



# Long-term exposure to air pollution and mortality in Scotland: A register-based individual-level longitudinal study

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## ABSTRACT

**Background:** Air pollution is associated with several adverse health outcomes. However, heterogeneity in the size of effect estimates between cohort studies for long-term exposures exist and pollutants like SO<sub>2</sub> and mental/behavioural health outcomes are little studied. This study examines the association between long-term exposure to multiple ambient air pollutants and all-cause and cause-specific mortality from both physical and mental illnesses.

**Methods:** We used individual-level administrative data from the *Scottish-Longitudinal-Study (SLS)* on 202,237 individuals aged 17 and older, followed between 2002 and 2017. The SLS dataset was linked to annual concentrations of NO<sub>2</sub>, SO<sub>2</sub>, and particulate-matter (PM<sub>10</sub>, PM<sub>2.5</sub>) pollution at 1 km<sup>2</sup> spatial resolution using the individuals' residential postcode. We applied survival analysis to assess the association between air pollution and all-cause, cardiovascular, respiratory, cancer, mental/behavioural disorders/suicides, and other-causes mortality.

**Results:** Higher all-cause mortality was associated with increasing concentrations of PM<sub>2.5</sub>, PM<sub>10</sub>, NO<sub>2</sub>, and SO<sub>2</sub> pollutants. NO<sub>2</sub>, PM<sub>10</sub>, and PM<sub>2.5</sub> were also associated with cardiovascular, respiratory, cancer and other-causes mortality. For example, the mortality hazard from respiratory diseases was 1.062 (95%CI = 1.028–1.096), 1.025 (95%CI = 1.005–1.045), and 1.013 (95%CI = 1.007–1.020) per 1 µg/m<sup>3</sup> increase in PM<sub>2.5</sub>, PM<sub>10</sub> and NO<sub>2</sub> pollutants, respectively. In contrast, mortality from mental and behavioural disorders was associated with 1 µg/m<sup>3</sup> higher exposure to SO<sub>2</sub> pollutant (HR = 1.042; 95%CI = 1.015–1.069).

**Conclusion:** This study revealed an association between long-term (16-years) exposure to ambient air pollution and all-cause and cause-specific mortality. The results suggest that policies and interventions to enhance air quality would reduce the mortality hazard from cardio-respiratory, cancer, and mental/behavioural disorders in the long-term.

## 1. Introduction

The air we breathe is crucial for health. Previous epidemiological cohort studies from Europe and North America have shown that long-term exposure to ambient air pollution is associated with poor health outcomes and increased mortality from all-causes, but mostly from cardiovascular, respiratory, and lung cancer diseases (Abed Al Ahad et al., 2022, 2023; Beelen et al., 2014; Beverland et al., 2012; Crouse Dan et al., 2012, 2015; Dehbi et al., 2017; Fischer et al., 2015; Hvidtfeldt et al., 2019; Pinault et al., 2017; Pope et al., 2019; Raaschou-Nielsen et al., 2020; Rodopoulou et al., 2022; So et al., 2020; Zhang et al., 2021). For example, the ELAPSE project across eight European cohorts followed

over an average of 20 years showed that higher exposures to particulate-matter with diameters of less than or equal to 2.5 µm (PM<sub>2.5</sub>), nitrogen dioxide (NO<sub>2</sub>), and black carbon are associated with increased risk of almost all mortality outcomes including stroke, lung cancer, and Chronic Obstructive Pulmonary Disease (COPD), and with a 13% increase in natural deaths per 5 µg/m<sup>3</sup> increase in PM<sub>2.5</sub> (Hvidtfeldt et al., 2021; Liu et al., 2021; Stafoggia et al., 2022; Strak et al., 2021; Wolf et al., 2021).

Likewise, studies from the Danish HERMES project have shown associations between long-term exposure to NO<sub>2</sub> and PM<sub>2.5</sub> pollutants and higher hazards of cardiovascular mortality outcomes including stroke, myocardial infarction, and diabetes (Poulsen et al., 2023a; Poulsen

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et al., 2023b; Sørensen et al., 2022). In contrast, little research has been conducted on the association between long-term exposure to ambient air pollution and mental and behavioural health outcomes as per a recent systematic literature review (Dominski et al., 2021).

Despite the increasing evidence on the adverse air pollution associations with mortality and health, heterogeneity in the size of effect estimates between cohort studies for long-term exposures has been identified as per several systematic literature reviews and meta-analyses (Abed Al Ahad et al., 2020; Chen and Hoek, 2020; Hoek et al., 2013; Pope et al., 2020; Vodonos et al., 2018). These heterogeneities might be related to differences in air pollution exposure assessments (e.g., residential versus residential plus workplace assessments, assessments using high versus low spatial resolution, or baseline versus yearly assessments), exposure levels, study location, population composition (e.g., age, sex, socioeconomic status), and study methodology (Chen and Hoek, 2020; Vodonos et al., 2018). The heterogeneities in estimates also suggest the need for further studies to obtain more conclusive evidence on the association between air pollution and health.

Furthermore, literature in the last decade has been mostly concentrated on the health impacts of exposure to NO<sub>2</sub> and particulate-matter with diameters of less than or equal to 10 µm (PM<sub>10</sub>) or 2.5 µm (PM<sub>2.5</sub>), whereas other pollutants such as sulphur dioxide (SO<sub>2</sub>) have been little studied (Abed Al Ahad et al., 2020; Pope et al., 2019; Vodonos et al., 2018). This could be due to the large decline in SO<sub>2</sub> emissions driven by the reduction in coal use in the energy sector and the desulfurization of cars and power plants in developed nations (O'Brien et al., 2023), making this pollutant of low priority compared to other pollutants. Regardless, studying the association between long-term exposure to SO<sub>2</sub> and mortality outcomes is important, given that SO<sub>2</sub> would still be harmful for human beings even at low exposures (O'Brien et al., 2023).

Taken together, this study investigates the association between long-term (16 years) exposure to four ambient air pollutants (NO<sub>2</sub>, SO<sub>2</sub>, PM<sub>10</sub>, and PM<sub>2.5</sub>) and all-cause and cause-specific mortality in Scotland. The associations between air pollution and mortality are examined distinguishing deaths related to cardiovascular, respiratory, cancer and other diseases as well as deaths related to mental/behavioural disorders/suicides. These associations are examined by linking longitudinal administrative individual-level data from a large Scottish cohort using the individuals' residential postcodes for each year between 2002 and 2017 to annual air pollution data modelled at 1 km<sup>2</sup> spatial resolution. Our study contributes to the evidence on air pollution and health by examining multiple air pollutants in relation to multiple mortality outcomes, providing insights into the specific mechanism of action of each pollutant in producing toxic effects.

