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ORIGINAL ARTICLE

Urban and transport planning, air pollution, and green space: health effects in three Belgian cities

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Abstract

Aim This study examines how urban planning in Belgium impacts public health by influencing air pollution and green space. It analyses the link between these environmental factors and health issues such as mortality, cardiovascular disease, diabetes, asthma, and depression.

Subject and methods This study quantifies the combined disease burden attributable to fine particulate natter ($PM_{2.5}$), NO_2, and insufficient green space in Brussels, Liège, and Mechelen using a multiplicative population preventable fraction (PF) approach with World Health Organization (WHO) exposure target values as counterfactual scenario.

Results Our findings reveal that reducing $PM_{2.5}$ and NO_2 and increasing green space to WHO-recommended levels could lower total mortality by 14.7% (95% CI 9.4–19.8) in Brussels, 11.7% (95% CI 7.5–16.3) in Liège, and 12.0% (95% CI 7.7–16.3) in Mechelen. Cardiovascular mortality could decrease by 13.4% (95% CI 5.8–21.0) in Brussels, 10.3% (95% CI 4.1–16.0) in Liège, and 11.0% (95% CI 5.1–16.6) in Mechelen. Diabetes reductions are estimated at 25.4% (95% CI 7.0–40.3) in Brussels, 21.6% (95% CI 7.2–37.0) in Liège, and 19.9 (95% CI 6.7–33.9) in Mechelen.
Focusing on background concentrations for NO_2 underestimates health effects. Traffic contributes 73.2–78.6% of NO_2, with local traffic accounting for 40.9–55.0% of concentrations.

Conclusion This study identifies a statistically significant link between elevated air pollution, limited green space, and the potential to reduce chronic disease prevalence by adhering to WHO guidelines. It underscores the importance of health-centred urban planning, advocating for green space expansion, air quality improvements, and more precise NO_2 source allocation to better pinpoint and mitigate pollution sources, ultimately fostering healthier communities.

Keywords Air pollution · Urban planning · Transport planning · Green space · Preventable fraction · WHO guidelines

Introduction

Urban planning, transportation, and mobility policies have a multifaceted impact on human health, influencing exposure to environmental stressors, access to green spaces, and levels of outdoor physical activity (Mueller et al. 2017; Rojas-Rueda et al. 2013). Traffic-related air pollution has been linked to respiratory diseases such as asthma, chronic obstructive pulmonary disease (COPD), diabetes, and cardiovascular diseases (Achakulwisut et al. 2019; Brunekreef et al. 2021; Collart et al. 2018; Requia et al. 2023). Additionally, a scarcity of urban green spaces can contribute to stress, mental health issues, reduced physical activity, and an increased risk of cardiovascular disease (Liu et al. 2022; White et al. 2013). Furthermore, urban heat islands, characterised by elevated temperatures in urban areas relative to

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less developed surrounding areas, can cause or exacerbate cardiovascular diseases (Huang et al 2020).

A study in Barcelona demonstrated that compliance with international recommendations could prevent 20% of mortality (Mueller et al. 2017). These recommendations involve increasing physical activity, reducing exposure to air pollution, noise, and heat, and enhancing access to green spaces. Another study in Barcelona estimated that replacing 40% of car trips with public transportation or active modes of transport could lead to a reduction in cardiovascular disease, diabetes, traffic injuries, dementia, breast cancer, and colorectal cancer (Rojas-Rueda et al. 2013).

While numerous studies have explored the link between various environmental factors and health outcomes (Rojas-Rueda et al. 2021), integrated health impact assessments (HIAs)—focusing on how policies affect health through their environmental, economic, and social impacts—remain scarce for urban and transport planning (Khreis and Nieuwenhuijsen 2019; Nieuwenhuijsen 2016).

Additionally, existing air pollution source allocation methods often underestimate the traffic-related exposure and subsequent health and environmental impacts. For example, the SHERPA tool, commonly used for air pollution source allocation, does not account for the local component of traffic-related emissions, leading to an underestimation of the associated health effects. These studies often provide the best available estimates for policy contexts, although most studies using this tool explicitly acknowledge its limitations and the likelihood of underestimation (Degraeuwe et al. 2021; Khomenko et al. 2023; Thunis et al. 2018).

