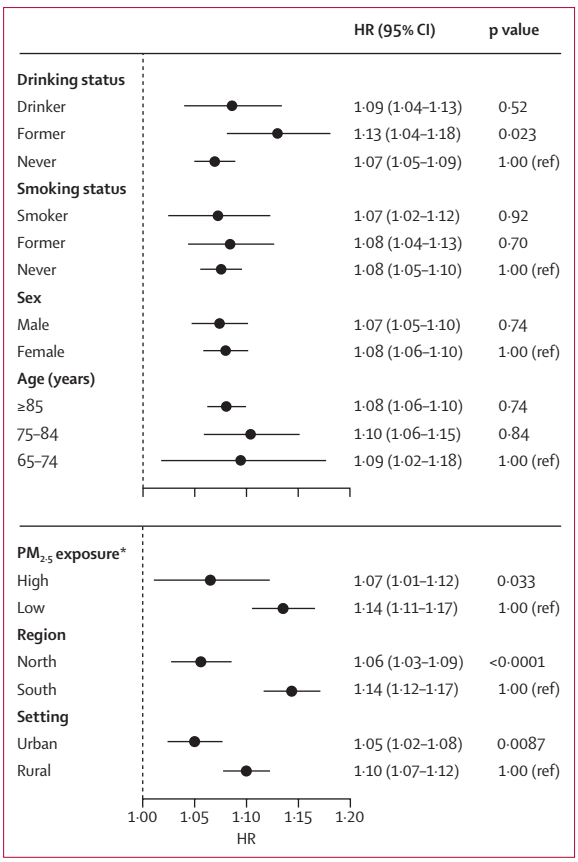


***Figure 3:*** **Risk of death associated with 10 μg/m³ increase in $PM_{2.5}$ by subgroup**
HR=hazard ratio. *High $PM_{2.5}$ exposure was ≥60·9 μg/m³ and low $PM_{2.5}$ exposure was <60·9 μg/m³.

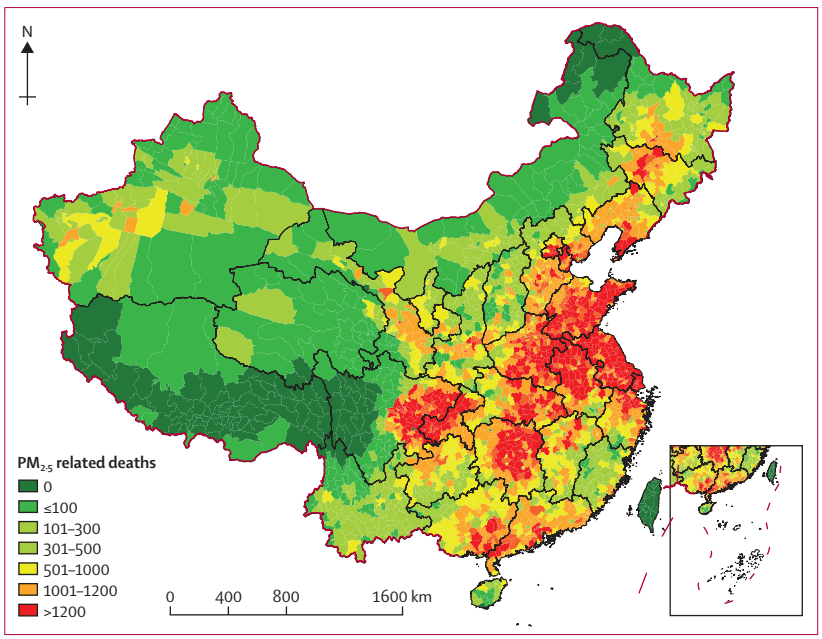


***Figure 4:*** **Estimated all-cause mortality related to $PM_{2.5}$ exposure in adults aged 65 years and older in China in 2010**
Islands in the South China Sea are shown in the box.

In the stratified analyses (figure 3), the effect of a 10 μg/m³ increase in average 3-year exposure to $PM_{2.5}$ on the risk of death did not differ by age, sex, or smoking status. However, effects differed according to drinking status, setting, region, and exposure to high versus low concentrations of $PM_{2.5}$. In particular, the risk of death was higher in rural than in urban counties, in southern than in northern counties, and in areas with ambient $PM_{2.5}$ concentrations of less than 60·9 μg/m³ than in those with concentrations of 60·9 μg/m³ or higher.

