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The association between PM_{2.5} and frailty: evidence from 122 cities in China and 7 countries in Europe

Yanchao Wen^{1,2}, Guiming Zhu^{1,2}, Kexin Cao^{1,2}, Jie Liang^{1,2}, Xiangfeng Lu³ and Tong Wang^{1,2*}

Abstract

Background The accelerated aging process worldwide is placing a heavy burden on countries. PM_{2.5} particulate matter exposure is a significant factor affecting human health and is crucial in the aging process.

Methods We utilized data from China Health and Retirement Longitudinal Study (CHARLS) and the Survey of Health, Aging, and Retirement in Europe (SHARE) to study the relationship between PM_{2.5} exposure and the frailty index. Acquire PM_{2.5} exposure data for China and Europe, match them according to geographic location within the database. Our study used frailty index to evaluate frailty, which comprises 29 items. We examined the association between PM_{2.5} and frailty index using fixed-effects regression models and Mendelian randomization (MR) analysis.

Results We first examined the association between PM_{2.5} and frailty index using fixed-effects regression models, revealing a notable positive link across populations in China (coefficient = 0.0003, $P = 0.0380$) and Europe (Coefficient = 0.0019, $P < 0.0001$). This suggests that PM_{2.5} exposure is a significant risk factor for frailty, leading to accelerated frailty. Moreover, our MR analysis uncovered a possible causal association (OR = 1.2933, 95%CI: 1.2045–1.3820, $P < 0.0001$) between PM_{2.5} exposure and the frailty index.

Conclusions Our findings indicate that long-term exposure to PM_{2.5} in the environment is a risk factor for physical frailty and may have a potential causal relationship. Given the rapid global aging trend, public health measures are needed to reduce PM_{2.5} concentrations and prevent frailty.

Keywords PM_{2.5}, Frailty index, Air pollution, Aging, Cohort study

Introduction

Aging is evolving into a worldwide trend, this is the development challenge faced by countries worldwide. The World Health Organization (WHO) projects that the elderly population will increase to 1.4 billion by 2030 and 2.1 billion by 2050 [1]. As a result, the rapid growth of the frail elderly population will exert significant pressure on global medical infrastructure and the elderly themselves [2]. Frailty, characterized by a weakened ability to withstand health stress, often impacts the elderly and is linked to numerous negative health outcomes, including all-cause mortality [3], newly diagnosed chronic diseases

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[4], depressive symptoms [5], and falls [6]. Additionally, research indicates that active improvement strategies can mitigate frailty [7, 8], improving the quality of life for the elderly.

Air pollution, as the greatest environmental risks to health, may contribute to the aging process and impact successful aging, which is especially significant in understanding aging [9]. In 2019, a staggering 99% of people worldwide resided in areas failing to comply with WHO's air quality criteria [10]. Air pollution's health impacts are diverse, encompassing respiratory diseases, allergic conditions [11], cardiovascular disorders [12], and brain health [13]. Epidemiological evidence indicates a potential link between these diseases and both long-term exposure to air pollution and frailty [14–17].

In recent years, several studies have investigated the impact of air pollution on frailty. For example, research has reported a 30% increase in the likelihood of frailty for every 10 $\mu\text{g}/\text{m}^3$ rise in $\text{PM}_{2.5}$ levels in rural areas [18]. A population-based quasi-experimental study, utilizing propensity score matching and double difference analysis, showed a significant decrease in individuals' FI scores and the frailty status improved following the implementation of the Clean Air Action in China [19]. However, current research, despite their contributions, exhibits several limitations. Firstly, the evidence predominantly stems from cross-sectional studies, which inherently lack the capacity to establish causal relationships. However, research has shown that cohort studies and Mendelian randomization (MR) studies provide more convincing evidence than cross-sectional studies [20]. Secondly, the research subjects are primarily concentrated within a single region, resulting in a scarcity of cross-regional studies. Consequently, there is a pressing need for cross-regional research to yield more universally applicable findings. Finally, there is a paucity of research examining the relationship between $\text{PM}_{2.5}$ exposure and the Frailty Index (FI). Studies have shown that the FI is widely recognized as a standard measure of aging and can be used to assess the burden of age-related clinically significant health deficits [4, 21]. Furthermore, the FI demonstrates strong predictive ability and is effective in forecasting adverse health outcomes [22].

