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Higher air pollution exposure in early life is associated with worse health among older adults: A 72-year follow-up study from Scotland

Gergő Baranyi [a,*], Lee Williamson [a,b], Zhiqiang Feng [a], Edward Carnell [c], Massimo Vieno [c], Chris Dibben [a]

[a] Centre for Research on Environment, Society and Health, School of GeoSciences, The University of Edinburgh, Edinburgh, United Kingdom
[b] Longitudinal Studies Centre - Scotland, School of GeoSciences, The University of Edinburgh, Edinburgh, United Kingdom
[c] UK Centre for Ecology & Hydrology, Bush Estate, Penicuik, United Kingdom

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ABSTRACT

Air pollution increases the risk of mortality and morbidity. However, limited evidence exists on the very long-term associations between early life air pollution exposure and health, as well as on potential pathways. This study explored the relationship between fine particle ($PM_{2.5}$) exposure at age 3 and limiting long-term illness (LLTI) at ages 55, 65 and 75 using data from the Scottish Longitudinal Study Birth Cohort 1936, a representative administrative cohort study. We found that early life $PM_{2.5}$ exposure was associated with higher odds of LLTI in mid-to-late adulthood (OR = 1.10, 95% CI: 1.06, 1.14 per 10 μg m^{-3} increment) among the 2085 participants, with stronger associations among those growing up in disadvantaged families. Path analyses suggested that 15–21% of the association between early life $PM_{2.5}$ concentrations and LLTI at age 65 (n = 1406) was mediated through childhood cognitive ability, educational qualifications, and adult social position. Future research should capitalise on linked administrative and health data, and explore causal mechanisms between environment and specific health conditions across the life course.

1. Introduction

Approximately 99% of the global population breaths air with pollutant concentrations exceeding WHO limits (World Health Organization, 2021). Poor air quality is an established risk factor of all-cause and cause-specific mortality (Chen and Hoek, 2020) and of a wide range of health conditions, including respiratory, cardiovascular, and brain diseases (Dominski et al., 2021; Boogaard et al., 2022; Fu and Yung, 2020). Although air pollution affects people across the entire life course, due to population ageing and higher prevalence of morbidity, older adults bear a disproportionate diseases burden translating into higher mortality and large economic costs (Yin et al., 2021). Long-term air pollution exposure increases the risk of limiting long-term illness (Al Ahad et al., 2022; Ju et al., 2022) and multimorbidity (Ronaldson et al., 2022), conditions particularly common among older adults.

Early life circumstances have been associated with functional limitations among older adults (Iveson et al., 2020), and it is plausible that air pollution during sensitive pre- and postnatal developmental windows, might have long-lasting effects on health (Bettiol et al., 2021; Isaevska et al., 2021; Klepac et al., 2018). While existing literature suggest high or moderate-to-high level of confidence in the associations between contemporary air pollution concentrations and different health conditions across the human life course (Boogaard et al., 2022), the very long-term impact of poor air quality experienced by older people in their earlier life remains relatively unexplored. It is plausible that toxic air contributes to age-related disorders through first influencing development and health in childhood. Early life air pollution exposure has been associated with change in BMI (Kim et al., 2018), lung development (Bettiol et al., 2021; Hsu et al., 2023; Yu et al., 2023), and brain development and cognition (Lubczyńska et al., 2021; Lopuszanska and Samardakiewicz, 2020), as well as cardiometabolic health (Johnson et al., 2021) in childhood; key determinants for later life health and longevity (e.g. (Calvin et al., 2011)). Although evidence suggests that childhood cognitive ability might mediate the association between $PM_{2.5}$ exposure at age 3 and mortality (Baranyi et al., 2023), likely acting through socioeconomic status and health literacy in mid-life

* Corresponding author. Centre for Research on Environment, Society and Health, School of GeoSciences, University of Edinburgh, Drummond Street, Edinburgh EH89XP, United Kingdom.
E-mail address: gergo.baranyi@ed.ac.uk (G. Baranyi).

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(Calvin et al., 2011), pathways operating across the life course remain relatively unexplored.

There are two key challenges when addressing the impact of early life air pollution exposure on healthy ageing. First, it requires cohort data following individuals throughout their life course. Although participants in several birth cohorts are now in their late adulthood, there is generally a lack of information on geocoded residential addresses, especially from birth onwards (Pearce et al., 2018; Desjardins et al., 2023). Second, national air quality monitoring networks were only established during the second half of the 20th century (in the 1950s for the United Kingdom) (Fowler et al., 2020); epidemiological studies on the long-term impact of toxic air quality on health and mortality rarely have a follow-up time stretching over 25 years (Hansell et al., 2016). Understanding the life-course impact of bad air quality experienced in early life requires historical air pollution estimates going back several decades, and only very few investigations were able to address these limitations (Baranyi et al., 2023; Hansell et al., 2016; Russ et al., 2021; Baranyi et al., 2022; Phillips et al., 2018; von Hinke and Sørensen, 2023; Bailey et al., 2018).

