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research article

Long-term exposure to air pollution and incidence of asthma and chronic obstructive pulmonary disease among adults aged 50 years and older: the English Longitudinal Study of Ageing

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Air pollution is a recognised risk factor for respiratory diseases, but its long-term effect on obstructive lung diseases in older adults remains inconclusive. We investigated the associations between long-term air pollution exposure and asthma and chronic obstructive pulmonary disease (COPD) incidence in 11,391 respondents (aged ≥ 50 years) in the English Longitudinal Study of Ageing, recruited in 2002–03 and followed up to 2017 for new asthma and COPD diagnoses or hospital records. Annual mean levels of fine particulate matter ($PM_{2.5}$), nitrogen dioxide (NO_2) and ozone (O_3) with a one-year lag were assessed at respondents' residential addresses. Time-varying Cox models adjusted for confounders were used and effect modifiers were tested. Over the 15 follow-up years, we observed 343 asthma diagnoses, 531 COPD diagnoses and 236 COPD hospitalisations. $PM_{2.5}$ was associated with asthma and COPD diagnosis and COPD hospitalisations. O_3 was associated with COPD diagnosis and hospitalisation. Stronger associations of $PM_{2.5}$ with asthma diagnosis were observed in ever-smokers, and stronger associations of $PM_{2.5}$ and NO_2 with COPD hospitalisation were observed in people aged 65+ and those in deprived areas; stronger associations of O_3 with COPD hospitalisation were observed in men. These findings underscore the significant risk of air pollution exposure in developing asthma and COPD in adults, highlighting the need for further studies to strengthen the evidence and identify susceptible populations.

Keywords long-term exposure to air pollution • asthma • chronic obstructive pulmonary disease • adult population • longitudinal cohort

Key messages

- Long-term exposure to $PM_{2.5}$ and O_3 is linked to higher asthma and COPD cases, while exposure to NO_2 shows no strong associations.
- Smokers, physically active individuals, older adults and those in deprived areas face higher risks.
- Reducing air pollution exposure is crucial to protecting public health, especially for at-risk populations.

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Introduction

Chronic respiratory diseases (CRDs) are among the leading causes of death, responsible for 4 million deaths and 455 million prevalence cases globally, and chronic obstructive pulmonary disease (COPD) and asthma are the two most prevalent CRDs (Momtazmanesh et al, 2023). Air pollution is a major risk factor for the development and aggravation of respiratory diseases (World Health Organization, 2016), causing about 1.7 million CRD deaths globally, second only to smoking (Momtazmanesh et al, 2023). Inhaled air pollutants trigger oxidative stress and chronic inflammation in the airways, disrupting epithelial barriers and promoting airway remodelling and hyperreactivity. Pathophysiological processes, such as impaired mucociliary clearance and heightened immune responses, provide a biological pathway for expecting elevated incidence of asthma and COPD in areas with polluted air (Kelly, 2003; Pope and Dockery, 2006). While strong epidemiological evidence links short-term air pollution exposure to increased CRD mortality and morbidity (Brunekreef and Holgate, 2002), findings on long-term exposure and disease incidence in adults aged 50 and over remain limited and inconsistent (Boogaard et al, 2022). Studies have suggested that nitrogen dioxide (NO_2) is associated with increased asthma (Andersen et al, 2012; Wu et al, 2020; Liu et al, 2021b; 2021d), though some found no effect (Gandini et al, 2018; Jeong et al, 2018). Findings for fine particulate matter ($PM_{2.5}$) have been mixed, with several studies reporting a positive association with asthma (Young et al, 2014; Lee et al, 2021; Liu et al, 2021b; 2021d; Zhang et al, 2021), while others found no significant link (Rice et al, 2015; Jeong et al, 2018; Salimi et al, 2018; Wu et al, 2020). Asthma has also been linked to increased exposure to ozone (O_3; Lee et al, 2021). Air pollutants $PM_{2.5}$ (Gan et al, 2013; Schikowski et al, 2014; Atkinson et al, 2015; Guo et al, 2018; Han et al, 2020; Clarke et al, 2021; Liu et al, 2021a; 2021c; Zhang et al, 2021; Lo et al, 2022; Wang et al, 2022b), NO_2 (Andersen et al, 2011; Doiron et al, 2021; Liu et al, 2021a; 2021c; Wang et al, 2022a) and O_3 (Shin et al, 2021) have been linked to varying degrees to increased risk of COPD outcomes. Some studies in the UK and London examined long-term air pollution exposure and COPD

hospitalisation (Atkinson et al, 2015; Carey et al, 2016; Chen et al, 2022). However, only the UK Biobank study (Chen et al, 2022) found a positive association with $PM_{2.5}$; other pollutants were not assessed. Moreover, most previous studies lack nationally representative samples with long follow-up periods (Andersen et al, 2012; Gan et al, 2013; Atkinson et al, 2015; Salimi et al, 2018; Han et al, 2020; Lee et al, 2021).

While older individuals tend to have decline in pulmonary reserve, reduced immune responses and a higher baseline prevalence of comorbidities, making them particularly susceptible to the adverse respiratory effects of air pollutants (Marengoni et al, 2011; Shumake et al, 2013), most previous research has not focused explicitly on older adults. With global populations ageing rapidly, understanding how long-term exposure to ambient air pollution is associated with CRD in older cohorts is critical for preparing future healthcare needs, guiding policy interventions and targeting preventive strategies to the most susceptible subgroups.

This study contributes to the literature by investigating associations between long-term exposure to $PM_{2.5}$, NO_2 and O_3 and asthma and COPD incidence in older adults. Using data from the nationally representative English Longitudinal Study of Ageing (ELSA), which includes detailed individual health behaviours and socioeconomic status, we analyse both hospitalisation records and self-reported doctor diagnoses from participants interviewed between 2002 and 2003 and followed up until 2017.

Materials and methods

Study population

We used data from ELSA, a nationally representative study of adults aged 50 years and over residing in private households in England. Details of the study are well documented elsewhere (Steptoe et al, 2013). Briefly, ELSA began in 2002–03 (Wave 1, baseline) to investigate various aspects of the ageing process and has continued biennially, with ten waves of data collection completed to date.