Using the concentration–response function estimated in model 10, we estimated that the total burden of all-cause mortality related to $PM_{2.5}$ exposure among the population aged 65 years and older in China in 2010 was 1765 820 people (figure 4). Regions where $PM_{2.5}$ exposure had the greatest effect on this population included the Beijing-Tianjin-Hebei region, the North China Plain in northern China, the Yangtze River Delta in eastern China, the Wuhan metropolitan region and the Changsha-Zhuzhou-Xiangtan region in central China, and the Sichuan Basin in southwestern China. A map of estimated deaths per 100 km² among people aged 65 years and older in China in 2010 is shown in the appendix.

## Discussion

In this community-based prospective cohort study in China, long-term exposure (previous 3 years) to $PM_{2.5}$ was associated with an increased risk of all-cause mortality in people aged 65 years and older after adjustment for known risk factors. In particular, there was a strong positive association between exposure to 7–113 μg/m³ $PM_{2.5}$ and all-cause mortality. We also found that the risk was higher in rural than in urban areas, in southern than in northern China, and with exposure to low versus high concentrations of $PM_{2.5}$. These results suggest that there are regional differences in the effect of $PM_{2.5}$ exposure on all-cause mortality in China.

Our finding of a positive effect of $PM_{2.5}$ on all-cause mortality is consistent with results from most North American and European cohorts.[1–4,6,8] The magnitude of the effect in this study was consistent with that of a US study[21] in Medicare recipients, despite the $PM_{2.5}$ exposure range being different between the two studies. Our finding was also consistent with a meta-analysis of long-term $PM_{2.5}$ exposure and all-cause mortality,[1,2,4,7] and a 2017 study[9] in a cohort of Chinese men, which reported a HR per 10 μg/m³ increase in $PM_{2.5}$ of 1·09 (95% CI 1·08–1·09) for non-accidental mortality. Some previous studies[22,23] from China have reported the long-term effects of air pollution on mortality; however, they were based on exposure metrics that included larger, non-respirable particles (ie, total suspended particulates and $PM_{10}$). In this study, we had access to nationwide survey data and a dataset of $PM_{2.5}$ concentrations (7–113 μg/m³) in China, which allowed a more robust analysis of the association between $PM_{2.5}$ and all-cause mortality.

Within a narrow range of reasonably low $PM_{2.5}$ concentrations, previous studies in North American and European cohorts found almost linear associations between $PM_{2.5}$ exposure and all-cause mortality.[1,3,5,8,21,24,25] However, a question remains regarding the validity of extrapolating these findings to higher concentrations of $PM_{2.5}$. To address this issue, GBD 2010[11,19] developed an integrated exposure–response (IER) model that integrates available relative risks (RRs) from long-term studies of ambient air pollution in the USA and Europe with studies of second-hand smoke, household solid cooking fuel, and active smoking to estimate the RR from long-term exposure to $PM_{2.5}$ at concentrations as high as 1000 μg/m³. However, in the published IER model, a data gap exists in the concentration range of 50–100 μg/m³ because of the scarcity of ambient $PM_{2.5}$ epidemiological evidence from LMICs. Our data suggest that the RR curve is non-linear from 7–113 μg/m³ and steeper at lower concentrations of $PM_{2.5}$. These findings support the IER concept, which assumes that effects level off at higher $PM_{2.5}$ concentrations. Our data might inform future studies investigating concentration–response relationships and estimating disease burden from $PM_{2.5}$ exposure in LMICs such as China.

We used the concentration–response function developed in this study to estimate burden from $PM_{2.5}$-related mortality in the population aged 65 years and older in 2010 in China. Our estimate of more than 1·7 million premature deaths related to $PM_{2.5}$ exposure was higher than the estimate in GBD 2010.[19] However, the regional pattern was consistent with previous estimates.[20,26–28] There are several possible explanations for the higher estimates in our study than in other studies. First, we based our calculations on a new concentration–response curve that was generated with data from a Chinese nationwide cohort; this curve was more representative of the Chinese population than those in previous studies.[20,26–28] Second, our exposure–mortality curve was for the population aged 65 years and older with all-cause mortality, which was different from a previous study[20,26–28] that used IER models for the entire population with five diseases. Third, previous studies[26–28] based their estimates on a gridded population that did not fully cover all of China. By contrast, we used more relevant age-specific, county-level population data from the Chinese census,[14] which captured more detailed and accurate information.

