Original Article

Long-Term Exposure to Low-Level Ambient Air Pollution and Mortality among 0.3 Million Chinese Older Adults*

Likun Liu[1,&], Xueli Yuan[2,&], Wenqing Ni[2], Jing Wei[3], Tingting Liu[4], Ruijun Xu[1], Yingxin Li[1], Zihua Zhong[1], Yi Zheng[1], Sihan Liang[1], Rui Wang[5], Jian Xu[2,#], and Yuewei Liu[1,#]

1. Department of Epidemiology, School of Public Health, Sun Yat-sen University, Guangzhou 510080, Guangdong, China; 2. Department of Elderly Health Management, Shenzhen Center for Chronic Disease Control, Shenzhen 518020, Guangdong, China; 3. Department of Atmospheric and Oceanic Science, Earth System Science Interdisciplinary Center, University of Maryland, College Park, MD 20740, USA; 4. Shenzhen Maternity & Child Healthcare Hospital, Shenzhen 518017, Guangdong, China; 5. LuoHu District Chronic Disease Hospital, Shenzhen 518024, Guangdong, China

Abstract

Objective Evidence that long-term exposure to ambient air pollution increases mortality among older adults, particularly those residing in low-level air pollution locations, remains scarce. This study investigated the potential links between long-term low-level air pollution exposure and mortality among Chinese older adults.

Methods A population-based study with 317,464 individuals aged ≥ 65 years was conducted in Shenzhen, China during 2018 and 2020. Logistic regression models were used to analyze the associations between long-term exposure to air pollution and all-cause mortality, as the primary outcome, as well as non-accidental, cancer and cardiovascular mortality.

Results Significant associations of PM_1, $PM_{2.5}$, PM_{10}, SO_2, CO, and O_3 exposures with a higher risk of all-cause mortality were found. Adjusted odds ratio (*OR*) for each 1 μg/m^3 increment was 1.49 [95% confidence interval (*CI*): 1.46, 1.53] for PM_1, 1.30 (1.27, 1.32) for $PM_{2.5}$, 1.05 (1.04, 1.06) for PM_{10}, 5.84 (5.39, 6.32) for SO_2, 1.04 (1.04, 1.05) for CO, and 1.02 (1.00, 1.03) for O_3, respectively. Long-term PM_1, $PM_{2.5}$, PM_{10}, SO_2, and CO exposures also elevated the risks of non-accidental, cancer and cardiovascular mortality.

Conclusion Long-term low-level air pollution exposure was associated with an increased mortality risk among Chinese older adults.

Key words: Ambient air pollution; Mortality; Older adults; Population-based study; Low-level concentrations

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#Correspondence should be addressed to Jian Xu, Professor of Medicine, Tel/Fax: 86-755-25506942, E-mail: anniexu73@126.com; Yuewei Liu, Associate Professor, Tel/Fax: 86-20-83653005, E-mail: liuyuewei@mail.sysu.edu.cn

Biographical notes of the first authors: Likun Liu, female, born in 1998, Master of public health, majoring in environmental epidemiology; Xueli Yuan, female, born in 1986, Master of Medicine, majoring in chronic diseases prevention and control.

INTRODUCTION

The health of older adults has emerged as a critical public health problem because the global population is rapidly aging. According to the Global Burden of Disease (GBD) 2019 Study, cardiovascular diseases and cancer were both major causes of death among individuals over 70 years old, which seriously threaten the life and health of older adults[1]. Since 99% of humanity lives with ambient air pollution at levels beyond the World Health Organization (WHO) Air Quality Guidelines limits, this issue has grown in concern for public health[2]. Recently, more and more studies have reported that long-term ambient air pollution exposure was correlated with a greater risk of mortality[3–6]. Studies were mainly focused on particulate matter with an aerodynamic diameter ≤ 2.5 μm ($PM_{2.5}$)[7,8], PM_{10}[4,9], nitrogen dioxide (NO_2)[10,11], and ozone (O_3)[5,12], whereas epidemiological evidence on PM_1, sulfur dioxide (SO_2), and carbon monoxide (CO) was limited. Due to their impaired physiological function, physical weakness, and pre-existing chronic diseases, older adults are more susceptible to air pollution[13]. Nevertheless, little is known about the negative effects of ambient air pollution on older adults.

In China, over 1.5 million people died from ambient air pollution in 2019, of whom 77.4% were 65 years or older[14]. Evidence in China has revealed the association between long-term air pollution exposure and mortality[3,15,16]. Most of these studies were conducted in northern China in regions with more severe air pollution concentrations, therefore less is known about the risks in regions in southern China with lower air pollution concentrations. Given recent cohorts from Europe and the United States of America have found that the chronic harmful effects of air pollution still exist at low concentrations, it is intriguing to find out the consequences of low-level air pollution in China.

Hence, our study aimed to evaluate the association between long-term exposure to PM_1, $PM_{2.5}$, PM_{10}, SO_2, NO_2, CO, and O_3 and mortality among individuals aged ≥ 65 years in southern China to better understand the impact of air pollution on mortality among Chinese older adults.

METHODS

Study Area

Shenzhen is situated in southern China and the ambient air quality is relatively good. It ranked sixth among 168 cities in China in 2020, and the annual mean $PM_{2.5}$ concentration of Shenzhen in 2020 (19.0 μg/m^3) has fallen below the WHO interim target 2 (25.0 μg/m^3). In addition, the total elderly population aged ≥ 65 years in Shenzhen city was 0.56 million in 2020. Benefiting from the well-developed economy, the construction of medical service systems for older adults in Shenzhen is relatively complete, which is conducive to carrying out epidemiologic studies among older adults.

