Association between ambient air pollution and skin health in middle-aged and older adults: the China Health and Retirement Longitudinal Study

Huiqin Chen, Le Liu, Shan Xu, Yan Li, Xiujing Ge, Xuexue Zhu, Huixian Wang, Bohua Ai*

Jiujiang Maternal and Child Health Care Hospital, Jiujiang, China

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***Corresponding author:**
Bohua Ai MD
Jiujiang Maternal and Child Health Care Hospital
Jiujiang, China
E-mail: 18379425025@163.com

Abstract

Introduction: Long-term exposure to ambient air pollution is a significant public health concern, yet its specific association with skin diseases in the Chinese middle-aged and older population remains insufficiently characterized, with limited evidence on the role of individual pollutants.

Material and methods: We analyzed prospective data from the China Health and Retirement Longitudinal Study (CHARLS), a nationally representative cohort. Participants aged ≥ 45 years from the 2011 baseline survey were followed through 2018. Annual average concentrations of $PM_{2.5}$, PM_{10}, NO_2, SO_2, and PM1 at participants' community residences were assessed using satellite-based models with 1-km resolution. Skin disease was defined by a self-reported physician diagnosis or related symptoms. Multivariable logistic regression models were used to estimate odds ratios (ORs), adjusting for demographic, socioeconomic, lifestyle, and health-related covariates. Sensitivity analyses included age-restricted (≥65 years) and incident-case cohorts, and propensity score matching.

Results: Among 7,894 participants, long-term exposure to higher levels of $PM_{2.5}$ (fully adjusted OR = 1.25, 95% CI: 1.13–1.38), PM_{10} (OR = 1.18, 95% CI: 1.04–1.32), and NO_2 (OR = 1.15, 95% CI: 1.02–1.29) was significantly associated with higher odds of skin disease. Associations for SO_2 and PM1 were not statistically significant. Results remained consistent across all sensitivity analyses, supporting the robustness of the findings.

Conclusions: This national cohort study provides evidence that long-term exposure to $PM_{2.5}$, PM_{10}, and NO_2 is associated with higher odds of skin diseases among Chinese middle-aged and older adults. These findings suggest that air pollution control may represent a potential intervention target for reducing the burden of skin disease in aging populations.

Key words: air pollution, skin diseases, China Health and Retirement Longitudinal Study (CHARLS).

Introduction

The skin, the body's largest organ, serves as a protective barrier between the internal environment and the external milieu, preventing the loss of water and electrolytes, limiting penetration of harmful substances, and resisting invasion by pathogenic microorganisms[1]. In addition to these barrier functions, the skin plays important roles in immune and metabolic processes, a complexity that renders it particularly vulnerable to environmental toxins [2]. Chemical constituents of air pollutants

can function as exogenous ligands that disrupt normal signaling pathways in skin cells [3]. At the same time, the skin is essential for maintaining cutaneous homeostasis; any damage to the skin – such as from ultraviolet radiation, seasonal changes, or air pollution – can impair local homeostasis, injure skin tissue, and compromise barrier function [4]. Global Burden of Disease estimates attribute 1.79% of the global disease burden to skin disorders, measured in disability-adjusted life years (DALYs) [5]. Previous studies estimate that approximately 25% of the population is affected by skin disease [6]. The spectrum of dermatological conditions in the elderly is broad, encompassing age-related disorders such as xerosis, pruritus, and benign tumors, as well as infection-prone diseases associated with immunosenescence, including herpes zoster and fungal infections [7]. These conditions are influenced by external factors such as environment, nutritional status, and individual lifestyle habits. Concurrently, age-related loss of skin collagen and elastin, along with other naturally occurring structural and physiological changes, reduces the viscoelastic properties of the skin and diminishes its capacity for defense and repair following injury, thereby markedly increasing susceptibility to dermatological diseases in older adults [8]. Moreover, certain inflammatory skin diseases have established pathophysiological links to systemic conditions such as cardiovascular disease and metabolic syndrome, thereby complicating comorbidity management [9–11]. Rapid population ageing in China has been accompanied by a rising prevalence of skin diseases among older adults. A survey of community-dwelling, middle-aged and older Chinese adults found that chronic pruritus was the most prevalent dermatologic condition and was strongly associated with older age, sex, higher educational attainment, alcohol consumption, hypertension, and hyperlipidemia [12].