## 2. Methods

### 2.1. Design and data

A prospective cohort longitudinal design was employed using individual-level data from the "Scottish Longitudinal Study (SLS)" (Scottish Longitudinal Study, Accessed November 08, 2021). The SLS is a large representative dataset of 5% of the Scottish population. It includes information from three linked censuses (1991, 2001, 2011) on individuals' socio-demographics and vital events including marriages, births, and mortality (up to 2013). The dataset includes central-register data on migration into or out of Scotland and on residential postcode histories (Scottish Longitudinal Study, Accessed November 08, 2021). To complement the SLS mortality data after 2013 through 2017, "Public Health Scotland" through the "Electronic Data Research and Innovation Service (eDRIS)" provided data on the year, month, and final underlying cause of death for SLS individuals from 2014 to 2017.

For this study, a cohort of 202,237 SLS individuals aged 17+ starting in 2002 was followed through 2017. Data was requested initially on all traced SLS individuals aged 16+ in the 2001 census (n = 205,732 individuals). We dropped 36 individuals because of missing gender and

1127 individuals (0.6%) because of missing postcode history. We also excluded observations for all individuals in 2001 due to no information on deaths that occurred prior to the census date on April 2001, which made the 2001 death rate incomparable to death rates in later years.

The SLS cohort structure relies on the 2001 and 2011 baseline Scottish censuses and on individuals' migration and mortality events. The possibilities of entering and exiting the cohort during the follow-up period (2002–2017) for 9 hypothetical individuals are illustrated in a Lexis-diagram (Fig. 1).

### 2.2. Variables

#### 2.2.1. Mortality

Year of death was used to indicate whether individuals died in that year. Codes from the 10th Revision of International Statistical Classification of Diseases (ICD-10) were used to classify the main underlying cause of death as follows: cardiovascular (I00–I99), respiratory (J00–J99), cancer (C00–C97; D00–D48), and mental/behavioural disorders/suicide (F00–F99; X60–X84; Y10–Y34; Y87.0; Y87.2) (Supplementary Material Table 1).

In data quality checks, we compared the age-specific mortality rates (starting at age 20) of our SLS cohort to mortality rates in Scotland published by the Human Mortality Database (HMD) for the period of 2002–2017. Age-specific mortality rates are slightly lower for the SLS cohort compared to HMD for ages below 65 and above 84 (rate-ratio between 0.8 and 0.95; the difference does not exceed 20%; Supplementary material: Fig. 1).

#### 2.2.2. Air pollution

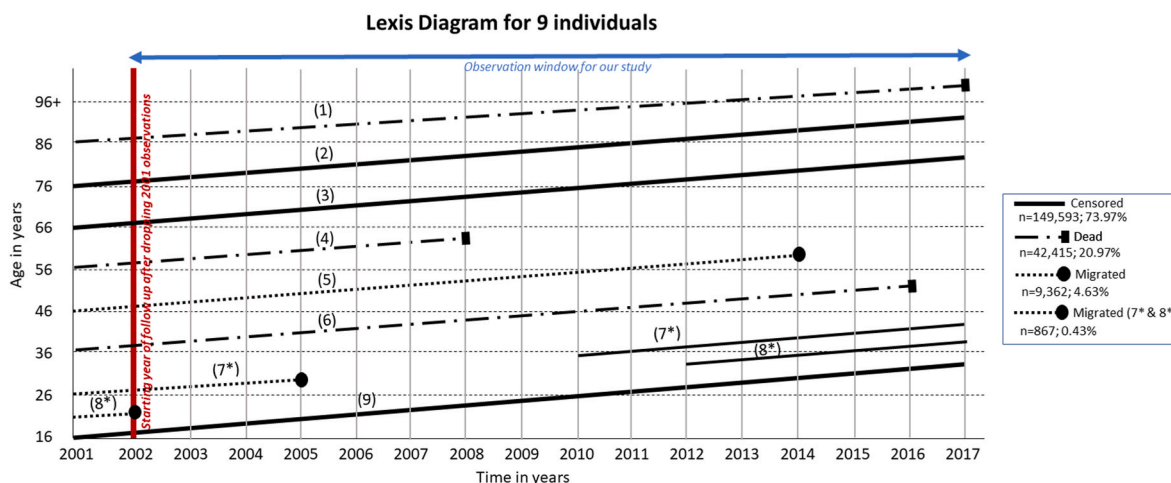
Yearly air pollution data that combine all sources of air pollution including road traffic and industrial/combustion processes for NO<sub>2</sub>, SO<sub>2</sub>, PM<sub>10</sub>, and PM<sub>2.5</sub> pollutants were obtained from the "Department-for-Environment-Food and Rural-Affairs (DEFRA)" (Department-for-Environment-Food-and-Rural-Affairs, 2020). These are raster data of mean annual concentrations of pollutants measured in µg/m<sup>3</sup>, estimated using air dispersion models at 1 km<sup>2</sup> resolution, and projected using the UK-National Grid (Department-for-Environment-Food-and-Rural-Affairs, 2020). These data were linked to postcodes in Scotland received from "National-Records-of-Scotland" (National-Records-of-Scotland, 2019) that fell within the 1 km<sup>2</sup> raster cells for each year between 2002 and 2017. Fig. 2 shows the concentrations of NO<sub>2</sub>, SO<sub>2</sub>, PM<sub>10</sub>, and PM<sub>2.5</sub> pollutants in 2017 across the residential postcodes in Scotland. In a second step, we linked the data file of matched yearly air pollution concentrations with postcodes with the data file of SLS real-time residential postcode histories. For individuals who changed their residential postcode within the same year, we kept the postcode with the highest monthly duration.

#### 2.2.3. Socioeconomic and contextual covariates

Our study included individual-level socioeconomic covariates collected at the 2001 and 2011 censuses: gender, ethnicity, country of birth, marital status, education, and occupation (Supplementary material: Table 2). We also included place of residence, a time-varying contextual variable that classifies the individuals' residential postcodes into six rural-urban categories based on the data-zone where each postcode is located (Scottish-Government, 2021). We accounted for these socioeconomic and place of residence covariates because they are available in the SLS administrative data and are considered by relevant literature to affect the association between air pollution and mortality (Abed Al Ahad et al., 2020; Beelen et al., 2008; Cesaroni et al., 2013; Strak et al., 2021).