To fill this research gap, we utilised a unique data-linkage study and explored the association between early life air pollution exposure and limiting long-term illness in mid-to-late adulthood, a valid and widely used indicator of morbidity (Manor et al., 2001). Based on a representative sample of Scottish adults born in 1936, we estimated the association between exposure to fine particles with the aerodynamic diameter of less than 2.5 μm ($PM_{2.5}$) in early life and limiting long-term illness (LLTI) at age 55, 65 and 75; effect modification by sex and parental social position as well as change over time were explored (*Objective 1*). Pathways between early life $PM_{2.5}$ and LLTI at age 65 were investigated including childhood cognitive ability, educational qualifications, and occupational social position (*Objective 2*). Due to availability of residential addresses, exposure to air pollution was measured at age 3; a time important for building up lifelong health and wellbeing.

2. Methods

This study utilises data from the Scottish Longitudinal Study Birth Cohort of 1936 (SLSBC 1936), a representative administrative sample of the Scottish population born in 1936. SLSBC1936 was assembled through data linkages across: (i) the 1939 National Identity Register, a census survey of the entire UK population on the September 29, 1939; (ii) the Scottish Mental Survey 1947 assessing general cognitive ability in almost all 1936-born children attending Scottish schools on the June 4, 1947; (iii) Scotland's National Health Services Central Register; (iv) and the Scottish Longitudinal Study, a 5% sample of the population present in any of the 1991, 2001 and 2011 Censuses. The linkage rates between the Scottish Mental Survey 1947 and the other sources were high (i.e., 97% traced in Scotland's National Health Services Central Register, 95% in the 1939 National Identity Register, and 87% in the Scottish Longitudinal Study). Participants tracked but not captured in the main SLSBC1936 sample either died or migrated from Scotland (Huang et al., 2017). The sample included SLSBC1936 participants with residential address and household information from the 1939 National Identity Register (age 3), general cognitive ability from the Scottish Mental Survey 1947 (age 11), and limiting long-term illness in the 1991 (age 55), 2001 (age 65) and 2011 (age 75) Censuses from the Scottish Longitudinal Study.

2.1. Early life $PM_{2.5}$ pollution exposure

Residential addresses for SLSBC1936 participants were digitised from the 1939 National Identity Register. Using the 1939 enumeration districts centroid and the 1939 street index datasets, addresses were geocoded with the Historical Address Geocoding – GIS software (Daras, 2015). Coordinates were then intersected with annual average of $PM_{2.5}$ concentrations in 1935, estimated using the EMEP4UK version 4.17 atmospheric chemistry transport model (Vieno et al., 2010, 2014, 2016) (see detailed information in (Baranyi et al., 2023)). Briefly, anthropogenic emission of pollutants (including $PM_{2.5}$) was estimated for 1950 and scaled down to 1935 using activity data research (Russ et al., 2021); the meteorological driver came from the Weather Research and Forecast model version 3.9.1.1 (Skamarock et al., 2008) used for the year of 2015. The EMEP4UK model employed a horizontal resolution of 0.037 × 0.037° (~3 km × 4 km) for the UK, nested within a greater European domain providing boundary conditions (0.5 × 0.5°). The EMEP4UK model has been evaluated showing good agreement between modelled and observed concentrations (Lin et al., 2017). For Scotland, modelled 1935 $PM_{2.5}$ concentrations ranged between 4.2 μg m^{-3} to 116.9 μg m^{-3}.

2.2. LLTI in mid-to-late adulthood

LLTI was assessed with a question on the presence or absence of health problem, illness, or disability, including age-related problems, lasting for a longer period, and limiting daily activities and work. LLTI often indicate the presence of serious health conditions such as cardiovascular, musculoskeletal or mental disorders (Payne and Saul, 2000). As questions and answer categories were comparable across the 1991, 2001 and 2011 Censuses (Supplementary Table 1), information was analysed longitudinally across all three censuses (Dearden et al., 2019); however, we also provided cross-sectional findings for sensitivity.

