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Long term exposure to ambient air pollution and hospital admission burden in Scotland: 16 year prospective population cohort study

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ABSTRACT

Objectives Air pollution is considered a major threat for global health and is associated with various health outcomes. Previous research on long term exposure to ambient air pollution and health placed more emphasis on mortality rather than hospital admission outcomes and was characterised by heterogeneities in the size of effect estimates between studies, with less focus on mental/behavioural or infectious diseases outcomes. In this study, we investigated the association between long term exposure to ambient air pollution and all cause and cause specific hospital admissions.

Design This was a prospective cohort study.

Setting Individual level data from the Scottish Longitudinal Study (SLS) were linked to yearly concentrations of four pollutants (nitrogen dioxide (NO_2), sulphur dioxide (SO_2), particulate matter diameter ≤10 µm (PM_{10}) and particulate matter diameter ≤2.5 µm ($PM_{2.5}$)) at 1 km^2 spatial resolution using the individual's residential postcode for each year between 2002 and 2017.

Participants The study included 202 237 adult individuals aged ≥17 years.

Outcome measures The associations between air pollution and all cause, cardiovascular, respiratory, infectious, mental/behavioural disorders and other cause hospital admissions were examined using multi-level, mixed effects, negative binomial regression.

Results Higher exposure to NO_2, PM_{10} and $PM_{2.5}$ was associated with a higher incidence of all cause, cardiovascular, respiratory and infectious hospital admissions before adjusting for the area of residence, and in fully adjusted models when considering cumulative exposure across time. In fully adjusted models, the incidence rate for respiratory hospital admissions increased by 4.2% (95% CI 2.1% to 6.3%) and 1.2% (95% CI 0.8% to 1.7%) per 1 µg/m^3 increase in $PM_{2.5}$ and NO_2 pollutants, respectively. SO_2 was mainly associated with respiratory hospital admissions (incidence rate ratio (IRR)=1.016; 95% CI 1.004 to 1.027) and NO_2 was related to a higher incidence of hospital admissions for mental/behavioural disorders (IRR=1.021; 95% CI 1.011 to 1.031). Average cumulative exposure to air pollution showed stronger positive associations with higher rates of hospital admissions.

Conclusions The results of this study support an association between long term (16 years) exposure to ambient air pollution and increased all cause and cause specific hospital admissions for both physical and mental/behavioural illnesses. The results suggest that interventions on air pollution through stricter environmental regulations could help ease the hospital care burden in Scotland in the long term.

STRENGTHS AND LIMITATIONS OF THIS STUDY

⇒ This study was based on 16 years (2002–17) of administrative, prospective, cohort individual level data from Scotland, linked to high resolution 1 km^2 air pollution data at the residential postcode.
⇒ The study investigated the association between long term exposure to air pollution and all cause and cause specific hospital admissions, focusing on admissions related to physical illness, such as cardiovascular, respiratory and infectious diseases, as well as on mental and behavioural disorders.
⇒ This study attempted to develop previous research by contributing to the existing evidence on air pollution and health through examination of multiple air pollutants (ie, nitrogen dioxide (NO_2), sulphur dioxide (SO_2), particulate matter diameter ≤10 µm (PM_{10}) and particulate matter diameter ≤2.5 µm ($PM_{2.5}$)) in relation to multiple hospital admission outcomes over a prolonged period of time.
⇒ The limitations of this study included following individuals for 16 years, starting in 2002; thus bias may result from earlier lifetime exposures to air pollution.
⇒ Assessment of air pollution exposure was done at the place of residence, but individuals are exposed to air pollution not only at the place of residence, but also at the workplace, during daily outdoor activities and through commuting patterns.
⇒ We could not account for some important lifestyle covariates (eg, smoking, alcohol consumption and exercise) because this information was not available in the administrative data.

INTRODUCTION

Air pollution is considered by the WHO as one of the largest environmental health risks in the 21st century.[1] Air pollution results in poor health and in increased hospital admissions, mortality and doctor visits, mostly for

cardiovascular, respiratory and cancer diseases.[2–8] For example, in Italy, long term exposure to NO_2 (nitrogen dioxide) and $PM_{2.5}$ (particulate matter diameter ≤2.5 µm) was associated with increased hospital admissions for circulatory diseases, myocardial infarction, lung cancer, kidney cancer and lower respiratory tract infections.[9] Results from the Effects of Low-Level Air Pollution: A Study in Europe (ELAPSE) project have shown elevated asthma, chronic obstructive pulmonary disease and stroke incidence with higher long term exposure to NO_2, $PM_{2.5}$ and black carbon pollutants,[10–12] and elevated lung cancer incidence with higher exposure to $PM_{2.5}$.[13] Similarly, the Danish Health Effects of Air Pollution Components, Noise and Socioeconomic Status (HERMES) project found an association between long term exposure to $PM_{2.5}$ and higher risks of stroke and myocardial infarction,[14–16] and between NO_2 pollutant and higher diabetes risk.[17] A similar association was also shown with short term exposures to PM_{10} (particulate matter diameter ≤10 µm) and $PM_{2.5}$ pollutants and increased respiratory hospital admissions in Poland.[18]

Although the association between long term exposure to air pollution and health is well documented, most research has focused on mortality outcomes.[4 7 19–32] This is because of the ease of access to mortality databases, the less strict ethical considerations and the straightforward analysis of mortality that occurs only once in the individual's life.[2]

Studies that have investigated the association between long term air pollution and health outcomes other than mortality often estimated a combined risk of mortality with other health indicators, such as hospital admissions or doctoral prescriptions, and focused only on analysing the first hospital admission.[12 14 16] Also, variations in the magnitude of effect estimates among studies have been identified in numerous systematic literature reviews and meta-analyses.[2 33–36] These discrepancies may be attributed to differences in the assessment of air pollution exposure, such as residential versus combined residential and workplace assessments, high versus low spatial resolution, baseline versus annual assessments or temporal resolution, including hourly, daily, weekly, monthly or yearly measurements. Other contributing factors include variations in exposure levels, outcome measurements, study locations, population characteristics (eg, age, sex and socioeconomic status) and study designs (eg, cohort, cross sectional, case crossover, case-control and ecological) and methodologies (eg, survival analysis, multi-level mixed effects modelling, structural equation modelling and difference in differences approach).[35 36] The differences in estimates also underscore the necessity for further research to obtain more conclusive evidence regarding the association between air pollution and health.