Air pollution is a recognized risk factor for adverse health outcomes. Major emission sources include industrial activities, vehicular traffic, and household biomass combustion. Pollutants arise from both anthropogenic and natural sources and encompass particulate matter (PM), nitrogen oxides (NOx), and other hazardous gases [13]. Evidence indicates that air pollution is a risk factor for adverse health outcomes; pollutants, including fine particulate matter (PM_1, $PM_{2.5}$, PM_{10}) and nitrogen dioxide (NO_2), have been linked to age-related diseases [14, 15]. Experimental and epidemiological studies suggest that cutaneous exposure to these pollutants can induce inflammation, disrupt the skin barrier, increase oxidative stress, and impair mitochondrial function [16–18]. Prospective cohort analyses using UK Biobank data have reported associations between air pollution and higher risks of incident atopic dermatitis and psoriasis [19, 20]. However, these studies are geographically limited and may not generalize to the Chinese population; most previous studies have examined single pollutants or specific skin conditions. For example, one epidemiological study reported an association between particulate matter and inflammatory skin disorders [21]. However, research assessing the joint effects of multiple pollutants on skin disease remains limited, which hampers assessment of the risks posed by complex exposures. Evidence specifically addressing older adults is also sparse. Older individuals – because of age-related skin degeneration, immunosenescence, and a higher burden of comorbidities – may be more vulnerable to pollution-induced skin injury and have reduced reparative capacity; yet this high-risk group is frequently overlooked. In addition, air-pollution health effects may be modified by individual behaviors and lifestyle factors. Evidence on interactions between air pollutant exposure and behavioral or lifestyle factors, and on underlying mechanisms in older Chinese adults, remains sparse.

Using data from the China Health and Retirement Longitudinal Study (CHARLS), this prospective cohort examined associations between multiple ambient air pollutants and skin disease among middle-aged and older Chinese adults. Analyses adjusted for sociodemographic, socioeconomic, behavioral, and health-related factors and applied multilevel statistical models to assess determinants across hierarchical levels. By examining interactions between socioecological exposure pathways and individual biological mechanisms, the study provides epidemiological evidence to inform targeted environmental-health interventions and skin-disease prevention strategies in the context of China's aging population.

Material and methods

Study population

Data for this study were obtained from CHARLS, a nationally representative longitudinal survey initiated by the National School of Development, Peking University. CHARLS covers 150 county-level administrative units across 28 Chinese provinces and collects data on demographic characteristics, household composition, health status, healthcare and insurance, employment and pensions, income and expenditures, housing, and laboratory tests. It is designed to systematically assess health status, socioeconomic characteristics, and demographic indicators among adults aged 45 years and older. CHARLS conducted its baseline survey in 2011, with follow-up waves in 2013, 2015, 2018, and 2020. The CHARLS project was approved by the

Biomedical Ethics Review Committee of Peking University (Approval No. IRB00001052–11015), and all participants provided written informed consent. We used the 2011 baseline wave and follow-up data through 2018. Participants with missing information on exposure, outcome, or key covariates were excluded.

Main variables and covariates

Concentrations of PM_1, $PM_{2.5}$, PM_{10}, NO_2, and SO_2 used in this study were obtained from the China High-resolution Air Pollutant Dataset (CHAP) [22, 23]. CHAP is a nationwide, high-accuracy air-pollutant dataset that employs artificial intelligence to fuse multisource data – including satellite observations, ground monitoring, atmospheric reanalysis, and emission inventories – to generate long time-series, high-spatial-resolution gridded pollutant data covering China from 2000 to 2023. This study used pollutant concentrations for 2011–2018; spatial resolutions were 1 km for PM_1, $PM_{2.5}$ and PM_{10} and 10 km for NO_2 and SO_2. The dataset was evaluated by ten-fold cross-validation, with coefficients of determination (R2) between predicted and measured concentrations ranging 0.74–0.93. For exposure assignment, we matched each participant to the pollutant grid of their city and used the previous year's average concentration as the estimate of long-term exposure.

Skin disease was defined based on self-reported physician diagnosis or related symptoms recorded during follow-up. It was ascertained through a standardized questionnaire with the question: "Did s/he have any skin problems?" [24–30].

Covariates included demographic, socioeconomic, lifestyle, and health-related factors. Sociodemographic variables included age, sex (female, male), residence (rural, urban), marital status (married and living with a spouse, married but living without a spouse, others), and education level (elementary school or below, middle school or above). Health-related factors included body mass index, smoking status (non-smoker, smoker), drinking status (non-drinker, drinking less than once a month, drinking more than once a month), triglycerides, high-density lipoprotein, fasting blood glucose, systolic and diastolic blood pressure, and C-reactive protein.