### 2.3. Analysis

We calculated risk-time (person-years at risk), all-cause and cause-specific death counts, and percentages by the main socioeconomic and



**Fig. 1.** A Lexis diagram showing the possibilities of entering and exiting the cohort for 9 hypothetical individuals.

Data source: Author's own calculations based on the Scottish Longitudinal Study data; The red line presents the starting year of follow-up (2002) after dropping the 2001 observations; Individuals can either be censored at the end of the study (individuals 2, 3, 9), or died (individuals 1, 4, 6), or migrated and didn't come back during the observation period of 2002–2017 (individual 5), or migrated and then came back to Scotland and thus to our study within the follow-up period (individuals 7 and 8). Thus, individual 7 was followed from 2002 to 2005 and then from 2010 to 2017 with years spent outside Scotland (2006–2009) due to migration being dropped from observation. Similarly, individual 8 was present in 2002 and then was followed from 2012 to 2017 with years spent outside Scotland (2003–2011) due to migration being dropped from observation. Thus, possibilities for entering and exiting the cohort can be summarised as follows: (1) individuals followed for the whole study period from 2002 to 2017 and then either censored, migrated or died in 2017; (2) individuals exit the cohort before 2017 because they died during the follow-up period (2002–2017); (3) individuals exit the cohort because of migrating outside of Scotland and not coming back during the follow-up period (2002–2017); and (4) individuals exit the cohort because of migrating outside of Scotland, but they come back to the cohort in later years within the follow-up period (2002–2017) due to returning to Scotland. In case individuals migrate out of Scotland and then return in the same year, this short-term migration is ignored because the individual stayed in Scotland for some months of the full calendar year. If an individual returns to Scotland from a previous year migration and then migrates again out of Scotland within the same year, the individuals' observation for that year is kept since some months out of the full calendar year have been spent in Scotland.

contextual covariates. We also calculated the correlation between the four pollutants. Given the high correlations between  $\text{NO}_2$ ,  $\text{PM}_{10}$ , and  $\text{PM}_{2.5}$  pollutants (Pearson's-coefficient  $\geq 0.7$ ; Table 2), the association of the four pollutants with all-cause and cause-specific mortality was assessed in separate models.  $\text{SO}_2$  showed weak correlations (Pearson's-coefficient  $< 0.5$ ) with the other pollutants (Table 2), which enabled us to assess in a sensitivity analysis the association between  $\text{NO}_2$ ,  $\text{PM}_{10}$ , and  $\text{PM}_{2.5}$  and mortality in two-pollutant models adjusting for the  $\text{SO}_2$  pollutant.

We used Cox proportional-hazards models with age as baseline to study the association between air pollution and all-cause and cause-specific (cardiovascular, respiratory, cancer, mental/behavioural disorders/suicide, and other causes) mortality.

We fitted three models. Model 1 included air pollution, gender, and dummies for calendar years. Model 2 additionally included socio-demographic and economic covariates collected in 2001 and 2011 censuses: ethnicity, country of birth, marital status, education, and occupation. Yearly varying place of residence was included in Model 3.

The association of all-cause and cause-specific mortality with the socioeconomic and contextual covariates is shown in Supplementary Material Tables 3–8. Results are presented as hazard ratios (HRs) with 95% confidence-intervals (CIs) per  $1 \mu\text{g}/\text{m}^3$  increase in pollutants, visualised in coefficient plots. The  $1 \mu\text{g}/\text{m}^3$  increase in air pollutants was chosen as a fixed increment to facilitate comparison with other studies (Poulsen et al., 2023a; Stafoggia et al., 2022; Vodonos et al., 2018; Zhang et al., 2021), given that other increment types (e.g., interquartile range) are specific to the population under study, and their comparison would be more complicated. Statistical significance is at a p-value of less than 0.05. Statistical analysis was conducted in STATA<sub>16</sub>. Coefficient

plots were constructed using ggplot in R-Studio. Spatial pre-processing was conducted in ArcGIS-Pro software.

