International Journal of Environmental Epidemiology

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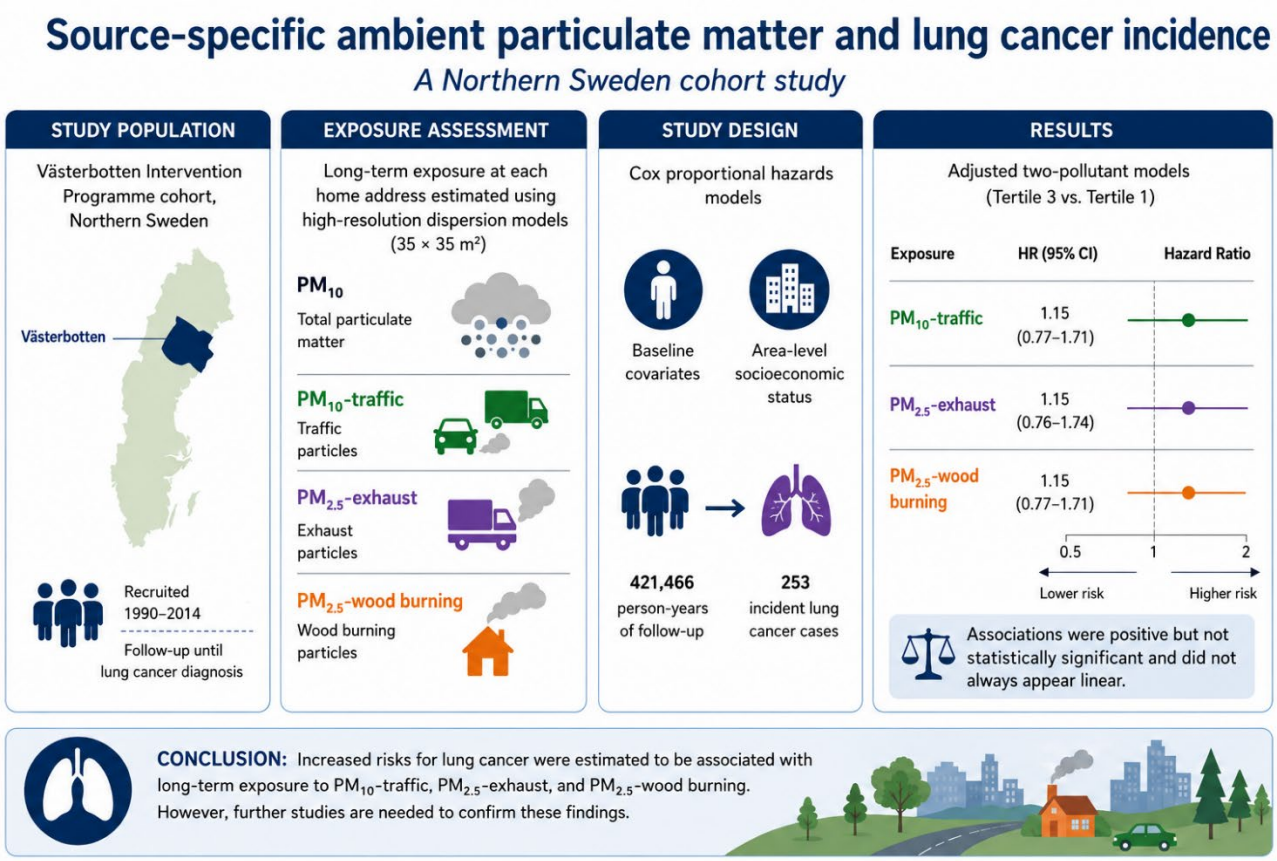
Received 13 October 2025; Revised 8 April 2026; Accepted 9 April 2026; Published 14 May 2026

https://doi.org/10.55092/ijee20260008

Graphical abstract



Highlights:

- Source-specific PM exposures and lung cancer were studied in Northern Sweden.
- High-resolution dispersion models estimated traffic, exhaust, and wood smoke PM.
- 253 lung cancer cases identified over 421,466 person-years of follow-up.
- Positive but non- statistically significant associations observed for all PM sources.
- Risk estimates were sensitive to individual and area-level confounder adjustment.

Source-specific ambient particulate matter and risk of incident lung cancer: evidence from a Northern Swedish cohort

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Abstract: **Objective:** To estimate the association between source-specific ambient particulate matter concentrations and lung cancer incidence in a northern Sweden cohort. **Methods:** Participants in the Västerbotten Intervention Programme cohort from Northern Sweden were recruited between January 1990 and December 2014 and followed until lung cancer diagnosis. Exposure to total particulate matter with aerodynamic diameter ≤ 10 µm (PM_{10}) and ≤ 2.5 µm ($PM_{2.5}$) as well as source-specific particulate matter (PM) concentrations from traffic (PM_{10}-traffic), exhaust ($PM_{2.5}$-exhaust) and wood burning ($PM_{2.5}$-wood burning) was estimated at each participant's home address using dispersion models with high spatial resolution (down to 35 × 35 m^2). Cox regression models were used to assess hazard ratios (HRs) and 95% Confidence Intervals (CIs) for the association between ambient particulate matter exposure and lung cancer incidence, adjusted for relevant potential confounding factors. **Results:** During 421,466 person-years of follow-up, 253 incident cases of lung cancer were observed. The risk estimates changed considerably when adjusting for individual-level baseline covariates and area level socioeconomic status. In adjusted two-pollutant models, HRs for incident lung cancer in the highest exposure tertile (tertile 3) compared to the lowest (tertile 1) were 1.15 (95% CI: 0.77–1.71) for PM_{10}-traffic, 1.15 (95% CI: 0.76–1.74) for $PM_{2.5}$-exhaust, and 1.15 (95% CI: 0.77–1.71) for $PM_{2.5}$-wood burning. **Conclusions:** Increased risks for lung cancer were estimated to be associated with long-term exposure to PM_{10}-traffic, $PM_{2.5}$-exhaust, and $PM_{2.5}$-wood burning. However, since these associations were not statistically significant and did not always appear linear, further studies are needed.