Building on previous HIAs, this study extends existing approaches by analysing small and mid-sized Belgian cities (Liège and Mechelen) at high spatial resolution, enabling neighbourhood-level assessment of preventable disease burden. It further advances prior work by quantifying the underestimation of traffic-related NO_2 in conventional source allocation models, and by linking environmental exposure to socio-economic deprivation indicators, thereby identifying inequalities in health risks and supporting more context-specific and actionable policy guidance for urban and transport planning.

Accurately quantifying this disease burden is essential for defining and implementing effective mitigation measures to reduce health impacts by achieving World Health Organization (WHO) guidelines for air pollution levels and residential green space availability. The objective of our research is to estimate the disease burden of asthma, diabetes, depression, and cardiovascular disease resulting from air pollution exposure and insufficient green space, as associated with urban and transport planning practices. Specifically, we aim to (1) estimate the total disease burden of the selected conditions that could be prevented if WHO recommendations for long-term air pollution exposure and residential green space availability are met, and (2) quantify the underestimation of the health burden associated with traffic-related air pollution by current source allocation methods and explore solutions to improve these methods to enhance the accuracy of HIAs.

Methodology and data

Study area

A list of inclusion criteria and an evaluation of Belgium's 15 largest cities regarding those criteria can be found in Appendix I: Selection of cities. We selected three Belgian cities. The study area includes the Brussels Capital Region (BCR), Liège, and Mechelen (Fig. 1).

Data

Environmental data

We used air pollution data from the ATMO-Street high-resolution (100 m including street canyon effects) model chain (Hooyberghs et al. 2022), open data obtained from reference measurement stations in the cities of Mechelen (one station, classified as 'traffic'), Brussels (two stations, one station classified as 'traffic' and one station as 'central urban zone'), and Liège (one station, classified as 'traffic'), and the SHERPA tool for information on source allocation of the background air pollution concentration (Degraeuwe et al. 2021; Pisoni et al. 2024). Both ATMO-Street air pollution model data and data from reference measurement stations are from the calendar year 2022. For green space, we use remote-sensing normalized difference vegetation index (NDVI) data from the Moderate Resolution Imaging Spectroradiometer (MODIS) satellites (Huete et al. 2002, 2010). We computed the yearly average for 2016 based on the 16-day NDVI images with a cell size of 250 m.

Socio-economic data

To describe the socio-economic status (SES) of each statistical sector, we use the Belgian Index of Multiple Deprivation (BIMD), combining six domains of deprivation (income, employment, education, housing, crime, and health). We also used the housing domain of the index of deprivation, as calculated by Otavova (2023), as the housing domain is closely linked to urban and transport planning. Both indices refer to the situation in year 2011, because no census data are available beyond 2011.