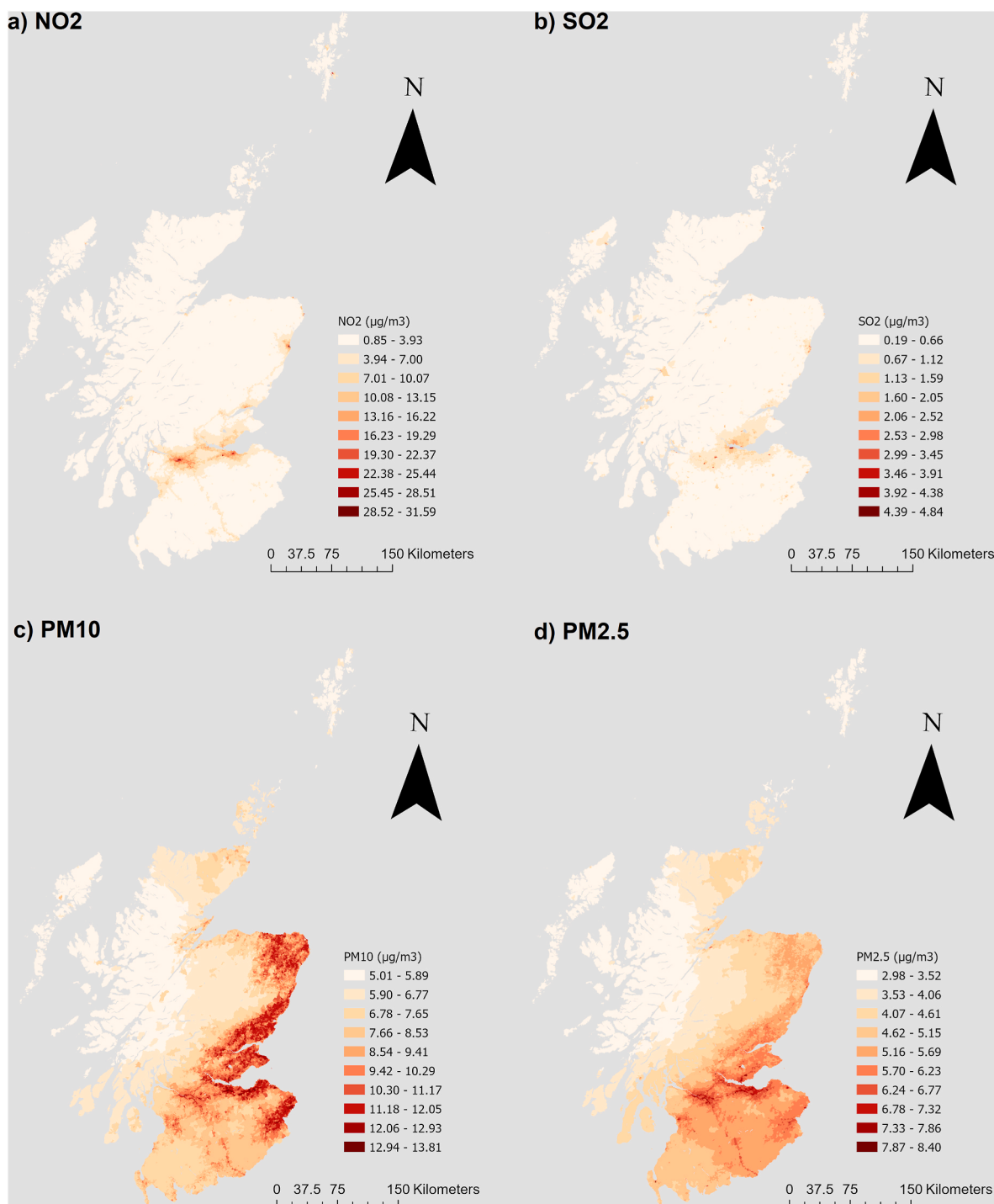
## 2.4. Ethics

Ethical approval was granted on May 14, 2020, by the Ethics Committee of the researchers' university. Access to SLS data was approved by SLS board and access to linked data from Public Health Scotland was approved following Public Benefit and Privacy Panel for Health and Social Care (HSC-PBPP) application. No consent was required for this study because it is based on secondary data analysis. Data cleaning, management, and analysis were performed in SLS safe settings, and the study conformed to the Declaration of Helsinki principles.

## 3. Results

### 3.1. Risk-time and deaths

Our study included 202,237 SLS adult individuals followed over 16 years (2002–2017) with a total of 2,821,344 person-years at risk and 42,415 deaths distributed as: 13,459 (32%) cardiovascular, 5418 (13%) respiratory, 12,069 (28%) cancer, 2815 (7%) mental/behavioural disorders/suicide, and 8654 (20%) other-causes deaths (Table 1). Most individuals were females (53%), belonged to white ethnicity (96%), were born in Scotland (87%), were married (54%), had post-school/university education (29%), worked in white-collar high skilled (30%) or white-collar low skilled (27%) jobs, and lived in large urban (35%) areas (Table 1).



**Fig. 2.** Maps showing mean annual concentrations of NO<sub>2</sub>, SO<sub>2</sub>, PM<sub>10</sub>, and PM<sub>2.5</sub> 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 of 2017 downloaded from the DEFRA online data repository (Department-for-Environment-Food-and-Rural-Affairs, 2020) and postcode boundaries shapefiles downloaded from the National Records of Scotland (National-Records-of-Scotland, 2019). Both DEFRA and National Records of Scotland shapefiles are governed under the Open Government Licence v.3.0.

### 3.2. Air pollution

Fluctuations in air pollutants were observed from one year to another (2002–2017) with higher concentrations in the first three years (2002–2004) compared to later years (Supplementary material: Fig. 2). Across the follow-up time, the average concentrations of NO<sub>2</sub>, SO<sub>2</sub>,

PM<sub>10</sub>, and PM<sub>2.5</sub> pollutants were 11.9 (SD = 6.4), 1.9 (SD = 1.5), 11.3 (SD = 2.1), and 7.2 (SD = 1.6)  $\mu\text{g}/\text{m}^3$ , respectively. High correlations (Pearson's-coefficient  $\geq 0.7$ ) were noted between NO<sub>2</sub>, PM<sub>10</sub>, and PM<sub>2.5</sub>, which could be related to the source of emission and chemical reactivity of pollutants (Table 2).