Additionally, over the past decade, most of the literature has concentrated on investigating the health implications of NO_2, PM_{10} and $PM_{2.5}$ exposure, while other pollutants, such as sulphur dioxide (SO_2), were less studied.[2 23 35] This lack of emphasis on SO_2 may be attributed to a substantial decrease in its emissions, driven by the diminished use of coal in the energy sector and the desulfurisation efforts in cars and power plants in developed nations.[37] Consequently, SO_2 has become a lower priority compared with other pollutants. Nonetheless, investigating the link between prolonged exposure to SO_2 and health outcomes, such as hospital admissions, remains crucial as even at reduced levels, SO_2 can still pose harm to human health.[37]

Finally, despite the literature availability on air pollution in relation to cardiovascular and respiratory diseases,[2 3] other health complications, such as mental/behavioural disorders, have not been thoroughly studied.[38 39] A recent study has also found an association between short term exposure to $PM_{2.5}$ and hospital admissions for rarely studied infectious diseases, such as sepsis, kidney failure, urinary tract infections and skin infections.[40] This suggests the need for more studies investigating the association between air pollution and less studied health outcomes, such as infectious diseases and mental/behavioural disorders.

Taken collectively, our study examined the association between long term exposure to air pollution and all cause and cause specific hospital admissions. We distinguished between hospital admissions related to cardiovascular, respiratory, infectious, mental/behavioural disorders and other illnesses. In examining these associations, we relied on administrative longitudinal individual level data from a large Scottish cohort that linked to yearly 1 km^2 air pollution data using individuals' residential postcodes for each year between 2002 and 2017. Our study tried to develop previous research by contributing to the existing evidence on air pollution and health through examination of multiple air pollutants (ie, NO_2, SO_2, PM_{10} and $PM_{2.5}$) in relation to multiple hospital admission outcomes over time.

METHODS

Design, sample and structure

We used individual level, longitudinal prospective cohort data from the Scottish Longitudinal Study (SLS). This is a representative dataset on 5% of the Scottish population that includes information from linked censuses (2001 and 2011 in the case of our study) on individuals' sociodemographics, vital events records on marriages, births and mortality (up to 2013), and migration and residential histories at the postcode level.[7] To supplement SLS mortality data after 2013 up to 2017, data on individuals' year and month of death were obtained from Public Health Scotland via the Electronic Data Research and Innovation Service.[7]

For this study, we followed 202 237 individuals aged ≥17 years, with a total of 2 810 414 person years between 2002 and 2017. Basic demographic information regarding sex, ethnicity, country of birth, marital status, education and occupation were based on the 2001 and 2011 censuses. Information from two censuses was essential because some of the demographic characteristics (eg, marital status, education and occupation) can change over time. Additionally, we only considered individuals aged ≥17

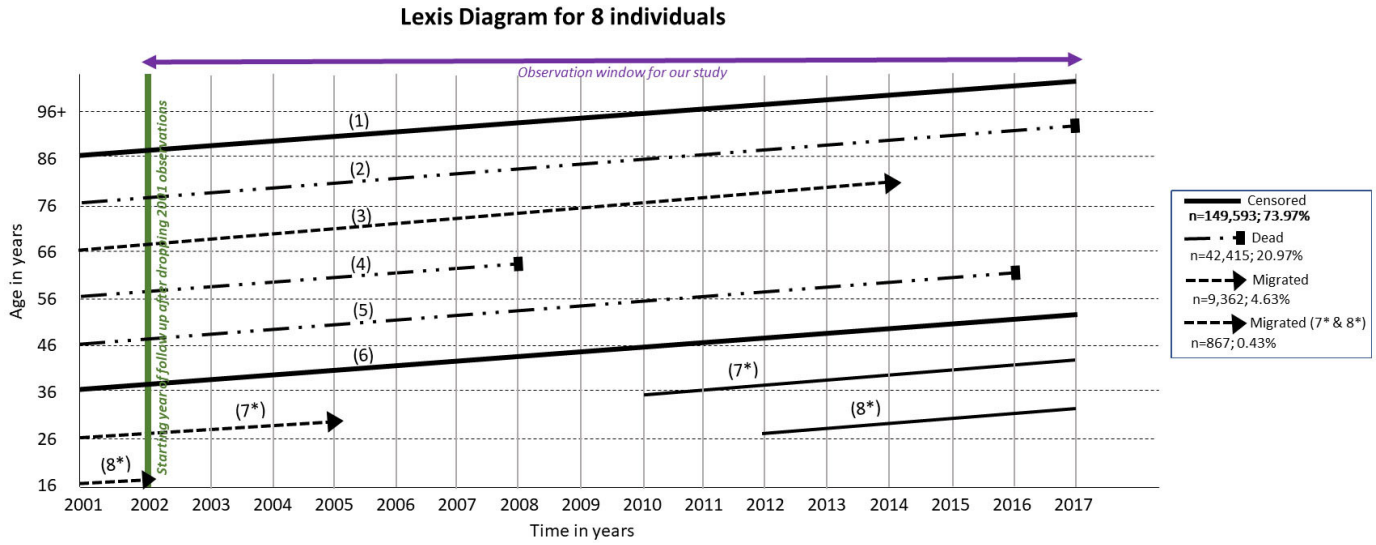

Figure 1 Possibilities of entering and exiting the cohort for eight hypothetical individuals demonstrated in a Lexis diagram. Numbers and percentages in the diagram key are the authors' own calculations based on data from the Scottish Longitudinal Study. The green vertical line represents the starting year of follow-up (2002) after excluding the 2001 observations. Individuals can be followed up until the last year of observation, which is 2017 when they are censored (eg, individual Nos 1 and 6), until they die (eg, individual Nos 2, 4 and 5), or until they migrate without returning to Scotland during the observation period 2002–17 (eg, individual No 3) or migrate and then return to Scotland and thus to our study within the follow-up period (eg, individual Nos 7 and 8). In this context, individual No 7 was followed between 2002 and 2005 and then between 2010 and 2017, inclusive. The years spent by individual No 7 outside of Scotland (2006–09) due to migration were excluded from the analysis. Similarly, individual No 8 was present in 2002 and then was followed from 2012 to 2017, with the years spent abroad (2003–11) due to migration excluded from the analysis. This Lexis diagram further reveals that individuals can enter and exit the cohort based on four scenarios. Firstly, individuals can be followed for the whole study period (2002–17) and then be censored, migrate, or die in 2017. Secondly, individuals can exit the cohort before 2017 due to death in any year during the follow-up period (2002–17). Thirdly, individuals can exit the cohort due to migration out of Scotland, without returning during the follow-up time (2002–17). Fourthly, individuals can exit the cohort due to out migration, but then they re-enter the cohort in later years due to returning to Scotland within the follow-up time (2002–17). If individuals migrate out of Scotland and then return in the same year, this short term migration is disregarded because the individual stayed in Scotland for some months of the full calendar year. If an individual comes back to Scotland from a previous year migration and then migrates out again within the same year, the individuals' observations for that year are kept because some months out of the full calendar year have been spent in Scotland.

years because the mechanisms by which air pollution impacts health may differ between adults and children. Future research would benefit from assessing the impact of air pollution on health among children.