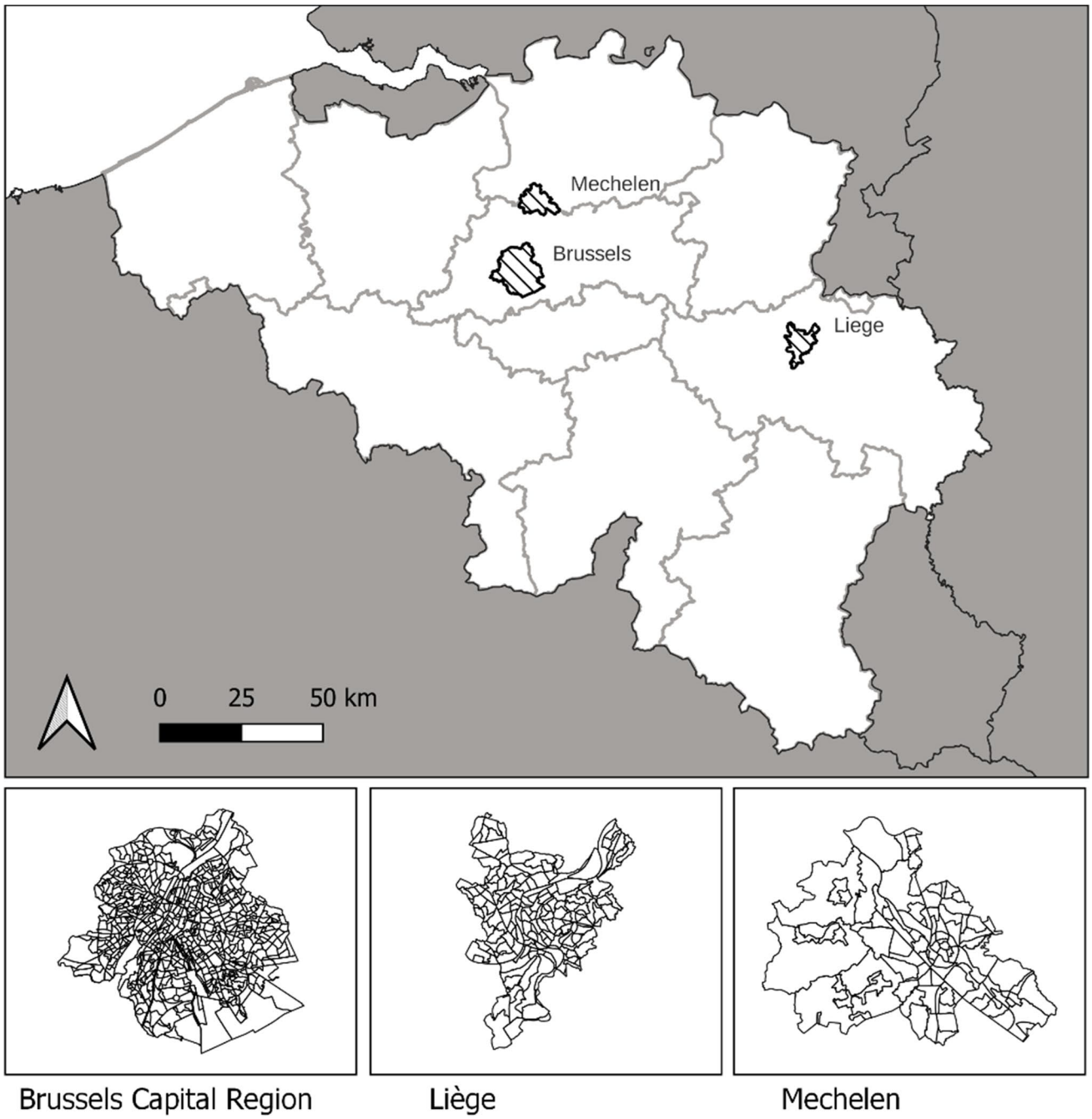


Fig. 1 Study area with the Belgian cities Brussels, Liège, and Mechelen. The zoomed-in-display for each of the cities shows the statistical sector boundaries within each urban area. Map by Eva De Clercq, data from StatBel

Selection of diseases

We reviewed meta-analyses of existing studies to determine which cause-specific diseases to include in our study, limited to diseases that could be affected by urban and transport planning, with the availability of epidemiological evidence and the existence of plausible biological mechanisms to explain the relationship. We excluded diseases for which the available evidence was inconsistent or inconclusive, meaning that the 95% confidence interval overlapped with an estimate of zero attributable disease (absence of statistically significant relation). For air pollution, we consider both fine particulate matter ($PM_{2.5}$) and NO_2, and for green space, we use NDVI values as a proxy, as it is the most commonly studied criterion in HIAs. We focused on cardiovascular diseases, diabetes, and asthma as air pollution-related

conditions, and included cardiovascular diseases and depression as conditions linked to a lack of residential and urban green space.

Exposure–response functions

The exposure-response functions (ERF) used in our HIA were derived from recent comprehensive meta-analyses. A summary of the diseases and the origin of the exposure–response relationships is provided in Table 1.