Analytical sample

Of the 11,391 respondents in ELSA Wave 1, we excluded 696 who had moved to their Wave 1 address in the interview year or had missing information on air pollution and other covariates, resulting in 10,695 subjects (details are provided in Supplementary Figure S1). Additionally, we excluded 1,246 respondents with previous asthma diagnoses and 696 COPD diagnoses at baseline, leaving a total sample of 9,449 individuals at risk of incident disease in the self-reported asthma group and 9,999 in the self-reported COPD group. For hospitalisation incidence outcomes, we excluded 1,596 and 1,674 participants from the self-reported asthma and COPD samples, respectively, due to a lack of consent for linkage to Hospital Episode Statistics (HES) and death registry. Lastly, we excluded two participants from the COPD hospitalisation sample due to a recorded COPD hospitalisation before baseline. As a result, the analytical sample of asthma hospitalisation consisted of 7,853 participants and the sample of COPD hospitalisation consisted of 8,325 participants.

Outcome definition

Asthma or COPD incidence was identified from two sources: (1) self-reported doctor diagnosis and (2) hospitalisation records. During the first interview, participants were asked whether a doctor or nurse had ever told them they had asthma or COPD. At each follow-up interview, they were asked if they had received a new diagnosis of asthma or COPD since the previous interview. For the purpose of this work, the date of a new diagnosis was assumed to be the survey date (the month and year of the interview), as ELSA did not collect the date when new diagnoses were received.

For hospitalisation records, data from participants who provided content were linked to the Admitted Patient Care dataset (available from 11 February 1997) and outpatient appointment data (available from 1 April 2003) within the HES. Incident hospitalisation was defined as the first hospitalisation with a primary diagnosis of asthma or COPD between baseline (2002 or 2003) and 31 December 2017, based on the International Classification of Diseases 10th Revision codes: J45–J46 for asthma and J41–J44 for COPD. The date of hospitalisation was obtained from HES records.

Air pollution exposure assessment

We assessed annual average exposure levels to $PM_{2.5}$, NO_2 and O_3 during the follow-up period using global spatio-temporal models linked to participants' residential addresses at each survey wave, as part of the Gateway to Global Aging Data project (https://g2aging.org/home; Di Gessa et al, 2025).

$PM_{2.5}$ levels were estimated monthly from 1998 using the V5.GL.04 model (van Donkelaar et al, 2021), which integrates data from ground-based monitoring stations, satellites, chemical transport models, meteorological models and area-level characteristics. The model provides high-resolution predictions (0.01° × 0.01° or ~1 km^2) and demonstrates strong agreement with ground-level observations (cross-validated $R^2 = 0.9$).

NO_2 concentrations (parts per billion air molecules – ppb) were estimated using a land-use regression model (Larkin et al, 2023) with a resolution of 50 m × 50 m. This model incorporates data from the Ozone Monitoring Instrument satellite, road networks, built environment characteristics and meteorological variables, providing moderate explanatory power (cross-validated $R^2 = 0.6$). NO_2 estimates were available starting in 2005; therefore, 2005 values were assigned to participants' residential addresses for Wave 1 (2002–03) and Wave 2 (2004–05).

O_3 levels (ppb) were estimated from 1990 using a Bayesian Maximum Entropy fusion model (Becker et al, 2023), which combines multiple atmospheric chemistry models with ground-level O_3 monitoring data. This model operates at a coarser spatial resolution (0.1° × 0.1° or ~11 km × 11 km) but maintains strong predictive performance (cross-validated $R^2 = 0.8$). O_3 exposure was defined as the highest six-month running average of daily eight-hour maximum concentrations.

For years when participants were not surveyed but for which we had hospital records, we assumed they continued to reside at the residential address recorded in their most recent available wave.

Covariates

Baseline characteristics (2002–03) included birth year and month, sex, marital status, smoking status, physical activity level and wealth. Marital status was categorised into a binary variable indicating whether or not people were living with a partner. Smoking status was defined as never-smoker versus ever-smoker or current smoker. Physical activity was categorised as inactive (no activity on a weekly basis or mildly intensive activity less than once a week) or active (at least moderately active at least once a week; Hamer et al, 2014). Wealth was used as a measure of the permanent socioeconomic status of older people (Zaninotto et al, 2020), and it was defined as the sum of net financial wealth and net housing wealth less all debts. Wealth tertiles were derived from the continuous measure. We also assessed area-level socioeconomic status using the 2004 Index of Multiple Deprivation (IMD) in England and categorised the score into tertiles. The IMD is a composite measure of relative deprivation of small areas in England, combining area-level socioeconomic status indicators from income, employment, education, health deprivation and disability, crime, barriers to housing and services, and living environment into a single score (Noble et al, 2004).

Statistical analysis

We used time-varying Cox models to assess the associations between long-term exposure to each pollutant, modelled individually, and asthma and COPD incidence, treating air pollutants as time-varying variables, while covariates were assessed at baseline. Age was used as the underlying timescale, and participants were censored at the age of their last study wave, death or the end of follow-up on 31 December 2017, whichever came first.

Associations were first examined using crude models. Model 1 was stratified by sex and adjusted for the calendar year using a spline term with four degrees of freedom. Model 1 was then further adjusted for individual health behaviours, including smoking status and physical activity level (Model 2a); marital status and wealth tertiles (Model 2b); and area-level socioeconomic status, measured using the IMD in tertiles (Model 2c). The main model, Model 3, included all covariates.

We presented a visual representation of the non-linear association between each air pollutant and the outcomes using a natural spline function with two degrees of freedom, selected based on the lowest Bayesian information criterion (BIC) from models tested with two to four degrees of freedom (BIC results are not shown). Deviation from linearity was assessed using likelihood ratio tests.

When a significant association between a pollutant and an outcome was found, we further examined potential effect modification by testing interactions between the pollutant and each variable, including sex, age, smoking status, physical activity, marital status, wealth and IMD. We used the likelihood ratio test to assess whether each variable acted as a potential effect modifier.