Initially, information was sought for all identified individuals in the SLS aged ≥16 years during the 2001 census (totalling 205 732 individuals). However, 36 individuals were excluded due to the absence of data on sex, and 1127 individuals (constituting 0.55%) were excluded due to missing postcode history. We also excluded 2001 observations (n=204 569) due to missing data on deaths that occurred before the census date (April 2001), which made the 2001 death rate incomparable with the death rates for later years.[7] The SLS cohort structure and the possibilities of entering and exiting the study between 2002 and 2017 are illustrated in a Lexis diagram (figure 1).

Variables

Hospital admissions

The month, year and main underlying cause of hospital admissions were obtained from Public Health Scotland and linked to SLS data by the Electronic Data Research and Innovation Service using the individual's unique identification number. We then calculated the yearly count of all cause and cause specific hospital admissions for each SLS individual. Individuals who did not go to hospital in a certain year were given a count of zero. The International Statistical Classification of Diseases, 10th revision (ICD-10) codes of the main underlying cause of hospital admission were used to determine the cause specific outcomes (online supplemental table 1) as follows: cardiovascular (I00-I99), respiratory (J00-J99), infectious (A00-B99) and mental/behavioural disorders (F00-F99). It should be noted that the infectious categorisation does not include respiratory viral infections covered within the respiratory categorisation of J00-J99.

Air pollution

Annual air pollution data encompassing all sources, including road traffic and industrial/combustion processes for NO_2, SO_2, PM_{10} and $PM_{2.5}$, was acquired from the Department for Environment Food and Rural Affairs.[41] These data consist of raster representations indicating the average annual concentrations of pollutants measured in $\mu g/m^3$. Air dispersion models were used to estimate these concentrations at a spatial

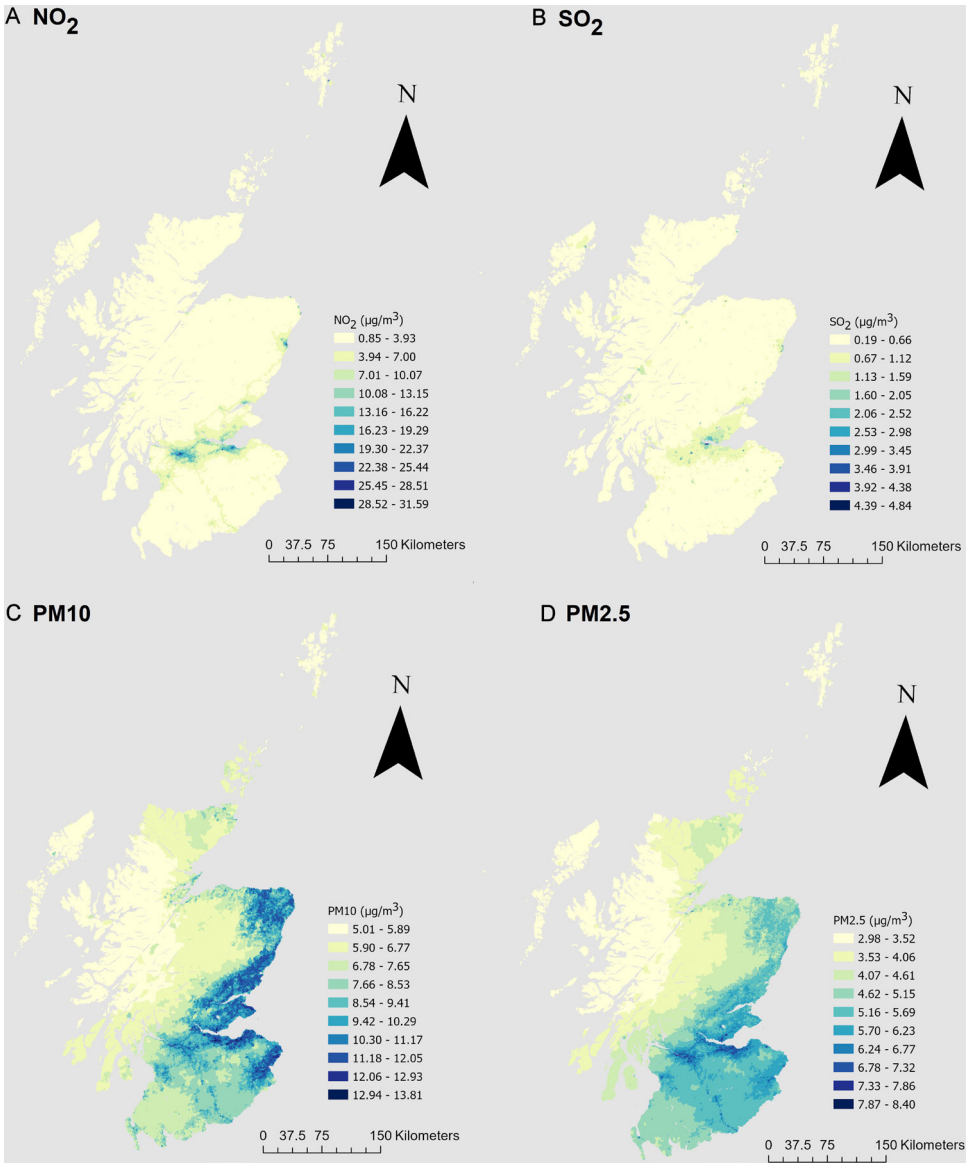

Figure 2 Four maps illustrating the concentrations of nitrogen dioxide (NO_2, A), sulphur dioxide (SO_2, B), particulate matter diameter ≤10 µm (PM_{10}, C) and particulate matter diameter ≤2.5 µm ($PM_{2.5}$, D) pollutants in 2017 across the residential postcodes in Scotland. The map was constructed by the authors in ArcGIS Pro software using air pollution shapefiles for the year 2017 downloaded from the Department for Environment Food and Rural Affairs (DEFRA) online data repository,[41] and postcode boundaries shapefiles obtained from the National Records of Scotland. Both DEFRA and National Records of Scotland shapefiles are governed under the Open Government Licence V.3.0.