Keywords: ambient particulate matter; lung cancer; road traffic; wood-burning; air pollution

1. Introduction

Among the leading causes of mortality and morbidity worldwide, lung cancer is a major public health problem. It accounted for 2.21 million cancer cases and 1.80 million deaths globally in 2020 and has even been labelled as the most common cause of cancer-related death [1]. In Europe, lung cancer is the second most commonly diagnosed form of cancer after prostate cancer among men and the third most common cancer after breast and colorectal cancer among women, comprising a leading cause of morbidity in the region [2]. Despite great advances in medical knowledge, such as screening and treatment, the prognosis for lung cancer patients remains poor [3]. It is thus imperative to identify modifiable risk factors to reduce the global burden of lung cancer. One such environmental risk factor, modifiable at the population level, is ambient air pollution [4]. Exposure to ambient air pollution is included among the leading risk factors for human ill health. In 2019, it was estimated to be responsible for 118 million disability-adjusted life years (DALYs) and 4.18 million deaths in Europe [5]. Tracheal, bronchus, and lung cancer accounted for a median of 55.5 DALYs per case and 39,500 deaths. Most of these adverse health effects were estimated to be attributed to fine particulate matter ($PM_{2.5}$), that is, airborne particles with an aerodynamic diameter of 2.5 µm or smaller.

Inflammation and oxidative stress are thought to be potential underlying mechanisms through which air pollution causes lung cancer. These adverse effects are triggered by the inhalation of particulate matter that translocate first to the pulmonary circulation and then to the general circulation. This may lead to DNA damage and increased cell turnover, promoting the proliferation of lung tissue [6]. Moreover, there may be a mediating effect of epigenetic changes in the genome, especially in relation to hypermethylation of promotor genes, on the association between air pollution and lung cancer [7].

Extensive epidemiological evidence on air pollution as a risk factor for lung cancer has been accumulated over the years. A review of 22 prospective studies showed a 16% and 23% increased risk of lung cancer per 10 µg/m^3 of $PM_{2.5}$ and particulate matter with aerodynamic diameter ≤ 10 µm (PM_{10}), respectively [8]. Still, limitations were identified. For example, the included studies differed in study characteristics,

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air pollution concentration, and their methods to estimate exposure. Additionally, several studies were at risk of bias due to exposure misclassification, as individual exposure was often inferred from ecological-level data, potentially leading to an underestimation of the true risk.

During recent years substantial evidence has also started to accumulate that show higher risk estimates for (mainly) mortality at low exposure level, compared with higher [9], but research about the effects of low-level air pollution on the risk of lung cancer is limited. In a pooled analysis of 7 European cohorts, a 5 μg/m^3 increment in $PM_{2.5}$ was associated with a 13% increased risk of lung cancer [10]. Despite the growing evidence linking total PM_{10} and $PM_{2.5}$ exposure to an increased risk of lung cancer, only a limited number of studies have examined source-specific pollutants. In a review of case–control and cohort studies, Chen *et al.* conclude that exposure to traffic-related pollutants, including $PM_{2.5}$, NO_2, and SO_2, elevates lung cancer risk [11]. Evidence from a pooled analysis of 14 European cohorts further supports the importance of the pollutant's composition: associations were observed between elemental components of particulate matter and lung cancer, with $PM_{2.5}$ from sulphur sources emerging as particularly influential [12]. Although still few in number, such studies suggest that lung cancer risk is attributable to a variety of emission sources and particulate matter (PM) components. Identifying the emission sources contributing to this association is crucial for designing effective local mitigation strategies, as pollutants emitted locally often have stronger health effects than those measured at urban background sites [13].

Studies conducted in regions with low air pollution levels are also largely lacking, despite their importance. Such settings enable the investigation of exposure–response relationships across the full concentration range, help determine whether there is a safe exposure threshold, inform air quality guidelines, and provide evidence relevant for countries and regions that have already achieved relatively clean air but continue to see adverse health effects.

The objective of this study is to estimate the association of total and source-specific concentrations of ambient particulate matter and lung cancer incidence in a low-level exposure setting.