To investigate the association and causal relationship between $\text{PM}_{2.5}$ exposure and FI, we utilized data from the China Health and Retirement Longitudinal Study (CHARLS), the Survey of Health, Ageing and Retirement in Europe (SHARE), and summary data from Genome-Wide Association Studies (GWAS). Employing fixed effect regression models and MR methods, we aimed to provide a comprehensive analysis of the potential impacts of $\text{PM}_{2.5}$ on FI.

Methods

Study design

The baseline survey of CHARLS, a nationally representative cohort study of Chinese residents aged 45 and above, began in 2011. It included 17,708 individuals and was followed up every two years [23]. The survey used multi-stage probability sampling to select respondents. In addition, data from SHARE, the largest longitudinal study in Europe, are available for people aged 50 years and older. We used data from three follow-up visits from 2011 to 2015. We obtained the following information from the two cohorts: gender, age, marital status, education, residence, smoking status, drinking status, retirement status, body mass index (BMI (kg/m^2)), and a variety of chronic diseases. In addition, information was obtained on variables related to activities of daily living (ADL), instrumental activities of daily living (IADL), physical function limitations, cognitive abilities, and depression. Figure 1 shows the data screening flowchart.

To ensure study consistency, we limited the age range of both cohorts to those over 50 years of age. During the research process, for CHARLS, we excluded participants who lacked information on key variables for constructing the FI, as well as participants data lacked geographical location information. Finally, we included 6,407 research subjects. The SHARE data is consistent with the CHARLS data above, and participants from European countries with land areas smaller than or comparable to the largest cities in China were retained (include Estonia, Belgium, Czech Republic, Austria, Switzerland, Denmark, Slovenia). Finally, 17,029 research subjects were included. Figure 2 shows the 122 cities in China and 7 countries in Europe included and the number of people surveyed. The darker the color, the more surveyed people are included.

Furthermore, MR analysis was performed using GWAS summary data to explore the causal association between FI and $\text{PM}_{2.5}$. The summary data for $\text{PM}_{2.5}$ was sourced from the IEU Open GWAS project (ID: ukb-b-10817). For the FI, GWAS data were derived from a study that investigated British individuals of European descent [24, 25]. This study encompassed a GWAS meta-analysis of the FI conducted on Biobank participants and Swedish twin genome participants. Calculations for FI utilized self-reported health-related information from the UK Biobank (UKB) and Twin Genome, encompassing 49 to 44 elements related to symptoms, disability, and identified illnesses.

Air pollution data

Ambient $\text{PM}_{2.5}$ data were obtained from previously established dataset through satellite-based remote sensing technology [26, 27]. The time span of air pollution data is consistent with that of the study population, which is