The inclusion criteria for each environmental stressor–outcome pair required that the ERF be adjusted for confounding factors such as smoking and SES. Additionally, studies had to focus on healthy populations and include high-spatial-resolution exposure assessment (e.g., 100 × 100 m). Priority was given to recent, extensive meta-analyses, especially those providing two- or multiple-pollutant estimates that adjust for the interrelated impacts of NO_2 and $PM_{2.5}$. This approach was consistently applied across outcomes such as natural mortality, cardiovascular mortality, and adult asthma (Table 1).

The use of two-pollutant models, where NO_2 is adjusted for $PM_{2.5}$, represents a conservative approach that helps avoid double-counting risks attributable to correlated pollutants. While this may slightly underestimate the full impact of traffic-related pollution, it prevents overestimation observed in single-pollutant models. An additional sensitivity analysis comparing NO_2-related health impacts using unadjusted versus $PM_{2.5}$-adjusted ERFs is provided in Appendix IV.

Potential interactions between air pollution and green space were not explicitly modelled. Most epidemiological studies report limited or negligible statistical interactions between these exposures. For example, James et al. (2016) found that $PM_{2.5}$ explained only about 4% of the association between green space and all-cause mortality, while the meta-analysis on cardiovascular mortality (Brunekreef et al 2021) already included air pollution adjustments in some of the studies included in the meta-analysis without major changes in effect estimates. The combined effects are therefore treated as additive, although some uncertainty remains, and the results should be interpreted with caution, as synergistic or antagonistic effects, if present, could modestly influence the estimated health impacts.

Health impact assessment

To calculate the preventable fraction (PF)—the fraction of disease that could be prevented by achieving the counterfactual scenario—for each of the selected diseases (see table), the following formula is used:

$$PF = \frac{(1 - RR)}{RR} \quad (1)$$

in which RR is the relative risk of exposure. For air pollution, the counterfactual scenario used is 5 µg/m^3 $PM_{2.5}$ and 10 µg/m^3 NO_2, corresponding to the WHO guideline values for long-term air pollution exposure. For the target value for NDVI, we used the calculation from an earlier study of 1000 cities by the ISGlobal institute, where they translated the WHO recommendations on green space to target NDVI values specific for each city (Barboza et al. 2021). This resulted in the use of a counterfactual target NDVI of 0.559 for Liège and 0.52 for Brussels. As Mechelen was not included in the 1000-city study, for Mechelen we used the value of the nearby and similar city Leuven, with a target NDVI of 0.56.

Table 1 Dose–response relationships extracted from recent meta-analyses

Exposure–response functions (ERFs)	NO_2 per 10 µg/m^3	$PM_{2.5}$ per 10 µg/m^3	Source	NDVI per 0.1 unit increment	Source
Natural mortality	1.050 [1.031–1.070]	1.083 [1.054–1.113] Traffic-specific: 1.212 [1.141–1.283]	(Brunekreef et al. 2021) (Chen et al. 2022)	0.96 [0.94–0.97]	(Rojas-Rueda et al. 2019)
Cardiovascular mortality	1.043 [1.007–1.079]	1.100 [1.053–1.150] Traffic-specific: 1.212 [1.071–1.389]	(Brunekreef et al. 2021) (Chen et al. 2022)	0.97 [0.96–0.99]	(X.-X. Liu et al. 2022)
Diabetes risk	1.07 [1.04–1.11] (prevalence)	1.08 [1.04–1.12] (prevalence)	(Yang et al. 2020)	0.919 [0.862–0.982] per 0.09 NDVI (IQR) (incidence)	(Ccami-Bernal et al. 2023)
Asthma incidence—children	1.125 [1.10–1.175]	*	(Khreis et al. 2017)	**	
Asthma incidence—adults	1.21 [1.11–1.32]	*	(S. Liu et al. 2021)	**	
Depression prevalence	**	**		0.931 [0.887–0.977]	(Z. Liu et al. 2023)

* No effect after correction for NO_2; ** no statistically significant effect. *IQR* interquartile range