All statistical tests were two-sided, and p-values of < 0.05 were used as statistical significance. Estimates of linear associations were expressed as hazard ratios (HRs) with 95 per cent confidence intervals (CIs) per the interquartile range increase of each pollutant, or per 5 $\mu g/m^3$ for $PM_{2.5}$ and 10 $\mu g/m^3$ for NO_2 and O_3, for comparison with previous literature. All analyses and figure presentations were undertaken in R, version 4.3.2.

We ran two sensitivity analyses to assess the robustness of the results. First, we repeated fully adjusted models, further adjusting for the presence of other pollutants. Second, we further adjusted main Model 3 for two additional individual socioeconomic status variables: educational attainment (college and above; A level; below A level) and occupational social class (using National Statistics Socio-Economic Classifications: professional and managerial; intermediate; manual and routine; other). A multicollinearity test was performed to ensure that these additional individual variables were not highly correlated with each other and with wealth. We also tested for effect modifications between each pollutant and educational attainment and social class.

Results

In Supplementary Table S1, we report the characteristics of participants included in the analytical sample compared to those who were excluded. Excluded participants were less likely to develop the studied outcomes, be physically active, have a partner, belong to the highest wealth tertile or reside in a less deprived area at baseline. Additionally, individuals excluded from the self-reported COPD and COPD hospitalisation cohorts were more likely to be older, be a current smoker and have been exposed to slightly higher levels of $PM_{2.5}$ and NO_2 in 2002–03.

Descriptive statistics

Among the 9,449 participants in the self-reported asthma sample, 343 were newly diagnosed over a mean follow-up period of 8.2 years. In the asthma hospitalisation sample, 14 out of 7,853 participants were newly hospitalised over a mean follow-up of 8.9 years; however, this group was excluded from the analyses due to the small case size. Among the 9,999 participants in the self-reported COPD sample, 531 were newly diagnosed over a mean follow-up of 8.3 years. In the COPD hospitalisation sample, 236 out of 8,325 participants were newly hospitalised over a mean follow-up of 8.9 years.

Participants diagnosed with asthma were younger and more likely to be a current smoker at baseline compared to those not diagnosed (Table 1). Those diagnosed with or hospitalised for COPD were more likely to be male, a current smoker, physically inactive, unpartnered, have lower wealth and reside in a more deprived area at baseline.

Association between air pollution and obstructive lung diseases

Table 3 presents the linear associations from models with different covariate adjustments. For $PM_{2.5}$, positive associations were observed with all outcomes in crude models. For example, per interquartile range of 1.6 $\mu g/m^3$, the estimated HR was 1.14 (95 per cent CI: 1.01–1.28) for asthma diagnosis. For COPD, we observed stronger associations for hospitalisation (HR: 1.16, 95 per cent CI: 1.00–1.34) than for diagnosis (HR: 1.08, 95 per cent CI: 0.96–1.19). These associations remained robust in the fully adjusted model. For NO_2, we found no evidence of associations with asthma or COPD diagnosis in crude models, while we observed a protective effect for COPD diagnosis after adjustment for covariates (Model 3). A positive association

Table 1: Baseline characteristics of the sample according to the diagnosis at the end of follow-up, England, 2002–17

Characteristics	Self-reported asthma cohort (N = 9,449)			Self-reported COPD cohort (N = 9,999)			COPD hospitalisation cohort (N = 8,325)		
	Cases (N = 343)	Non-cases (N = 9,106)	*p*[a]	Cases (N = 531)	Non-cases (N = 9,468)	*p*[a]	Cases (N = 236)	Non-cases (N = 8,089)	*p*[a]
Age (years), mean ± SD	64.2 ± 9.2	65.8 ± 10.3	0.01	64.8 ± 9.0	65.6 ± 10.3	0.13	67.5 ± 9.0	64.9 ± 10.0	< .01
Sex, N (%)			0.93			0.01			0.02
Male	161 (46.9)	4,239 (46.6)		272 (51.2)	4,284 (45.2)		127 (53.8)	3,727 (46.1)	
Female	182 (53.1)	4,867 (53.4)		259 (48.8)	5,184 (54.8)		109 (46.2)	4,362 (53.9)	
Smoking status, N (%)			< .01			< .01			< .01
Never-smoker	93 (27.1)	3,279 (36.0)		97 (18.3)	3,554 (37.5)		17 (7.2)	2,990 (37.0)	
Ever-smoker	156 (45.5)	4,250 (46.7)		224 (42.2)	4,428 (46.8)		89 (37.7)	3,816 (47.2)	
Current smoker	94 (27.4)	1,577 (17.3)		210 (39.5)	1,486 (15.7)		130 (55.1)	1,283 (15.9)	
Physical activity, N (%)			0.12			< .01			< .01
Inactive	96 (28.0)	2,199 (24.1)		159 (29.9)	2,245 (23.7)		84 (35.6)	1,799 (22.2)	
Active	247 (72.0)	6,907 (75.9)		372 (70.1)	7,223 (76.3)		152 (64.4)	6,290 (77.8)	
Marital status, N (%)			0.77			0.01			< .01
Partnered	234 (68.2)	6,295 (69.1)		340 (64.0)	6,598 (69.7)		136 (57.6)	5,762 (71.2)	
Unpartnered	109 (31.8)	2,811 (30.9)		191 (36.0)	2,870 (30.3)		100 (42.4)	2,327 (28.8)	

(*Continued*)

Table 1: Continued

Characteristics	Self-reported asthma cohort (N = 9,449)			Self-reported COPD cohort (N = 9,999)			COPD hospitalisation cohort (N = 8,325)		
	Cases (N = 343)	Non-cases (N = 9,106)	p^a	Cases (N = 531)	Non-cases (N = 9,468)	p^a	Cases (N = 236)	Non-cases (N = 8,089)	p^a
Tertile of wealth, N (%)			0.20			< .01			< .01
Poorest	119 (34.7)	2,937 (32.3)		234 (44.1)	2,935 (31.0)		136 (57.6)	2,465 (30.5)	
Middle	122 (35.6)	3,036 (33.3)		177 (33.3)	3,214 (33.9)		67 (28.4)	2,781 (34.4)	
Richest	102 (29.7)	3,133 (34.4)		120 (22.6)	3,319 (35.1)		33 (14.0)	2,843 (35.1)	
IMD, N (%)			0.08			< .01			< .01
Least deprived	105 (30.6)	3,073 (33.7)		133 (25.0)	3,264 (34.5)		37 (15.7)	2,813 (34.8)	
Middle deprived	107 (31.2)	3,079 (33.8)		180 (33.9)	3,183 (33.6)		71 (30.1)	2,757 (34.1)	
Most deprived	131 (38.2)	2,954 (32.4)		218 (41.1)	3,021 (31.9)		128 (54.2)	2,519 (31.1)	