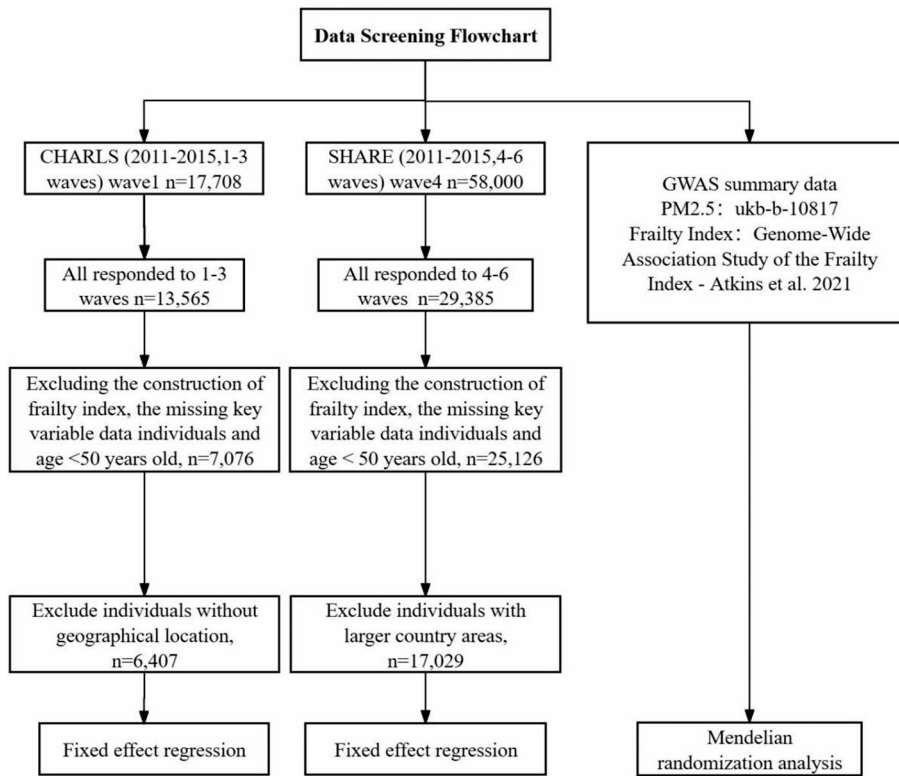


Fig. 1 Data screening flowchart

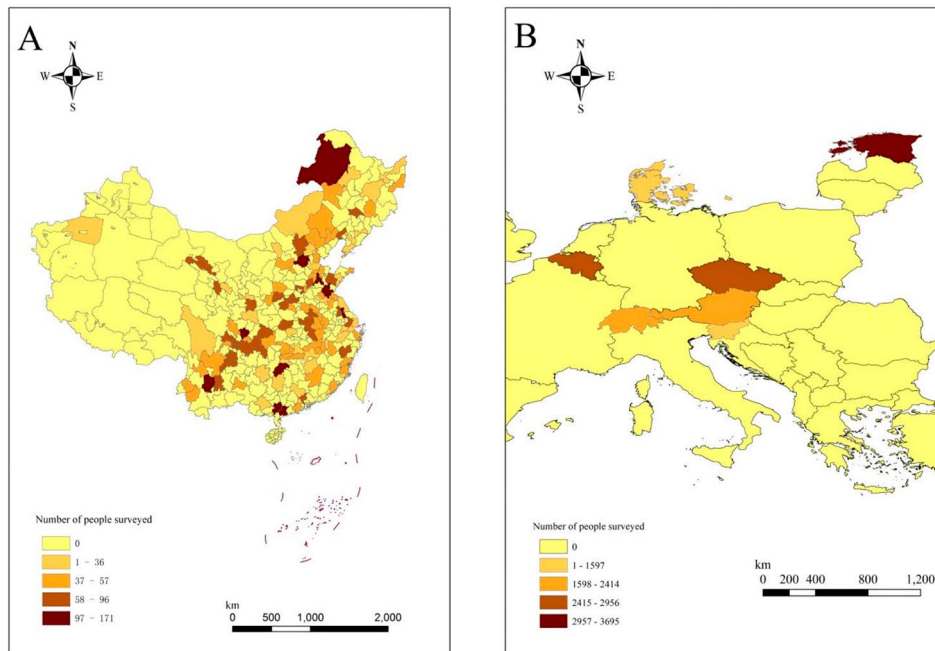


Fig. 2 122 Chinese cities and 7 European countries included and the number of respondents. The darker the color, the more respondents were included. A: China; B: Europe

2011–2015. Specifically, air pollution data for China were sourced from the China High-Resolution, High-Quality Near Surface Air Pollutant dataset published by Wei et al. [26]. This dataset employs a comprehensive approach, integrating big data from various sources such as ground-based measurements, satellite remote sensing products, atmospheric reanalysis, and model simulations. By incorporating artificial intelligence, the dataset effectively generates $PM_{2.5}$ data while accounting for the spatiotemporal heterogeneity of air pollution. Notably, the dataset boasts a spatial resolution of $1\text{ km} \times 1\text{ km}$, offering enhanced accuracy and predictive capability compared to previously reported datasets. To integrate the mean concentration of each grid cell with geographic data, ArcGIS software (ESRI Inc.) was utilized. This process involved using a geographic shapefile that contained the boundaries of prefecture-level cities in mainland China. Therefore, we were able to match and calculate the average $PM_{2.5}$ concentration levels across 122 cities. These calculations were based on data collected from three follow-up visits conducted between 2011 and 2015.