Study Population

Study participants were recruited from Shenzhen Healthy Ageing Research, which was established based on the older adult health management project of the National Basic Public Health Service. The population sample was selected from the lists of all individuals who signed up at community health centers in Shenzhen. The recruitment process has been described in detail in the previous study[17]. According to the specification of Shenzhen Healthy Ageing Research, people aged ≥ 65 years were eligible to take free health examinations once a year. The health examination included a face-to-face questionnaire about basic health status and lifestyles, anthropometric tests, biochemical index measurements, and health status assessments. With an exemption of informed consent, the Shenzhen Center for Chronic Disease Control Human Ethics Committee approved our study (No. SZCCC-2021-061-01-PJ).

A total of 361,329 subjects were enrolled in Shenzhen Healthy Ageing Research from 2018 to 2020, which included 63.9% of older adults aged ≥ 65 years in Shenzhen. By strictly excluding participants who (1) did not complete the questionnaire or physical examinations, and (2) had missing information on air pollutants, we included 317,464 subjects with complete information in the final analyses (Supplementary Figure S1, available in www.besjournal.com). All subjects were followed from recruitment to the date of mortality or the study's finish (December 31, 2020), whichever came first.

Outcomes

To verify the vital status of each participant, we performed three steps. First, family physicians telephoned the participant to follow up on deaths. Second, we linked the health examination data to the China Population Death Information Registration System to determine the date and cause of death[18].

The International Statistical Classification of Diseases and Related Health Problems 10th Revision (ICD-10) was applied to code underlying causes of death. With regard to some people who were registered by the family physicians but did not have a record in the national death registration system, we further checked their vital status by telephone in the third step. In this study, all-cause mortality (A00-Z99), non-accidental mortality (A00-R99), cancer mortality (C00-C97), and cardiovascular mortality (I00-I99) were considered as outcomes.

Exposure Assessment

Daily gridded data of air pollutants were calculated from the ChinaHighAirPollutants (CHAP) dataset at 10 km^2 resolution. The CHAP dataset was produced using artificial intelligence algorithms in conjunction with big data[19]. The cross-validation results demonstrated that the accuracy of the CHAP dataset was high, where the cross-validation coefficient of determination (R^2) for PM_1, $PM_{2.5}$, PM_{10}, SO_2, NO_2, CO, and O_3 was 0.77, 0.90, 0.86, 0.84, 0.84, 0.80, and 0.87, respectively[20–22]. Depending on the geocoded residential address, we retrieved the 24-hour mean PM_1, $PM_{2.5}$, PM_{10}, SO_2, NO_2, CO, and maximum 8-hour mean O_3 concentrations for each individual. Then we calculated 3-year averages before the end date of follow-up as the main exposure.

Covariates

Referring to previous studies and available data, a series of potential confounders were taken into account[23,24]. Individual-level variables were gathered from the baseline data and categorized for sex (male, female), education (illiteracy, primary school: ≤ 6 years, middle school: 7–12 years, college and above: > 12 years), marital status [married, other (unmarried or widowed or divorced)], physical activity (never, once in a while, more than once a week, daily), smoking (never, quit or current), drinking (never, quit or current). Weight (in kilograms) divided by the square of height (in meters) was used to calculate body mass index (BMI). According to the WHO guidelines, we classified BMI into four groups: underweight, normal weight, overweight, and obese (< 18.5 kg/m², 18.5–24.9 kg/m^2, 25.0–29.9 kg/m^2, ≥ 30.0 kg/m^2)[25].

History of chronic diseases included hypertension and diabetes. We repeatedly measured blood pressure in both arms of each subject twice and used the side with the higher blood pressure value for our study. Hypertension was determined as (1) diastolic blood pressure ≥ 90 mmHg and systolic blood pressure ≥ 140 mmHg[26]; (2) taking the anti-hypertension medicine; or (3) self-reported physician-diagnosed hypertension. Subjects are required to fast for 8 hours before venous blood collection. Diabetes was determined as (1) fasting blood glucose (FBG) ≥ 7.0 mmol/L[27]; (2) taking the anti-diabetes medicine; or (3) self-reported physician-diagnosed diabetes.

Statistical Analysis

The correlation between air pollutants was assessed using Spearman's correlation analysis. The association of 3-year exposure to ambient air pollution with all-cause, non-accidental, cancer and cardiovascular mortality was quantitatively assessed using logistic regression models. Models were adjusted for baseline age, sex, education, marital status, physical activity, smoking, drinking, history of hypertension, and history of diabetes. Additionally, we used categorical analyses to assess the trend of mortality risk, while the exposure level of each air pollutant was grouped by its quartiles, and the first quartile was used as the reference group. The linear trend was examined by putting the median of each quartile spacing in the model and setting them as continuous variables. The calculated *P* value of the air pollutant was used to determine whether the trend of the odds ratio (*OR*) was statistically significant.

Stratified analyses were carried out by sex (male, female), age (aged < 75 years, aged ≥ 75 years), BMI (underweight and normal weight, overweight and obese), physical activity frequency (not daily, daily), smoking (never, quit or current), history of hypertension (no, yes), and history of diabetes (no, yes). For each subgroup, we first assessed the association of each air pollutant with different mortality outcomes and then compared the association across stratified variables by two-sample *z* tests. In each adjusted model, the stratified variable was excluded from the adjustments.