resolution of 1 km^2 on the UK national grid.[41] These data were in turn linked to postcodes in Scotland obtained from the National Records of Scotland that fell within the 1 km^2 raster cells for each year between 2002 and 2017. Figure 2 describes the concentrations of NO_2, SO_2, PM_{10} and $PM_{2.5}$ pollutants in 2017 across the residential postcodes in Scotland. In a second step, we linked the data file of matched annual air pollution concentrations with postcodes with the data file of SLS residential postcode histories. Where individuals changed residential postcodes during a certain year, the postcode with the lengthiest monthly duration within that year was the one used.[7]

Study covariates

The association between air pollution and hospital admissions is influenced by several factors: (1) socioeconomic (eg, age, sex, income, education, occupation, ethnicity, country of birth and economic activity)[2 8 42]; (2) individual lifestyle (eg, pre-existing diseases, smoking, alcohol consumption, exercise and obesity)[8 43]; (3) contextual (eg, neighbourhood, deprivation and rural–urban classifications)[43 44]; and (4) environmental (eg, season, temperature, humidity, rainfall and wind).[45]

Accordingly, the following individual level baseline socioeconomic covariates collected at the 2001 and 2011 censuses were included in our study: age, squared age

(age^2; to account for possible non-linear age effects), sex, ethnicity, country of birth, marital status, education and occupation (online supplemental table 2). We additionally included a yearly varying place of residence variable that classified individuals' residential postcodes into six rural–urban categories, based on the data zone where the postcode was located. Calendar year dummies were also included for each year between 2002 and 2017 to control for the time trend.

Analysis

All cause and cause specific hospital admission counts, and socioeconomic and place of residence covariates were described using frequencies, percentages, means, variances and SD. The mean and correlation between the four pollutants were also calculated. Given the high correlations between NO_2, PM_{10} and $PM_{2.5}$ (Pearson's coefficient ≥0.7), the association of the four pollutants with all cause and cause specific hospital admissions was assessed in separate models. SO_2 showed weak correlations (Pearson's coefficient <0.5) with the other pollutants, which enabled us to assess in a sensitivity analysis the association between NO_2, PM_{10} and $PM_{2.5}$ and hospital admissions in two pollutant models adjusting for SO_2.

We used multi-level, mixed effects, negative binomial regression with a random intercept for individuals to study the association between air pollution and all cause and cause specific (cardiovascular, respiratory, infectious, mental/behavioural disorders and other causes) hospital admissions. The negative binomial model was used because about 85% of annual individual observations did not include a hospital admission (a skewed distribution with a high number of zeros). Therefore, the variance of the count of hospital admissions was greater than the mean (table 1), and the overdispersion parameter (α) for all hospital admission outcomes was greater than zero (online supplemental tables 4–9). This makes the negative binomial regression that can account for overdispersion in hospital admission counts more appropriate than Poisson regression.[46] In a sensitivity analysis, we treated the hospital admission outcomes as binary variables (0=not admitted to hospital, 1=admitted to hospital) and used multi-level, mixed effects logistic regression for analysis.

Three stepwise models were developed: model 1 included the air pollution independent variable and controlled for age, age^2, sex and calendar year dummies; model 2 controlled additionally for ethnicity, country of birth, marital status, education and occupation; and model 3 included the place of residence covariate.

The association of all cause and cause specific hospital admissions with the socioeconomic and place of residence covariates is shown in online supplemental tables 4–15. Results of the multi-level, negative binomial regression are presented as incidence rate ratios (IRRs) with 95% confidence intervals (CIs) per 1 µg/m^3 increase in pollutants and visualised in coefficient plots. Statistical significance was p<0.05. Statistical analysis was conducted in STATA$_{16}$. Coefficient plots using the ggplot package were performed in R Studio. Spatial preprocessing was conducted in ArcGIS-Pro software.

Table 1 Description of hospital admissions (n=2 810 414 observations, study period 2002–17)

	Frequency	%	Mean	Variance	SD
All cause hospital admission			0.33	1.64	1.28
0	2 388 584	85			
1	241 717	8.6			
2	80 813	2.9			
≥3	99 300	3.5			
Cardiovascular hospital admission			0.04	0.14	0.37
0	2 753 992	98			
1	29 432	1.1			
2	13 091	0.5			
≥3	13 899	0.5			
Respiratory hospital admission			0.03	0.10	0.32
0	2 773 192	98.7			
1	19 685	0.7			
2	9538	0.3			
≥3	7999	0.3			
Infectious hospital admission			0.005	0.01	0.10
0	2 802 217	99.7			
1	5174	0.2			
2	1937	0.1			
≥3	1086	0.04			
Mental/behavioural disorders hospital admission			0.003	0.01	0.10
0	2 804 737	99.8			
1	3521	0.1			
2	1263	0.04			
≥3	893	0.03			
Hospital admission for other causes			0.26	1.18	1.09
0	2 448 665	87.1			
1	225 980	8.0			
2	66 976	2.4			
≥3	68 793	2.5			

Data source was the authors' own calculations based on data from the Scottish Longitudinal Study.

Calculation of IRR was as follows:

$$IRR_{ti} = e^{\beta_0 + U_{0i} + \beta 1 \text{ overall pollutant concentration}_{ti} + \beta n \text{ Covariates}_{ti} + \varepsilon_{ti}}$$

Where *IRRti* is the incident rate ratio for the hospital admission outcome for individuals *i* at year *t*; $\beta 1$ is the air pollution coefficient, βn represents coefficients of the other study covariates and $\beta 0$ is the fixed intercept; U_{0i} is level 2 random intercept of individuals; and ε_{ti} are the model residuals.

Additional analysis

To assess the impact of cumulative air pollution (CAP) exposure from year to year and across the different places of residence between 2002 and 2017, we repeated models 1–3 replacing the yearly air pollution variable with average CAP exposure. Following the methods of Bentayeb *et al*,[47] the CAP variable was calculated as the average of cumulative yearly exposure before censoring or death. Thus for every individual, we computed the mean pollutant concentration from the baseline year (2002) to each year of follow-up (eg, exposure in 2004 was calculated as the average of annual concentrations from 2002 to 2004; in 2005, from 2002 to 2005, etc).