European $PM_{2.5}$ concentrations were obtained from the Atmospheric Composition Analysis Group Web site at Washington University, which is a $1\text{ km} \times 1\text{ km}$ global model developed by HAMMER et al. [27]. HAMMER and colleagues combined aerosol optical depth (AOD) retrievals from NASA's MODIS, MISR, SeaWiFS, and VIIRS with the GEOS-Chem chemical transport model. They then used a residual convolutional neural network (CNN) to calibrate these results against global ground

observations, estimating annual and monthly surface $PM_{2.5}$ from 2000 to 2019. Similar to China, to facilitate the matching of $PM_{2.5}$ data for seven European countries, country-level geographic location information was obtained from SHARE. Figure 3 shows the average $PM_{2.5}$ concentrations from 2011 to 2015 across 122 cities in China and 7 European countries. The numbers represent the number of people surveyed included from each provincial-level unit in China or each European country.

In addition to $PM_{2.5}$ concentration, NO_2 concentration and temperature levels are considered influential factors. Therefore, global NO_2 concentrations (with a spatial resolution of $1\text{ km} \times 1\text{ km}$) [28] and temperature data (<https://cds.climate.copernicus.eu/cdsapp#!/dataset/reaanalysis-era5-pressure-levels>) were incorporated as control variables in the model. This comprehensive approach ensures a thorough examination of the impact of $PM_{2.5}$ concentration while accounting for potential confounding effects of NO_2 concentration and temperature levels.

Assessment of the frailty index

In our study, we used the FI to evaluate frailty, which is characterized by the accumulation of various age-related health problems. During the follow-up period between 2011 and 2015, three repeated measurements of FI were taken for individual. The FI was developed according to established procedures and principles outlined by Searle SD [29, 30], and informed by relevant previous research [3, 31]. Based on data from CHARLS and SHARE, a total of 29 items were selected for the construction of the FI.

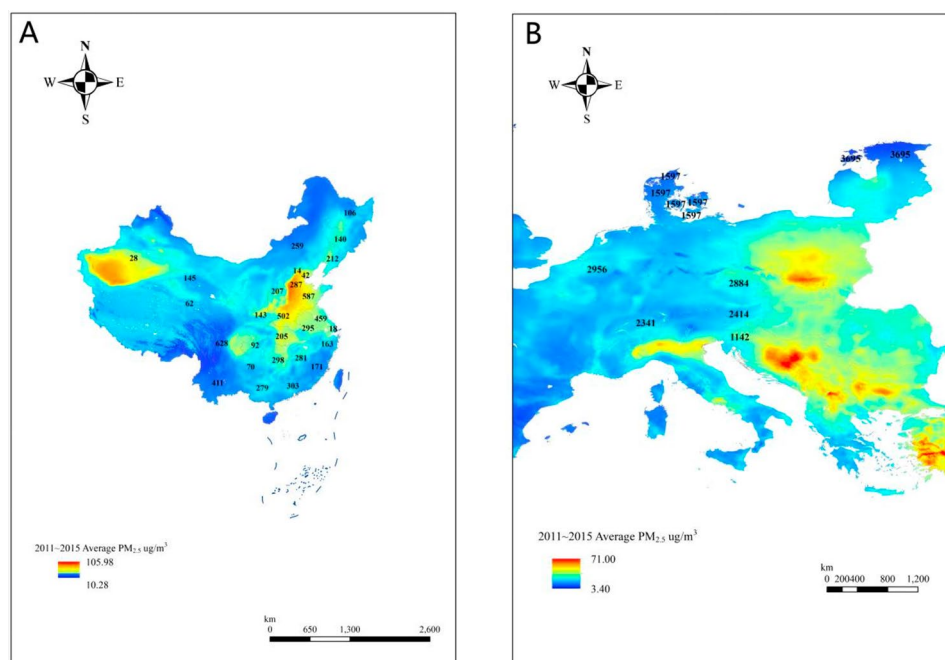


Fig. 3 The average $PM_{2.5}$ concentrations from 2011 to 2015 across 122 cities in China and 7 European countries. The numbers represent the number of people surveyed included from each provincial-level unit in China or each European country. **A:** China; **B:** Europe

These items included illness, disability in ADL and disability in IADL, physical functioning, depression, and cognition. Each item was scored as 1 (deficit present) or 0 (no deficit), respectively. The scores for items 1–29 were summed to obtain the FI score, which ranged from 0 to 29. Items 28 and 29 representing cognitive and depression scores, are continuous variables, their values spanning from 0 to 1. Currently, research focuses on categorizing FI. However, existing studies have revealed discrepancies in the classification of FI and in identifying older adults as “frail”, thereby limiting our understanding of frailty. Maintaining FI as a continuous variable may be beneficial until further research establishes the optimal FI category for this population [32]. Therefore, the FI for each participant is calculated by dividing the total current health deficits by 29. As a continuous variable, FI ranges from 0 to 1, with higher values indicating greater frailty. The variables for constructing the FI are shown in the Supplementary table (Supplementary Table 1).