2. Materials and methods

2.1. Study population

We conducted a prospective cohort study of adults living in Västerbotten region of Northern Sweden who were recruited into the still ongoing Västerbotten Intervention Programme (VIP) between 1990 and 2014. The Västerbotten region had a population of 281,138 in 2024. Covering an area of 55,186 km^2, the county has a low population density of about 5 inhabitants per km^2. The population is unevenly distributed, with most residents living along the Baltic Sea coast, while the western mountainous areas are very sparsely populated. A detailed description of VIP is available elsewhere [14]. In brief, VIP is a large population-based health screening and intervention programme that has been collecting anonymous individual-level data since 1985, with the goal of improving public health and wellbeing through preventive measures focused on lifestyle changes. All inhabitants in the Västerbotten region are invited to the VIP during the year they turn 40, 50, and 60 years old. The participation rate has varied over time with an average of 60%. Participation in VIP includes a visit to their local primary health care centre for a health examination and counselling. Participants also complete a comprehensive survey on their health, diet, and lifestyle. Written informed consent are obtained from all VIP participants prior to enrolment. In the present study, residential address histories of participants were collected by linking personal identification numbers with the Swedish Taxation Authority. These addresses are geocoded using the Swedish Mapping Cadastral and Land regression Authority databases.

For the present study, we restricted the cohort to include only those individuals who had no previous history of lung cancer as of January 1, 1990. To eliminate prevalent cases of lung cancer, we also excluded individuals who had received a lung cancer diagnosis prior to recruitment, as well as those who had been prescribed lung cancer medication one year prior to recruitment. This resulted in a study population of 51,064 VIP participants, who were followed until December 31, 2014.

2.2. Exposure assessment

A thorough description of the exposure assessment has been published previously [15], see also Figure 1. In short, source-specific exposure to PM_{10} and $PM_{2.5}$ was estimated using a combination of monitoring data and dispersion modelling. Contributions from long-range transport (*i.e.*, sources outside the assessment area) were represented using monitoring data and were treated as a separate source category. Because long-range transport concentrations based on directs measurements were available for each year, interpolation was not required.

In contrast, contributions from emission sources within the assessment area were calculated using Gaussian dispersion models. Dispersion modelling was carried out on an hourly basis for the years 1990, 2000, and 2011, but only annual average concentrations were used to represent exposure. The different emission source categories road traffic (exhaust and non-exhaust), residential wood combustion, shipping, industry and energy production, and other sources, were modelled separately to enable source-specific attribution. For these local sources, annual concentrations for intermediate years were linearly interpolated between modelling years, assuming a gradual change in emissions. These estimates were further adjusted using a meteorological index to account for year-to-year meteorological variability. To resolve concentration gradients within the urban environment, especially near major roads, a quadtree receptor grid was applied, achieving a spatial resolution down to 35 × 35 m^2.

Particular attention was given to two local source-categories: traffic-related emissions (PM_{10}-traffic and $PM_{2.5}$-exhaust) and residential wood combustion ($PM_{2.5}$-wood burning). Both

were modelled using a bottom-up approach, meaning that each single road or wood-firing appliance were represented explicitly. Emissions from roads were calculated for different vehicle types, speeds, and driving conditions using the Handbook on Emission Factors for Road Traffic (version 3.1) [16]. Traffic volumes for individual roads were obtained from the Municipality of Umeå as well as the Swedish Traffic Administration. Fleet composition for different types of roads were based on statistics from the Swedish Transport Administration. Non-exhaust emissions from road, brake, and tyre wear were assumed to follow the same spatial distribution as exhaust emissions, with a relative ratio derived according to Omstedt *et al.* [17]. Emissions from residential wood combustion were characterised using chimney-sweeper registers of stoves and boilers, together with survey data on typical fuel consumption and firing practises [15].

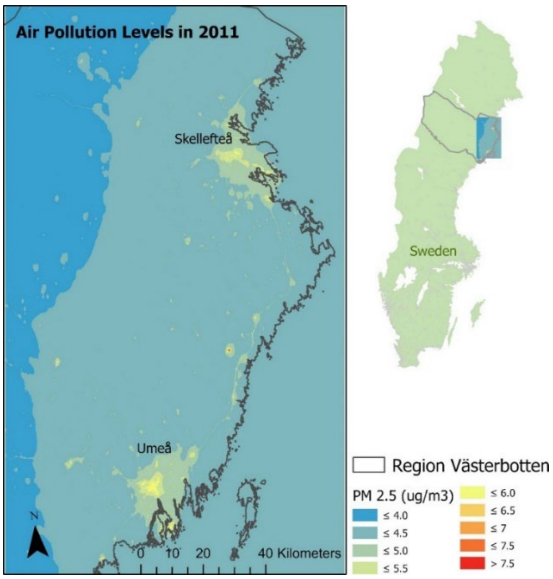

Figure 1. Modelled annual average concentrations of total $PM_{2.5}$ in the study area for year 2011 [15].

Larger industrial sources and energy production facilities were represented as point-sources in the model. Emissions from shipping were also included, using a similar method as described by Jalkanen *et al.* [18]. Finally, individual exposure classification was performed by extracting annual average concentrations at each participant's residential address, which were available through a linkage between VIP and a nation-wide Swedish register. We focused on PM_{10}, $PM_{2.5}$, PM_{10}-traffic, $PM_{2.5}$-exhaust, and $PM_{2.5}$-wood burning because these fractions capture the main sources and size ranges relevant to our study area [15,19]. PM_{10} represents the total coarse particulate matter, while $PM_{2.5}$ represents the fine fraction. Source-specific metrics, including PM_{10}-traffic, $PM_{2.5}$-exhaust, and $PM_{2.5}$-wood burning, allow us to distinguish between contributions from traffic (non-exhaust particles), exhaust, and residential wood combustion, which differ in particle size, composition, and potential health impacts. This focus enables a more detailed assessment of exposure-response relationships in our population.