2.3. Mediators

Potential pathways were chosen based on literature (Iveson et al., 2020; Baranyi et al., 2023). *Childhood general cognitive ability* was assessed with the Moray House Test No.12 as part of Scottish Mental Survey (1947) (Deary et al., 2004). The paper and pencil test included 71 items with tasks such as following directions, work classification, reasoning, spatial items and sypher decoding. The scores ranged between 0 and 76 and had high correlation with the Stanford-Binet test indicating good criterion validity (Deary et al., 2004).

Highest educational qualification (No qualification, Level 1, Level 2, Level 3, and Level 4; see Supplementary Table 2) was derived by the census team from a question listing all qualifications held. As the same question was asked in the 2001 and 2011 Censuses, we took for each participant the earliest available data.

Occupational social position: Occupations were classified using the Standard Occupational Classification 1990 coding system. For each participant, we took the earliest reported occupation between 1991 and 2001 Censuses and converted it into occupational social position using the Cambridge Social Interaction and Stratification (CAMSIS) scale. CAMSIS is a social interaction distance scale providing a continuous measure of social inequality based on occupations (Lambert and Griffiths, 2018). The score mean is 50 with a standard deviation of 15; values range between 1 and 99 with greater numbers indicating a higher position.

2.4. Covariates

Confounders are presented in graphs (Supplementary Figs 1A and 1B). Age (in years) and sex (male, female) were derived from the Scottish Mental Survey 1947. Mother's age (in years), mother's marital status (married, not married), and parental occupational social position were from the 1939 National Identity Register. Similar to participants' own adult occupational social position, parental occupational codes, based on the Historical International Classification of Occupations coding system, were transformed into social stratification using the Historical CAMSIS (HISCAM) scale created for occupations during the 19th and early 20th centuries (Lambert et al., 2013). HISCAM has the same structure as CAMSIS and is therefore comparable. Father's (or, if not available, mother's) HISCAM score was taken. Where neither parent had a valid score, we calculated average score from all other household

members (e.g., siblings, grandparents).

2.5. Statistical analysis

For Objective 1, longitudinal associations between early life $PM_{2.5}$ exposure and LLTI in mid-to-late adulthood were explored using generalised linear mixed effects models with a random intercept for participants. Associations were expressed as Odds Ratios (OR) with 95% Confidence Intervals (CI) per 10 μg m^{-3} $PM_{2.5}$ increase. Model 1 controlled for age, sex, and census wave; Model 2 additionally included parental occupational social position, mother's age, and mother's marital status (see equations in Supplementary Table 3). We explored effect modification by sex and parental occupational social position by adding interaction terms to Model 2. In addition to focussing on the level of LLTI across waves, we explored rate of change in LLTI during follow-up by adding an interaction term (i.e., exposure interacting with age at census wave) to the fully adjusted model. Continuous covariates were standardized before entering them in the models. Findings were visualised by plotting the predicted values of LLTI, where discrete covariates were held constant at their proportions and continuous ones at their means.

Objective 2 investigated pathways between early life $PM_{2.5}$ exposure and LLTI at age 65. Within structural equation modelling framework utilising complete cases, we first fitted a probit regression for binary outcome to estimate the total effect while controlling for all confounders (i.e., age, sex, parental occupational social position, mother's age, mother's marital status). Then, we added paths leading from early life $PM_{2.5}$ exposure to LLTI through childhood cognitive ability, highest educational qualifications and occupational social position (Iveson et al., 2020); direct path from $PM_{2.5}$ to adult occupational social position was not specified in the model. Regression coefficients (*b*) with their 95% CI were presented in a path diagram. Continuous covariates were standardized before entering them in the model, highest educational qualification was treated as ordinal, and $PM_{2.5}$ exposure was expressed as 10 μg m^{-3} increase. The outcome was LLTI at age 65, as detailed educational qualification breakdowns were not available before the 2001 Census.

Multiple sensitivity analyses were carried out. As there are differences in how LLTI was operationalised, we fitted generalised linear models separately for the 1991, 2001 and 2011 Censuses (S1). For Objective 1, models were also fitted using air pollution quintiles as a categorical exposure (S2). To avoid bias towards complete cases, missing variables were imputed in 25 datasets based on sex (no missing), age (no missing), parental occupational social position, mother's marital status, mother's age, childhood cognitive ability, highest educational qualifications, occupational social position, and limiting long-term illness (not imputed). Multiple imputation by chained equations was carried out using normal distribution for mother's age and childhood cognitive ability, and predictive mean matching for the other variables; for Objective 1, we also considered the multilevel structure of the data. Results were pooled by Rubin's rule (S3). Although LLTI at 65 was selected as main outcome for the mediation analysis, we provided path models for LLTI at age 55 and 75 (S4). Finally, all models were rerun after adjusting for area of residence in 1939, determined as residing in urban (i.e., Aberdeen, Dundee, Edinburgh, or Glasgow) versus rural council areas (S5).