Statistical analysis

After obtaining air pollution exposure data and FI outcome data, we matched them based on the geographic location information provided by the CHARLS and SHARE databases. The data for each individual included three PM_{2.5} concentration data and FI measurements, as well as a series of covariate data. First, to reduce the impact of missing values on the analysis, we excluded individuals with missing information on key variables in the construction of the FI. For the remaining individuals,

we performed multiple imputation of covariates to obtain complete data for analysis [33, 34]. The proportion of missing variables is shown in the supplementary table (Supplementary Table 2). Fixed effects regression was used to evaluate the longitudinal data. Fixed effects regression can account for both observed and unobserved time-invariant confounding variables [35]. Consequently, fixed effect regression is deemed more robust than conventional regression models in investigating the correlation between predictor alterations and outcome variations.

The fixed effect regression model is as follows

$$Y_{it} = \alpha + \alpha_i + \beta_1 X_{1it} + \beta_2 X_{2it} + \dots + \beta_k X_{kit} + \epsilon_{it}$$

Y_{it} is the dependent variable (the FI in this study), X_{kit} is the independent variable and the covariate that needs to be controlled (such as PM_{2.5} and BMI in this study), α is the intercept term, α_i is the individual fixed effect, ϵ_{it} is the error bar, and β_k is the regression coefficient.

European SHARE data was employed to explore the impact of regional variances on the link between PM_{2.5} and FI. The following covariates were controlled for in the analysis: age, marital status, education, smoking status, drinking status, retirement status, BMI, mean annual NO₂ concentration and temperature.

Furthermore, to explore the causal association between the two variables, we performed MR analysis using GWAS summary data. In this analysis, we initially filtered out outlier single nucleotide polymorphisms (SNPs), retaining only those SNPs deemed reliable for further investigation. The MR analysis was primarily conducted employing the inverse variance weighted with modified weights (MW-IVW) method [36] as the main analytical approach. Additionally, sensitivity analyses were performed using the Inverse Variance Weighted (IVW) [37] and weighted median (WM) methods [38] to assess the robustness of the findings. To test for pleiotropy, the MR-Egger method was employed, and the F statistic was applied to gauge the strength of instrumental variables in the study [39, 40].

The statistical evaluations were performed utilizing R (version 4.2.3), ArcGIS (version 10.8), Stata 16, and SPSS 25 software, considering a *P*-value less than 0.05 as statistically significant.

Results

Basic characteristics of study participants

The baseline basic characteristics of CHARLS and SHARE participants are presented in Table 1. The mean age (SD) of 6,407 participants in CHARLS was 60.67 years (7.11 years), with males comprising 49.9%. And the proportion of elderly people aged 65 and above is

Table 1 Fundamental traits of the population studied

Characteristic	CHARLS (N (%))	SHARE (N (%))
Total sample (N)	6,407	17,029
Males	3,194 (49.9%)	6,992 (41.1%)
≥ 65 years old	1,706 (26.63%)	7,940 (46.63%)
Age (Mean ± SD, years)	60.67 ± 7.11	65.06 ± 9.13
BMI (Mean ± SD, years)	23.42 ± 3.84	27.11 ± 4.80
Marital status (married)	5,964 (88.9%)	11,062 (65.0%)
Educational level		
Less than lower secondary	5,740 (89.9%)	5,003 (29.4%)
Upper secondary & vocational training	594 (9.3%)	7,785 (45.7%)
Tertiary	72 (1.1%)	4,341 (24.9%)
Residence (Rural)	4,178 (65.2%)	5,993 (35.2%)
Smoking status (Smoker)	2,009 (32.8%)	3,348 (19.7%)
Drinking status (Drinker)	2,191 (34.2%)	8,081 (47.1%)
Frailty Index in 2011 (Mean ± SD)	0.1268 ± 0.1078	0.1037 ± 0.1031
PM _{2.5} in 2011 (Mean ± SD, µg/m ³)	58.1757 ± 17.8567	14.8600 ± 3.6226
NO ₂ in 2011 (Mean ± SD, µg/m ³)	7.1756 ± 4.5943	5.3475 ± 2.3394
Temperature in 2011 (Mean ± SD, °C)	13.9071 ± 5.0140	8.2235 ± 1.6146