2.3. Outcome assessment

Lung cancer incident cases were identified according to the criteria of International Classification of Diseases, Tenth Revision (ICD-10) code C34.90. Only primary cases of lung cancer were included; metastatic cancers to the lung were excluded. To ascertain case status, we linked cohort data to population-based health administrative databases using each participant's unique personal identification number. These databases included the National Patient Register and the Cause of Death Register, maintained by the Swedish National Board of Health and Welfare (https://www.socialstyrelsen.se/en/statistics-and-data/registers/). Sweden manages a comprehensive system of high-quality national registers, which provide an exceptional resource for scientific research. These registers cover the entire population and include detailed information on demographics, socioeconomic factors, health care utilisation, diagnoses, prescribed medications, births, deaths, and more. Moreover, they are continuously updated, standardised, and subjected to rigorous quality control, ensuring reliable and complete data. Each citizen and resident in Sweden is assigned a unique personal identity number, allowing precise linkage across multiple registers and over time. This infrastructure enables population-based, longitudinal, intergenerational, and multilevel studies with minimal selection bias, making Swedish registers among the most powerful tools globally for epidemiological and public health research.

2.4. Potential confounders

Potential confounders were pre-selected based on prior research in comparable studies [10–12]. These included calendar year and sex, as well as the following individual-level covariates: smoking status (current, former, never smoker), alcohol consumption (daily, weekly, seldom, never), leisure time physical activity (sedentary, moderate, intermediate or vigorous), marital status (single, married or living with partner, missing), education level

(primary school or lower, up to secondary school or equivalent, university degree or higher, missing), and employment status (gainfully employed, unemployed/not gainfully employed, retired, missing). Area-level socioeconomic status, estimated from mean neighbourhood income at each cohort member's residence, was also incorporated. This was based on registry data from 1994 and defined according to Small Areas for Market Statistics (SAMS).

2.5. Statistical methods

Participants were followed from recruitment until diagnosis of lung cancer, death, emigration, or end of follow-up. Age was used as the time axis in the Cox proportional hazards models, but survival time was not defined from birth. Instead, we applied delayed entry, so that each participant entered the risk set at their age at cohort enrolment (≥ 40 years) and was followed until the age at event or censoring. Thus, survival time was defined from age at baseline to age at event/diagnosis/death/emigration/end of follow-up, and lifetime exposure data from birth were not required. An exposure time window of 1–10 years preceding the event (lag 1–10), as a moving average, was applied. Associations between lung cancer and long-term exposure to particles from different sources were estimated using Cox-proportional hazard models with age as the underlying time scale. Both single pollutant and two pollutant models were fitted and estimates of particle concentration exposure are reported in tertiles. Associations were adjusted for calendar year, baseline information on individual covariates, and area level socioeconomic status. Results are presented as hazard ratios (HRs) and 95% confidence intervals (CI). Interaction analyses were conducted to assess effect modification by sex, smoking status, and educational level.

3. Results

3.1. Participant characteristics

The study included 51,064 individuals, with a median age at recruitment of 40 years. Of these, 53% were women; 22% were current smokers and 28% were former smokers; 42% were sedentary (did not engage in exercise that required training clothes); 55% consumed alcohol every week; 20% did not live with a partner; 44% had primary school education or less; and 5% were unemployed (Table 1). In the highest tertile of PM_{10}-traffic concentrations, there was a somewhat larger proportion of never smokers, a larger proportion of individuals with a higher education level, and a larger proportion of gainfully employed participants. Over 421,466 person-years of follow-up, 253 incident cases of lung cancer were identified. The median length of follow-up among lung cancer cases was 5 years, with quartile limits (2, 9). Among non-cases, the median length of follow-up was 9 years, with quartile limits (6, 12).

Table 1. Baseline socio-demographic and lifestyle characteristics of study participants.

Variables	Categories	All participants	In tertiles of PM_{10}-traffic (μg/m³)		
			< 0.21	0.21–0.34	> 0.34
Participants (n)		51,064	17,022	17,021	17,021
Women		53%	50%	54%	54%
BMI (kg/m²; mean ± standard deviation)		25 (4.3)	26 (4.0)	25 (3.8)	25 (3.7)
Smoking status	Current smoker	22%	20%	23%	22%
	Former smoker	28%	27%	28%	28%
	Never smoker	44%	44%	44%	45%
	Missing data	6%	9%	5%	4%
Leisure time physical activity	Sedentary	42%	46%	42%	39%
	Moderate	43%	41%	43%	46%
	Intermediate and vigorous	13%	12%	14%	14%
	Missing data	1%	1%	1%	1%
Alcohol consumption	Daily	39%	40%	41%	35%
	Weekly	16%	15%	16%	15%
	Seldom	45%	45%	41%	49%
	Never	0%	0%	1%	0%
	Missing data	< 1%	< 1%	< 1%	< 1%
Married/living with partner	Yes	80%	81%	79%	80%
	Missing data	< 1%	< 1%	< 1%	< 1%
Education level	Primary school or less	44%	48%	44%	40%
	Up to secondary school or equivalent	26%	26%	27%	25%
	University degree & more	24%	18%	24%	30%
	Missing data	6%	8%	5%	4%
Occupation	Gainfully employed	80%	77%	80%	82%
	Unemployed/not gainfully employed	6%	5%	6%	5%
	Retired	5%	5%	6%	4%
	Missing data	10%	12%	9%	9%
Mean neighbourhood income (Swedish Krona, SEK)		121,555	113,757	121,118	129,854