Statistical analysis

Continuous variables with a normal distribution are presented as mean and standard deviation (SD), and categorical variables are presented as percentages. One-way analysis of variance (ANOVA), the Kruskal-Wallis H test, or the chi-square test was used to compare air pollutant levels and the incidence of skin disease. The main analysis was conducted in the full eligible cohort using multivariable logistic regression models. Three hierarchical models were fitted: Model 1 adjusted for age and sex; Model 2 additionally adjusted for marital status, education and residence; and Model 3 further included smoking, drinking status, BMI, and baseline health-related factors (triglycerides, high-density lipoprotein, fasting blood glucose, systolic blood pressure, diastolic blood pressure, C-reactive protein).

To evaluate the robustness of the findings, three sensitivity analyses were conducted. The first restricted the sample to adults aged ≥ 65 years at baseline to reduce heterogeneity related to age-dependent physiological skin characteristics. The second restricted the cohort to incident skin disease cases, excluding individuals who reported skin disease within two years prior to baseline. This approach minimized potential reverse causality and removed those with pre-baseline symptom onset. The third sensitivity analysis applied propensity score matching (PSM) to further mitigate confounding. Propensity scores were estimated from demographic, socioeconomic, behavioral, and health-related variables, and 1 : 1 nearest-neighbor matching with a caliper of 0.02 was used. Covariate balance was examined using standardized mean differences. All analyses were performed using R version 4.3.0.

Results

Population characteristics

Table I summarizes the characteristics of the study population. The sample comprised 7,894 participants (median age 63.4 years; 3,852 men and 4,042 women). The mean age of the skin-disease group (64.1 ±9.8) was significantly higher than that of the non-skin-disease group (63.3 ±9.4) (p = 0.015). BMI in the skin-disease group (24.1 ±3.6) was significantly higher than in the non–skin-disease group (23.8 ±3.5) (p = 0.032). There was no significant difference in sex distribution between the two groups (p = 0.245). Compared with participants without skin disease, those with skin disease were less likely to live in rural areas (p = 0.002), and were more likely to be current smokers (p = 0.006). No significant between-group differences were observed in marital status (p = 0.612), educational attainment (p = 0.521), or alcohol consumption (p = 0.788). Regarding clinical biomarkers, the skin disease group had higher mean fasting blood glucose (p = 0.018), systolic blood pressure (p = 0.045), and C-reactive protein levels (p < 0.001); triglycerides (p = 0.354), high-density lipoprotein

Table I. Characteristics of the participants at baseline (*N* = 7894)

Parameter	Total (*N* = 7894)	Skin diseases (*N* = 912)	Non-skin disease (*N* = 6982)	*P*-value
Age, mean (SD)	63.4 (9.5)	64.1 (9.8)	63.3 (9.4)	0.015
BMI, mean (SD)	23.8 (3.6)	24.1 (3.8)	23.8 (3.5)	0.032
Sex, *n* (%)				0.245
Female	4,042 (51.2)	483 (53.0)	3,559 (51.0)	
Male	3,852 (48.8)	429 (47.0)	3,423 (49.0)	
Residence, *n* (%)				0.002
Rural	4,973 (63.0)	532 (58.3)	4,441 (63.6)	
Urban	2,921 (37.0)	380 (41.7)	2,541 (36.4)	
Marital status, *n* (%)				0.612
Married and living with a spouse	6,078 (77.0)	693 (76.0)	5,385 (77.1)	
Married but living without a spouse	316 (4.0)	38 (4.2)	278 (4.0)	
Education status, *n* (%)				0.521
Elementary school or below	5,289 (67.0)	602 (66.0)	4,687 (67.1)	
Middle school or above	2,605 (33.0)	310 (34.0)	2,295 (32.9)	
Smoking status, *n* (%)				0.006
Non-smoker	5,605 (71.0)	611 (67.0)	4,994 (71.5)	
Smoker	2,289 (29.0)	301 (33.0)	1,988 (28.5)	
Drinking status, *n* (%)				0.788
Drinking less than once a month	5,052 (64.0)	575 (63.0)	4,477 (64.1)	
Drinking more than once a month	1,421 (18.0)	164 (18.0)	1,257 (18.0)	
Non-drinker	1,421 (18.0)	173 (19.0)	1,248 (17.9)	
Triglycerides, mean (SD)	1.48 (0.92)	1.51 (0.95)	1.48 (0.91)	0.354
High-density lipoprotein, mean (SD)	1.32 (0.35)	1.30 (0.36)	1.32 (0.35)	0.112
Fasting blood glucose, mean (SD)	5.94 (1.85)	6.08 (1.92)	5.92 (1.84)	0.018
Systolic blood pressure, mean (SD)	134.5 (21.2)	135.8 (22.0)	134.3 (21.1)	0.045
Diastolic blood pressure, mean (SD)	77.8 (11.5)	78.1 (11.8)	77.7 (11.4)	0.32
C-reactive protein, mean (SD)	2.45 (3.10)	2.85 (3.45)	2.40 (3.05)	< 0.001

(p = 0.112), and diastolic blood pressure (p = 0.32) were similar between groups.