RESULTS

Hospital admissions

About 15% of person years involved a hospital admission The mean of all cause hospital admissions was 0.3 (variance=1.6; SD=1.3) with 8.6% of yearly individual observations including one hospital admission, 2.9% including two admissions and 3.5% including three or more admissions. The mean of cardiovascular, respiratory, infectious, mental/behavioural disorders and other cause hospital admission count were 0.04 (variance=0.14; SD=0.37), 0.03 (variance=0.1; SD=0.32), 0.005 (variance=0.01; SD=0.1), 0.003 (variance=0.01; SD=0.1) and 0.26 (variance=1.18; SD=1.1), respectively (table 1).

Study covariates

Most individuals were women (53% vs 47% men), of white ethnicity (~95%), were born in Scotland (~87%), were married (~55%), had a post-school/university education (24% in 2002–10; 34% in 2011–17), worked in white collar high skilled (27% in 2002–10; 33% in 2011–17) or white collar low skilled (25% in 2002–10; 29% in 2011–17) jobs and lived in large urban (35%) areas (table 2).

Air pollution

Fluctuations in air pollutant levels were observed across the years 2002–17, with higher concentrations recorded in the initial 3 years (2002–04) compared with subsequent years (online supplemental figure 1). Over the period 2002–17, mean (±SD) concentrations of NO_2, SO_2, PM_{10} and $PM_{2.5}$ pollutants were 11.9±6.4, 1.9±1.5, 11.3±2.1 and 7.2±1.6 µg/m^3, respectively (online supplemental table 3). The average annual mean concentrations for NO_2, PM_{10} and $PM_{2.5}$ pollutants were lower than the 2005 WHO guidelines of 40 µg/m^3 for NO_2, 20 µg/m^3 for PM_{10} and 10 µg/m^3 for $PM_{2.5}$, but the concentrations of NO_2 and $PM_{2.5}$ were higher than the most recent 2021 WHO guidelines of 10 µg/m^3 for NO_2 and 5 µg/m^3 for $PM_{2.5}$.[48] Significant correlations (Pearson's coefficient ≥0.7) were observed among NO_2, PM_{10} and $PM_{2.5}$ (online supplemental table 3), potentially attributed to the atmospheric chemical reactions involving these pollutants.[19]

Association of air pollution with hospital admissions

Our results revealed higher incidence rate ratios for cardiovascular (except for model 3), respiratory and infectious hospital admissions with increasing concentrations of NO_2, PM_{10} (except for model 3) and $PM_{2.5}$ pollutants. After adjusting for socioeconomic variables and place of residence (model 3), the incidence rate for respiratory hospital admissions increased by 4.2% (95% CI 2.1% to 6.3%) and 1.2% (95% CI 0.8% to 1.7%) per 1 µg/m^3 increase in $PM_{2.5}$ and NO_2, respectively. Higher exposure to SO_2 was associated with higher rates of respiratory hospital admissions only in models 1 and 2. Hospital admissions for mental/behavioural disorders were associated with higher exposure to NO_2 (IRR=1.021; 95% CI 1.011 to 1.031). Contrary to our expectations, higher exposure to SO_2 was associated with lower incidence rates for all cause, mental/behavioural disorders and other cause hospital admissions in models 2 and 3 (figure 3).

In a sensitivity analysis considering hospital admissions as binary (yes/no) outcomes, we observed similar results; higher exposure to NO_2, PM_{10} and $PM_{2.5}$ pollutants was associated with higher odds of cardiovascular, respiratory and infectious hospital admissions. An exception was the absence of an association between NO_2 and $PM_{2.5}$ pollutants and all cause hospital admission treated as a binary (yes/no) outcome (online supplemental figure 3). Similar results were observed in two pollutant models, which included SO_2 and one of the other three pollutants in the same model (online supplemental figures 2 and 4).

Average CAP results

Stronger positive associations were noticed in the analysis of average CAP effect on hospital admissions compared with the analysis of yearly air pollution effects. Higher average CAP concentrations for NO_2, PM_{10} and $PM_{2.5}$ pollutants were associated with higher incidence rate ratios for all cause, cardiovascular, respiratory, infectious, mental/behavioural disorders and other cause hospital admissions. Cumulative exposure to SO_2 was associated with higher rates of respiratory hospital admissions in all three models. The incidence rate for respiratory hospital admissions increased by 12.6% (95% CI 9.9% to 15.5%), 6.8% (95% CI 5.1% to 8.5%), 2.8% (95% CI 1.1% to 4.6%) and 2.1% (95% CI 1.7% to 2.6%) per 1 µg/m^3 increase in average cumulative exposure to $PM_{2.5}$, PM_{10}, SO_2 and NO_2 pollutants, respectively. Higher cumulative exposure to SO_2 was not associated anymore with a lower incidence of hospital admissions for mental/behavioural disorders (figure 4). Similar results were noted when hospital admissions were treated as binary (yes/no) outcomes (online supplemental figure 5). This shows that long term average cumulative exposure to air pollution has a greater effect on both physical and mental health outcomes, resulting in higher rates of hospital admissions.

DISCUSSION

In this study, we used a large and representative census based, individual level cohort data linked to 1 km^2

Table 2 Description of socioeconomic study covariates (n=2 810 414 observations, study period 2002–17)