Sensitivity analyses were performed to check the stability of our results. Firstly, we assessed the association by using the 1-year exposure level before the end of follow-up. Secondly, by separately adding each air pollutant into the model, we performed two-pollutant models and compared the estimates using likelihood ratio tests. To avoid collinearity, Spearman's correlation coefficient between the pair < 0.9 was allowed to develop two-pollutant models. Thirdly, we excluded subjects who died within 1 year after joining Shenzhen Healthy Ageing

Research to disregard those who were seriously ill. Finally, we only included subjects whose follow-up time was less than the average follow-up time (1.5 years). All statistical analyses were performed in R (version 4.1.1). A two-sided *P* less than 0.05 was regarded as statistically significant.

RESULTS

From 2018 to 2020, 3,975 deaths were reported, including 3,822 from non-accidental causes, 1,425 from cancer, and 1,555 from cardiovascular diseases (Table 1). By $PM_{2.5}$ exposure categories, the number of deaths increased with increasing $PM_{2.5}$ exposure levels. Among total participants, the mean age was 70.9 years [standard deviation (SD): 5.6 years], 55.5% were female, 45.4% had middle school degrees, most of them (96.3%) were married, 62.0% had a normal BMI, 64.4% had daily physical activities. Never-smokers and never-drinkers accounted for 83.7% and 84.7% of participants, respectively. 59.9% and 27.0% of participants had a

Table 1. Baseline characteristics of the study population from 2018 to 2020

Characteristics	Overall (*n* = 317,464)	$PM_{2.5}$ exposure, (μg/m^3)				*P* value
		Quartile 1 (*n* = 79,365)	Quartile 2 (*n* = 79,317)	Quartile 3 (*n* = 79,354)	Quartile 4 (*n* = 79,427)	
Follow-up, years	1.5 ± 0.9	1.5 ± 0.9	1.5 ± 1.0	1.5 ± 0.9	1.4 ± 0.9	< 0.001
All-cause mortality	3,975	228	435	1,229	2,083	
Non-accidental mortality	3,822	219	421	1,183	1,999	
Cancer mortality	1,425	69	169	444	743	
Cardiovascular mortality	1,555	93	157	481	824	
Age, years	70.9 ± 5.6	71.3 ± 5.8	71.2 ± 5.7	70.4 ± 5.2	70.7 ± 5.6	< 0.001
Female	176,098 (55.5)	43,971 (55.4)	44,052 (55.5)	44,097 (55.6)	43,978 (55.4)	0.820
Education						< 0.001
Illiteracy	24,897 (7.8)	4,267 (5.4)	4,069 (5.1)	6,087 (7.7)	10,474 (13.2)	
Primary school	121,115 (38.2)	23,046 (29.0)	26,704 (33.7)	32,587 (41.1)	38,777 (48.8)	
Middle school	144,063 (45.4)	40,435 (50.9)	40,169 (50.6)	36,086 (45.5)	27,373 (34.5)	
College and above	27,389 (8.6)	11,617 (14.6)	8,375 (10.6)	4,594 (5.8)	2,803 (3.5)	
Married	305,580 (96.3)	75,673 (95.3)	76,725 (96.7)	77,129 (97.2)	76,052 (95.8)	< 0.001
BMI						< 0.001
Underweight	11,476 (3.6)	2,513 (3.2)	2,606 (3.3)	2,895 (3.6)	3,462 (4.4)	
Normal	196,986 (62.0)	48,892 (61.6)	48,433 (61.1)	49,329 (62.2)	50,332 (63.4)	
Overweight	97,975 (30.9)	25,069 (31.6)	25,381 (32.0)	24,436 (30.8)	23,088 (29.1)	
Obese	11,027 (3.5)	2,891 (3.6)	2,897 (3.7)	2,694 (3.4)	2,545 (3.2)	
Physical activity						< 0.001
Never	56,300 (17.7)	12,269 (15.5)	11,454 (14.4)	13,179 (16.6)	19,398 (24.4)	
Once in a while	23,201 (7.3)	5,946 (7.5)	6,565 (8.3)	5,426 (6.8)	5,264 (6.6)	
More than once a week	33,668 (10.6)	8,614 (10.9)	9,710 (12.2)	8,213 (10.3)	7,131 (9.0)	
Daily	204,295 (64.4)	52,536 (66.2)	51,588 (65.0)	52,536 (66.2)	47,634 (60.0)	
Never-smoker	265,723 (83.7)	67,130 (84.6)	68,706 (86.6)	65,531 (82.6)	64,356 (81.0)	< 0.001
Never-drinker	268,827 (84.7)	66,853 (84.2)	68,624 (86.5)	66,479 (83.8)	66,870 (84.2)	< 0.001
Hypertension	190,038 (59.9)	48,661 (61.3)	49,367 (62.2)	45,759 (57.7)	46,251 (58.2)	< 0.001
Diabetes	85,852 (27.0)	22,658 (28.5)	22,164 (27.9)	20,609 (26.0)	20,420 (25.7)	< 0.001

Note. Values are *n*, *n* (%) or mean ± SD. $PM_{2.5}$, particulate matter with an aerodynamic diameter ≤ 2.5 μm; BMI, body mass index.

history of hypertension and diabetes, respectively. Supplementary Figure S2 shows the spatial distribution of all participants.