Associations of air pollution with skin disease

In the three models, higher levels of NO_2, $PM_{2.5}$, and PM_{10} were consistently associated with higher odds of skin disease. In the age- and sex-adjusted model (Model 1), exposure to $PM_{2.5}$, PM_{10}, and NO_2 was associated with a higher odds of skin disease: $PM_{2.5}$ OR = 1.30 (95% CI: 1.18–1.43, p < 0.001), PM_{10} OR = 1.22 (95% CI: 1.08–1.38, p = 0.004), and NO_2 OR = 1.25 (95% CI: 1.10–1.43, p = 0.002). SO_2 showed a borderline association (OR = 1.10, 95% CI: 1.00–1.20, p = 0.042), whereas PM_1 was not statistically significant (OR = 1.10, 95% CI: 0.98–1.24, p = 0.085). After further adjustment for marital status, education, residence, and household income (Model 2), associations for $PM_{2.5}$, PM_{10}, and NO_2 persisted but were slightly attenuated: $PM_{2.5}$ OR = 1.28 (95% CI: 1.16–1.41, p < 0.001), PM_{10} OR = 1.20 (95% CI: 1.06–1.35, p = 0.008), and NO_2 OR = 1.18 (95% CI: 1.05–1.32, p = 0.015). In Model 2, neither SO_2 (OR = 1.08, 95% CI: 0.98–1.19, p = 0.088) nor PM_1 (OR = 1.08, 95% CI: 0.96–1.21, p = 0.168) reached statistical significance. In the fully adjusted model, the ORs were 1.15 (p = 0.021, 95% CI: 1.02–1.29) for NO_2, 1.25 (p = 0.0005, 95% CI: 1.13–1.38) for $PM_{2.5}$, and 1.18 (p = 0.019, 95% CI: 1.04–1.32) for PM_{10}. SO_2 and PM_1 showed weaker associations, and their estimates did not reach statistical significance in Model 3 (Table II, Figure 1).

Sensitivity analysis

The first sensitivity analysis, restricting the sample to adults aged ≥ 65 years, yielded slightly stronger estimates. Across three progressively adjusted models, long-term exposure to $PM_{2.5}$, PM_{10}, and NO_2 was consistently associated with higher

odds of incident skin disease, and the estimates remained robust after additional covariate adjustment. In Model 1, $PM_{2.5}$, PM_{10}, and NO_2 were each significantly associated with higher odds of skin disease: $PM_{2.5}$ OR = 1.35 (95% CI: 1.10–1.50, p = 0.00003), PM_{10} OR = 1.25 (95% CI: 1.11–1.41, p = 0.0004), and NO_2 OR = 1.28 (95% CI: 1.12–1.46, p = 0.0006). SO_2 showed a borderline association (OR = 1.12, 95% CI: 1.01–1.23, p = 0.03), and PM_1 was significant (OR = 1.15, 95% CI: 1.02–1.29, p = 0.022). In Model 2 (further adjusted), associations for $PM_{2.5}$, PM_{10}, and NO_2 persisted but were slightly attenuated: $PM_{2.5}$ OR = 1.31 (95% CI: 1.18–1.46, p = 0.0001), PM_{10} OR 1.22 (95% CI: 1.08–1.38, p = 0.001), and NO_2 OR = 1.23 (95% CI: 1.08–1.40, p = 0.002). In this model, neither SO_2 (OR = 1.09, 95% CI: 0.99–1.20, p = 0.071) nor PM_1 (OR = 1.12, 95% CI: 0.99–1.26, p = 0.064) reached statistical significance. In Model 3, $PM_{2.5}$ (OR = 1.28, 95% CI: 1.15–1.43, p = 0.0003), PM_{10} (OR = 1.20, 95% CI: 1.06–1.36, p = 0.005), and NO_2 (OR = 1.20, 95% CI: 1.05–1.37, p = 0.007) remained significantly associated with incident skin disease. SO_2 and PM_1 were not statistically significant (Table III, Figure 2).