**Table 1**  
Person-years at risk and the number of death events by the study covariates.

Covariates	Risk Time	%	All-cause death		Cardiovascular death		Respiratory death		Cancer death		Mental/ behavioural disorders/ suicide death		Other-causes death	
			Deaths	%	Deaths	%	Deaths	%	Deaths	%	Deaths	%	Deaths	%
<b>Age</b>														
<40	741537	26.3	671	1.6	70	0.2	15	0.0	94	0.2	198	0.5	294	0.7
40–44	283663	10.1	495	1.2	88	0.2	24	0.1	108	0.3	84	0.2	191	0.5
45–49	295294	10.5	768	1.8	143	0.3	34	0.1	254	0.6	89	0.2	248	0.6
50–54	284779	10.1	1158	2.7	260	0.6	58	0.1	411	1.0	86	0.2	343	0.8
55–59	266092	9.4	1729	4.1	456	1.1	140	0.3	708	1.7	65	0.2	360	0.9
60–64	240244	8.5	2477	5.8	637	1.5	233	0.6	1032	2.4	80	0.2	495	1.2
65–69	212892	7.6	3742	8.8	1048	2.5	387	0.9	1569	3.7	65	0.2	673	1.6
70–74	176893	6.3	5037	11.9	1523	3.6	626	1.5	1962	4.6	120	0.3	806	1.9
75–79	140549	5.0	6595	15.6	2144	5.1	884	2.1	2175	5.1	261	0.6	1131	2.7
80–84	98432	3.5	7546	17.8	2629	6.2	1101	2.6	1875	4.4	491	1.2	1450	3.4
85–89	53647	1.9	6521	15.4	2363	5.6	953	2.3	1248	2.9	617	1.5	1340	3.2
90–94	21341	0.8	4115	9.7	1539	3.6	665	1.6	507	1.2	474	1.1	930	2.2
95+	5981	0.2	1561	3.7	559	1.3	298	0.7	126	0.3	185	0.4	393	0.9
<b>Year</b>														
2002	201948	7.2	2755	6.5	1058	2.5	307	0.7	724	1.7	120	0.3	546	1.3
2003	197896	7.0	2865	6.8	1043	2.5	356	0.8	762	1.8	154	0.4	550	1.3
2004	194046	6.9	2711	6.4	988	2.3	344	0.8	691	1.6	155	0.4	533	1.3
2005	190522	6.8	2660	6.3	922	2.2	337	0.8	743	1.8	142	0.3	516	1.2
2006	187002	6.6	2634	6.2	886	2.1	331	0.8	745	1.8	151	0.4	521	1.2
2007	183570	6.5	2767	6.5	909	2.1	348	0.8	783	1.9	178	0.4	549	1.3
2008	180183	6.4	2716	6.4	845	2.0	355	0.8	753	1.8	171	0.4	592	1.4
2009	176723	6.3	2548	6.0	805	1.9	310	0.7	763	1.8	154	0.4	516	1.2
2010	173710	6.2	2563	6.0	797	1.9	340	0.8	708	1.7	182	0.4	536	1.3
2011	170814	6.1	2447	5.8	788	1.9	312	0.7	693	1.6	173	0.4	481	1.1
2012	168036	6.0	2571	6.1	767	1.8	337	0.8	763	1.8	187	0.4	517	1.2
2013	164831	5.8	2505	5.9	740	1.7	337	0.8	775	1.8	193	0.5	460	1.1
2014	162183	5.8	2581	6.1	715	1.7	324	0.8	783	1.9	203	0.5	556	1.3
2015	159473	5.7	2689	6.3	784	1.9	364	0.9	773	1.8	206	0.5	562	1.3
2016	156533	5.6	2632	6.2	706	1.7	364	0.9	757	1.8	211	0.5	594	1.4
2017	153874	5.5	2771	6.5	706	1.7	352	0.8	853	2.0	235	0.6	625	1.5
<b>Gender</b>														
Male	1323609	46.9	19935	47.0	6361	15.0	2397	5.7	6126	14.4	1173	2.8	3878	9.1
Female	1497735	53.1	22480	53.0	7098	16.7	3021	7.1	5943	14.0	1642	3.9	4776	11.3
<b>Ethnicity</b>														
White	2695450	95.5	40061	94.5	12671	29.9	5089	12.0	11555	27.2	2658	6.3	8088	19.1
Not-white	125894	4.5	2354	5.6	788	1.9	329	0.8	514	1.2	157	0.4	566	1.3
<b>Country of birth</b>														
Born in Scotland	2448385	86.8	37491	88.4	11850	27.9	4838	11.4	10773	25.4	2496	5.9	7534	17.8
Born in rest of UK	257551	9.1	3244	7.7	1050	2.5	362	0.9	915	2.2	198	0.5	719	1.7
Born outside UK	115408	4.1	1680	4.0	559	1.3	218	0.5	381	0.9	121	0.3	401	1.0
<b>Marital status</b>														
Married	1517571	53.8	18534	43.7	5658	13.3	2061	4.9	6506	15.3	903	2.1	3406	8.0
Single never married	758808	26.9	5129	12.1	1512	3.6	599	1.4	1136	2.7	505	1.2	1377	3.3
Divorced/separated/widowed	536131	19.0	18292	43.1	6129	14.5	2697	6.4	4344	10.2	1371	3.2	3751	8.8
No response	8834	0.3	460	1.1	160	0.4	61	0.1	83	0.2	36	0.1	120	0.3
<b>Education</b>														
No educational qualification	849560	30.1	18473	43.6	5470	12.9	2454	5.8	5684	13.4	1151	2.7	3714	8.8
Intermediate school qualification	640152	22.7	3909	9.2	992	2.3	374	0.9	1368	3.2	292	0.7	883	2.1
High school qualification	377028	13.4	1795	4.2	475	1.1	157	0.4	650	1.5	120	0.3	393	0.9
Post-school/university qualification	814050	28.9	4667	11.0	1288	3.0	382	0.9	1744	4.1	301	0.7	952	2.2
*Still a student/Not recoded/No response	140554	5.0	13571	32.0	5234	12.3	2051	4.8	2623	6.2	951	2.2	2712	6.4
<b>Occupation</b>														
White collar high skilled	843133	29.9	6027	14.2	1723	4.1	569	1.3	2194	5.2	357	0.8	1184	2.8
White collar low skilled	765422	27.1	6711	15.8	1846	4.4	736	1.7	2284	5.4	438	1.0	1407	3.3
Blue collar high skilled	312312	11.1	4161	9.8	1255	3.0	467	1.1	1344	3.2	249	0.6	846	2.0
Blue collar low skilled	649796	23.0	9517	22.4	2731	6.4	1252	3.0	3068	7.2	599	1.4	1867	4.4
Not applicable: students/never worked	209152	7.4	13037	30.7	4933	11.6	2009	4.7	2341	5.5	1034	2.4	2720	6.4
No response	41529	1.5	2962	7.0	971	2.3	385	0.9	838	2.0	138	0.3	630	1.5
<b>Place of residence</b>														
Large Urban areas	982209	34.8	15251	36.0	4726	11.1	2025	4.8	4343	10.2	1019	2.4	3138	7.4
Other urban areas	817733	29.0	12442	29.3	3905	9.2	1659	3.9	3504	8.3	863	2.0	2511	5.9
Accessible small towns	207817	7.4	3059	7.2	994	2.3	387	0.9	868	2.1	169	0.4	641	1.5
Remote small towns	68031	2.4	1265	3.0	413	1.0	151	0.4	351	0.8	84	0.2	266	0.6
Accessible Rural areas	530751	18.8	7185	16.9	2323	5.5	844	2.0	2078	4.9	480	1.1	1460	3.4
Remote rural areas	214803	7.6	3213	7.6	1098	2.6	352	0.8	925	2.2	200	0.5	638	1.5
<b>Total</b>	<b>2821344</b>	<b>100</b>	<b>42415</b>	<b>100</b>	<b>13459</b>	<b>31.7</b>	<b>5418</b>	<b>12.8</b>	<b>12069</b>	<b>28.5</b>	<b>2815</b>	<b>6.6</b>	<b>8654</b>	<b>20.4</b>

Data source: Author’s own calculations based on the Scottish Longitudinal Study data; \*Still a student/Not recoded/no response: The counts and percentages of the two categories of Still a student and Not recoded/no response were combined due to low counts; Percentages might not add up to 100 due to rounding.

**Table 2**  
Description of air pollution exposure.

	NO <sub>2</sub> (µg/m <sup>3</sup> )	SO <sub>2</sub> (µg/m <sup>3</sup> )	PM10 (µg/m <sup>3</sup> )	PM2.5 (µg/m <sup>3</sup> )
Mean	11.9	1.9	11.3	7.2
Standard deviation	6.4	1.5	2.1	1.6
Median	11.2	1.6	11.2	7.1
Interquartile Range	9.4	1.3	2.8	2.3
Correlation matrix of air pollutants				
	NO <sub>2</sub> (µg/m <sup>3</sup> )	SO <sub>2</sub> (µg/m <sup>3</sup> )	PM10 (µg/m <sup>3</sup> )	PM2.5 (µg/m <sup>3</sup> )
NO <sub>2</sub> (µg/m <sup>3</sup> )	1.0			
SO <sub>2</sub> (µg/m <sup>3</sup> )	0.3	1.0		
PM10 (µg/m <sup>3</sup> )	<b>0.7</b>	0.4	1.0	
PM2.5 (µg/m <sup>3</sup> )	<b>0.7</b>	0.3	<b>0.9</b>	1.0

Strong correlations with correlation coefficient ≥0.7 are highlighted in bold; Data source: Author’s own calculations based on the Scottish Longitudinal Study data.