3.2. Particle concentrations

Mean lag 1–10 concentrations of total PM_{10}, total $PM_{2.5}$, PM_{10}-traffic, $PM_{2.5}$-exhaust, and $PM_{2.5}$-wood burning during the follow-up period were 9.49, 5.60, 0.49, 0.07, and 0.76 μg/m^3, respectively (Table 2). Total PM_{10} concentrations demonstrated moderate correlation with PM_{10}-traffic, $PM_{2.5}$-exhaust, and $PM_{2.5}$-wood burning (Spearman's correlation coefficients, r_s = 0.48, 0.59, and 0.40, respectively, all p-values < 0.05). In contrast, correlations between PM_{10}-traffic and $PM_{2.5}$-wood burning were weaker (r_s = 0.23, p-value < 0.05). PM_{10}-traffic accounted for 5.2% of total PM_{10}, and $PM_{2.5}$-exhaust and $PM_{2.5}$-wood burning constituted 1.3% and 13.6% of total $PM_{2.5}$, respectively.

Table 1. Ambient particulate matter concentrations during follow-up, presented as means (SD), quartiles, and interquartile ranges (IQRs). PM_{10}: particulate matter ≤ 10 μm; $PM_{2.5}$: particulate matter ≤ 2.5 μm; PM_{10}-traffic: traffic-related PM_{10}; $PM_{2.5}$-exhaust: exhaust-related $PM_{2.5}$; $PM_{2.5}$-wood burning: $PM_{2.5}$ from residential wood combustion.

PM	Mean (SD)	Median (1st Quartile–3rd Quartile)	IQR
PM_{10}	9.49 (1.04)	9.36 (8.81–10.06)	1.25
$PM_{2.5}$	5.60 (0.62)	5.55 (5.20–5.96)	0.77
PM_{10}-traffic	0.49 (0.58)	0.34 (0.21–0.60)	0.39
$PM_{2.5}$-exhaust	0.07 (0.09)	0.05 (0.03–0.09)	0.06
$PM_{2.5}$-wood burning	0.76 (0.31)	0.74 (0.60–0.89)	0.29

3.3. Associations with lung cancer incidence

Non-statistically significant risk increases for incident lung cancer were observed in association with total PM_{10}, total $PM_{2.5}$, PM_{10}-traffic, $PM_{2.5}$-exhaust, and $PM_{2.5}$-wood burning (Table 3). The risk estimates changed considerably after adjusting for individual-level baseline covariates as well as area-level socioeconomic status. For instance, the adjusted HR for incident lung cancer per 1 μg/m^3 increase in $PM_{2.5}$-exhaust was 1.20 (95% CI: 0.65–2.23), while the corresponding HR per 1 μg/m^3 increase in $PM_{2.5}$-wood burning was 1.05 (95% CI: 0.70–1.57). The analysis of exposure tertiles provided some evidence of associations between lung cancer incidence and concentrations of total PM_{10}, total $PM_{2.5}$, PM_{10}-traffic, $PM_{2.5}$-exhaust, and $PM_{2.5}$-wood burning. However, clear dose-response associations did not seem evident, as the estimates for tertiles 2 and 3 were very similar (Table 4). In the two-pollutant models using tertiles of exposure, there were tendencies for risk increases in relation to PM_{10}-traffic, $PM_{2.5}$-exhaust, and $PM_{2.5}$-wood burning, but not for total PM_{10} and $PM_{2.5}$ concentrations. As reflected in the wide confidence intervals, these estimates were imprecise (Table 4). No clear evidence of effect modification by sex, smoking status, or educational level was observed. Similar patterns were found for all pollutants; results are shown for PM_{10}-traffic in Table 5.

Table 2. Single-pollutant associations between ambient particulate matter and lung cancer. Model 1 is adjusted for calendar year and sex, Model 2 is further adjusted for individual-level baseline confounders, and Model 3 (main model) is additionally adjusted for area-level socioeconomic status.

Cases, person years and PM	Increment	Model 1	Model 2	Model 3
		HR (95% CI)	HR (95% CI)	HR (95% CI)
Lung cancer cases		253	186	186
Person-years		421,466	339,900	339,629
PM_{10}	10 μg/m^3	2.13 (0.76–6.02)	1.47 (0.58–3.75)	1.31 (0.48–3.57)
$PM_{2.5}$	5 μg/m^3	1.58 (0.43–5.86)	1.48 (0.44–5.00)	1.35 (0.39–4.71)
PM_{10}-traffic	1 μg/m^3	1.10 (1.01–1.20)	1.04 (0.94–1.15)	1.02 (0.92–1.14)
$PM_{2.5}$-exhaust	1 μg/m^3	1.84 (1.14–2.95)	1.30 (0.74–2.28)	1.20 (0.65–2.23)
$PM_{2.5}$-wood burning	1 μg/m^3	0.97 (0.67–1.41)	1.05 (0.70–1.56)	1.05 (0.70–1.57)

Table 3. Associations between tertiles of ambient particulate matter and lung cancer in single- and two-pollutant models including PM-wood burning. Main model (Model 3) is adjusted for calendar year, sex, and potential individual- and area-level confounders.