Notes: IMD – Index of Multiple Deprivation; N – number; SD – standard deviation; NO_2 – nitrogen dioxide; O_3 – ozone; $PM_{2.5}$ – Particulate matter aerodynamic diameter < 2.5 µm.
[a] *p*-values are from a *t*-test for numeric variables and a chi-square test for categorical variables to test the difference of baseline characteristics between cases and non-cases.
The annual mean levels of air pollutants across the studied samples are 12.4–12.5 µg/m³ (interquartile range [IQR]: 1.6 µg/m³) for $PM_{2.5}$, 12.5–12.6 ppb (IQR: 5.00 ppb) for NO_2 and 34.7–34.8 ppb (IQR: 4.11–4.24 ppb) for O_3 (Table 2). $PM_{2.5}$ shows a weak positive correlation with NO_2 (Spearman's rank correlation coefficient, $r = 0.2$) but no correlation with O_3 ($r = 0.0$), while NO_2 is negatively correlated with O_3 ($r = -0.3$).

Table 2: Descriptive statistics of each pollutant according to asthma and COPD outcomes, England, 2002–17

Outcome	Air pollutants	Mean ± SD	IQR	Min	Percentiles			Max	Correlation[a]	
					25th	50th	75th		With $PM_{2.5}$	With NO_2
Asthma diagnosis	$PM_{2.5}$ ($\mu g/m^3$)	12.5 ± 1.5	1.60	7.2	11.6	12.4	13.2	21.7	-	0.23
	NO_2 (ppb)	12.6 ± 3.5	5.00	2.0	10.0	13.0	15.0	27.0	0.23	-
	O_3 (ppb)	34.8 ± 3.5	4.24	16.4	32.7	34.8	37.0	50.9	0.01	–0.26
COPD diagnosis	$PM_{2.5}$ ($\mu g/m^3$)	12.5 ± 1.5	1.60	6.8	11.6	12.4	13.2	21.7	-	0.23
	NO_2 (ppb)	12.6 ± 3.5	5.00	2.0	10.0	13.0	15.0	27.0	0.23	-
	O_3 (ppb)	34.8 ± 3.5	4.23	16.4	32.7	34.8	37.0	50.9	0.02	–0.26
COPD hospitalisation	$PM_{2.5}$ ($\mu g/m^3$)	12.4 ± 1.5	1.60	6.7	11.5	12.3	13.1	21.7	-	0.22
	NO_2 (ppb)	12.5 ± 3.5	5.00	2.0	10.0	13.0	15.0	27.0	0.22	-
	O_3 (ppb)	34.7 ± 3.4	4.11	16.4	32.7	34.7	36.8	50.9	0.06	–0.27

Notes: IQR – interquartile range; SD – standard deviation; ppb – parts per billion air molecules; $\mu g/m^3$ – micrograms per cubic metre of air; NO_2 – nitrogen dioxide; O_3 – ozone; $PM_{2.5}$ – particulate matter aerodynamic diameter < 2.5µm.
[a] Spearman's rank correlation coefficient.

with COPD hospitalisation was found in the crude model, which became weak and statistically non-significant in the fully adjusted model, especially when adjusting for area-level socioeconomic status (Model 2c). For O_3, positive associations were observed only with COPD diagnosis and hospitalisation (HR: 1.08, 95 per cent CI: 0.97–1.21 per 4.23 ppb for diagnosis; HR: 1.28, 95 per cent CI: 1.07–1.52 per 4.11 ppb for hospitalisation) in fully adjusted models.

We did not observe evidence of deviation from linearity of associations for any combination of air pollutants and outcomes (Figure 1), except for $PM_{2.5}$ and COPD diagnosis presenting a steeper upward-sloping curve at the higher exposure range ($p < 0.01$).

Effect modifications

Figure 2 presents the results of the effect modifiers in the associations between $PM_{2.5}$ and asthma diagnosis, and $PM_{2.5}$ and O_3 and COPD diagnosis (numerical results are provided in Supplementary Table S2). An increased risk of asthma diagnosis was observed in ever-smokers exposed to higher levels of $PM_{2.5}$ (HR: 1.22, 95 per cent CI: 1.07–1.39) and a lower non-significant risk was observed in never-smokers at baseline ($p = 0.07$). No effect modification by physical activity was observed for $PM_{2.5}$ and COPD diagnosis, while those who were physically active and exposed to higher levels of O_3 were at higher risk of COPD diagnosis than those who were physically inactive (HR: 1.16, 95 per cent CI: 1.02–1.32).

Figure 3 presents the effect modifiers of the associations of $PM_{2.5}$, NO_2 and O_3 with COPD hospitalisation (numerical results are shown in Supplementary Table S3).