### 3.3. Air pollution and mortality associations

Results showed higher hazard ratios of all-cause mortality with increasing concentrations of PM2.5 (HR = 1.035; 95%CI = 1.023–1.047), PM10 (HR = 1.016; 95%CI = 1.009–1.023), NO<sub>2</sub> (HR = 1.009; 95%CI = 1.007–1.012), and SO<sub>2</sub> (HR = 1.009; 95%CI = 1.002–1.016) pollutants (Fig. 3). Higher hazard ratios for cardiovascular, respiratory, cancer and other-causes mortality were also shown with increasing concentrations of NO<sub>2</sub>, SO<sub>2</sub> (only in models 1 and 2), PM10 and PM2.5 pollutants. For example, the mortality hazard for respiratory diseases in model 3 was 1.062 (95%CI = 1.028–1.096), 1.025 (95%CI = 1.005–1.045), and 1.013 (95%CI = 1.007–1.020) per 1 µg/m<sup>3</sup> increase in PM2.5, PM10, and NO<sub>2</sub> pollutants, respectively. Mortality from mental/behavioural disorders/suicides was mainly associated with higher exposure to SO<sub>2</sub> (HR = 1.042; 95%CI = 1.015–1.069) pollutant (Fig. 3). Separating mental/behavioural disorders from suicide mortality still showed a similar pattern of associations; though the association between suicide mortality and SO<sub>2</sub> was not significant due to the low

suicides count (n = 417; Supplementary material: Fig. 3).

Similar results were noticed in two-pollutant models. The exceptions were cardiovascular and cancer mortality that did not show a significant association with PM2.5 after adjusting for SO<sub>2</sub> (Supplementary material: Fig. 4). Thus, the effect of PM2.5 on cardiovascular and cancer mortality may largely be the consequence of exposure to other pollutants that co-exist with PM2.5 in the atmosphere such as SO<sub>2</sub> (Ku et al., 2017) and their post-inhalation interactions within the human body (Gładka et al., 2018).

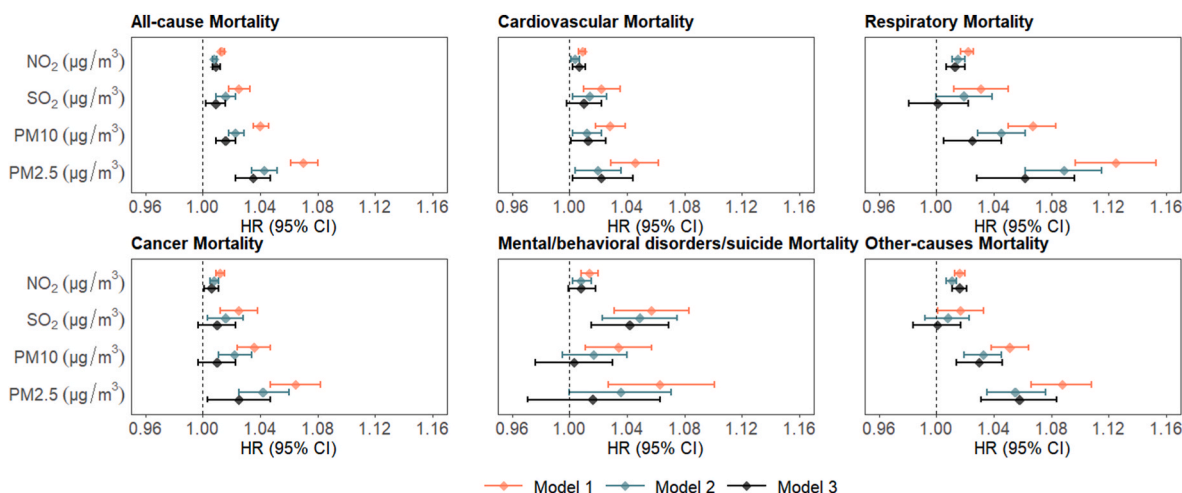
#### 3.3.1. Sensitivity analysis

In a sensitivity analysis, we refitted models 1–3 replacing yearly air pollution with average cumulative air pollution (CAP) that was calculated following the methods of Bentayeb et al. (2015) (Bentayeb et al., 2015). For each individual, we calculated the average pollutant concentration from the baseline year (2002) to each year of follow-up before death or censoring (e.g., exposure in 2006 was calculated as the mean of annual concentrations from 2002 to 2006; in 2007, from 2002 to 2007).

We observed positive associations between higher exposure to average CAP and all-cause and cause-specific mortality. However, stronger associations were noted for average CAP, which supports that long-term cumulative exposures have an exacerbating effect on health (Supplementary material: Fig. 5). Nevertheless, the differences between average CAP and yearly pollution effects were not large and this could be related to the low mobility of individuals during the 16 years of follow-up: 54% of individuals did not change the postcode and 32% moved within the same data-zone or council area.

## 4. Discussion

Using large individual-level administrative data linked to 1 km<sup>2</sup> air pollution data at the individuals’ residential postcode between 2002 and 2017, this study supports an association between long-term exposure to NO<sub>2</sub>, SO<sub>2</sub>, PM10 and PM2.5 pollutants and all-cause, cardiovascular, respiratory, cancer, mental/behavioural disorders, suicides, and other-causes mortality in Scotland. The highest death rates were observed from respiratory causes, especially with higher exposure to long-term PM2.5, which was expected given that air pollution enters mainly the



**Fig. 3.** The association of all-cause and cause-specific mortality with NO<sub>2</sub>, SO<sub>2</sub>, PM10, and PM2.5 air pollutants in separate Cox Proportional-Hazard models (N = 202,237 individuals and 2,821,344 person-years at risk).

Data source: Author’s own calculations based on the Scottish Longitudinal Study data; The dashed line is placed at HR = 1 as a cut-off for statistically insignificant results; Model 1 is adjusted for gender and calendar year dummies; Model 2 = Model 1 + ethnicity + country of birth + marital status + education + occupation; Model 3 = Model 2 + Place of residence. Results are presented as HRs with 95%CI per 1 µg/m<sup>3</sup> increase in air pollutants. Models 1 to 3 include the same individuals (N = 202,237 individuals and 2,821,344 person-years at risk).

human body through air-breathing, causing damage to the nasal cavity, upper respiratory airways, and lung tissues (Abed Al Ahad et al., 2020; Manisalidis et al., 2020).