		Census fixed socioeconomic covariates (n=202 237 individuals)			
		2002–10		2011–17	
		Frequency	%	Frequency	%
Sex	Men	94 859	46.9	80 282	46.9
	Women	107 314	53.1	90 753	53.1
Ethnicity	White	192 485	95.2	163 571	95.6
	Not white	9688	4.8	7464	4.4
Ethnicity: 3 categories	White	192 485	95.2	163 571	95.6
	Pakistani/Bangladeshi/Indian	1525	0.8	1323	0.8
	Other ethnicities	8163	4.0	6141	3.6
Country of birth	Born in Scotland	173 229	85.7	149 018	87.1
	Born in rest of UK	19 649	9.7	15 340	9.0
	Not born in UK	9295	4.6	6677	3.9
Marital status	Married	104 386	51.6	93 867	54.9
	Single, never married	58 396	28.9	38 979	22.8
	Divorced/separated/widowed	38 481	19.0	37 770	22.1
	No response	910	0.5	419	0.2
Education	No educational qualification	60 311	29.8	53 223	31.1
	Intermediate school	44 854	22.2	36 561	21.4
	High school	27 844	13.8	20 343	11.9
	Post-school/university	48 251	23.9	58 781	34.4
	Still a student	688	0.3	53	0.0
	No response/not recoded	20 225	10.0	2074	1.2
Occupation	White collar high skilled	54 299	26.9	55 595	32.5
	White collar low skilled	50 325	24.9	49 202	28.8
	Blue collar high skilled	20 530	10.2	20 500	12.0
	Blue collar low skilled	46 718	23.1	38 173	22.3
	Not applicable: students/never worked	25 535	12.6	5793	3.4
	No response	4766	2.4	1772	1.0
Total		202 173	100	171 035	100
	Yearly varying covariates (n=2 810 414 observations)				
Age (years) (mean (SD))	52.53 (17.57)				
Place of residence	Large urban areas	977 697	34.8		
	Other urban areas	815 048	29.0		
	Accessible small towns	207 083	7.4		
	Remote small towns	67 776	2.4		
	Accessible rural areas	528 929	18.8		
	Remote rural areas	213 881	7.6		

Data source is the authors' own calculations based on data from the Scottish Longitudinal Study.
Z test to compare the percentages between 2002–10 and 2011–17 was 0.0046, with a p value of 0.996, indicating that there was no significant difference in the percentages between the two study periods.

resolution air pollution at the postcode level between 2002 and 2017. Our analysis supported an association between long term exposure to NO_2, SO_2, PM_{10} and $PM_{2.5}$ pollutants and higher rates of all cause, cardiovascular, respiratory, infectious, mental/behavioural disorders and other cause hospital admissions. The direction of these positive associations was concordant with previous studies investigating the long term effects of different air pollutants on all cause,[8 49 50] cardiovascular,[9 12 16 49] respiratory[9–11 50] and mental/behavioural disorder[51] hospital admissions.

Given the differences in population size, study location, air pollution exposure assessment and level, and outcomes measurement and methodology, we cannot

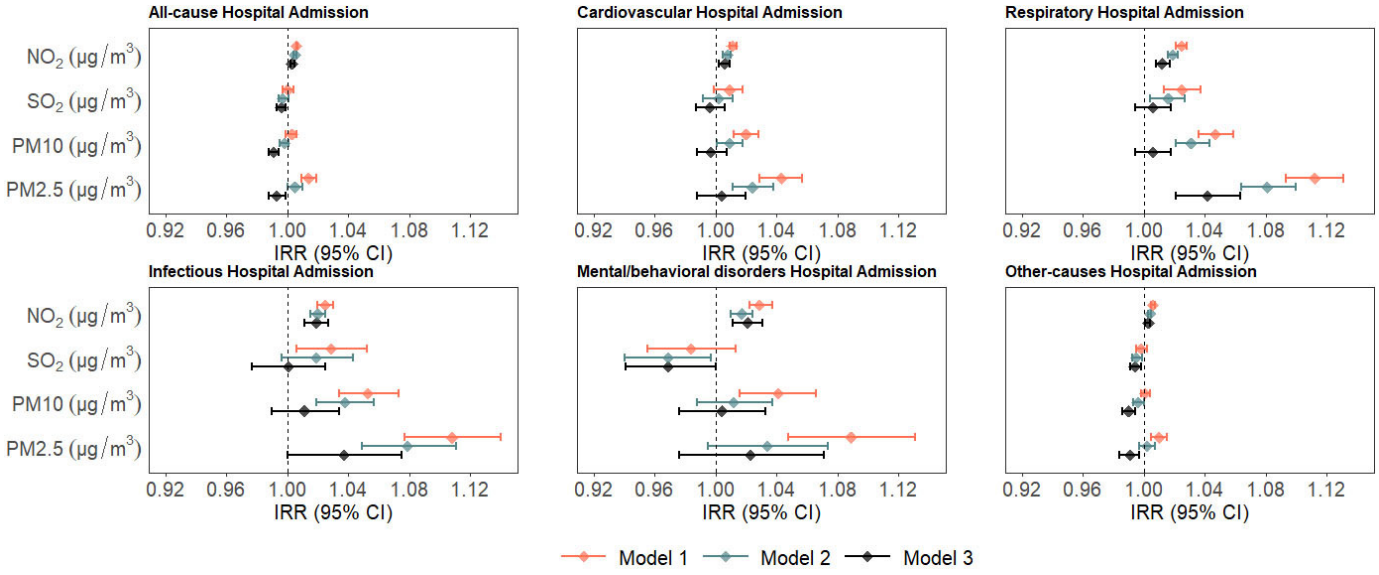

Figure 3 Association of all cause and cause specific hospital admissions with nitrogen dioxide (NO_2), sulphur dioxide (SO_2), particulate matter diameter ≤10 µm (PM_{10}) and particulate matter diameter ≤2.5 µm ($PM_{2.5}$) air pollutants in separate multi-level, mixed effects negative binomial models (n=202 237 individuals and 2 810 414 observations). Data source was the authors' own calculations based on data from the Scottish Longitudinal Study. The broken line is placed at incidence rate ratio (IRR)=1 as a cut-off for statistically insignificant results. Model 1 is adjusted for age, age^2, sex and calendar year dummies; model 2 is additionally adjusted for ethnicity, country of birth, marital status, education and occupation; and model 3 is additionally adjusted for place of residence.

compare directly the magnitude of our estimates with estimates from other studies. Rather, we can conclude that our findings are in line with what the previous literature has suggested, with some noted heterogeneities. For example, Yazdi *et al* found that with every 1 µg/m^3 increase in $PM_{2.5}$ and NO_2 pollutants, the risk of hospital admission for stroke increased by 0.0091% and 0.00059%, respectively.[49] Gandini *et al* found higher hazard ratios of 1.05/1.05, 1.03/1.02, 1.15/1.14, 1.18/1.20, 1.24/1.20, 1.06/1.06 and 1.10/1.05 for circulatory system diseases, respiratory system diseases, stroke, lung cancer, kidney cancer, all cancers excluding lung cancer and lower respiratory tract infections first ever hospital admissions with every 10 µg/m^3 higher exposure to $PM_{2.5}$/NO_2