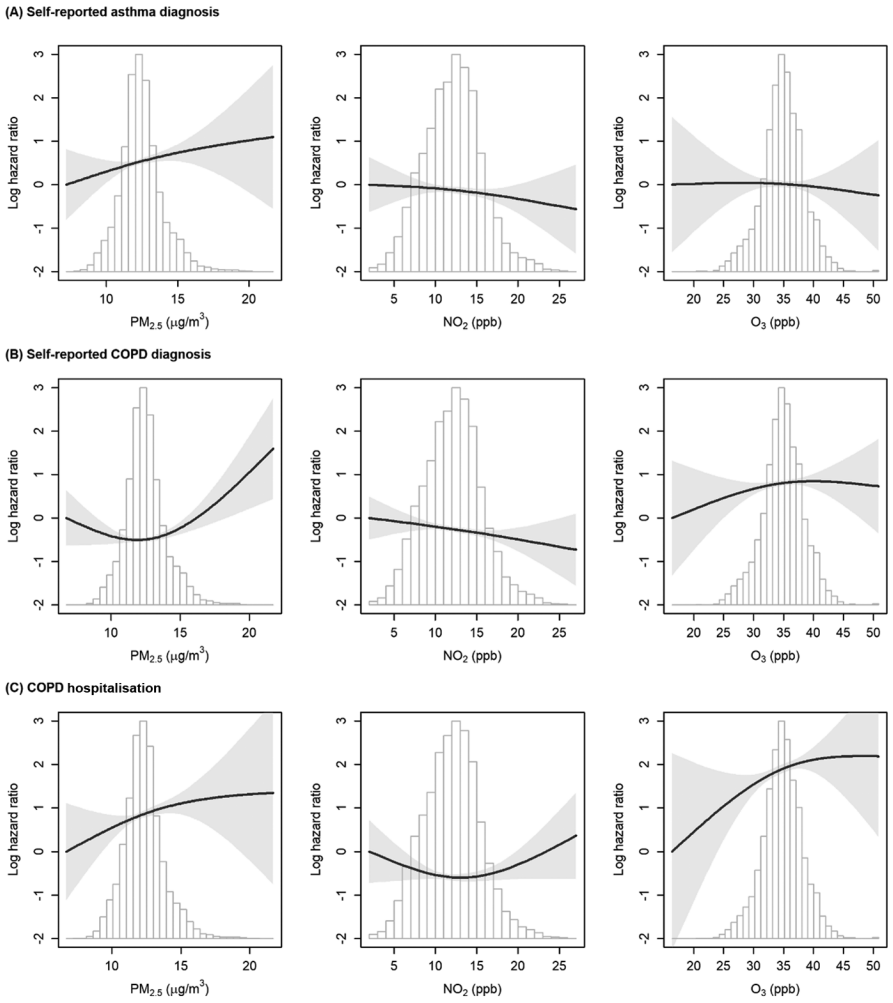
Table 3: Hazard ratios for the impact of exposure to each pollutant (interquartile range increase) on incidence of asthma and COPD, England, 2002–17

Outcomes	Pollutant	Unit (IQR)	Model 1[a] HR (95% CI)	Model 2a[b] HR (95% CI)	Model 2b[c] HR (95% CI)	Model 2c[d] HR (95% CI)	Model 3[e] HR (95% CI)
Self-reported asthma diagnosis (343 cases among 9,449)	$PM_{2.5}$	1.60 µg/m^3	1.14 (1.01, 1.28)	1.13 (1.00, 1.27)	1.15 (1.03, 1.30)	1.14 (1.01, 1.29)	1.14 (1.01, 1.29)
	NO_2	5.00 ppb	1.01 (0.87, 1.18)	0.96 (0.83, 1.12)	0.96 (0.82, 1.12)	0.93 (0.79, 1.09)	0.90 (0.77, 1.06)
	O_3	4.24 ppb	0.91 (0.80, 1.03)	0.93 (0.82, 1.06)	0.95 (0.83, 1.09)	0.95 (0.83, 1.08)	0.97 (0.85, 1.10)
Self-reported COPD diagnosis (531 cases among 9,999)	$PM_{2.5}$	1.60 µg/m^3	1.08 (0.98, 1.19)	1.07 (0.97, 1.17)	1.12 (1.01, 1.23)	1.09 (0.99, 1.20)	1.10 (0.99, 1.21)
	NO_2	5.00 ppb	1.03 (0.91, 1.17)	0.94 (0.83, 1.06)	0.92 (0.81, 1.04)	0.91 (0.81, 1.04)	0.87 (0.76, 0.99)
	O_3	4.23 ppb	0.95 (0.85, 1.06)	1.00 (0.90, 1.12)	1.06 (0.95, 1.19)	1.03 (0.92, 1.15)	1.08 (0.97, 1.21)
COPD hospitalisation (236 cases among 8,325)	$PM_{2.5}$	1.60 µg/m^3	1.16 (1.00, 1.34)	1.14 (0.99, 1.32)	1.22 (1.06, 1.40)	1.18 (1.02, 1.36)	1.18 (1.03, 1.36)
	NO_2	5.00 ppb	1.36 (1.13, 1.63)	1.17 (0.97, 1.40)	1.18 (0.98, 1.42)	1.09 (0.91, 1.32)	1.02 (0.84, 1.25)
	O_3	4.11 ppb	1.02 (0.86, 1.21)	1.12 (0.94, 1.32)	1.19 (1.00, 1.41)	1.20 (1.01, 1.42)	1.28 (1.07, 1.52)

Notes: CI – confidence interval; HR – hazard ratio; IQR – interquartile range; ppb – parts per billion air molecules; µg/m^3 – micrograms per cubic metre of air; NO_2 – nitrogen dioxide; O_3 – ozone; $PM_{2.5}$ – particulate matters with aerodynamic diameters of less than 2.5 µm.
Results are presented as HRs and 95 per cent CI per unit of the interquartile range increment for each pollutant within each cohort.
[a] Model 1 is accounted for age (underlying timescale) and sex (strata) and adjusted for calendar year (spline term with degrees of freedom = 4).
[b] Model 2a is Model 1 further adjusted for individual lifestyle factors, including smoking status and level of physical activity.
[c] Model 2b is Model 1 further adjusted for individual socioeconomic status, including marital status and wealth.
[d] Model 2c is Model 1 further adjusted for area-level deprivation.
[e] Model 3 is Model 1 further adjusted for all covariates mentioned.