PM	Tertiles	Single-pollutant model	Two-pollutant model	
			Adjusted for $PM_{2.5}$-wood burning	$PM_{2.5}$-wood burning
		HR (95% CI)	HR (95% CI)	HR (95% CI)
PM_{10}	T2	0.99 (0.67–1.46)	0.92 (0.61–1.39)	1.19 (0.81–1.75)
	T3	1.10 (0.69–1.74)	0.95 (0.54–1.65)	1.20 (0.77–1.85)
$PM_{2.5}$	T2	1.08 (0.74–1.57)	1.00 (0.66–1.51)	1.16 (0.78–1.73)
	T3	1.14 (0.71–1.82)	1.01 (0.56–1.83)	1.16 (0.71–1.90)
PM_{10}-traffic	T2	1.17 (0.80–1.72)	1.14 (0.77–1.70)	1.13 (0.78–1.64)
	T3	1.14 (0.77–1.69)	1.10 (0.73–1.66)	1.15 (0.77–1.71)
$PM_{2.5}$-exhaust	T2	1.17 (0.81–1.71)	1.15 (0.78–1.68)	1.11 (0.77–1.62)
	T3	1.18 (0.80–1.75)	1.15 (0.76–1.74)	1.15 (0.77–1.71)
$PM_{2.5}$-wood burning	T2	1.16 (0.82–1.66)		
	T3	1.17 (0.79–1.74)		

T2 = Second tertile of ambient PM exposure compared to first tertile;
T3 = Third tertile of ambient PM exposure compared to first tertile.

Table 4. Interaction analysis for PM_{10}-traffic. High and low exposure were defined as values above and below the median, respectively. Estimates were adjusted according to Model 3.

Modifier	Association	HR	95% CI
Gender	High exposure	0.762	0.488–1.188
	Gender (women *vs.* men)	0.811	0.524–1.257
	Interaction	1.498	0.822–2.728
	High exposure among men	0.762	0.488–1.188
	High exposure among women	1.141	0.736–1.769
Smoking	High exposure	0.937	0.669–1.312
	Smoking (never smoker)	0.124	0.064–0.240
	Interaction	1.061	0.409–2.755
	High exposure among ever smokers	0.961	0.686–1.345
	High exposure among never smokers	1.055	0.426–2.614
Education	High exposure	0.899	0.633–1.277
	Education (university)	0.901	0.463–1.753
	Interaction	1.252	0.541–2.898
	Exposure among those without university education	0.890	0.411–1.929
	Exposure among those with university education	1.089	0.767–1.547

4. Discussion

This large population-based cohort study with detailed exposure assessment provides risk estimates for lung cancer in relation to long-term exposure to PM_{10}-traffic, $PM_{2.5}$-exhaust, and $PM_{2.5}$-wood burning, but these risk estimates were not statistically significant. Despite inconclusive findings, the study contributes to the existing evidence base on air pollution and lung cancer by providing source-specific risk estimates for local particulate matter exposure. These results are valuable for future meta-analyses. Notably, the risk estimates, although uncertain, tended to be higher for local emissions from traffic and wood burning compared to total particulate matter concentrations that included regional background levels, particularly in the two-pollutant models.

The potential mechanisms by which inhaled particles cause lung cancer include oxidative stress, inflammation, DNA damage, epigenetic regulation, metabolism, and related signal transduction pathways [20]. Inhaled PM may induce oxidative stress by releasing reactive oxygen species, which then trigger different markers of inflammation [21]. These inflammatory responses, including the release of cytokines, TNF-α (tumour necrosis factor), and IL-1 (interleukin 1), can further lead to the development of lung cancer [22]. Exposure to PM may also lead to DNA damage by altering nucleotide sequence during replication, potentially inducing tumour promoting genes (oncogenes) or inactivating tumour supressing genes [23]. Additionally, PM might also cause gene dysregulation through epigenetic alterations, such as DNA methylation, histone modifications, and non-coding RNA expression [24,25]. Furthermore, exposure to PM may alter signal transduction pathways and, thereby, affect the cell cycle, metabolism, and apoptosis [26]. There is also evidence supporting inflammation as a direct mechanism. In an experimental study, for example, mice carrying mutations in the epidermal growth factor receptor gene that were exposed to particles developed more lung tumours compared to unexposed controls [27]. Notably, there was no increase in the number of DNA mutations in the lung cells of the mice. Instead, a sustained inflammatory response several weeks after the exposure ended suggested that air pollution may promote lung cancer by creating a pro-inflammatory environment that fosters the growth of cells with pre-existing cancer-causing mutations, rather than by directly inducing new mutations.

Previous studies suggest that particulate matter is among the most important and abundant of pollutants in urban air, exposing the lungs to high levels of inhaled particles. While residential wood combustion has been proposed to have detrimental effects on respiratory and cardiovascular health, similar to what has been observed for traffic-related air pollution, its role in lung cancer risk has not been extensively investigated in many epidemiological studies. More literature exists on air pollution exposure from road traffic and lung cancer, but studies investigating traffic-related PM specifically are still limited. Overall, most previous research on air pollution and lung cancer has focused on total PM rather than source-specific exposure [28].