The distribution of 3-year exposure to ambient air pollutants is shown in Table 2. The average exposure level of PM_1, $PM_{2.5}$, PM_{10}, SO_2, NO_2, CO, and O_3 was 15.1 μg/m^3, 25.3 μg/m^3, 44.0 μg/m^3, 6.9 μg/m^3, 31.7 μg/m^3, 0.68 mg/m^3, and 94.5 μg/m^3, respectively. PM_1, $PM_{2.5}$, PM_{10}, SO_2, NO_2, and CO had positive correlations with each other, while O_3 was negatively correlated with SO_2 and NO_2 (Supplementary Table S1, available in www.besjournal.com).

In the main models, 3-year exposure to PM_1, $PM_{2.5}$, PM_{10}, SO_2, and CO were significantly associated with increased risks of all-cause, non-accidental, cancer and cardiovascular mortality, and O_3 was significantly associated with increased risks of all-cause and non-accidental mortality (Table 3). For all-cause mortality, the *OR* for each 1 μg/m^3 increase in exposure to PM_1, $PM_{2.5}$, PM_{10}, SO_2, CO, and O_3 was 1.49 [95% confidence interval (*CI*): 1.46, 1.53], 1.30 (1.27, 1.32), 1.05 (1.04, 1.06), 5.84 (5.39, 6.32), 1.04 (1.04, 1.05), and 1.02 (1.00, 1.03), respectively. Outcomes for non-accidental mortality were similar to those for all-cause mortality. In terms of cancer mortality, the *OR* each 1 μg/m^3 increment in exposure to PM_1, $PM_{2.5}$, PM_{10}, SO_2, and CO was 1.46 (95% *CI*: 1.40, 1.52), 1.30 (1.26, 1.35), 1.04 (1.02, 1.06), 6.34 (5.55, 7.25), and 1.04 (1.04, 1.05), respectively. Regarding cardiovascular mortality, the *OR* per 1 μg/m^3 increase in exposure to PM_1, $PM_{2.5}$,

Table 2. Distribution of 3-year exposure to ambient air pollutants before the end date of follow-up

Air pollutant	Mean	SD	Min	Percentile			Max
				25th	50th	75th	
PM_1, μg/m^3	15.1	1.6	12.9	13.6	14.8	16.6	26.7
$PM_{2.5}$, μg/m^3	25.3	2.0	19.0	23.8	24.9	27.0	35.8
PM_{10}, μg/m^3	44.0	3.9	31.0	40.5	42.9	48.3	55.4
SO_2, μg/m^3	6.9	0.5	4.8	6.6	6.8	7.3	10.6
NO_2, μg/m^3	31.7	4.7	11.8	28.5	31.2	35.9	45.3
CO, mg/m^3	0.68	0.02	0.61	0.67	0.68	0.69	0.85
O_3, μg/m^3	94.5	2.6	80.0	92.7	94.7	95.8	102.9

Note. PM_1, particulate matter with an aerodynamic diameter ≤ 1 μm; $PM_{2.5}$, particulate matter with an aerodynamic diameter ≤ 2.5 μm; PM_{10}, particulate matter with an aerodynamic diameter ≤ 10 μm; SO_2, sulfur dioxide; NO_2, nitrogen dioxide; CO, carbon monoxide; O_3, ozone; SD, standardized deviation; Min, minimum; Max, maximum.

Table 3. Adjusted *OR*s and 95% *CI*s for per 1 μg/m^3 increase in 3-year exposure to ambient air pollution with risk of all-cause and cause-specific mortality

Air pollutant	All-cause mortality	Non-accidental mortality	Cancer mortality	Cardiovascular mortality
PM_1	1.49 (1.46, 1.53)	1.49 (1.45, 1.52)	1.46 (1.40, 1.52)	1.47 (1.42, 1.53)
$PM_{2.5}$	1.30 (1.27, 1.32)	1.29 (1.27, 1.32)	1.30 (1.26, 1.35)	1.30 (1.26, 1.34)
PM_{10}	1.05 (1.04, 1.06)	1.05 (1.04, 1.06)	1.04 (1.02, 1.06)	1.04 (1.03, 1.06)
SO_2	5.84 (5.39, 6.32)	5.87 (5.42, 6.37)	6.34 (5.55, 7.25)	5.33 (4.69, 6.07)
NO_2	1.01 (1.00, 1.01)	1.01 (1.00, 1.01)	1.01 (1.00, 1.02)	1.01 (1.00, 1.02)
CO	1.04 (1.04, 1.05)	1.04 (1.04, 1.05)	1.04 (1.04, 1.05)	1.04 (1.04, 1.05)
O_3	1.02 (1.00, 1.03)	1.02 (1.00, 1.03)	1.00 (0.98, 1.02)	1.02 (0.99, 1.04)

Note. Values are *OR* (95% *CI*), and adjusted for age, sex, race, education, marital status, BMI, physical activity, smoking, drinking, hypertension, and diabetes. PM_1, particulate matter with an aerodynamic diameter ≤ 1 μm; $PM_{2.5}$, particulate matter with an aerodynamic diameter ≤ 2.5 μm; PM_{10}, particulate matter with an aerodynamic diameter ≤ 10 μm; SO_2, sulfur dioxide; NO_2, nitrogen dioxide; CO, carbon monoxide; O_3, ozone; *OR*, odds ratio; *CI*, confidence interval.