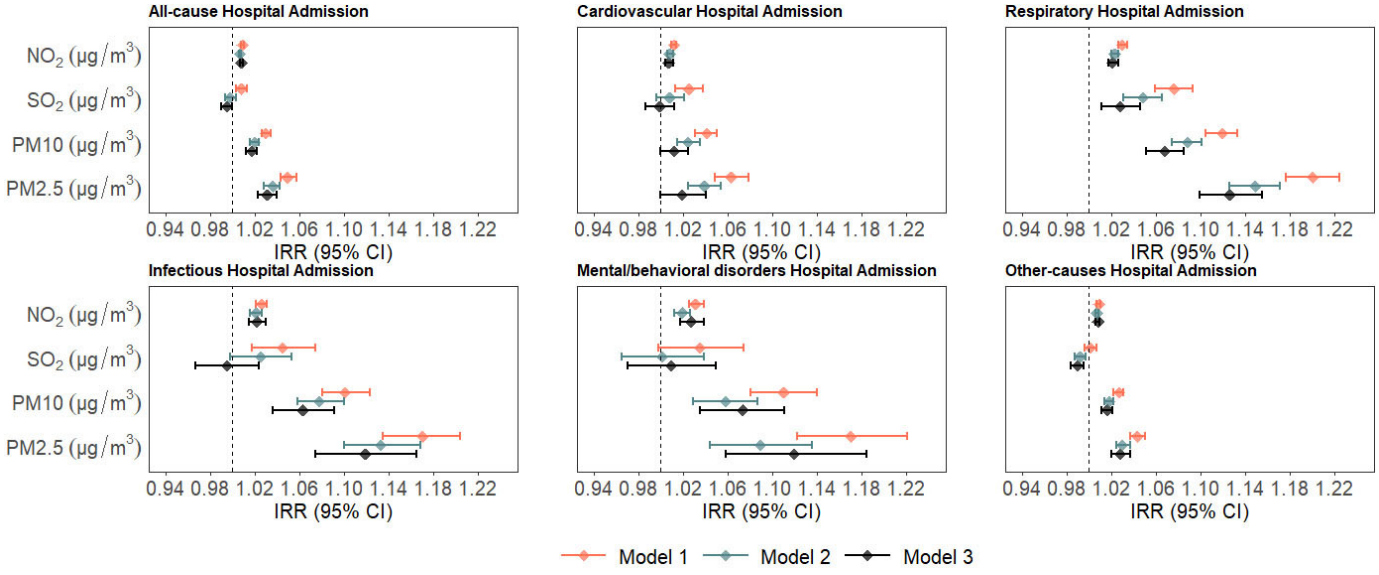

Figure 4 Association of all cause and cause specific hospital admissions with average cumulative exposure to nitrogen dioxide (NO_2), sulphur dioxide (SO_2), particulate matter diameter ≤10 µm (PM_{10}) and particulate matter diameter ≤2.5 µm ($PM_{2.5}$) air pollutants (n=202 237 individuals and 2 810 414 observations). Data source is the authors' own calculations based on data from the Scottish Longitudinal Study. The broken line is placed at incidence rate ratio (IRR)=1 as a cut-off for statistically insignificant results. Model 1 is adjusted for age, age^2, sex and calendar year dummies; model 2 is additionally adjusted for ethnicity, country of birth, marital status, education and occupation; and model 3 is additionally adjusted for place of residence.

pollutants.[9] Similarly, Liu *et al* found 1.17 and 1.11 higher hazard ratios for chronic obstructive pulmonary disease first ever hospital admission with every 5 µg/m^3 and 10 µg/m^3 higher exposure to $PM_{2.5}$ and NO_2 pollutants, respectively.[11]

Some of these heterogeneities might be due to residual confounding from unobserved factors. For example, our models adjusted only for sociodemographic and economic factors of individuals (eg, age, age squared, sex, education, marital status, ethnicity, country of birth and occupation), as well as for the time trend (ie, year dummies) and place of residence (ie, rural–urban area classifications). However, lifestyle covariates at the individual level, such as smoking, exercise, alcohol consumption or body mass index, which could influence the relationship between air pollution and hospital admissions, were not considered due to their unavailability in the SLS register based data. Accounting for these lifestyle covariates could result in a slight adjustment of association estimates, typically within the range of a 1–2% increase or decrease, depending on the specific outcome, as indicated by other studies.[4 6 25 52–54] Similarly, our models did not incorporate environmental factors at the place of residence, such as noise pollution or the absence of green spaces. However, the impact on association magnitudes is anticipated to be minimal, with estimates showing an attenuation of 0–3% increase or decrease, as documented in previous studies.[17 54 55]

The adjustment of our models for the individual level socioeconomic and rural–urban covariates might also absorb some of the residual confounding due to the interconnections between individuals' socioeconomic circumstances, their lifestyle and their surrounding environment. Additionally, our models were not adjusted for weather factors, such as temperature, humidity, rainfall or wind, which might impact the association between air pollution and hospital admissions. Nevertheless, the air pollution data used in this study were modelled yearly data using air dispersion models, and meteorological factors, such as temperature and wind, were accounted for within the modelling framework.[41]

Regarding the positive association between long term exposure to NO_2, PM_{10} and $PM_{2.5}$ and infectious hospital admissions, the literature on this outcome is scarce. But one study examining the association between short term exposure to $PM_{2.5}$ air pollution and hospital admissions for infectious diseases, such as sepsis, urinary tract and skin infections, corroborate our findings.[40]

Our study showed that yearly exposures to different air pollutants can be associated with different hospital admission outcomes. For example, NO_2 was associated with all hospital admission outcomes, SO_2 was related to respiratory hospital admissions, while PM_{10} and $PM_{2.5}$ were associated with respiratory and infectious hospital admissions. This could be related to the mechanisms of action of specific pollutants in producing toxic effects. Gaseous pollutants (eg, NO_2 and SO_2) are irritants of the respiratory system that can penetrate deep into the lungs, inducing respiratory irritation, mucus production, coughing, wheezing, bronchoconstriction, airways inflammation, bronchospasm and pulmonary oedema.[2 56 57] Long term exposure to NO_2 is also related to weakening of the immune system and to cardiovascular problems, such as ventricle hypertrophy.[56] Additionally, NO_2 exposure can trigger neuronal injury and neurological disorders through the formation of reactive oxygen species and free radicals.[58 59]