Several previous studies have shown an association between long-term total particle concentrations and the risk of lung cancer in the general population. In the European Study of Cohorts for Air Pollution Effects (ESCAPE), Raaschou-Nielsen *et al.* used land use regression modelled PM_{10} and $PM_{2.5}$ and found increased risks of 22% (95% CI: 3%–45%) per 10 μg/m^3 PM_{10} and 18% (95% CI: −4%–46%) per 5 μg/m^3 $PM_{2.5}$ in a meta-analysis of 17 European cohorts [29]. A more recent meta-analysis by Ciabattini *et al.*, which included 15 high quality studies from across the world that were adequately adjusted for potential confounders, observed even higher risk estimates [8]. Specifically, the analysis reported a 16% (95% CI: 9%–23%) increase in lung cancer risk per 5 μg/m^3 $PM_{2.5}$ and a 23% (95% CI: 5%–40%) increased risk per 10 μg/m^3 PM_{10}. These risk increases were more pronounced for lung cancer mortality, compared with incidence, and were particularly elevated in studies conducted in Asia. Furthermore, in a pooled analysis of seven European cohorts within the Effects of Low-level Air Pollution: a Study in Europe (ELAPSE), a 13% (95% CI: 5%–23%) increased risk for incident lung cancer was reported per 5 μg/m^3 $PM_{2.5}$, even at concentrations below WHO Air Quality Guidelines [10].

Despite major advances in knowledge on the association between air pollution and lung cancer, questions related to the importance of source and type of air pollutants remain unanswered. Results from the limited number of studies estimating the risk of lung cancer in relation to source-specific particle concentrations have been somewhat inconsistent. For instance, a meta-analysis of cohort, case-control, and nested case-control studies conducted largely in Europe and North America reported an increase in the risk of lung cancer in relation to traffic-related $PM_{2.5}$, with an odds ratio of 1.11 (95% CI: 1.00–1.22). For NO_2, used as a marker of traffic-related air pollution, the odds ratio in the same study was 1.06 (95% CI: 0.99–1.13). Although NO_2 is not classified as a carcinogen, it is frequently used as a proxy for combustion-related emissions, particularly from traffic [30–34]. Some studies also use proxy measures, such as proximity to the nearest major road, to depict traffic-related exposure. For example, in the Netherlands Cohort Study on Diet and Cancer (n ≈ 110,000), a non-statistically significant increased risk of lung cancer was associated with traffic intensity on the nearest road and living near a major road [35].

Among studies investigating the association between source-specific $PM_{2.5}$ and the risk of lung cancer, some have focused specifically on mortality. In an analysis of the US Medicare cohort of elderly participants investigating the effect of long-term $PM_{2.5}$ components and their sources, a positive, but statistically non-significant, association with lung cancer mortality was found for traffic-related $PM_{2.5}$ (HR: 1.03, 95% CI: 0.99–1.06 per IQR increase), whereas no association was observed for $PM_{2.5}$ from biomass burning [36]. The large-scale American Cancer Society Cancer Prevention Study reported an increased risk of lung cancer mortality by 4% increased risk per 1.6 μg/m^3 of near-source (primarily traffic-related) $PM_{2.5}$ [37].

Although our findings were not statistically significant, the magnitude of our estimates was larger than those reported in most previous studies. These discrepancies may be explained by differences in population characteristics, differences in pollutants sources as well as differences in the assessments of air pollution exposure. In Northern Sweden, traffic-related emissions, including those from combustion, road wear, and brake abrasion, as well as residential wood burning are the two most significant local sources of PM [15]. Concerning exposure assessment, the ESCAPE study assessed air pollution at residential addresses with reasonable precision; however, it did not account for the change in addresses, potentially introducing misclassification. Furthermore, the meta-analysis by Ciabattini *et al.* included studies that measured average concentrations of $PM_{2.5}$ and PM_{10} exposure at an ecological level, which may have led to non-differential misclassification and an underestimation of risk [8]. In contrast, our study incorporated complete residential address histories and updated exposure models over the follow-up period to capture temporal changes in pollution levels. Moreover, we estimated ambient particulate matter with high spatial resolution using dispersion modelling, allowing for more accurate characterisation of long-term exposure, especially for locally emitted $PM_{2.5}$.

In this study, we focused on exposures occurring 1–10 years prior to the event to capture long- to medium-term effects while minimising potential bias from very recent exposures that might be influenced by preclinical disease or diagnostic processes. We acknowledge that longer latency periods may also be relevant for lung cancer aetiology, as exposures occurring earlier in life could contribute to disease risk. While longer lag periods were considered, this approach resulted in a substantially higher proportion of missing exposure data, which could have introduced bias and reduced statistical power. Thus, the chosen exposure window represents a balance between capturing relevant aetiological exposure and maintaining sufficient data coverage for robust analyses. Future studies with extended follow-up or more complete historical exposure data could further explore the impact of longer latency periods on lung cancer risk.