PM_{10}, SO_2, and CO was 1.47 (95% *CI*: 1.42, 1.53), 1.30 (1.26, 1.34), 1.04 (1.03, 1.06), 5.33 (4.69, 6.07), and 1.04 (1.04, 1.05), respectively. In the categorical analyses, the mortality risks for SO_2 and CO demonstrated a monotonic increment, while the mortality risks for PM_1, $PM_{2.5}$ and PM_{10} increased at lower concentrations but levelled off or decreased at higher concentrations. The mortality risk for O_3 increased at high-level concentrations (Figure 1, Supplementary Table S2, available in www.besjournal.com). There was no significant association between NO_2 and any mortality outcomes.

In stratified analyses, we observed effect modifications by age, BMI, physical activity, smoking, and history of diabetes for different air pollutant exposures and all-cause mortality (*P* for difference < 0.05) (Table 4). With regard to age, stronger associations of PM_1 and SO_2 were found in participants younger than 75 years. For BMI, a higher association of SO_2 exposure was observed in overweight and obese subjects. For physical activity, stronger associations of PM_1, $PM_{2.5}$, PM_{10}, SO_2, CO, and O_3 exposure were observed in subjects who did physical activities daily. For smoking, a higher association of CO exposure was observed in never-smokers. For diabetes, we investigated a stronger association of SO_2 exposure in participants with a history of diabetes. Stratified analyses for non-accidental, cancer and cardiovascular mortality displayed similar trends (Supplementary Tables S3–S5, available in www.besjournal.com).

In the sensitivity analysis, using the 1-year exposure level did not change the main results visibly, despite the significant association for CO disappeared, while the significant association was found for NO_2 (Supplementary Table S6, available in www.besjournal.com). In two-pollutant models, although estimates were changed after adjustment, the association of exposure to PM_1, SO_2, and CO

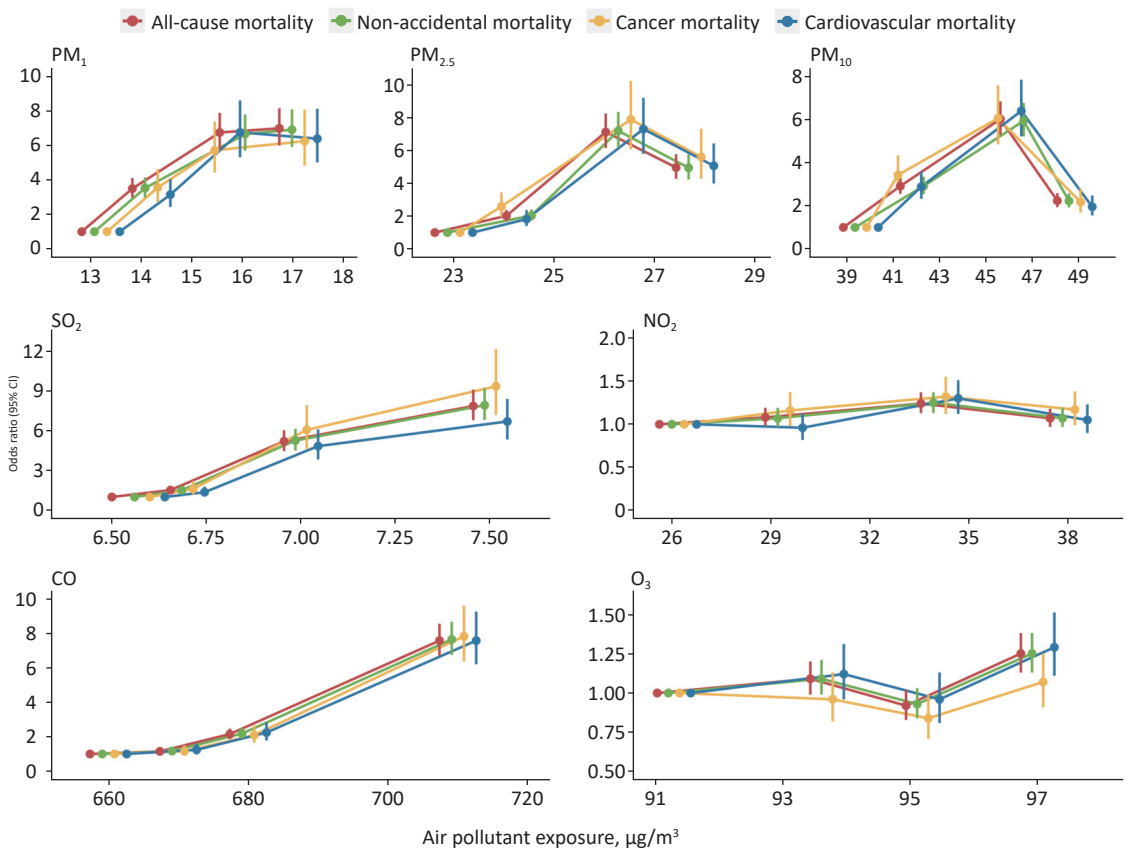

Figure 1. *OR*s and 95% *CI*s of all-cause and cause-specific mortality associated with 3-year exposure to ambient air pollution in categorical analyses. The first quartile was considered as a reference group. PM_1, particulate matter with an aerodynamic diameter ≤ 1 μm; $PM_{2.5}$, particulate matter with an aerodynamic diameter ≤ 2.5 μm; PM_{10}, particulate matter with an aerodynamic diameter ≤ 10 μm; SO_2, sulfur dioxide; NO_2, nitrogen dioxide; CO, carbon monoxide; O_3, ozone; *OR*, odds ratio; *CI*, confidence interval.

remained stable (Supplementary Tables S7–S10, available in www.besjournal.com). The analysis excluding the subjects who died within 1 year after entering Shenzhen Healthy Ageing Research yielded similar trends (Supplementary Table S11, available in www.besjournal.com). Results in the analysis with a shorter mortality follow-up also remained stable (Supplementary Table S12, available in www.besjournal.com).