25.63%. The mean annual exposure concentration of PM_{2.5} in 2011 at baseline was 58.1757 µg/m³, the average FI in 2011 was 0.1268. The mean age (SD) of all 17,029 participants in SHARE was 65.06 years (9.13 years), with males comprising 41.2% and over 65 years old 46.63%. The mean annual exposure concentration of 14.8600 µg/m³ of PM_{2.5} in 2011 at baseline, the average FI in 2011 was 0.1037.

Association between PM_{2.5} and Frailty Index in cohort study

In Table 2, the association between PM_{2.5} and the FI is displayed. Initially, a significant positive correlation was identified without controlling for potential confounding variables: The FI increases by 0.0030 for every 10 µg/m³ rise in PM_{2.5} concentration, $P=0.0290$. Upon adjusting for these confounding factors, a positive relationship between PM_{2.5} and the FI persisted (coefficient=0.0003, $P=0.0380$). This suggests that with each 10 µg/m³ rise in PM_{2.5} concentration in the atmosphere, the FI also increases by 0.0030. In the SHARE data, we observed similar results. Prior to adjustment: coefficient=0.0022, $P<0.0001$, after adjustment: coefficient=0.0019, $P<0.0001$. This indicates that the FI was found to increase by 0.0190 for every 10 µg/m³ increase in PM_{2.5} concentration. Limiting the study to individuals aged 65 years and older revealed that PM_{2.5} remained a risk factor for frailty in the European population. While a positive association was observed in the Chinese population, it was not statistically significant ($P=0.3140$). Additionally, to further explore the urban-rural associations. CHARLS data revealed a notable positive relationship between PM_{2.5} and the FI in rural areas: coefficient=0.0004, $P=0.0250$. However, no such association was found in urban areas: coefficient=0.0001, $P=0.6220$. In the SHARE, the impact of PM_{2.5} on the FI remains consistent, indicating that it serves as a risk factor for frailty.

Casual association between PM_{2.5} and Frailty Index in MR

The F-statistic (MeanF=23.9521) and MR-Egger results ($P=0.5290$) demonstrated that the analysis was not influenced by weak instrumental variables and pleiotropy. The MR results indicated that MWIVW: Odds ratio (OR)=1.2933, 95% CI: 1.2045–1.3820, $P<0.0001$; the results indicated that PM_{2.5} was a significant risk for the FI. Sensitivity analyses showed similar results: IVW: OR=1.2820, 95% CI: 1.1874–1.3767, $P<0.0001$; WM: OR=1.2630, 95% CI: 1.1271–1.3989, $P=0.0008$. Our findings demonstrate that higher PM_{2.5} levels expedite the frailty process., with each standard deviation increase in PM_{2.5} associated with a 29.33% rise in frailty risk.

Table 2 Main results

Method	CHARLS		SHARE	
	Coefficient	P	Coefficient	P
Fixed effects regression (Model 1)	0.0003	0.0290	0.0022	<0.0001
Fixed effects regression (Model 2)	0.0003	0.0380	0.0019	<0.0001
Fixed effects regression (Rural)	0.0004	0.0250	0.0031	<0.0001
Fixed effects regression (Urban)	0.0001	0.6220	0.0016	0.0005
Fixed effects regression (≥ 65 years old)	0.0003	0.3140	0.0025	<0.0001
MR	OR = 1.2933, 95%CI: 1.2045–1.3820, $P<0.0001$			

Note: Model 1 is not adjusted. Model 2 adjusted for age, marital status, education, smoking status, drinking status, retirement status, BMI, mean annual NO₂ concentration and temperature. OR: Odds ratio; CHARLS: the China Health and Retirement Longitudinal Study; SHARE: the Survey of Health, Aging and Retirement in Europe; MR: Mendelian randomization

Discussion

The longitudinal study demonstrated a significant positive association between PM_{2.5} exposure and FI, and MR results indicated a causal association. Our study investigated the association between PM_{2.5} and FI through a follow-up design. It is noteworthy that a generally accepted instrument was used to measure frailty, and the FI was measured using a combination of 29 items. Through cross-ethnic studies, we obtained similar results in European populations, indicating that exposure to PM_{2.5} can accelerate frailty in people of different ethnic groups. Our study revealed that exposure to PM_{2.5} may speed up the aging process, similar findings were observed across different regions, and suggesting a possible causal relationship.