Analyses were carried out using *lme4* (Bates et al., 2015), *lavaan* (Rosseel, 2012) and *mice* (van Buuren and Groothuis-Oudshoorn, 2011) packages in R4.2.2 (R Core Team. R, 2022).

3. Results

The analytical sample for Objective 1 included 2085 individuals with 93% participating in the 1991, 73% in the 2001 and 66% in the 2011 Census. The percentage of individuals with LLTI increased during follow-up (1991: 40%; 2001: 45%; 2011: 53%). Objective 2 was explored in a smaller sample (n = 1406) as all included individuals had to be present at the 2001 Census. Characteristics for both samples are shown in Table 1.

3.1. *Objective 1: $PM_{2.5}$ exposure and LLTI*

As shown in Table 2, being exposed to 10 μg m^{-3} higher $PM_{2.5}$ concentrations in early life was associated with 10% (95% CI: 1.07, 1.14) higher odds of reporting LLTI during mid-to-late adulthood after adjusting for age, sex, and census wave (Model 1). After further adjustment for parental occupational social position, mother's age, and mother's marital status (Model 2) the association remained the same (OR = 1.10, 95% CI: 1.06, 1.14).

There was no interaction between $PM_{2.5}$ exposure and sex (p = 0.391); confidence intervals were overlapping for males (OR = 1.11, 95% CI: 1.06, 1.17) and females (OR = 1.08, 95% CI: 1.03, 1.14). We found significant effect modification by parental occupational social position (p = 0.002): associations between $PM_{2.5}$ concentrations and LLTI in mid-to-late adulthood were smaller among individuals growing up in advantaged families (OR = 0.94, 95% CI: 0.90–0.98) (Fig. 1A; Supplementary Table 4).

After adding the interaction term of $PM_{2.5}$ exposure by age at census wave, findings did not suggest different associations at ages 65 (2001 Census: OR = 1.00, 95% CI: 0.95, 1.06) or 75 (2011 Census: OR = 0.95, 95% CI: 0.89, 1.01) in comparison to age 55 (i.e., 1991 Census). Despite non-significant rate of change at age 75 (p = 0.100), visually inspecting the predicted values of the top and bottom 20% $PM_{2.5}$ exposure groups showed converging values (Fig. 1B; Supplementary Table 4).

Table 1
Sample characteristics for Objective 1 and Objective 2 using the Scottish Longitudinal Study Birth Cohort of 1936.

Characteristics	Objective 1[a] (*n* = 2085)	Objective 2[b] (*n* = 1406)
	mean ± SD/n (%)	mean ± SD/n (%)
1939 National Identity Register		
$PM_{2.5}$ exposure (in μg m^{-3}), mean ± SD	31.5 ± 33.1	29.9 ± 31.4
Parental occupational social position (HISCAM score), mean ± SD	53.5 ± 9.1	54.1 ± 9.6
Mother's marital status, n (%)		
Married	2016 (96.7%)	1359 (96.7%)
Not married	69 (3.3%)	47 (3.3%)
Mother's age in years, mean ± SD	32.2 ± 6.2	32.1 ± 6.1
Scottish Mental Survey 1947		
Age in years, mean ± SD	11.0 ± 0.2	11.0 ± 0.25
Sex, n (%)		
Male	1019 (48.9%)	703 (50.0%)
Female	1066 (51.1%)	703 (50.0%)
Moray House No.12 test score, mean ± SD	NA	38.3 ± 14.8
1991/2001/2011 Censuses		
Occupational social position (CAMSIS score), mean ± SD	NA	46.0 ± 14.3
Highest qualifications, n (%)		
No qualification	NA	878 (62.5%)
Level 1		216 (15.4%)
Level 2		99 (7.0%)
Level 3		42 (3.0%)
Level 4		171 (12.2%)

[a] Participants were included with valid outcome for at least 2 census waves and complete data for covariates.
[b] Participants were included with valid outcome for the 2001 Census and complete data for covariates.
Source: Scottish Longitudinal Study. CAMSIS=Cambridge Social Interaction and Stratification; HISCAM= Historical CAMSIS; NA=Not applicable as variable not used for Objective 1.