The RR at the computed average value for each statistical sector (*RRexposure*) is converted from the default unit per 10 μg/m^3 increase to the relevant exposure unit, using

$$\mathrm{RRexposure} = \exp((\ln(\mathrm{RR10})/10) \times (\mathrm{CON})) \quad (2)$$

in which RR10 is the RR for an increase of 10 μg/m^3 in air pollution or a 0.1-unit decrease in NDVI starting from the counterfactual scenario of the target NDVI, and CON is the actual mean concentration of the statistical sector of the air pollutant under consideration. A Monte Carlo simulation (10,000 iterations) assuming a log-linear distribution is applied on the reported ERFs in order to obtain a more likely distribution of the confidence intervals of the ERFs for both air pollution and green spaces. For each of the results, we estimated combined health effects. To combine the different PF estimates into an overall PF, we use the formula for the multiplicative PF (Eq. 3).

$$\mathrm{PF\ Multiplciative} = 1 - \prod_{j=1}^{k}(1 - \mathrm{PF(xj)})\mathrm{PF\ Multiplciative} = 1 - \prod_{j=1}^{k}(1 - \mathrm{PF(xj)}), \quad (3)$$

where PF(xj) is the PF for each individual risk factor j, k is the total number of risk factors, and the term x represents the individual exposure levels or factors that contribute to the overall PF.

For the PF calculation, we used meta-analyses for air pollution that accounted for the overlap between $PM_{2.5}$ and NO_2. Additionally, all the meta-analyses we used corrected for socio-economic factors and other confounders like smoking. We then compared the combined PF of air pollution and green space (corrected for socio-economic factors) with socio-economic indicators such as the BIMD and housing deprivation. Since the ERFs for environmental factors already adjust for socio-economic factors, our analysis shows whether high environmental stress from suboptimal urban and transport planning overlaps with a high disease burden from socio-economic factors.

Source allocation of air pollution: the traffic component

High-resolution air pollution models provide a robust estimation of overall air pollution levels at individual locations within Belgium. These models consist of a foundational RIO (residual Interpolation optimized for ozone) component, complemented by additional local and street canyon components. Together, it is called 'ATMO-Street'. In accordance with the passage of the FAIRMODE (Forum for Air Quality Modeling) criterion, a measure for the discrepancy between measured and modelled concentrations based on the root mean square error (RMSE), the ATMO-Street model substantiates its suitability for informing policy decisions (Hooyberghs et al. 2022).

However, these models exhibit a limitation in their inability to facilitate model decomposition, rendering retro calculation of air pollution concentrations for each sector unfeasible through the existing model tools. Although SHERPA stands out as one of the most frequently employed tools for source allocation across Europe, it is constrained by its exclusive focus on background air pollution concentrations and thus misses the contribution of local traffic emissions on air pollution estimates. This limitation is similar to considering only the RIO background component within the ATMO-Street model chain.

Given that the disparity between background and total air pollution is not just primarily but entirely attributable to traffic and certain minor industrial sites, it can be asserted that at spatial points devoid of industrial facilities, the difference between background and total concentrations may be considered as the local traffic component of air pollution (Hooyberghs et al. 2022; Lefebvre et al. 2011, 2013). This can be expressed by Eqs. 4 and 5.

$$\mathit{Local\ Traffic}\left(LT, \mathit{absolute\ concentration}, \frac{\mu g}{m^3}\right) = \mathit{Total\ concentration} - \mathit{Background\ Concentration}, \quad (4)$$

$$\mathit{Total\ Traffic}\left(TT, \mathit{absolute\ concentration}, \frac{\mu g}{m^3}\right) = (\mathit{Background\ concentration} * \mathit{Fraction\ of\ background\ concentration\ originating\ from\ traffic}) + \mathit{Local\ Traffic}. \quad (5)$$

The background concentration, including the proportion of the background concentration originating from traffic, is derived from the SHERPA tool, and the total concentration and the derived local traffic contribution are obtained from the reference measurement stations. In a final calculation, we quantify the underestimation of traffic-related air pollution using the SHERPA background method by comparing the SHERPA background concentrations with the values of total concentrations measured in the reference measurement stations.

In Appendix II, a theoretical simulation is included demonstrating how not taking into account the local traffic component can have considerable consequences for HIA and mitigating disease burden.