In the second sensitivity analysis, the incident-only cohort had fewer events but showed a consistent pattern. After excluding all cases with skin disease reported within two years prior to baseline, the sensitivity analysis showed that

Table II. Associations between long-term exposure to air pollutants and odds of skin disease in the full cohort

Parameter	Model 1		Model 2		Model 3	
	OR (95% CI)	*p*	OR (95% CI)	*p*	OR (95% CI)	*p*
SO_2	1.10 (1.00, 1.20)	0.042	1.08 (0.98, 1.19)	0.088	1.05 (0.95, 1.17)	0.263
$PM_{2.5}$	1.30 (1.18, 1.43)	0.0001	1.28 (1.16, 1.41)	0.0003	1.25 (1.13, 1.38)	0.0005
PM_1	1.10 (0.98, 1.24)	0.085	1.08 (0.96, 1.21)	0.168	1.05 (0.94, 1.17)	0.332
PM_{10}	1.22 (1.08, 1.38)	0.004	1.20 (1.06, 1.35)	0.008	1.18 (1.04, 1.32)	0.019
NO_2	1.25 (1.10, 1.43)	0.002	1.18 (1.05, 1.32)	0.015	1.15 (1.02, 1.29)	0.021

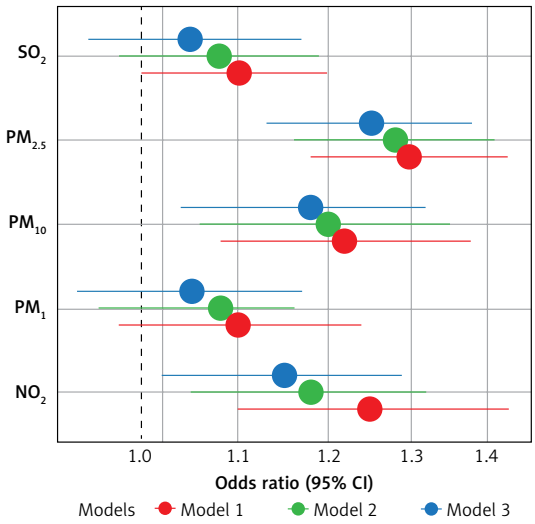

Figure 1. Association between long-term exposure to air pollutants and skin disease risk in the full cohort

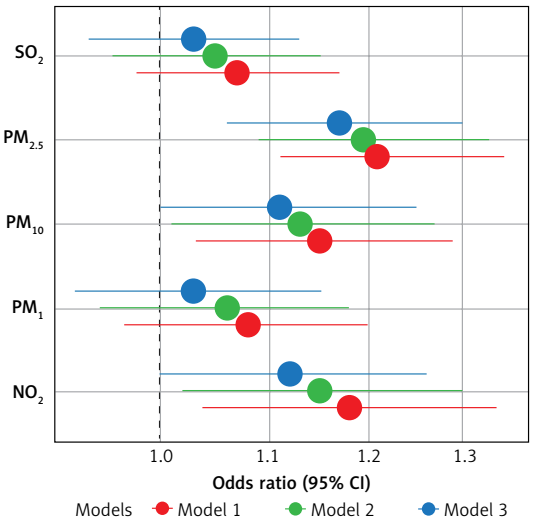

Figure 2. Association between long-term exposure to air pollutants and skin disease risk in the subgroup aged ≥ 65 years

Table III. Associations between long-term exposure to air pollutants and odds of skin disease in the subgroup aged ≥ 65 years

Parameter	Model 1		Model 2		Model 3	
	OR (95% CI)	*p*	OR (95% CI)	*p*	OR (95% CI)	*p*
SO_2	1.12 (1.01, 1.23)	0.03	1.09 (0.99, 1.20)	0.071	1.07 (0.96, 1.18)	0.182
$PM_{2.5}$	1.35 (1.22, 1.50)	0.00003	1.31 (1.18, 1.46)	0.0001	1.28 (1.15, 1.43)	0.0003
PM_1	1.15 (1.02, 1.29)	0.022	1.12 (0.99, 1.26)	0.064	1.10 (0.97, 1.24)	0.146
PM_{10}	1.25 (1.11, 1.41)	0.0004	1.22 (1.08, 1.38)	0.001	1.20 (1.06, 1.36)	0.005
NO_2	1.28 (1.12, 1.46)	0.0006	1.23 (1.08, 1.40)	0.002	1.20 (1.05, 1.37)	0.007