DISCUSSION

This large population-based study observed that long-term PM_1, $PM_{2.5}$, PM_{10}, SO_2, and CO exposures

Table 4. Adjusted *OR*s and 95% *CI*s for per 1 μg/m^3 increase in 3-year exposure to ambient air pollution with risk of all-cause mortality in stratified analyses

Subgroup	PM_1	$PM_{2.5}$	PM_{10}	SO_2	NO_2	CO	O_3
Sex							
Male	1.50 (1.45, 1.55)	1.32 (1.28, 1.35)	1.06 (1.04, 1.07)	5.94 (5.35, 6.59)	1.01 (1.00, 1.02)	1.04 (1.04, 1.05)	1.02 (1.00, 1.04)
Female	1.49 (1.44, 1.55)	1.27 (1.23, 1.31)	1.05 (1.03, 1.06)	5.76 (5.09, 6.51)	1.00 (0.99, 1.01)	1.04 (1.04, 1.05)	1.01 (0.99, 1.03)
P for difference[a]	0.860	0.100	0.450	0.830	0.070	0.410	0.950
Age, years							
< 75	1.52 (1.46, 1.58)	1.28 (1.24, 1.32)	1.05 (1.03, 1.07)	6.31 (5.61, 7.10)	1.01 (1.00, 1.02)	1.04 (1.04, 1.05)	1.02 (1.00, 1.04)
≥ 75	1.46 (1.41, 1.51)	1.29 (1.25, 1.32)	1.05 (1.03, 1.06)	5.38 (4.84, 6.00)	1.00 (0.99, 1.01)	1.04 (1.04, 1.05)	1.00 (0.98, 1.02)
P for difference[a]	0.030	0.300	0.610	0.004	0.100	0.670	0.480
BMI, kg/m^2							
< 25.0	1.49 (1.44, 1.53)	1.29 (1.26, 1.32)	1.05 (1.04, 1.06)	5.84 (5.33, 6.41)	1.01 (1.00, 1.01)	1.04 (1.04, 1.05)	1.02 (1.00, 1.03)
≥ 25.0	1.52 (1.45, 1.59)	1.32 (1.27, 1.37)	1.06 (1.04, 1.08)	5.89 (5.04, 6.88)	1.01 (1.00, 1.03)	1.05 (1.04, 1.05)	1.02 (0.99, 1.05)
P for difference[a]	0.150	0.250	0.840	0.020	0.870	0.480	0.640
Physical activity							
Not daily	1.48 (1.43, 1.53)	1.25 (1.21, 1.29)	1.04 (1.03, 1.06)	5.14 (4.57, 5.77)	1.01 (1.00, 1.02)	1.04 (1.04, 1.04)	1.00 (0.98, 1.02)
Daily	1.51 (1.46, 1.57)	1.35 (1.31, 1.39)	1.06 (1.05, 1.08)	6.61 (5.93, 7.37)	1.01 (1.00, 1.02)	1.05 (1.04, 1.05)	1.03 (1.01, 1.05)
P for difference[a]	0.002	< 0.001	< 0.001	0.010	0.060	< 0.001	0.040
Smoking							
Never	1.49 (1.45, 1.53)	1.30 (1.27, 1.33)	1.05 (1.04, 1.06)	6.01 (5.51, 6.57)	1.01 (1.00, 1.02)	1.05 (1.04, 1.05)	1.01 (1.00, 1.03)
Quit or current	1.54 (1.45, 1.63)	1.29 (1.23, 1.36)	1.06 (1.03, 1.08)	5.14 (4.26, 6.20)	1.01 (0.99, 1.03)	1.04 (1.04, 1.04)	1.03 (1.00, 1.06)
P for difference[a]	0.416	0.587	0.933	0.079	0.954	0.004	0.810
Hypertension							
No	1.50 (1.44, 1.56)	1.31 (1.26, 1.36)	1.06 (1.04, 1.07)	5.78 (5.08, 6.59)	1.00 (0.99, 1.01)	1.05 (1.04, 1.05)	1.01 (0.98, 1.03)
Yes	1.49 (1.45, 1.54)	1.29 (1.25, 1.32)	1.05 (1.04, 1.06)	5.86 (5.30, 6.48)	1.01 (1.00, 1.02)	1.04 (1.04, 1.04)	1.02 (1.00, 1.04)
P for difference[a]	1.000	0.650	0.690	0.840	0.250	0.080	0.180
Diabetes							
No	1.48 (1.44, 1.53)	1.28 (1.25, 1.32)	1.05 (1.03, 1.06)	5.48 (4.97, 6.03)	1.00 (0.99, 1.01)	1.04 (1.04, 1.05)	1.02 (1.00, 1.03)
Yes	1.52 (1.46, 1.58)	1.32 (1.27, 1.37)	1.06 (1.04, 1.08)	6.74 (5.84, 7.77)	1.02 (1.00, 1.03)	1.04 (1.04, 1.05)	1.01 (0.99, 1.04)
P for difference[a]	0.360	0.210	0.140	0.030	0.050	0.190	0.910

Note. Values are *OR* (95% *CI*), and adjusted for age, sex, race, education, marital status, BMI, physical activity, smoking, drinking, hypertension, and diabetes. [a]*P* for difference was compared with another stratified group. Abbreviations: BMI, body mass index; PM_1, particulate matter with an aerodynamic diameter ≤ 1 μm; $PM_{2.5}$, particulate matter with an aerodynamic diameter ≤ 2.5 μm; PM_{10}, particulate matter with an aerodynamic diameter ≤ 10 μm; SO_2, sulfur dioxide; NO_2, nitrogen dioxide; CO, carbon monoxide; O_3, ozone; *OR*, odds ratio; *CI*, confidence interval.

were significantly positively associated with all-cause, non-accidental, cancer and cardiovascular mortality, and O_3 was significantly positively associated with all-cause and non-accidental mortality. Varying by subpopulations, a stronger association was detected among participants who were younger (< 75 years), overweight and obese, engaged in daily physical activities, never smoked, and had a history of diabetes.