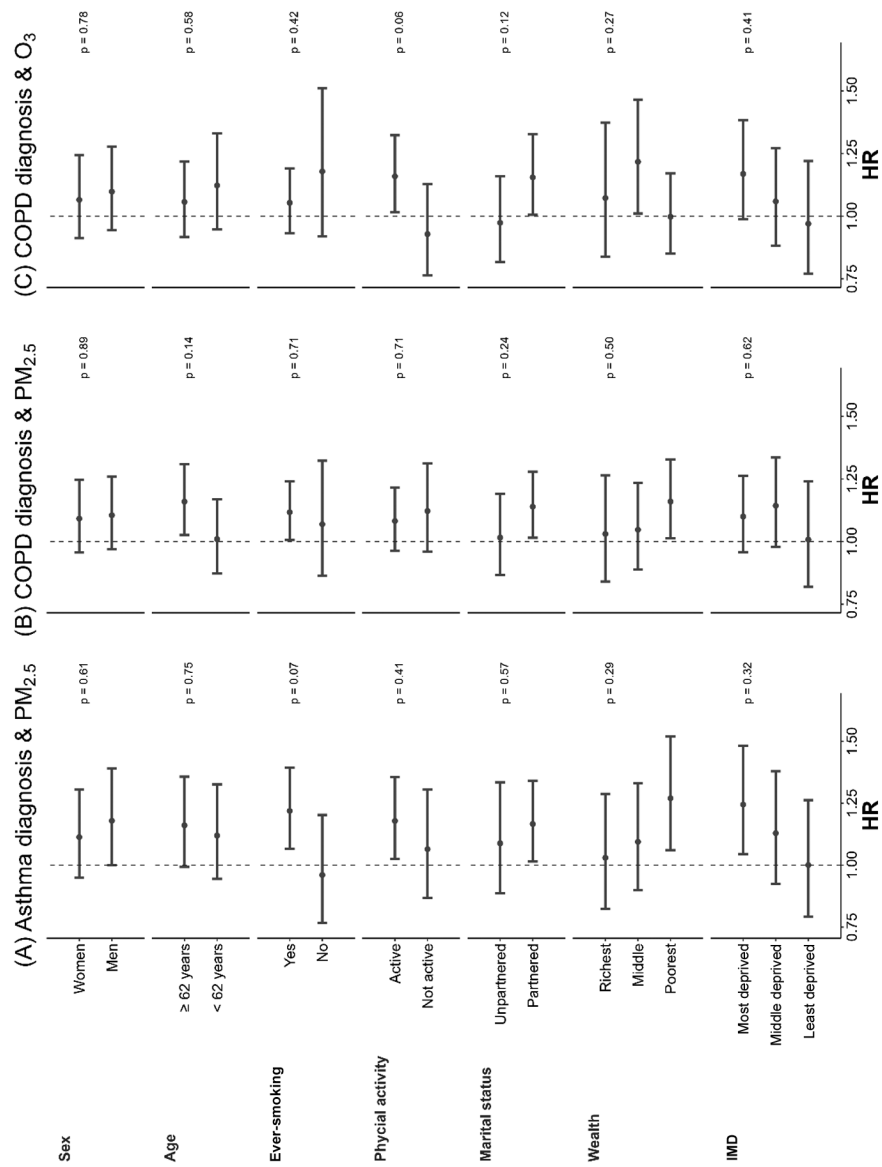
Those at or over the median age of 62 years at baseline and exposed to higher levels of $PM_{2.5}$ and NO_2 were at higher risk of COPD hospitalisation. Furthermore, men exposed to O_3 had a higher risk of COPD hospitalisation. The related part for the results of effect modification were also modified accordingly. Those living in the most deprived and middle deprived areas and exposed to higher levels of $PM_{2.5}$ and NO_2 also had higher risks of COPD hospitalisation.

Figure 1: Exposure-response curve for the associations of long-term exposure to $PM_{2.5}$, NO_2 and O_3 with (A) self-reported asthma diagnosis, (B) self-reported COPD diagnosis and (C) COPD hospitalisation



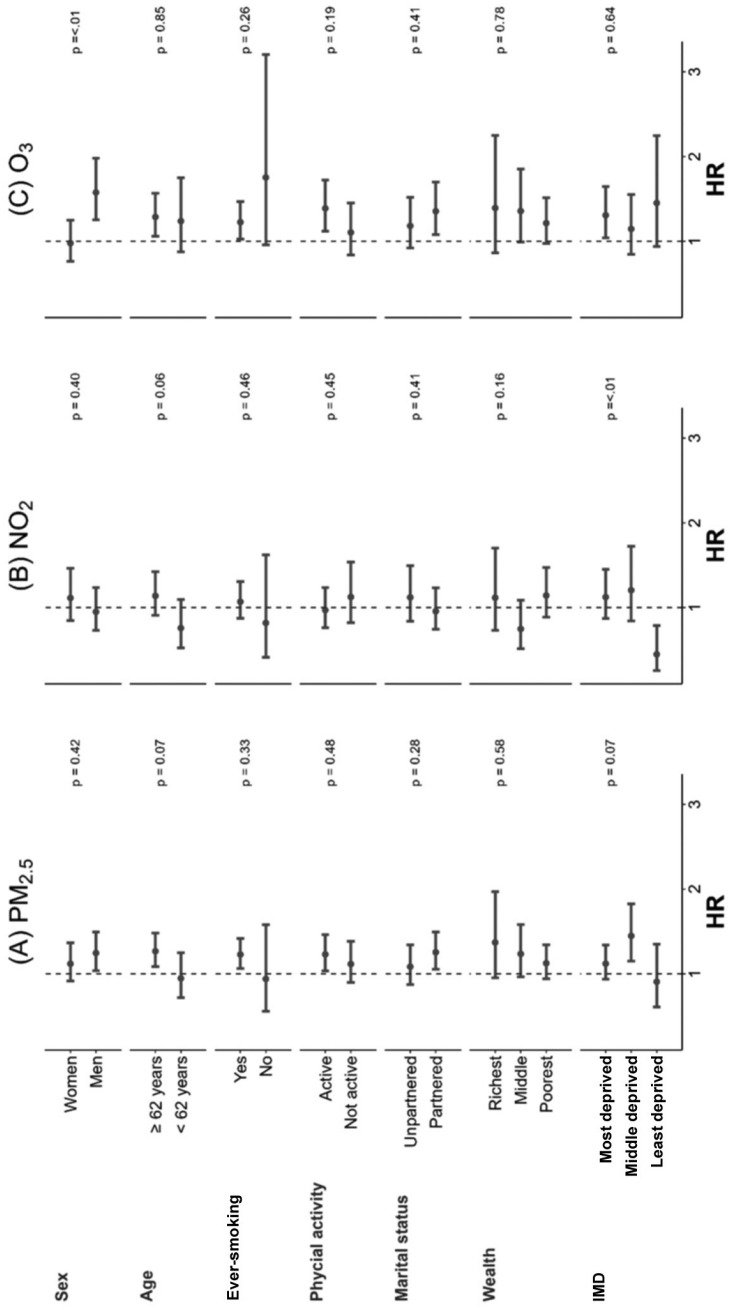
Notes: COPD – chronic obstructive pulmonary disease; NO_2 – nitrogen dioxide; O_3 – ozone; $PM_{2.5}$ – particulate matters with aerodynamic diameters of less than 2.5 μm.
The associations are expressed in a solid spline line with 95 per cent confidence interval (grey shading). The y-axis in plots indicates log hazard ratios at certain levels of pollutants compared to the risk at minimum levels of exposure. Histograms show the distribution of each pollutant. Models are accounted for age (underlying timescale) and sex (strata) and adjusted for calendar year, smoking status, level of physical activity, marital status, wealth and area-level deprivation.