positive associations of $PM_{2.5}$, PM_{10}, and NO_2 with incident skin disease remained robust and were similar to the main analysis. In Model 1, $PM_{2.5}$ (OR = 1.30, 95% CI: 1.17–1.45, $p < 0.001$), PM_{10} (OR = 1.20, 95% CI: 1.07–1.35, $p = 0.002$), and NO_2 (OR = 1.22, 95% CI: 1.06–1.40, $p = 0.004$) were associated with higher odds of incident skin disease; SO_2 and PM_1 were not statistically significant. In Model 2, associations persisted but were slightly attenuated: $PM_{2.5}$ OR = 1.27 (95% CI: 1.14–1.42, $p < 0.001$), PM_{10} OR = 1.17 (95% CI: 1.04–1.32, $p = 0.01$), and NO_2 OR = 1.18 (95% CI: 1.03–1.36, $p = 0.017$); SO_2 and PM_1 remained non-significant. In Model 3, $PM_{2.5}$ (OR = 1.24, 95% CI: 1.11–1.39, $p < 0.001$), PM_{10} (OR = 1.15, 95% CI: 1.02–1.30, $p < 0.001$), and NO_2 (OR = 1.15, 95% CI: 1.00–1.33, $p = 0.049$) remained associated with higher odds of incident skin disease, with NO_2 showing borderline statistical significance; SO_2 and PM_1 estimates were not statistically significant (Table IV, Figure 3).

The third sensitivity analysis, using PSM, demonstrated that covariate balance improved markedly after matching, with all standardized mean differences reduced to below 0.1. The matched analysis produced estimates comparable to the main analysis, though slightly attenuated, suggesting that confounding may have been reduced. In the propensity-score–matched sample, with baseline characteristics balanced between

Table IV. Associations between long-term exposure to air pollutants and odds of incident skin disease

Parameter	Model 1		Model 2		Model 3	
	OR(95%CI)	*p*	OR(95%CI)	*p*	OR(95%CI)	*p*
SO_2	1.08 (0.98, 1.20)	0.118	1.06 (0.95, 1.17)	0.258	1.04 (0.93, 1.15)	0.421
$PM_{2.5}$	1.30 (1.17, 1.45)	0.00006	1.27 (1.14, 1.42)	0.0002	1.24 (1.11, 1.39)	0.0006
PM_1	1.10 (0.98, 1.24)	0.102	1.07 (0.95, 1.21)	0.224	1.05 (0.93, 1.19)	0.368
PM_{10}	1.20 (1.07, 1.35)	0.002	1.17 (1.04, 1.32)	0.01	1.15 (1.02, 1.30)	0.024
NO_2	1.22 (1.06, 1.40)	0.004	1.18 (1.03, 1.36)	0.017	1.15 (1.00, 1.33)	0.049

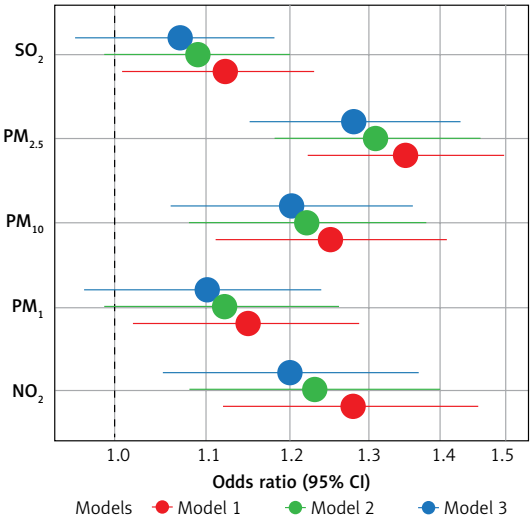

Figure 3. Association between long-term exposure to air pollutants and incident skin disease risk (excluding pre-baseline cases)

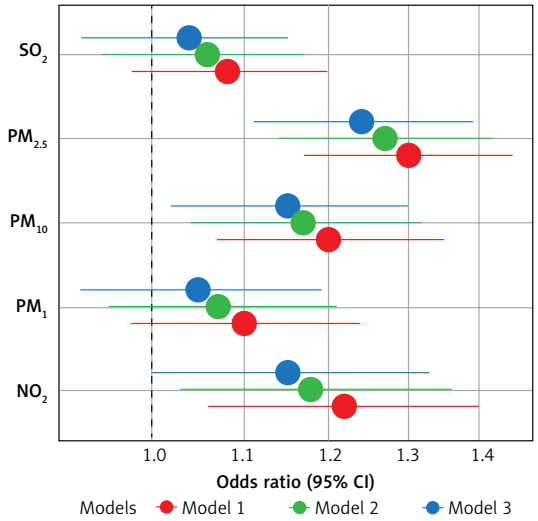

Figure 4. Association between long-term exposure to air pollutants and skin disease risk after propensity score matching

Table V. Associations between long-term exposure to air pollutants and odds of skin disease in the propensity-score-matched cohort