Table 2
Associations between $PM_{2.5}$ exposure in early life and limiting long-term illness in mid-to-late adulthood in the Scottish Longitudinal Study Birth Cohort of 1936.

Covariates	Model 1		Model 2	
	OR (95% CI)	*p*-value	OR (95% CI)	*p*-value
$PM_{2.5}$ in early life (per in 10 μg m^{-3})	1.10 (1.07, 1.14)	<0.001	1.10 (1.06–1.14)	<0.001
Age (per 1 SD increase)	1.01 (0.90, 1.13)	0.826	1.00 (0.90–1.12)	0.932
Sex				
Male	ref		ref	
Female	0.66 (0.53–0.83)	<0.001	0.66 (0.53–0.83)	<0.001
Census wave				
1991 (age 55)	ref		ref	
2001 (age 65)	6.87 (5.48–8.60)	<0.001	6.88 (5.49–8.63)	<0.001
2011 (age 75)	13.51 (10.37–17.61)	<0.001	13.50 (10.36–17.59)	<0.001
Parental occupational social position (per 1 SD increase)			0.73 (0.65–0.82)	<0.001
Mother's marital status				
Married			ref	
Not married			0.78 (0.42–1.47)	0.448
Mother's age (per 1 SD increase)			1.00 (0.90–1.12)	0.992

Source: Scottish Longitudinal Study. 2085 participants with 4945 observations were included in the analysis. Models were fitted with generalised linear mixed-effects regression with random intercept for participants. Abbreviation: OR = adjusted Odds Ratio; SD=Standard Deviation.

3.2. ***Objective 2:*** *mediation via cognitive and socioeconomic factors*

We found a modest positive association between higher early life $PM_{2.5}$ exposure and LLTI at age 65 after adjusting for confounders (b = 0.041 per 10 μg m^{-3} increment, 95% CI: 0.019, 0.063). This total association was reduced by approximately 15% when adding mediating pathways (b = 0.035, 95% CI: 0.015, 0.056) (Fig. 2). First, early life $PM_{2.5}$ exposure decreased childhood general cognitive ability (b = −0.021, 95% CI: 0.035, −0.006), which directly contributed to LLTI (b = −0.129, 95% CI: 0.221, −0.037). Second, higher childhood general cognitive ability was also linked to higher educational qualifications (b = 0.599, 95% CI: 0.554, 0.644), and higher occupation social position (b = 0.257, 95% CI: 0.198, 0.316); higher occupation social position was negatively associated with LLTI at age 65 (b = −0.107, 95% CI: 0.186, −0.028). Model fit indices suggested good fit to the data (Hu and Bentler, 1999).

3.3. *Sensitivity analysis*

Analysing LLTI cross-sectionally suggested that higher $PM_{2.5}$ concentrations in early life were associated in Model 2 with higher odds of LLTI in 1991 (OR = 1.08; 95% CI 1.05–1.11) and 2001 (OR = 1.06; 95% CI 1.03–1.10), but not in 2011 (OR = 1.03; 95% CI 1.00–1.07), indicating some converging over time (S1: Supplementary Table 5). Operationalising $PM_{2.5}$ exposure in quintiles revealed that there were increased odds of LLTI only in the highest (Q5) exposure group (Model 2: OR = 2.21; 95% CI 1.56–3.14) (S2: Supplementary Table 6). Imputing data with multiple imputation did not change the findings for Objective 1 (S3.1: Supplementary Table 7) and suggested the same indirect pathways as in the main model (21% of the total effect was mediated) (S3.2: Supplementary Fig. 2). Conducting mediation analysis with LLTI measured at different Censuses showed that early life $PM_{2.5}$ exposure was only directly associated with LLTI at age 55, while there was no association with LLTI at age 75 (S4: Supplementary Figs. 3–4). Finally, after adding area of residence in 1939 (i.e., urban versus rural) to the models, findings remained comparable for Objective 1 (S5.1: Supplementary Table 8) and Objective 2 (19% mediated) (S5.2: Supplementary Fig. 5).