Figure 2: Effect modifiers of the association between long-term exposure to air pollution and (A) asthma diagnosis and (B, C) COPD diagnosis, England, 2002–17



Notes: HR – hazard ratio; IMD – Index of Multiple Deprivation; O_3 – ozone; $PM_{2.5}$ – particulate matters with aerodynamic diameters of less than 2.5 µm. Associations are from models accounted for age (underlying timescale) and sex (strata) and adjusted for calendar year, smoking status, level of physical activity, marital status, wealth and area-level deprivation.

Figure 3: Effect modifiers of the association between long-term exposure to air pollution and COPD hospitalisation, England, 2002–17



Notes: HR: hazard ratio; IMD – Index of Multiple Deprivation; NO_2 – nitrogen dioxide; O_3 – ozone; $PM_{2.5}$ – particulate matters with aerodynamic diameters of less than 2.5 µm.
Associations are from models accounted for age (underlying timescale) and sex (strata) and adjusted for calendar year, smoking status, level of physical activity, marital status, wealth and area-level deprivation.

Sensitivity analyses

Our findings were robust to adjustment for other pollutants (Supplementary Table S4), except for the association of NO_2 with COPD hospitalisation, which, after adjustment for $PM_{2.5}$ and O_3, increased in magnitude (HR increased from 1.02, 95 per cent CI: 0.84, 1.25 per 5.00 ppb in the model with only NO_2 to 1.10, 95 per cent CI: 0.90, 1.34 per 5.00 ppb in the model adjusted for other pollutants). In Supplementary Table S5, we present the baseline characteristics of educational attainment and occupational social class for each outcome. Results of Model 3, presented in Table 3, were robust to further adjustment for educational attainment and occupational social class, as shown in Supplementary Table S6. There was no evidence of effect modification by educational attainment or by occupational social class for any of the pollutants.

Discussion

In this nationally representative sample of people aged 50 and over living in private households in England, we found significant increased risks of obstructive lung diseases from long-term air pollution exposure, particularly for $PM_{2.5}$ and O_3. $PM_{2.5}$ was positively associated with asthma and COPD diagnoses, with stronger associations observed for COPD hospitalisations. Long-term exposure to NO_2 showed no association with asthma or COPD diagnoses, though an inverse relationship was suggested for COPD. Long-term exposure to O_3 was linked to increased risk of COPD diagnosis and hospitalisation. Effect modification analyses indicated that ever-smokers exposed to higher $PM_{2.5}$ levels had an increased risk of asthma, while physically active individuals were more susceptible to O_3-related COPD. Additionally, older adults, men, and individuals in deprived areas appeared more vulnerable to air pollution exposure, particularly for COPD hospitalisation.

Comparison with previous studies

Alongside the US Sister Study (Young et al, 2014), our study is one of only two that find an association between $PM_{2.5}$ exposure and self-reported doctor-diagnosed asthma. In contrast, other studies on people aged 50+ that also used self-reported diagnoses reported weaker or null associations (Jeong et al, 2018). Our study is the first to find a positive association between long-term exposure to $PM_{2.5}$ and self-reported incidence of COPD among older people. The only other study that included older people in the sample – those aged 18 to 93 in rural provinces of the Netherlands – generally reported null or non-significant associations, probably due to the fact that the study focused on rural areas. Our findings on the effects of long-term exposure to $PM_{2.5}$ and COPD hospitalisation are instead in line with other studies which included older adults (Liu et al, 2021a; 2021c; Wang et al, 2022a).

For NO_2, our study finds no association with asthma or COPD, and some findings contradict our hypothesis. This finding is in line with other studies that generally report a short follow-up time (Gan et al, 2013; Atkinson et al, 2015; Carey et al, 2016; Gandini et al, 2018; Hooper et al, 2018; Salimi et al, 2018; Doiron et al, 2021). In contrast to our finding, a recent systematic review of asthma onset in adults, which analysed seven studies, reports a risk ratio of 1.10 (95 per cent CI: 1.01–1.21) per

10 μg/m^3 increase for long-term NO_2 exposure (Boogaard et al, 2022). More recent studies also report strong positive associations (Wu et al, 2020; Liu et al, 2021b; 2021d; Zhang et al, 2021), regardless of whether outcomes are based on hospital records or self-reported diagnoses. However, some of these inconsistencies between our findings and those reported in the above-mentioned studies could be due to the nature of the dataset, the covariates controlled for in the analyses and the length of the follow-up. Our study used a nationally representative prospective cohort of people aged 50+ and controlled for several individual and area characteristics that the Ontario, Canada, study by Zhang et al (2021) failed to account for. However, unlike the Danish nurse study by Liu et al (2021d), which had a long follow-up, ours, similar to ELAPSE (Effects of Low-Level Air Pollution: A Study in Europe; Liu et al, 2021a) and the Canadian study (Zhang et al, 2021), had 15 years of follow-up.