Influence of source-specific particulate matter dose–response relationships on health impact assessment

For $PM_{2.5}$, we quantify the impact of using global versus source-specific concentrations (accounting for chemical composition and distinct biological effects of different particulate matter components) on the health burden of traffic-related $PM_{2.5.}$ based on an analysis of suitable measurement stations in the cities of Brussels, Liège, and Mechelen. A traffic-specific ERF is available for $PM_{2.5}$ for natural mortality and cardiovascular mortality.

Integrated assessment of urban and transport planning-associated disease burden

For the selected point locations for which we have reference measurement stations available, we estimate the reduction in disease burden for the counterfactual scenario where (a) traffic-related NO_2 is reduced to 0 $\mu g/m^3$, (b) traffic-related $PM_{2.5}$ is reduced to 0 $\mu g/m^3$, and (c) green space exposure is increased up to the level of the city-specific target NDVI, still using the dose–response relationships as in Table 2 and the HIA methodology as in Sect. 2.3.

Results

Health impact assessment

If the counterfactual values of 5 $\mu g/m^3$ for $PM_{2.5}$ exposure, 10 $\mu g/m^3$ for NO_2 exposure, and city-specific target NDVI values (see the methodology section) are achieved, it is estimated that 11.96%, 12.04%, and 14.68% of total mortality in Liège, Mechelen, and Brussels, respectively, could be prevented (Table 2). Regarding specific causes of death, 10.26%, 10.98%, and 13.36% of cardiovascular mortality could be prevented in these cities (Table 2). For diabetes, reaching the recommended exposure targets could prevent 21.6%, 19.9%, and 25.4% of cases in Liège, Mechelen, and Brussels, respectively (Table 2). Additionally, meeting NO_2 targets would notably reduce asthma incidence in both adults and children, while increasing green space exposure to target values could reduce depression prevalence by 10.9%, 9.8%, and 12.6% in Liège, Mechelen, and Brussels (Table 2). At the inter-city level, spatial variations are evident: $PM_{2.5}$ contributions are most important in Mechelen, while NO_2 exposure and lack of green spaces have a larger impact in Brussels and Liège. At the intra-city level, distinct patterns emerge: mortality related to NDVI is concentrated in the city centres of Mechelen and Liège, NO_2 values display heterogeneous spatial patterns with generally higher values in the suburbs adjacent to city centres, and $PM_{2.5}$ values follow a more gradual distribution. To illustrate the importance of the spatial patterns, a sample of figures for Liège, Mechelen, and Brussels is presented in Fig. 2 for total mortality. Additional figures, including the spatial patterns for other selected diseases for all three cities, can be found in Appendix III.

Analysis of socio-economic inequalities, case study: cardiovascular mortality

Additional analyses correlate cardiovascular mortality with SES, incorporating data on the BIMD and housing deprivation. Figure 3 illustrates these relationships, with mean BIMD values recorded as 6.2 for Mechelen, 2.7 for Brussels, and 1.7 for Liège. In most cases, the PF decreases for increasing BIMD value (= less deprivation), and this is more pronounced for the overall BIMD than for the housing component in particular (Fig. 3a, b, c).

In general, the strongest trends are observed for green space, followed by NO_2, with $PM_{2.5}$ being characterised by the weakest trend or no correlation at all with BIMD.

Notably, Brussels shows a strong positive correlation between overall deprivation level and preventable cardiovascular mortality, with a PF of around 0.15 for the most deprived decile and 0.05 for the least deprived decile. In Mechelen, a similar trend can be observed, while in Liège the trend is more complex and irregular (Fig. 3).