Our study has several notable strengths. First, it benefits from a large sample size and long follow-up period, which enhances the statistical power and reliability of the findings. Lung cancer cases were identified using Sweden's national patient and cause of death registers, which are known for their comprehensive coverage and high-quality data [38–40], thereby minimizing the risk of misclassification. A major strength of our study lies in the use of state-of-the-art ambient particulate matter modelling for exposure assessment. We estimated both total and source-specific ambient particle concentrations with high spatial resolution. The modelling incorporated a range of factors affecting the air pollutants' local emission and dispersion, including meteorological conditions, traffic volumes (including vehicle types and speed, which affect exhaust and wear emissions), the width of the street, and the height of surrounding buildings. Importantly, changes in participants' residential address were also accounted for. Moreover, the analyses were adjusted for a comprehensive set of confounders at both the individual and area level, further strengthening the validity of the results.

The present study also has several limitations that should be highlighted. First, exposure assessment at participants' residential addresses is prone to misclassification. In reality, true exposure is not restricted to ambient concentrations at the home address, but also includes indoor exposure, workplace exposure, and exposure during commuting. However, recent simulation studies seem to suggest that the bias caused by attributing exposure to residential address only may be quite limited [41]. Second, we cannot rule out the possibility that non-participation of individuals with low socioeconomic status at recruitment might lead to an underestimation of the true effect of air pollutionambient particulate matter on the risk of lung cancer. This would be the case if low socioeconomic status were to be associated with higher concentrations of abmient particulate matter, since this group also has a higher incidence

of lung cancer. In our study area, however, the correlation between ambient particulate matter and socioeconomic status is quite low, which might mitigate this concern. Third, the VIP cohort lacks information on noise and access to green spaces. While emering evidence suggests that these environmental exposures may independently influence lung cancer risk, the findings remain limited and somewhat inconsitent [42,43]. Similarly, occupational exposures, also potentially contributing to lung cancer [44], were not included. The extent to which occupational exposure correlates with ambient particlate matter exposure for this population has not yet been investigated. Fourth, the VIP cohort does not contain information on exposure throughout participants' life course. Furthermore, information on potential confounders was only available at recruitment. Different factors, such as disease incidence, injury, or a health screening intervention, occuring throughout follow-up may influence these baseline variables, potentially introducing bias in the adjustment for confounders.

5. Conclusions

This study estimated increased risks for lung cancer associated with long-term exposure to PM_{10}-traffic, $PM_{2.5}$-exhaust, and $PM_{2.5}$-wood burning. However, the associations were not statistically significant and did not consistently demonstrate linear relationships. Notably, the observed trends occurred at concentrations below the current WHO air quality guidelines, reinforcing evidence that there may be no safe threshold for the harmful health effects of ambient particulate matter. These findings underscore the need for further research on source-specific ambient particulate matter exposure and its effects on the incidence of lung cancer. They also highlight the importance of multi-pollutant models and studies that distinguish between local pollution sources to better understand their combined and independent effects. Such evidence is critical to inform more targeted and effective public health policies and air pollution abatement strategies.

Ethical statement

The study was approved by the regional ethical review board in Umeå (reference 2015/16-31Ö).

Data availability statement

The data supporting the findings of this study, including individual-level data on outcomes, exposures, and covariates, are not publicly available due to ethical and legal restrictions. However, the data may be made available from the corresponding author upon reasonable request and subject to appropriate ethical and institutional approvals.

Declaration of generative AI and AI-assisted technologies

During the preparation of this manuscript, the authors used ChatGPT for limited sections of the manuscript and for language polishing only. The authors take full responsibility for the content of the manuscript.

Authors' contribution

JS: formal analysis, investigation, methodology, writing—original draft preparation, writing—review and editing; WR: investigation, methodology, writing—original draft, writing—review and editing; EF: writing—original draft preparation, writing—review and editing; DOÅ: writing—review and editing; AO: conceptualisation, data curation, funding acquisition, investigation, methodology, interpretation, project administration, resources, writing—original draft preparation, writing—review and editing. All authors have read and agreed to the published version of the manuscript.

Acknowledgments

We are grateful to the participants who contributed to the Västerbotten Intervention Programme and to Kajsa Pira for editing language in the final stage. Funding: This research was supported by Formas under grant agreement number 2017-00898 (How is our health affected by particles from wood burning? Anna Oudin). This work has received funding from the European Union's Horizon Europe research and innovation programme under grant agreement No. 101095430 (Marches project).

Abbreviations

Terms	Abbreviations
Confidence Interval	CI
European Study of Cohorts for Air Pollution Effects	ESCAPE
Hazard Ratio	HR
particulate matter in ambient air (particles with an aerodynamic diameter of 10 µm)	PM_{10}
fine particulate matter in ambient air (particles with an aerodynamic diameter of 2.5 µm)	$PM_{2.5}$
particulate matter in ambient air (particles with an aerodynamic diameter of 10 µm) with road traffic as source	PM_{10}-traffic
fine particulate matter in ambient air (particles with an aerodynamic diameter of 2.5 µm) with road traffic vehicle exhaust as source	$PM_{2.5}$-exhaust
fine particulate matter in ambient air (particles with an aerodynamic diameter of 2.5 µm) with small-scale residential heating, mainly wood-burning in the study area, as source	$PM_{2.5}$-wood burning
Västerbotten Intervention Programme	VIP

Conflicts of interest

Anna Oudin holds the position of Editorial Board Member for *International Journal of Environmental Epidemiology* and has not peer reviewed or made any editorial decisions for this paper.

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