Numerous research efforts have focused on exploring the link between air pollution and frailty among older adults. Research involving prospective cohorts revealed a correlation between air pollutants, like PM_{2.5}, and a heightened likelihood of frailty [41]. Consistent with the finding, a population-based study involving 220,079 UKB participants revealed that higher exposure to PM_{2.5} was associated with an elevated risk of frailty [42]. Likewise, studies derived from the Chinese Longitudinal Healthy Longevity Survey revealed an increased occurrence of frailty linked to heightened exposure to air pollution in the year preceding the interview [43]. Notably, frailty scores were significantly higher in older adults residing in areas with severe air pollution. This implies that air contamination could be a major factor in shaping the progression of healthy aging [43]. Furthermore, after China implemented the air pollution control policy, namely the Clean Air Action Plan, the FI scores of healthy individuals were significantly reduced by 0.0205, while the FI scores of pre-frail individuals were significantly reduced

by 0.0114 [19]. Although these studies support the adverse effects of ambient air pollution on frailty, they do not include cross-regional research or explore causal relationships. Building on a long-term cohort study, we incorporated a MR study, resulting in findings that are more robust compared to existing results. Our research corroborates the negative impact of environmental air pollution on frailty. Exposure to $PM_{2.5}$ is positively correlated with the FI, thereby accelerating the aging process. Interestingly, when we restricted our study subjects to individuals aged 65 and above, we observed a positive correlation between $PM_{2.5}$ exposure and FI. However, in the sample of the elderly population in China, this association did not reach statistical significance. This phenomenon may be related to significant lifestyle differences between elderly populations in China and Europe, which could influence the level of $PM_{2.5}$ exposure among those aged 65 and above and differences in genetic backgrounds and physiological characteristics might also contribute to this phenomenon [44–46]. In addition, our investigation brought to light disparities in results between China rural and urban settings. One potential explanation for this variation is that rural regions heavily rely on traditional energy sources like biomass burning, leading to higher levels of both outdoor and indoor air pollution [18]. Moreover, compared to urban residents, rural areas lack proper housing and transportation planning, which may exacerbate environmental exposures for rural residents and result in their limited understanding of the significance of air pollution prevention and control [47, 48].

Exposure to air pollution is widely recognized for causing a range of detrimental health impacts, including inflammatory reactions, oxidative stress, metabolic disorders, and epigenetic modifications. For instance, by upsetting mitochondria, air pollution can cause pro-inflammatory reactions in different immune cells, and since inflammation is thought to be a possible source of weakness, thus collectively leading to the onset of weakness [49, 50]. Moreover, air pollutants may disrupt the body's balance and reduce its ability to handle stress, hastening the decline in functional abilities and capacities associated with aging levels of cells, organs, and the entire system, ultimately resulting in frailty [7]. Clearly, air pollution plays a role in frailty to a certain extent, making the reduction of air pollution crucial for diminishing frailty in the elderly. With the rapid development of the global economy, environmental pollution and population aging have emerged as two critical issues impacting public health. Air pollution, a pervasive and increasingly severe environmental hazard, has long been a significant factor contributing to chronic diseases such as respiratory disorders, cardiovascular diseases, and cancer [51]. This issue is particularly pronounced in regions

experiencing rapid industrialization, where declining air quality poses a substantial threat to public health. Currently, the global population is aging at an accelerated pace due to advancements in healthcare and declining birth rates, leading to an increasing proportion of elderly individuals [52]. This demographic shift not only alters the population structure but also presents challenges in healthcare, social security, and lifestyle adjustments. The elderly population typically faces a higher burden of chronic diseases, and as they age, their immune and recovery capacities diminish, heightening their sensitivity to environmental pollution [53]. The interplay between air pollution and aging exacerbates public health pressures. Therefore, based on current research findings, it is imperative to implement measures to mitigate the impact of air pollution on the aging population. Such measures could include the development of effective public health policies by governments, enhancement of urban green spaces [54], dissemination of knowledge regarding air pollution and health risks to the public, and provision of personalized healthcare services for the elderly [55].