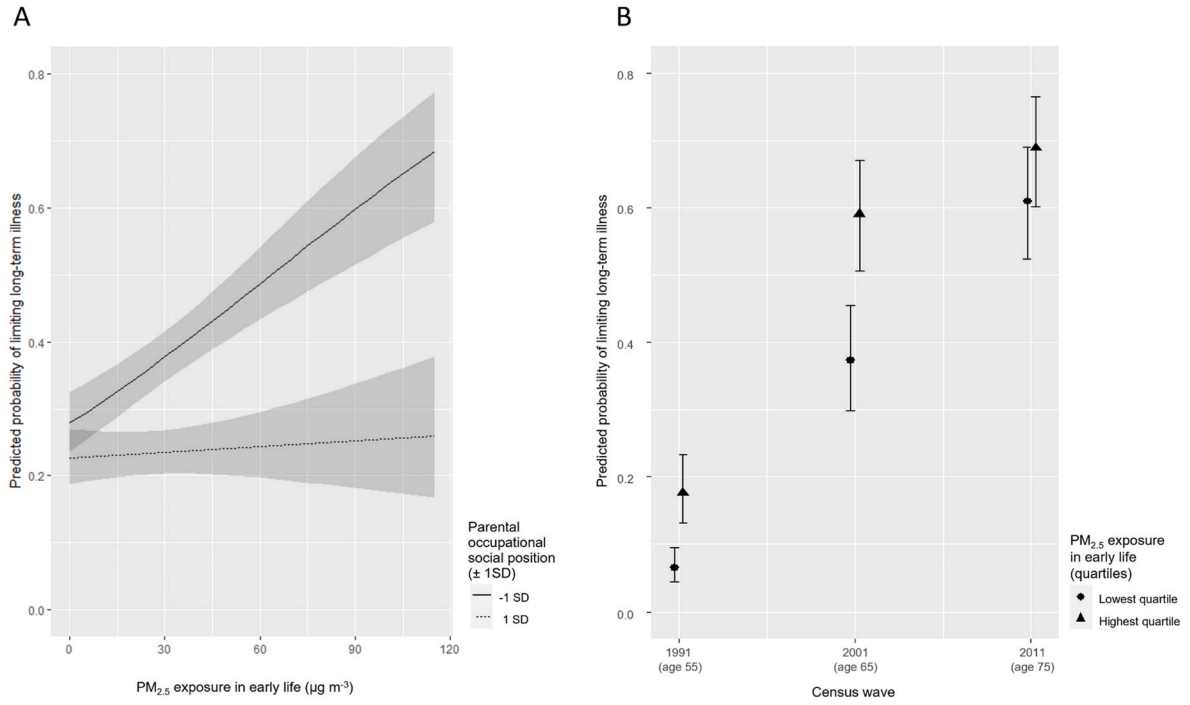

Fig. 1. Early life $PM_{2.5}$ exposure and predicted probability of limiting long-term illness in mid-to-late adulthood A) by parental occupational social position, and B) across the 1991, 2001, and 2011 Censuses. Models were fitted with generalised linear mixed-effects regression with random intercept. 2085 participants with 4945 observations were included in the analysis. Predicted values are presented in Supplementary Table 4. Source: Scottish Longitudinal Study. Abbreviation: SD = standard deviation.