Long-term exposure to low-level $PM_{2.5}$, NO_2, and O_3 have been linked with mortality among older adults in some large studies, especially in Australia and the United States of America. In Australia, Yu and colleagues[28] evaluated the association between low-level $PM_{2.5}$ (< 9 μg/m^3) and mortality among 0.27 million registered deaths (≥ 65 years). They discovered that the risk of non-accidental, and cardiovascular mortality increased by 5.02% (95% *CI*: 3.46%, 6.50%) and 4.08% (2.22%, 6.08%), respectively, for each 1 μg/m^3 increment in $PM_{2.5}$. In the United States of America, Yazdi and colleagues[29] developed a cohort study among more than 40 million Medicare beneficiaries (≥ 65 years) from 2000–2016 who had been restrictedly only in contact with low concentrations of $PM_{2.5}$ (< 12 μg/m^3), NO_2 [53 parts per billion (ppb), –100 μg/m^3], and O_3 (50 ppb, –107 μg/m^3). They observed an increased risk of all-cause mortality of 0.073% (95% *CI*: 0.071%, 0.076%), 0.0016% (0.0016%, 0.0021%), and 0.038% (0.037%, 0.039%) per 1 μg/m^3 increment in $PM_{2.5}$, NO_2, and O_3, respectively. Qian and colleagues[30] found similar findings in a cohort study of over 9 million Medicare beneficiaries (≥ 65 years). They observed an increased risk of all-cause mortality of 0.22% (95% *CI*: 0.21%, 0.24%) per 1 μg/m^3 increment for NO_2 and its concentration was below the WHO interim target 1 (40 μg/m^3). In contrast to recent epidemiological researches, we did not observe a significant association between NO_2 exposure and mortality. The inconsistency may be related to unadjusted confounders, such as dietary intake and socioeconomic status (SES), which could affect the results[31]. However, the estimates implied that there may be adverse effects, and we discovered the detrimental effects of NO_2 when changing the exposure window to 1 year before the end of follow-up.

Generally, compared with previous studies, the estimates in our study were larger. Evidence has shown the adverse impact of air pollution is elevated with the increasing concentration of air pollutants[32]. The concentration of air pollutants in Shenzhen was higher than that in Western countries. The concentration range of $PM_{2.5}$ was 19.0–35.8 μg/m³ in our study, whereas in Western countries, $PM_{2.5}$ concentrations were normally below 15 μg/m³. Additionally, researchers have suggested that the particular matter composition may have a more deleterious effect on health than its concentration[33]. The composition and sources of air pollutants differ among countries. Compared to other countries, China is the only country that has all the industrial categories, and the industrial scale of Shenzhen ranks at the top in China. Therefore, the composition of air pollutants in this region is more complex and may contain more harmful components.

Few studies have investigated the associations of long-term PM_1, SO_2, and CO exposure with mortality among older adults[34,35]. We observed significant associations of long-term PM_1, SO_2, and CO exposure with all-cause, non-accidental, cancer and cardiovascular mortality, which were robust in sensitive analyses. The estimates of PM_1 were higher than those of $PM_{2.5}$ and PM_{10}. PM_1 is smaller, more toxic, can reach deeper into the lung and rapidly enter the bloodstream and cells[36]. For SO_2, the relatively small range in SO_2 exposure in our study (4.8–10.6 μg/m^3) made the estimates higher than another cohort study conducted in China (*OR*: 1.008; 95% *CI*: 1.002, 1.014; each 10 μg/m^3 increment), which covered a concentration range of 16.75–189.00 μg/m^3[34]. Besides, we observed that in most studies conducted in Shenzhen, the estimates of SO_2 were much higher than that of other air pollutants[37–39]. Similar to our study, the SO_2 exposure varied in a very narrow range. However, the *OR*s of those studies conducted in areas with a higher level or wider range of SO_2 concentrations in China were not excessively elevated or markedly different from those of other pollutants. Therefore, we speculate that the main reason for the excessively high estimates of SO_2 in our study was the narrow range of concentration variation. In addition, potential exposure misclassification cannot be disregarded.

Previous studies found nonlinear associations between long-term exposure to particulate matter and mortality, with the risk rising in the range of low concentrations and leveling off at higher concentrations, which is generally consistent with our results[23,40,41]. The highly polluted environment will limit older adults from going outside, thereby reducing their exposure to air pollutants. Additionally, we observed monotonically increasing trends between SO_2 and CO exposures and mortality within a narrow range of fairly low SO_2 and CO

concentrations, which is in line with the existing evidence. Studies in developed western countries have shown a steep linear trend between long-term exposure to low-level air pollution and mortality[42–44].