Our results in terms of the direction of the associations are consistent with previous studies from North America, Europe, and South-Korea (Beelen et al., 2014; Carey et al., 2013; Crouse Dan et al., 2015; Crouse Dan et al., 2012; Dehbi et al., 2017; Fischer et al., 2015; Hansell et al., 2016; Hvidtfeldt et al., 2021; Hvidtfeldt et al., 2019; Lepeule et al., 2012; Liu et al., 2021; Min et al., 2018; Pinault et al., 2017; Pope et al., 2019; Poulsen et al., 2023a; Poulsen et al., 2023b; Pun et al., 2017; Raaschou-Nielsen et al., 2020; Rodopoulou et al., 2022; So et al., 2020; Stafoggia et al., 2022; Strak et al., 2021; Wolf et al., 2021; Yuchi et al., 2020; Zhang et al., 2021) and with systematic literature reviews and meta-analyses that investigated the long-term effect of air pollution on all-cause and cause-specific mortality (Chen and Hoek, 2020; Hoek et al., 2013; Pope et al., 2020; Vodonos et al., 2018). However, some of our estimates (e.g., HRs for the association between PM<sub>2.5</sub> and all-cause, cardiovascular, and respiratory mortality) were larger than what other studies and systematic literature reviews have found. For example, in a systematic literature review of 75 studies from around the world, Pope et al. (2020) found that the random effects meta-estimates for all-cause mortality per 10 µg/m<sup>3</sup> increase in PM<sub>2.5</sub> correspond to a hazard ratio of 1.09 (95%CI 1.07–1.11) in North America, 1.12 (95%CI 1.06–1.19) in Europe, and 1.07 (95%CI 1.04–1.11) in Asia (Pope et al., 2020). Similarly, a systematic literature review by Vodonos et al. (2018) reported for every 1 µg/m<sup>3</sup> increase in PM<sub>2.5</sub>, a 1.29% (95%CI 1.09–1.50), 1.46% (95%CI 1.25–1.67), and 1.13% (95%CI 0.85–1.41) increase in all-cause, cardiovascular, and respiratory mortality, respectively (Vodonos et al., 2018). On the other hand, the hazard ratios for the association between NO<sub>2</sub> and all-cause mortality were comparable to the ELAPSE project findings (HR 1.09; 95%CI 1.07–1.10 per 10 µg/m<sup>3</sup> increase in NO<sub>2</sub>) (Strak et al., 2021) and the hazard ratios for the association between NO<sub>2</sub> and respiratory diseases mortality were comparable to a National English Cohort study (HR 1.15; 95%CI 1.10–1.20 per 10.7 µg/m<sup>3</sup> increase in NO<sub>2</sub>) (Carey et al., 2013).

The elevated estimates in our study could be attributed to the differences in population size, study location, air pollution exposure assessment and level, and methodological differences such as residual confounding (Chen and Hoek, 2020). For example, our models adjusted only for the socio-demographic and economic characteristics of individuals (e.g., age, gender, education, marital status, ethnicity, country of birth, and occupation) and for the time trend (i.e., year dummies) and place of residence (i.e., rural-urban area classifications). However, our models did not account for individual lifestyle covariates such as smoking, exercise, alcohol consumption, or body mass index that may affect the association between air pollution and mortality (Beelen et al., 2008; Stafoggia et al., 2022; Strak et al., 2021; Weichenthal et al., 2014) due to the unavailability of this information in register-based data. Adjusting for these lifestyle covariates would likely alter the magnitude of association estimates slightly in the range of 1–2% increase or decrease, depending on the cause of death as shown in other studies (Abed Al Ahad et al., 2023; Carey et al., 2013; Chen et al., 2013; Crouse Dan et al., 2015; Fischer et al., 2015; Nieuwenhuijsen et al., 2018). Similarly, our models did not account for environmental factors at the place of residence such as noise pollution or the lack of green spaces. Nevertheless, this is unlikely to have a tremendous effect on the association between air pollutants and mortality outcomes with estimates being little attenuated by 0–3% increase or decrease as reported by previous studies (Nieuwenhuijsen et al., 2018; Sørensen et al., 2022; Yuchi et al., 2020).

Our study also supported differences in the effect of the four pollutants on cause-specific mortality. Whilst NO<sub>2</sub> was associated with all mortality causes, particulate-matter, especially PM<sub>2.5</sub> was mainly related to respiratory, cardiovascular and cancer mortality, while SO<sub>2</sub> was associated with mental and behavioural disorders mortality. This could be attributed to the specific mechanism of action of each pollutant

in producing toxic effects depending on its chemical and physical properties, which results in different diseases and mortality outcomes. Literature has shown that NO<sub>2</sub> is an irritant of the respiratory system that can penetrate deeply into the lungs (Costa et al., 2014; Manisalidis et al., 2020). Exposure to high concentrations of NO<sub>2</sub> (>2 ppm) affect the immune system by altering the function of T-lymphocytes and natural-killer cells (Manisalidis et al., 2020). Long-term exposure to NO<sub>2</sub> is also related to ventricular-hypertrophy in humans (Manisalidis et al., 2020). This increases the risk for cardiovascular, respiratory, and lung cancer mortality as shown in our study and other observational studies (Cesaroni et al., 2013; Fischer et al., 2015; Liu et al., 2021; Wolf et al., 2021). Exposure to NO<sub>2</sub> is also linked to mental and behavioural disorders due to the formation of reactive-oxygen species (ROS) and free radicals, which results in neuronal injury and neurological disorders (Kilian and Kitazawa, 2018; Yan et al., 2015).

Particulate-matter with larger diameters (e.g., PM<sub>10</sub>) will be deposited in nasal cavities and upper airways following inhalation. Particulate-matter with smaller diameters (e.g., PM<sub>2.5</sub>) may penetrate more deeply the respiratory system reaching alveoli and bloodstream (Costa et al., 2014; Manisalidis et al., 2020). Once inside the body, particulate-matter will initiate oxidative stress and the formation of ROS, resulting in respiratory, cardiovascular, immune, and central nervous system dysfunctions (Costa et al., 2014; Manisalidis et al., 2020).

Chronic exposure to SO<sub>2</sub> can disrupt the signal transmission and synaptic communications between neurons and initiate oxidative stress resulting in neurodegeneration and neuronal dysfunction (Ku et al., 2017; Manisalidis et al., 2020). This leads to various neurodegenerative, psychological, and behavioural complications including Alzheimer's and Parkinson's diseases (Ku et al., 2017; Manisalidis et al., 2020), cognitive impairment (Kilian and Kitazawa, 2018), and depression (Borroni et al., 2022).