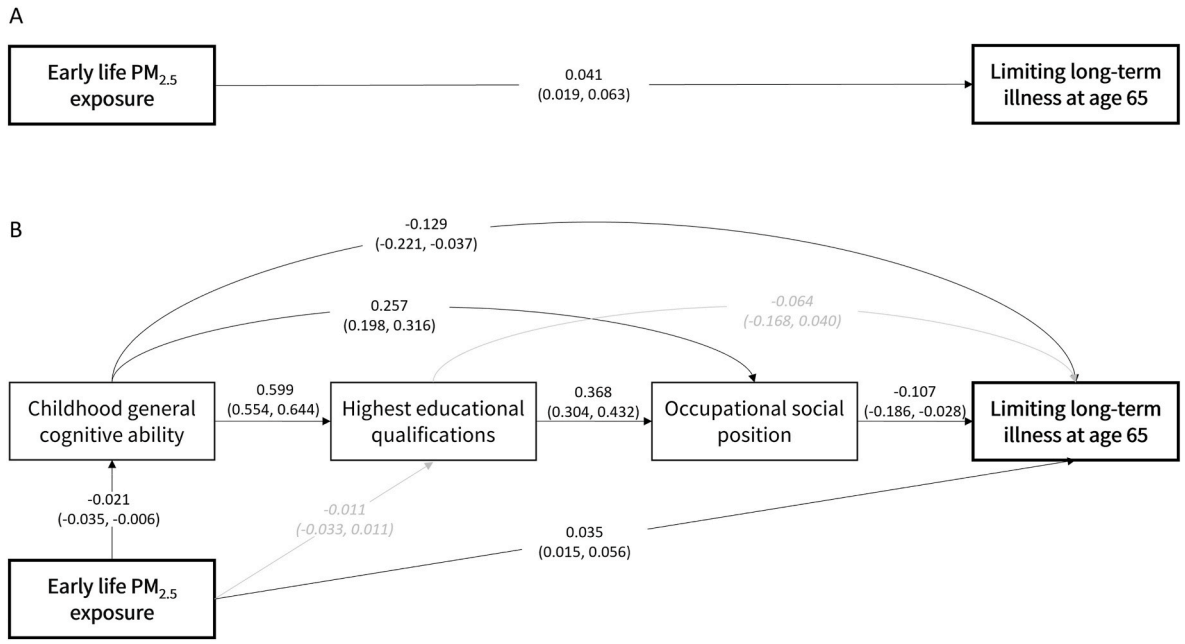

Fig. 2. Direct (A) and indirect (B) pathways between early life $PM_{2.5}$ exposure (per in 10 μg m^{-3}) and limiting long-term illness at age 65 year. Probit model was fitted within structural equation modelling framework with complete data. Regression coefficients (and their 95% confidence intervals in parentheses) are presented. Confounders (i.e., age, sex, parental occupation social position, mother's age, mother's marital status) are not shown for simplicity. Black solid lines represent significant ($p < 0.05$), grey dashed lines non-significant associations. The variable 'highest educational qualifications' was treated as ordinal in the path analysis; childhood general cognitive ability and occupational social position were standardized. Analysis was based on a sample size of 1406. Model fit indices: Root Mean Square Error of Approximation = 0.000, Standardized Root Mean Square Residual = 0.032. Source: Scottish Longitudinal Study.

4. Discussion

This study based on a representative sample from Scottish administrative data suggested that higher $PM_{2.5}$ exposure in early life was associated with increased odds of having limiting long-term illness in mid-to-late adulthood. First, we found that air pollution at age 3 was associated with higher level of LLTI between age 55 and 75; associations were stronger among individuals growing up in families with lower occupational social position but there were no differences between males and females. Change of association over time showed mixed results. Second, path analyses with different specifications suggested that 15–21% of the total association between early life $PM_{2.5}$ exposure and LLTI at age 65 was mediated through lower childhood cognitive ability, educational qualifications, and occupational social position.

David Baker's *developmental origins of health and disease* hypothesis proposes that detrimental exposures during early development may have lifelong implications for health (Heindel and Vandenberg, 2015). Exposure to air pollution is associated with foetal growth (Clemens et al., 2017), respiratory conditions (Bettiol et al., 2021) and cognitive development (Chiu et al., 2016) in children. There is also some evidence showing different DNA-methylation patterns in genes involved in oxidative stress, inflammation, and foetal development in newborns (Isaevska et al., 2021; Saenen et al., 2019); biological ageing presents a potential pathway between air pollution, early development, and health in later life (Isaevska et al., 2021; Baranyi et al., 2022; Saenen et al., 2019). More recently, studies that followed individuals throughout most of their life course found that poor air quality around the time of birth was associated with faster biological ageing (Baranyi et al., 2022), lower cognitive function (Russ et al., 2021; von Hinke and Sørensen, 2023), higher risk of respiratory conditions (von Hinke and Sørensen, 2023) as well as increased all-cause and case-specific mortality in late adulthood (Baranyi et al., 2023). The scarring effect of air pollution has also been shown to impact the physical development of British children growing up in the 1890s (Bailey et al., 2018). Our findings expand on this literature by demonstrating that differences in general morbidity can be detected several decades after early-life air pollution exposure, and that they are socially patterned. Individuals who grew up in socioeconomically disadvantaged families were more affected by early life exposure, which corresponds with health inequalities in air pollution effects found in recent investigations (Di et al., 2017).

Analysing trajectories of limiting long-term conditions during mid-to-late adulthood given different air pollution exposures led to mixed findings; although main analyses were not statistically significant, we observed converging probability of LLTI between high and low exposed groups, which was supported by cross-sectional findings in the sensitivity analyses. This difference might be due to a comparatively smaller sample size used in the main analysis, as the longitudinal model required at least two measures of LLTI. Change in association over time would be in line with the *age-as-leveler* hypothesis where health differences based on previous exposure diminish in older age due to mortality selection (Dupre, 2007). While the current investigation suggested declining strength of association between age 55 and age 75, a recent study using the same sample demonstrates how the risk of mortality increased during the same period when exposed to higher early life $PM_{2.5}$ concentrations (Baranyi et al., 2023). These combined findings highlights not only the narrowing health inequalities in later life, when the risk of mortality starts to increase (Baranyi et al., 2023), but also how mortality selection in older ages (after 65) might lead to underestimating the impact of early life $PM_{2.5}$ exposure on health outcomes. Studies with longer follow-up time on health (e.g., 2022 Census, NHS health records) are required to determine trajectories. Additionally, owing to the nature of the Scottish Longitudinal Study (including SLSBC 1936) as a record linkage study there is no information on LLTI prior to the 1991 Census, which prevents an understanding of when health differences first emerged during the life course.