Parameter	Model 1		Model 2		Model 3	
	OR (95% CI)	*p*	OR (95% CI)	*p*	OR (95% CI)	*p*
SO_2	1.07 (0.98, 1.17)	0.128	1.05 (0.96, 1.15)	0.226	1.03 (0.94, 1.13)	0.412
$PM_{2.5}$	1.22 (1.11, 1.35)	0.0004	1.20 (1.09, 1.33)	0.0011	1.17 (1.06, 1.30)	0.0028
PM_1	1.08 (0.97, 1.20)	0.162	1.06 (0.95, 1.18)	0.271	1.03 (0.93, 1.15)	0.478
PM_{10}	1.15 (1.03, 1.29)	0.016	1.13 (1.01, 1.27)	0.032	1.11 (1.00, 1.25)	0.046
NO_2	1.18 (1.04, 1.34)	0.011	1.15 (1.02, 1.30)	0.024	1.12 (1.00, 1.26)	0.049

groups, long-term exposure to $PM_{2.5}$, PM_{10}, and NO_2 remained significantly associated with higher odds of skin disease, whereas associations for SO_2 and PM_1 were not statistically significant. In Model 1, $PM_{2.5}$ (OR = 1.22, 95% CI: 1.11–1.35, p = 0.0004), PM_{10} (OR = 1.15, 95% CI: 1.03–1.29, p = 0.016), and NO_2 (OR = 1.18, 95% CI: 1.04–1.34, p = 0.011) were significantly associated with higher odd of skin disease; SO_2 and PM_1 were not statistically significant. In Model 2, associations persisted but were slightly attenuated: $PM_{2.5}$ (OR = 1.20, 95% CI: 1.09–1.33, p = 0.0011), PM_{10} (OR = 1.13, 95% CI: 1.01–1.27, p = 0.032), and NO_2 (OR = 1.15, 95% CI: 1.02–1.30, p = 0.024); SO_2 and PM_1 remained non-significant. In Model 3, $PM_{2.5}$ (OR = 1.17, 95% CI: 1.06–1.30, p = 0.0028), PM_{10} (OR = 1.11, 95% CI: 1.00–1.25, p = 0.046), and NO_2 (OR = 1.12, 95% CI: 1.00–1.26, p = 0.049) remained significantly associated with higher odds of skin disease, with the NO_2 association borderline statistically significant; associations for SO_2 and PM_1 were not statistically significant (Table V, Figure 4).

Across all analyses – including the main cohort, the ≥ 65 subgroup, the incident-case cohort, and the PSM-matched population – the pollutants $PM_{2.5}$, NO_2, and PM_{10} consistently showed positive associations with skin disease. The robustness of these findings across multiple analytic strategies strengthens the evidence for an association between air pollution and skin disease among middle-aged and older Chinese adults.

Discussion

Using a nationally representative cohort of middle-aged and older Chinese adults, this prospective study evaluated the longitudinal association between long-term ambient air pollution exposure and incident skin disease. Exposure to higher concentrations of $PM_{2.5}$, PM_{10}, and NO_2 was independently associated with higher odds of incident skin disease; these associations remained robust after adjustment for sociodemographic factors (age, sex, residence), socioeconomic status, health behaviors (smoking, alcohol use), and baseline health-related factors. Findings were consistent in sensitivity analyses restricted to incident cases and after propensity score matching to reduce potential reverse causation and residual confounding, strengthening confidence in the observed associations. In fully adjusted models, however, long-term exposure to sulfur dioxide (SO_2) or ultrafine particles (PM_1) were not significantly associated with the odds of skin disease. By focusing on China's large, understudied older population and applying a multi-pollutant framework with rigorous confounder control, this study clarifies the effects of complex pollutant mixtures on skin health in older adults. These findings provide important epidemiological evidence on the impact of ambient air pollution on skin health among middle-aged and older adults.

In this study, $PM_{2.5}$ and PM_{10} showed robust positive associations with skin disease, a pattern consistent with the physicochemical properties of these pollutants. Particulate matter can penetrate intact skin (likely via hair follicles), disrupt the epidermal barrier, and induce cutaneous inflammation in mouse models. PM also adsorbs harmful compounds such as polycyclic aromatic hydrocarbons (PAHs), which act as ligands for the aryl hydrocarbon receptor (AhR). AhR is a widely expressed transcription factor that functions as an environmental sensor and regulator of immune responses, cell differentiation, metabolic homeostasis, and tissue equilibrium [31, 32]. Activation of AhR by PAHs elevates expression of cytochrome P450 1A1 (CYP1A1) and related genes [32], increasing reactive oxygen species (ROS) production; ROS then promote keratinocyte release of proinflammatory cytokines (e.g., IL-6, TNF-α) and cellular injury [33]. Air pollutants, including PAHs in PM, can also directly stimulate NADPH oxidases (NOX), particularly NOX2 and NOX4, thereby enhancing ROS generation. NOX complexes on the plasma membranes of phagocytes, endothelial cells, and epithelial cells are assembled and activated by cytokines, bacterial lipopolysaccharide, and similar stimuli, catalyzing formation of superoxide anion and hydrogen peroxide. These ROS trigger lipid peroxidation, DNA damage, and release of proinflammatory mediators, directly impairing skin barrier function and exacerbating inflammation [34–37]. In the present study, NO_2 was also significantly associated with skin disease. As a traffic-related ambient pollutant [38], nitrogen dioxide increases oxidative stress and compromises barrier integrity, producing observable alterations of the skin surface [39].