The emission source of the four pollutants can also explain the observed differences. Traffic exhaust is the main source of NO<sub>2</sub> (Grazuleviciene et al., 2004). Particulate-matter results from both traffic and industrial processes as well as from domestic sources such as wood burning and wood stoves and from natural processes (e.g., forest fires and dust). Particulate-matter can stay for long time in the atmosphere due to their tiny dimensions and can spread over long distances through atmospheric transport, depending on factors related to weather, topography, coastal areas, and proximity to natural sources, among others (Jyethi, 2016; Malek et al., 2006; Manisalidis et al., 2020; Wood et al., 2023). Thus, higher concentrations of NO<sub>2</sub> are expected in urban areas and around busy roads in general rather than being restricted to certain industrial neighbourhoods, and particulate-matter pollution is expected to be scattered, covering large areas beyond its emission sources. In contrast, SO<sub>2</sub> originates from industries and power plants (Vestreng et al., 2007), which are restricted to specific urban and rural areas. Fig. 2 supports these expectations by showing higher concentrations of NO<sub>2</sub> and SO<sub>2</sub> in the urban Central Belt and in busy coastal cities like Aberdeen, while PM<sub>10</sub> and PM<sub>2.5</sub> are scattered across the whole of Scotland. Fig. 2 also shows some clustering of SO<sub>2</sub> in rural areas in places of local industries and energy production facilities. This further suggests that the place of residence (urban versus rural) plays a crucial role in the intensity and duration of exposure to ambient air pollution and its associated morbidity and mortality. Adjusting our analysis for the place of residence (model 3), reduced the magnitude of associations between mortality and all the four pollutants. This also indicates that living near factories and power plants could play a role in the observed association between SO<sub>2</sub> and mental/behavioural disorders mortality. Some studies have shown that areas near industries and power plants are more deprived and are disproportionately occupied by people of colour and lower socioeconomic status (Downey and Van Willigen, 2005). This is because those areas have high air and noise pollution, and lack positive environmental factors such as parks, trees, and open spaces (Downey and Van Willigen, 2005). This increases the risk of mental and

behavioural illness in a direct way due to higher exposure to air and noise pollution and indirectly due to the anxiety experienced from deprivation, individuals' perceptions of neighbourhood disorder, and personal powerlessness (Downey and Van Willigen, 2005; Marques and Lima, 2011).

#### 4.1. Strengths and limitations

An important strength of this article is that it used a large administrative individual-level cohort from Scotland over a long period of 16 years to investigate the association between mortality and air pollution exposure assessed and linked at 1 km<sup>2</sup> spatial scale. Administrative data are usually of high quality, and they possess advantage over other types of data (e.g., surveys) by providing continuous longitudinal information on residential histories at the postcode level, on emigrations and immigrations, and on mortality and births vital events. Knowing the exact postcode histories of individuals over the 16 years of follow-up is essential in the assessment of individual's air pollution exposure. Besides, the linkage of modelled air pollution data at 1 km<sup>2</sup> resolution provides a uniform assessment of exposure across the postcodes in Scotland, which would not be the case for unmodelled station-based pollution data. Furthermore, the SLS administrative-based data consists of a 5% representative sample of the total Scottish population based on the three censuses of 1991, 2001, and 2011, which ensures representation of all population sub-groups, reduces selection bias, and provides enough statistical power to observe associations. However, the usage of administrative data comes with its limitations related to the unavailability of important lifestyle covariates that were shown to affect the association between air pollution and mortality such as smoking, alcohol consumption, body-mass-index, and exercise. This may contribute to residual confounding as discussed previously. To overcome this limitation, future research would have to investigate the possibility of linking administrative data to survey data that contains information on lifestyle factors. Nevertheless, we adjusted for several commonly used potential confounders including socio-demographic, economic, and rural-urban confounders, which reduces the confounding by other risk factors.

Another strength of the present study is that it investigated the association between multiple air pollutants including the less studied SO<sub>2</sub> pollutant and several cause-specific mortality outcomes. This is important to show that air pollution is associated with both physical and mental illnesses mortality and to reveal differences in mortality outcomes based on exposures to specific pollutants. For example, our study showed that SO<sub>2</sub> is mainly associated with mortality from mental/behavioural disorders, while the other three pollutants were mainly associated with mortality from cardiovascular, respiratory, and cancer diseases. Furthermore, our study revealed that despite the relatively low concentrations of SO<sub>2</sub> in Scotland, it is still associated with an increased mortality hazard, particularly for mental and behavioural diseases, which warrant further investigation.

Finally, bias from exposure misclassification and selection cannot be excluded. First, our cohort included a 5% representative sample of all adult individuals (age 16+) who were present in Scotland at the 2001 census. This may introduce selection bias by excluding individuals who became adults after 2001 census or who entered Scotland at later years through immigration. However, following a cohort design by including adult individuals present at the onset of the study and not allowing new entries allows a lengthier follow-up time for everyone and reduces heterogeneities between individuals and the bias that may arise from periods of rapid population change. Second, the SLS cohort was followed over a period of 16 years starting the year 2002; thus bias may result from previous lifetime exposures to air pollution at different residences. Third, we assessed the individuals' exposure to ambient air pollution using the residential postcode, which does not necessarily equate to the true personal exposure. In reality, an exposure scenario is more complex involving exposure indoors, at workplace, during leisure activities and

through commuting patterns. Thus, future research would benefit from integrating air pollution exposure at the residence and workplace (e.g., by using census data on journey-to-work flows or real-time GPS data) and from considering both ambient and indoor air pollution exposures. Finally, we assessed the association between air pollution and mortality on a yearly rather than monthly or daily basis, which did not allow for seasonal variations in air pollution.

## 5. Conclusion

This study contributed to the evidence that long-term exposure to ambient air pollution is associated with mortality. The results suggest that policies and interventions to enhance air quality would reduce the mortality hazard from both physical (e.g., cardiovascular, respiratory, and cancer) and mental/behavioural illnesses in the long-term.

### Author contribution statement

Mary Abed Al Ahad: Conceptualization, Investigation, Methodology, Data curation, Formal Analysis, Writing-Original Draft, Writing-Review and Editing, Visualization, Project administration. Hill Kulu, Urška Demšar and Frank Sullivan: Conceptualization, Funding acquisition, Supervision, Writing-Review and Editing.

### Ethical considerations

This paper is part of a PhD project that was granted ethical approval on the May 14, 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 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 have already obtained all the needed consent and approvals for the data processing and analysis. Based on the SLS data policy and the sensitivity of the used data, all data cleaning, management, and analysis was performed in the safe settings of the SLS in Ladywell House in Edinburgh to ensure individuals' confidentiality and safe and secure data storage and access.

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### Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

### Data availability

The authors do not have permission to share data.



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## Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.envres.2023.117223>.

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