Despite the harmful effect of gaseous air pollutants (eg, NO_2 and SO_2) on health, particulate matter, especially particles with smaller diameters (eg, $PM_{2.5}$), have the greatest effect, as shown in our study, and particularly on respiratory health. Particulate matter can penetrate deeply into the respiratory system through air breathing, reaching the alveoli and bloodstream. This will initiate the oxidative stress mechanism and the production of reactive oxygen species affecting various systems in the human body, including the respiratory, cardiovascular, immune and neural systems.[2 56 57]

We also observed elevated estimates for average cumulative compared with yearly air pollution exposures in relation to hospital admissions, particularly for admissions for mental and behavioural disorders. While yearly exposure to SO_2 showed an unexpected negative association for hospital admissions for mental/behavioural disorders, which could be attributed to the small variation in the yearly concentrations of SO_2 across time (online supplemental figure 1) or to the residual confounding from unobserved factors, average cumulative exposure to SO_2 did not show this association. Higher exposure to cumulative PM_{10} and $PM_{2.5}$ pollutants was also associated with a higher incidence of hospital admissions for mental/behavioural disorders, while this association was not observed for yearly PM_{10} and $PM_{2.5}$ exposures. This shows that the average accumulation of air pollution exposures across time and for different places of residence had a greater effect on health, especially for mental and behavioural complications that take time to show up.

Finally, adjusting our analysis for place of residence (model 3) reduced the magnitude of associations between hospital admission outcomes and all four pollutants. This suggests that place of residence (urban vs rural) has a crucial role in the intensity and duration of exposure to ambient air pollution and its associated illness. For example, air pollution emission sources are more abundant in urban areas and factors that can absorb the air pollution emissions (eg, green spaces) are less available. Figure 2 confirms this, showing higher concentrations of traffic related (ie, NO_2) and industrial (ie, SO_2) air pollution in the urban central belt of Scotland and in large cities. Figure 2 also shows high concentrations of PM_{10} and $PM_{2.5}$ in major cities and along the east coast because particulate matter pollution originates from both traffic and industrial sources and can travel for long distances based on the wind direction and other meteorological and topological factors.[60–62]

Our study had some limitations. Firstly, individuals were followed for 16 years (2002–17) and thus bias may

result from previous lifetime exposures to air pollution at different residences. As shown in our analysis, average CAP exposure across the 16 years of the study had a greater effect on hospital admission rates compared with yearly exposures, particularly for mental/behavioural disorders.

Secondly, individuals' exposure to ambient air pollution was assessed at a yearly rather than monthly or daily basis, which did not allow for seasonal variations, and the residential postcode was used as a proxy for individuals' exposure to air pollution. This does not necessarily equate to true personal exposure, which can happen indoors, at the workplace, during daily outdoor activities and through commuting patterns. However, the emergence of real time GPS data would create an opportunity for future research to analyse real time exposure to ambient air pollution by knowing the real time location of individuals.

Finally, we could not account for some important lifestyle covariates (eg, smoking or exercise), as discussed previously, due to the unavailability of this information in the SLS census based data. However, using administrative data has its advantages, including high quality large representative data, less selection bias and the provision of continuous longitudinal information on residential postcode histories, emigrations and immigrations, and mortality and birth vital events. Knowing the exact postcode histories of individuals between 2002 and 2017, as provided in the SLS census based data, was also essential to obtain accurate assessments of individuals' residential air pollution exposure.

CONCLUSIONS

The results of this study support an association between long term exposure to air pollution and all cause and cause specific hospital admissions. Air pollution was associated with higher rates of hospital admissions for both physical (eg, respiratory, cardiovascular and infectious) and mental/behavioural diseases. Policies and interventions on air pollution through stricter environmental regulations, long term planning and the shifting towards renewable energy could eventually help ease the hospital care burden in Scotland in the long term. Specifically, policies aimed at making the zero emission zones (ie, small areas where only zero emission vehicles, pedestrians and bikes are permitted) more abundant in Scotland, especially in the central belt of Scotland where busy and more polluted cities such as Glasgow and Edinburgh are located, would improve the air quality and in turn lower the hospital care burden in those cities. For future research, it is also recommended to study the impact of air pollution on health outcomes synergically alongside other environmental issues, such as weather fluctuations and climate change.

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Contributors MAAA: conceptualisation, investigation, methodology, data curation, formal analysis, writing-original draft, writing-review and editing, visualisation and project administration. HK, UD and FS: conceptualisation, funding acquisition, supervision, writing-review and editing. The guarantor of the study is MAAA who accepts full responsibility for the finished work and/or the conduct of the study, had access to the data and controlled the decision to publish.

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Competing interests None declared.

Patient and public involvement The research conducted was carried out on the Scottish Longitudinal Study (SLS) quantitative data. A summary of the main findings of this study was shared with the SLS team and published on their website. Individuals in the SLS dataset are anonymised, and therefore it is impossible to share the data directly with each individual participant. Patients were not involved in the direct creation or carrying out of the research study. Instead, we had access to the month and year of mortality and hospital admissions records of each anonymised individual within the SLS dataset.

Patient consent for publication Not applicable.

Ethics approval This paper was part of a PhD project (id=2019006) that was granted ethical approval on 14 May 2020 by the School of Geography and Sustainable Development Ethics Committee, acting on behalf of the University Teaching and Research Ethics Committee (UTREC) at the University of St Andrews. Access to the Scottish Longitudinal Study (SLS) data was approved by the SLS manager following a detailed application, and access to the linked data from Public Health Scotland via the Electronic Data Research and Innovation Service (eDRIS) was approved following a detailed Public Benefit and Privacy Panel for Health and Social Care (HSC-PBPP) application. The SLS team had already obtained all of the necessary consent and approvals for the data processing and analysis. Based on the SLS data policy and the sensitivity of the data used, all data cleaning, management and analysis were performed in the safe settings of the SLS, Ladywell House, Edinburgh, to ensure individuals' confidentiality, and safe and secure data storage and access.

Provenance and peer review Not commissioned; externally peer reviewed.

Data availability statement Data may be obtained from a third party and are not publicly available. Data underlying this study are confidential and not publicly available due to ethical and legal restrictions. We used the Scottish Longitudinal Study dataset, which contains linked census, vital events and education data for a 5% sample of the population of Scotland. These data are protected by a copyright license and only available for licensed researchers in the UK following a detailed application and security checks. Researchers must also pass a Safe Researcher Training which equip them with the necessary knowledge and information to analyse data in safe settings. Further information on how to access the SLS data are available here: https://sls.lscs.ac.uk/.

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