Stratification by setting, region, and $PM_{2.5}$ concentration suggested that people in rural settings, southern China, and areas with low concentrations of $PM_{2.5}$ are more sensitive to $PM_{2.5}$ than are people in urban settings, northern China, and areas with high concentrations of $PM_{2.5}$. $PM_{2.5}$ concentrations in rural areas and southern China were slightly lower than those in urban areas and northern China (appendix), which suggests greater sensitivity at lower $PM_{2.5}$ concentrations. The health effect differences between areas might be caused by differences in $PM_{2.5}$ exposure concentrations, time spent outdoors, access to health care, baseline population health statuses, population structures, or composition of $PM_{2.5}$ (due to different sources). For example, a greater proportion of $PM_{2.5}$ in rural than in urban settings is probably derived from consumption of biomass fuels. Our results differed from the Chinese cohort study in men,[9] which found a lower HR for non-accidental mortality in rural and southern areas than in urban and northern areas.[9] One reason for this difference might be the different age structures of the cohorts: the age of participants in our study ranged from 65 years to 116 years, whereas the population in Yin and colleagues' study[9] had a mean age of 54·8 years (SD 10·7). Additionally, Yin and colleagues did not control for household income, which might bias effect estimates.

Although cumulative average $PM_{2.5}$ concentrations ranging from 2 years to 7 years were all significantly associated with all-cause mortality in our study, the 3-year average $PM_{2.5}$ concentration had the strongest association with all-cause mortality in this high-pollution setting. Previous studies[4,29–32] have suggested that $PM_{2.5}$ exposure windows of 2 years or less are generally associated with cardiovascular health endpoints, although at much lower concentrations than observed in this study.[4] Overall, these findings indicate that prompt action to improve China's air quality might have substantial health benefits in a reasonably short time.

One strength of our cohort was the availability of extensive covariate data obtained through face-to-face surveys. We selected a cohort of people aged 65 years and older that covered most of China's territory and reflected different $PM_{2.5}$ concentrations across China to enhance power and geographical stability. We found that $PM_{2.5}$ concentrations differed substantially across regions and for urban versus rural settings, and that effect estimates differed across different concentration ranges. Because residents were scattered geographically, we could quantify differences in $PM_{2.5}$ exposures using data resolved to a 1×1 km grid. We found that 60·9 μg/m³ was an inflection point in the exposure–response relationship curve, below which the gradient of the HR slope became steeper. This finding indicated that after reducing the $PM_{2.5}$ concentration to less than 60·9 μg/m³, continued effort to reduce the concentration further would yield increasing health benefits.

This study has several limitations. First, data on specific causes of death were not available. However, we believe that this study is an important contribution to the literature, considering the paucity of cohort studies of ambient particulate matter in China. Additionally, previous research has shown that $PM_{2.5}$ exposure increases risk of mortality from a range of causes that account for the majority of deaths worldwide, including cardiovascular diseases, lung cancer, and respiratory diseases, and from all causes combined.[2,5,7] Our estimate of all-cause mortality related to $PM_{2.5}$ exposure in 2010 is larger than the well known GBD estimates. It would be of

interest to ascertain whether this difference is due to a difference in the slope of the concentration–response function or because all-cause mortality includes additional causes of death that might have been missed in GBD, which included only five specific causes of death. Second, residential address information, which was used to estimate the exposure concentration for each participant, could be obtained only for 2008. However, all participants were aged 65 years and older, and the likelihood of them moving in the years after 2008 was very small. Third, we did not measure indoor $PM_{2.5}$ exposure and so biomass cookstoves—an important indoor source of air pollution—were not included in our survey. However, biomass cookstoves are used more in rural than in urban areas and, in the stratified analysis, we split the data by rural versus urban areas. A previous study[33] that adjusted for biomass consumption did not find that this factor changed the mortality effect. Fourth, we did not have access to data about the number of cigarettes consumed, only whether the participant was a current, former, or never smoker. Most previous studies have used the prevalence of smoking, rather than the number of cigarettes, as the control variable.[34] Fifth, we assessed only $PM_{2.5}$ because data for other ambient pollutants are scarce for China. Nevertheless, a previous study[21] showed that the chronic mortality effect of $PM_{2.5}$ remains robust regardless of whether other air pollutant confounders are considered. Finally, our data were limited to 6 years, and a longer follow-up would be preferable.

Despite its limitations, this study provides crucial information about the mortality risk associated with long-term exposure of people aged 65 years and older to $PM_{2.5}$ in China.

#### Contributors

TL, XS, YZe, HC, and PLK contributed to the concept and design of the study. ZY and JL collected the survey data. YZh, JW, DX, and YLv prepared and cleaned the data. YLi contributed to interpretation of the exposure data. TL did the statistical analysis and drafted the article. All authors contributed to the interpretation of results and critically revised the draft.

#### Declaration of interests

We declare no competing interests.

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