Source allocation of air pollution

For the reference measurement stations used in this study, we observe that focusing solely on background concentrations in source allocation studies leads to an underestimation of total NO_2 concentrations. This is because, across all reference stations, the local traffic component contributes a similar proportion (approximately 50%) as the combined contribution of background traffic and background other sources. Specifically, the local traffic component accounts for 51.3%, 52.2%, 50.0%, and 40.9% at the four reference

Table 2 Population-weighted PF values in the cities of Liège, Mechelen, and Brussels for the selected diseases

Population-Weighted averages	Liège	Mechelen	Brussels
Total Mortality NDVI	0.060 [0.036- 0.085]	0.057 [0.034- 0.008]	0.067 [0.041- 0.095]
Total Mortality $PM_{2.5}$	0.019 [0.013- 0.026]	0.0436 [0.029- 0.058]	0.041 [0.027- 0.055]
Total Mortality NO_2	0.045 [0.028- 0.0615]	0.0253 [0.016- 0.035]	0.046 [0.029- 0.063]
Total Mortality Multiplicative PF	0.117 [0.075 - 0.163]	0.1204 [0.077- 0.163]	0.147 [0.094- 0.198]
Cardiovascular Mortality NDVI	0.044 [0.02- 0.068]	0.040 [0.020- 0.062]	0.051 [0.025- 0.079]
Cardiovascular Mortality $PM_{2.5}$	0.023 [0.012- 0.033]	0.0519 [0.028- 0.075]	0.049 [0.027- 0.071]
Cardiovascular Mortality NO_2	0.039 [0.007- 0.070]	0.0218 [0.004- 0.039]	0.039[0.0073 0.071]
Card. Mor Multiplicative PF	0.103 [0.041- 0.16]	0.1098 [0.051- 0.166]	0.134 [0.058- 0.210]
Diabetes Incidence NDVI	0.148 [0.031- 0.288]	0.1334 [0.029- 0.257]	0.171 [0.036- 0.332]
Diabetes Prevalence $PM_{2.5}$	0.019 [0.0097- 0.027]	0.0421 [0.022-0.062]	0.040 [0.021- 0.059]
Diabetes Prevalence NO_2	0.062 [0.033- 0.0900]	0.035 [0.018- 0.051]	0.063 [0.019- 0.051]
Diabetes Multiplicative PF	0.216 [0.072- 0.370]	0.199 [0.067- 0.339]	0.254 [0.07- 0.403]
Asthma incidence NO_2 *Children*	0.105 [0.077- 0.132]	0.060 [0.077 - 0.132]	0.107 [0.078- 0.134]
Asthma incidence NO_2 *Adults*	0.164 [0.093- 0.228]	0.100 [0.053 - 0.135]	0.166 [0.095- 0.231]
Depression Prevalence NDVI	0.109 [0.033- 0.194]	0.099 [0.030- 0.174]	0.126 [0.039- 0.224]

Brackets contain the 95% CI. $<2.50\%$ = light green, 2.50–4.99% = yellow, 5.00–9.99% = orange, $\geq 10.00\%$ = red

measurement stations in Brussels, 53.1% at the station in Liège, and 55.0% at the reference measurement station in Mechelen (Fig. 3). The fraction of NO_2 that can be attributed to traffic (= local traffic + background traffic) varies between 73.2% at the station in Mechelen and 78.6% at the Kunst-Wet station in Brussels (Fig. 4). For $PM_{2.5}$, the local traffic component is negligible in most stations, with the exception of Kunst-Wet in Brussels, where local traffic is responsible for 4.3% of the total $PM_{2.5}$ concentrations (not shown).