SO_2 and PM_1 showed weaker associations with skin disease in this study. This finding may reflect the rapid atmospheric conversion of SO_2 to sulfate particles [40, 41], which would limit SO_2's direct irritant effects on the skin. A study also found that a higher sulfate fraction in $PM_{2.5}$ was associated with increased risk of autoimmune skin disease [42]. Greater uncertainty in PM_1 exposure assessment, or differences in the biological pathways by which PM_1 induces skin pathology compared with $PM_{2.5}$, may also explain the attenuated association. These possibilities warrant evaluation in future research using more refined exposure-assessment methods and mechanistic studies.

After sequentially adjusting for demographic, socioeconomic, lifestyle, and health-related confounders, the estimates for the primary pollutants remained stable and statistically significant, indi-

cating that the associations were independent of these known factors. Three sensitivity analyses, each addressing different potential biases, further supported the robustness of the findings. When the analysis was restricted to participants aged ≥ 65 years to reduce heterogeneity associated with age, the associations persisted and were slightly stronger, suggesting that older adults may be more susceptible to the effects of air pollution. Excluding participants with skin disease during the two years preceding baseline to create an incident-case cohort reduced the potential for reverse causation and strengthened the temporal ordering of pollutant exposure and subsequent disease onset. Finally, propensity score matching generated a sample balanced across baseline covariates; results were consistent with the primary analysis, suggesting that measured confounding may have been reduced. Overall, the consistency of these analyses strengthens the evidence for an association between air pollution and skin disease, although causality cannot be established.

This study has several strengths: a prospective cohort design with a large, nationally representative sample; high-spatial-resolution satellite models to estimate individual long-term pollutant exposure; and rigorous confounding control with multiple robustness checks, including multivariable adjustment and several sensitivity analyses (subgroup analyses, incident-case analysis, and propensity score matching).

Nevertheless, several limitations merit consideration. First, skin-disease ascertainment relied primarily on self-report and may be subject to misclassification. Although we combined physician diagnosis and relevant symptoms to improve validity, mild cases that did not seek medical care may have been missed. Second, despite using high-quality exposure models, exposure assignment based on residential or community location cannot fully capture individual mobility or indoor exposures, which may introduce exposure measurement error. Third, residual confounding from unmeasured or imprecisely measured factors (for example, ultraviolet exposure, occupational exposures, and skincare practices) may persist despite adjustment for numerous covariates. Finally, the study population comprised middle-aged and older adults in China; caution is warranted when generalizing findings to other age groups or ethnic populations.

In conclusions, using a large, nationally representative prospective cohort, we found that exposure to ambient air pollutants – particularly $PM_{2.5}$, PM_{10}, and NO_2 – was associated with higher odds of skin disease among middle-aged and older Chinese adults. These associations remained robust after extensive multivariable adjustment and sensitivity analyses, supporting air pollution as a potentially modifiable environmental factor associated with skin disease. In contrast, SO_2 and PM1 were not significantly associated with the odds of skin disease; this may reflect differences in pollutant composition, limitations in exposure assessment, or distinct biological mechanisms, which warrant further investigation. The study provides strong epidemiological evidence that air pollution may be an important environmental factor associated with skin diseases among middle-aged and older adults. Limitations include possible outcome misclassification from self-report and exposure measurement error due to exposure assignment that may not capture individual activity patterns. Future research should apply objective diagnostic criteria, incorporate individualized dynamic exposure monitoring, and investigate pollutant-specific pathways as well as gene–environment and other environmental interactions to better clarify links between air pollution and skin health and to inform targeted prevention and intervention strategies.

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Ethical approval

The CHARLS project was approved by the Biomedical Ethics Review Committee of Peking University (Approval No. IRB00001052–11015), and all participants provided written informed consent.

Conflict of interest

The authors declare no conflict of interest.

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