The present study has several significant strengths. Firstly, a cohort study design was employed to investigate the longitudinal association between $PM_{2.5}$ and FI in depth. During the research process, the effects of factors such as temperature, NO_2 and important covariates were carefully controlled to ensure the reliability of the results. Secondly, we constructed a comprehensive FI, taking into account multiple factors, including disease, physical functional limitations, disability in ADL, disability in IADL, physical function, depression, and cognition, to comprehensively assess the FI. Finally, we utilized data from CHARLS and SHARE. Moreover, we ensured consistency in variables used to construct the FI between SHARE and CHARLS, with a consistent data timeframe from 2011 to 2015. Through cross-regional observations, we obtained consistent results. Additionally, we further established causal relationship through MR analysis. Therefore, our study findings are generalizable and demonstrate the impact of $PM_{2.5}$ on frailty, providing robust support for the credibility of our research.

Although our study yielded some important findings, its limitations must also be acknowledged. First, the pollutant data used in the study are based on city-level data, which may not fully capture small changes within cities. The lack of consideration for variability within the city may introduce bias into the results, as detailed geographic locations could more accurately capture the study subjects' exposure levels, leading to more precise research outcomes. Second, we used a validated tool to detect FI, but we adapted it based on information available in the research database used. The use of existing data may introduce bias from the original version. Third, to ensure the maximum inclusion of the sample size, we

performed multiple imputation of cognitive variables required to construct FI in the SHARE database (such as: Orient variable: missing proportion: wave 4: 28.32%, wave 5: 99.62%, wave 6: 0.23%), which may have some impact on the results. However, the multiple imputation method we employed is widely recognized for addressing missing data in cohort studies, and its effectiveness is well-documented [34, 56]. Finally, we acknowledge that the data from CHARLS and SHARE may not fully represent populations in other regions with different socioeconomic or environmental context. However, our research indicates that exposure to PM_{2.5} accelerates aging in both China and Europe, suggesting that the adverse health effects of PM_{2.5} are widespread to some extent. Therefore, it is important to be cautious in interpreting the findings and to address these limitations in future studies.

Conclusion

In conclusion, our research indicates that prolonged exposure to PM_{2.5} is a risk factor for frailty and has a potential causal relationship. Therefore, in the context of an aging population, it is crucial to address the adverse health effects of air pollution. Effective public health measures should be implemented to reduce the concentration of environmental particulates, such as PM_{2.5}, while also mitigating the aging process to enhance overall public health.

Supplementary Information

The online version contains supplementary material available at <https://doi.org/10.1186/s12889-024-21121-4>.

Supplementary Material 1

Acknowledgements

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Author contributions

Yanchao Wen: Data Curation, Writing-Original Draft, Writing-Review & Editing, Visualization. Guiming Zhu: Data Curation, Writing-Review & Editing, Visualization. Kexin Cao: Data Curation, Writing-Review & Editing. Jie Liang: Writing-Review & Editing, Funding acquisition. Xiangfeng Lu: Writing-Review & Editing. Tong Wang: Writing-Review & Editing, Supervision, Funding acquisition.

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Data availability

The China Health and Retirement Longitudinal Study (CHARLS): <https://charls.charlsdata.com/pages/data/111/en.html>. The Survey of Health, Ageing and Retirement in Europe (SHARE): <http://www.share-project.org/data-access.html>. Harmonized data for CHARLS and SHARE can be accessed via: <https://g2aging.org/hrd/get-data>. GWAS summary data for PM_{2.5}: <https://gwas.mrcieu.ac.uk/datasets/ukb-b-10817/>. GWAS summary data for frailty index: <https://figshare.c>

[om/articles/dataset/Genome-Wide_Association_Study_of_the_Frailty_Index_-_Atkins_et_al_2019/9204998](https://doi.org/10.1186/s12889-024-21121-4)

Declarations

Ethical approval

CHARLS received ethical approval from the Biomedical Ethics Review Committee of Peking University (IRB00001052-11015) and all participants provided informed written consent. SHARE received ethical approval from the Ethics Council of the Max Planck Society and all participants provided informed written consent. GWAS summary data and air pollution data are public data and do not require ethical approval.

Consent for publication

All authors approved the final manuscript and the submission to this journal.

Competing interests

The authors declare no competing interests.

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