Children who grow up in areas with poor air quality had lower cognitive function at age 11 (Chiu et al., 2016), which is a powerful indicator of health and longevity (Calvin et al., 2011; Baranyi et al., 2023). Part of the association is likely mediated through educational attainment and social position (Iveson et al., 2020). Our study found that

around 80–85% of the total association between early life air pollution exposure and limiting long-term illness at age 65 could not be explained by cognitive and socioeconomic factors, requiring further investigations. Accelerated biological ageing may provide a mechanistic link (Baranyi et al., 2022); lower physical activity has been also proposed as a mediator (Hautekiet et al., 2022), with health behaviours established during childhood as well as childhood general health presenting further pathways.

4.1. Strengths and limitations

This study is based on an administrative data birth cohort with air pollution exposure at age 3 and morbidity assessed between age 55 and 75, providing an exceptionally long follow-up time. SLSBC1936 is a representative study with high quality of data linkages, it is only marginally affected by selection and attrition bias, and provides rich information on key life-course confounders and mediators (Huang et al., 2017). However, there are limitations. First, information on early life residential addresses was only available in the 1939 National Identity Register. Although early life residential mobility for this birth cohort is relatively low (Falkingham et al., 2016), we cannot ascertain that age 3 residential location was the same as *in utero* or at birth, prohibiting to identify sensitive/critical periods. Multiple addresses across the life course are required to properly understand the very long-term impact of air pollution on healthy ageing and disentangle the effects of earlier exposures from later ones. Second, LLTI was self-reported which is useful to monitor population health and plan healthcare provision but lacks information on specific conditions and is likely affected by reporting bias. Third, although we provided sensitivity analysis by running analysis separately for the 1991, 2001 and 2011 Censuses, differences in the operationalisation of LLTI variables (e.g. from the Census questionnaire wording using 'handicap' in 1991 versus 'disability' in 2001/2011; adding a 12-month timeframe in 2011) raise concerns of comparability (Iveson et al., 2020). Fourth, atmospheric chemistry models are routinely validated against present-day observations, showing good agreement between estimations and observations (Lin et al., 2017). However, there are larger uncertainties when estimating historical air pollution levels, especially in terms of their volumes. Nonetheless, their relative distribution can be considered sufficiently accurate. Fifth, data was determined by availability of administrative records. Socioeconomic variables were either available in early life or after the age of 55, with education assessed later (2001/2011 Censuses) than occupation social position (1991/2001 Censuses). Some life-course confounders (e.g., maternal smoking, childhood health) were not available in the dataset likely introducing bias.

5. Conclusions

This study found that early life $PM_{2.5}$ exposure was associated with limiting long-term illness in mid-to-late adulthood, especially among people growing up in socioeconomically disadvantaged families, and that part of this association was mediated through childhood cognitive ability, education, and social position. We demonstrated the feasibility, merit and challenges of life-course place and health research using administrative data. Future studies should explore the associations in larger cohort studies and with specific health conditions by utilising linked health service use data. Disentangling the effect of early life physical and social environment (e.g., air pollution, population density, area deprivation), investigating potential causal mechanisms and underlying pathways requires further attention. High air pollution concentrations in early life might have long-term implications for population health and healthy ageing despite decades of air pollution mitigations; understanding this relationship requires life-course investigations with representative high-quality datasets.

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Ethics approval

This study was approved by the Research Ethics and Integrity Committee Secretary (2019–332) at the University of Edinburgh, School of Geosciences.

CRediT authorship contribution statement

Gergő Baranyi: Conceptualization, Formal analysis, Methodology, Visualization, Writing – original draft. **Lee Williamson:** Conceptualization, Data curation, Writing – review & editing. **Zhiqiang Feng:** Data curation, Writing – review & editing, Conceptualization. **Edward Carnell:** Software, Writing – review & editing, Resources. **Massimo Vieno:** Software, Writing – review & editing, Resources. **Chris Dibben:** Conceptualization, Funding acquisition, Supervision, Writing – review & editing, Methodology.

Declaration of competing interest

No competing interest declared.

Data availability

The data that has been used is confidential.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.healthplace.2024.103208.

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