In terms of effect modification, the associations of PM_1 and SO_2 with all-cause, non-accidental, and cardiovascular mortality were stronger in subjects aged < 75 years. Compared to the subjects aged ≥ 75 years, adults aged < 75 years may spend more time outdoors and be more vulnerable to being affected by air pollution[30]. We also found stronger associations of SO_2 with all-cause and non-accidental mortality in overweight and obese subjects. Evidence has substantiated that overweight and obesity could damage normal physiological functions of the body, so these individuals are more vulnerable to air pollution[45]. Besides, we found stronger associations of air pollution with all-cause and cause-specific mortality among more physically active subjects. Individuals may inhale more air pollutants when performing physical activities outside because of incremental lung ventilation, which aggravates the harmful impact of air pollution[46]. However, the estimates across subgroups were similar. The information on physical activity in this study only includes frequency, which is a relatively rough indicator. Besides, since the older adults are now retired, the demographic characteristics and lifestyles of them in the same region are close. Therefore, we are unsure of whether there is a significant difference between the estimates for the categories of physical activity. Moreover, our results showed that the estimates of CO on all-cause and non-accidental mortality in never-smokers were slightly higher than that in quit or current smokers. Similar to physical activity, the reasons for our smoking-specific observation are unclear and require further investigation. In addition, we found the association of SO_2 with all-cause and non-accidental mortality was stronger in diabetic patients. Evidence has demonstrated that diabetic patients exposed to SO_2 for a long time have greater levels of inflammatory biomarkers than non-diabetic patients[47], which has been linked to a higher risk of macrovascular events and mortality.

Oxidative stress, inflammatory responses, and epigenetic changes are now the main explanations for biological mechanisms related to air pollution and mortality. During inhalation, air pollutants are deposited on the walls of the bronchi and lungs. These foreign substances can trigger inflammatory responses and increase oxidative stress, which can lead to the destruction of the cell's structure, and finally induce or expedite the development of atherosclerosis, cardiovascular diseases, and other chronic diseases[48]. Particular carcinogens and their mixtures, such as polycyclic aromatic hydrocarbon (PAHs) and sulfur-containing compounds, can cause molecular changes through oxidative stress and low-grade, chronic inflammation, involving inactivation of tumor suppressor genes and the activation of oncogenes, abnormal induction of somatic cells, and finally leading to cancer[49].

The present research has a few advantages. Firstly, the large sample size (over 0.3 million older adults) facilitated the statistical efficacy to produce reliable results. Secondly, we obtained air pollutants data from the CHAP dataset, which completely covered the study area in space and time, and performed an exposure assessment based on individual levels. It provided a more accurate exposure assessment than previous studies that obtained data from monitoring stations. Thirdly, by conducting research among older adults in an area where air pollutant concentrations are one of the lowest in China, our study adds to the knowledge about the association between long-term low-level air pollution exposure and mortality.

However, our study also has limitations to be noted. Firstly, the extrapolation of the results was limited. The subjects in our study were aged ≥ 65 years, thus the results may not be appropriate for extrapolation to younger groups. Subjects were recruited using convenience sampling. This is an important barrier in extrapolating the results to the general population. This study was conducted in a single city with a narrow range of air pollutants, so generalizing the findings to other areas or nations should be made with caution. Secondly, the resolution of the CHAP dataset was coarse, and we used pollutant concentrations at each subject's residential address to represent individual exposure, which may have led to exposure misclassifications. However, these misclassifications are more likely to be non-differential and only underestimate the effect of air pollution on mortality towards the null[50]. Nonetheless, a possible differential misclassification cannot be ruled out. Thirdly, the exposure assessment was performed according to the residential address reported by the subject at baseline while we did not consider changes during the study period. This might have introduced bias into the results if a change in a residential address was related to the exposure level. Fourthly, information on covariates was obtained from

questionnaires completed by participants at baseline, and some of that, such as lifestyles, might have changed during the study period, thus affected the results. In addition, we did not have any information about indoor air pollution. However, Shenzhen has a high level of modernization that clean fuels have basically been used for home cooking. Therefore, we consider the bias of indoor air pollution on our results is negligible. Besides, underreporting of mortality data and inaccurate coding of the cause of death are unavoidable problems. Nonetheless, the official investigation has shown that the completeness of death registration in Guangdong Province, where Shenzhen is located, is relatively high (86.7%)[51]. The occurrence rate of coding errors in the system is low (2.73%), and the errors are mostly in underdeveloped regions[18]. Therefore, it had little impact on our findings. Finally, due to the short follow-up period, the sample size on specific causes of death was relatively small. Hence, we did not consider specific diseases as outcomes in this study. A longer follow-up would collect more deaths.

CONCLUSION

We found that among Chinese older adults, long-term exposure to low-level ambient air pollution was associated with increased mortality risks.

AUTHORS' CONTRIBUTIONS

Likun Liu and Xueli Yuan: investigation, formal analysis, and writing – original draft. Wenqing Ni: data curation, writing - review & editing. Jing Wei: data curation. Tingting Liu, Ruijun Xu, Yingxin Li, Zihua Zhong, Yi Zheng, Sihan Liang, and Rui Wang: writing - review & editing. Jian Xu: conceptualization, data curation, supervision, validation, writing - review & editing, and funding acquisition. Yuewei Liu: conceptualization, data curation, supervision, validation, writing - review & editing, and project administration.

COMPETING INTERESTS

There are no conflicts of interest among any of the authors.

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DATA AVAILABILITY

The CHAP dataset can be found at https://weijing-rs.github.io/ product.html. The Shenzhen Healthy Ageing Research and mortality data are private.

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