Our finding of an association between long-term O_3 exposure and COPD incidence is in line with the Canadian study (Zhang et al, 2021) but contrasts with findings from the UK Clinical Practice Research Datalink study reported by Atkinson et al (2015) and the ELAPSE project, which analysed three cohorts from Denmark and Sweden (Liu et al, 2021a). These discrepancies may stem from differences in the sample (for instance, 93 per cent of ELAPSE participants were under 65 years old) and in exposure assessment or misclassification, since accurately assessing exposure to O_3, a strong oxidative pollutant, remains challenging (Mudway, 2000). The limited epidemiological evidence on O_3 and COPD underscores the need for further research and improved exposure assessments.

Finally, our study is the first to identify an increased risk of asthma diagnosis in ever-smokers aged 50 and overexposed to higher levels of PM_2. In contrast, a prospective Australian cohort study of individuals aged 45+ and over (Salimi et al, 2018) and a study of Danish nurses (Liu et al, 2021d) found no such associations in ever-smokers, most likely due to the non-representativeness of the samples. Additionally, we observe stronger associations between O_3 exposure and COPD outcomes among physically active individuals. This is in line with findings from a small non-representative study based on the Danish Diet, Cancer and Health cohort (Fisher et al, 2016) that the protective effect of physical activity on COPD risk is diminished by residential NO_2 exposure. However, ELSA does not have information on whether respondents exercise outdoors or indoors.

Interpretation and conceptual meaning

Our findings highlight the persistent vulnerability of older adults to ambient air pollution, even after accounting for individual lifestyle factors and socioeconomic disparities. The stronger associations observed for COPD hospitalisations suggest a cumulative burden of long-term exposure that exacerbates existing health inequalities in later life. This supports a life-course perspective, where environmental exposures interact with ageing-related physiological decline, leading to increased susceptibility to respiratory diseases. The observed modification by smoking and physical activity further suggests that air pollution may amplify pre-existing vulnerabilities, compromising the protective effects of healthy behaviours in older populations. These results also underscore the need to refine current conceptual models of environmental determinants of healthy ageing, integrating pollution exposure as a critical, yet often overlooked, factor influencing respiratory health trajectories in older adults.

Policy and public health implications

The evidence from our study points to several avenues for preventive action. At the individual level, older adults and in particular those with existing respiratory conditions or living in high-exposure areas, may benefit from targeted interventions such as the promotion of indoor air filtration systems (Guo et al, 2021) and educational campaigns to raise awareness of pollution-related health risks.

At a policy level, our findings reinforce the urgency of enforcing and tightening air quality regulations, particularly for fine particulate matter and O_3, which remain persistent challenges even in high-income countries with existing air quality standards. Investments in urban green infrastructure, the expansion of low-emission zones and stricter emission controls on transport and industry are critical to reducing exposure, especially in urban settings where older adults are concentrated (Fichera et al, 2023).

From a global perspective, these results hold particular relevance for rapidly ageing populations in low- and middle-income countries, where urbanisation and industrialisation are increasing exposure to air pollutants, often in contexts with weaker regulatory frameworks. The intersection of demographic ageing and environmental degradation poses a compounded risk to population health, making air pollution control a key priority for global ageing and health agendas.

Despite regional variations in pollution sources and population characteristics, the biological mechanisms underpinning pollution-related respiratory damage are universally applicable, making these findings highly transferable. Our study thus contributes valuable evidence to inform international efforts to promote healthy ageing through environmental health policies.

Strengths and limitations

Our study has several strengths, including the use of a nationally representative sample of older adults in England, detailed data on lifestyle behaviours and socioeconomic status, and longitudinal exposure assessments that account for residential mobility. Unlike studies relying solely on self-reported diagnoses with cross-sectional designs, the longitudinal structure of ELSA enabled us to track new diagnoses over time and compare the effects of air pollution using both self-reported data and hospital records. Additionally, we employed high-resolution exposure models for $PM_{2.5}$ (1 km × 1 km) and NO_2 (50 m × 50 m), enhancing the accuracy of our pollution estimates.

However, our study also has some limitations. The follow-up period of approximately eight years may have contributed to some null or non-significant findings. Additionally, the spatial resolution of our O_3 exposure model (11 km × 11 km) was relatively coarse compared to other studies (1 km × 1 km or 100 m × 100 m), which may have led to exposure misclassification, particularly in urban areas or near major roads. Another limitation is the lack of data on air pollution exposure in other environments, such as workplaces or during commuting, as well as exposure prior to baseline. Lastly, while the NO_2 land use regression model used in our study varies in accuracy between rural and urban areas due to limited monitoring stations in rural regions, this is unlikely to explain the null associations observed, as 75 per cent of our participants resided in urban areas.

Conclusion

This study provides robust evidence that long-term exposure to $PM_{2.5}$ and O_3 is associated with increased risks of asthma and COPD among older adults in England, which has important implications for public health and healthy ageing. By identifying subgroups at greater risk, such as ever-smokers, physically active individuals and those living in deprived areas, our findings highlight the need for targeted interventions to protect vulnerable populations.

These results advance our understanding of how environmental exposures interact with individual and sociodemographic factors to shape respiratory health in later life, offering valuable insights for gerontology research, clinical practice and environmental health policy. Crucially, they underscore the importance of integrating air quality improvements into strategies aimed at promoting healthy ageing.

Beyond contributing to an emerging body of evidence, this study calls for urgent policy action to mitigate air pollution exposure and prioritise environmental health as a key pillar of healthy ageing initiatives. Future research should continue to refine exposure assessments, explore life-course impacts and examine how cumulative environmental risks influence ageing trajectories globally.

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Data availability statement

Data and materials are archived at the UK Data Service (https://beta.ukdataservice.ac.uk/datacatalogue/series/series?id=200011), the Gateway to Global Aging Data (https://g2aging.org/home) and the Longitudinal Linkage Collaboration (https://ukllc.ac.uk).

Research ethics statement

The English Longitudinal Study of Ageing (ELSA) Wave 1 received ethical approval from the London Multi-Centre Research Ethics Committee on 7 February 2002 (MREC/01/2/91). Participants gave their informant consent to participate in the study and to have their data linked to Hospital Episode Statistics and the Mortality registry.

Conflict of interest

The authors declare that there is no conflict of interest.

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