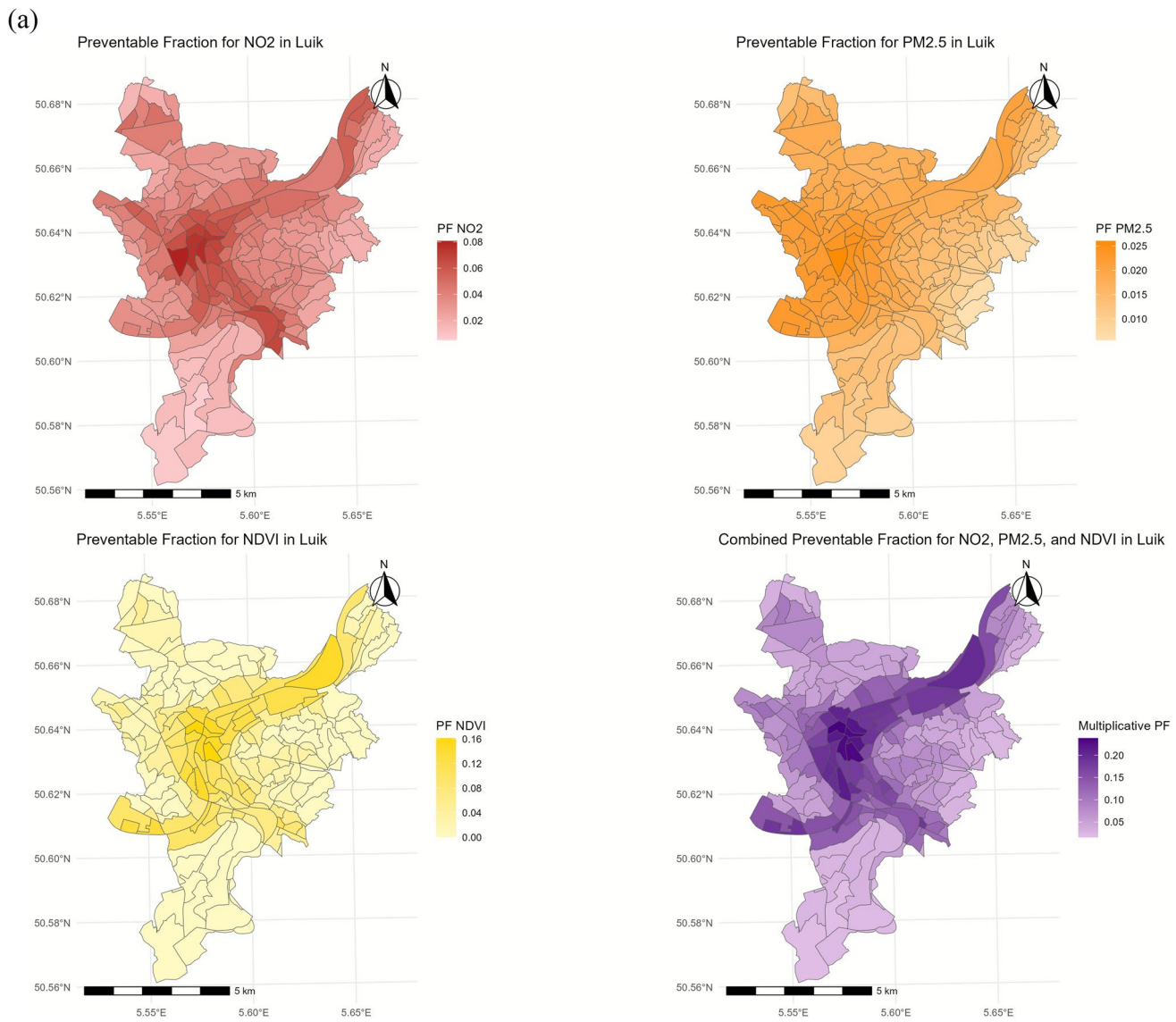


Fig. 2 Preventable fraction of total mortality if counterfactual target values of NDVI, NO_2, and $PM_{2.5}$ are achieved: spatial patterns in the cities of Liège (**a**), Mechelen (**b**), and Brussels (**c**)

Source-specific dose–response functions for particulate matter

Applying source-specific particulate matter dose–response functions accounting for chemical and physical composition of particulate matter, and distinct health effects originating from there, results in an increased burden of traffic-related particulate matter, with 1.5–2.1% of total mortality and 1.8–2.5% of cardiovascular mortality attributable to traffic-related $PM_{2.5}$ applying global dose–response functions, versus 3.7–5.2% of total mortality and 3.7–5.2% of cardiovascular mortality attributable to traffic-related $PM_{2.5}$ at the point locations of reference measurement stations in Mechelen, Liège, and Brussels (Fig. 5). For total mortality, this relation is highly significant, as there is no overlap in confidence intervals (Fig. 5). For cardiovascular mortality, where the confidence intervals are much wider, this relation is not statistically significant (Fig. 5).

Integrated assessment of urban and transport planning-associated disease burden

If air pollution is source-allocated and we only consider traffic-related air pollution, then NO_2 is clearly associated

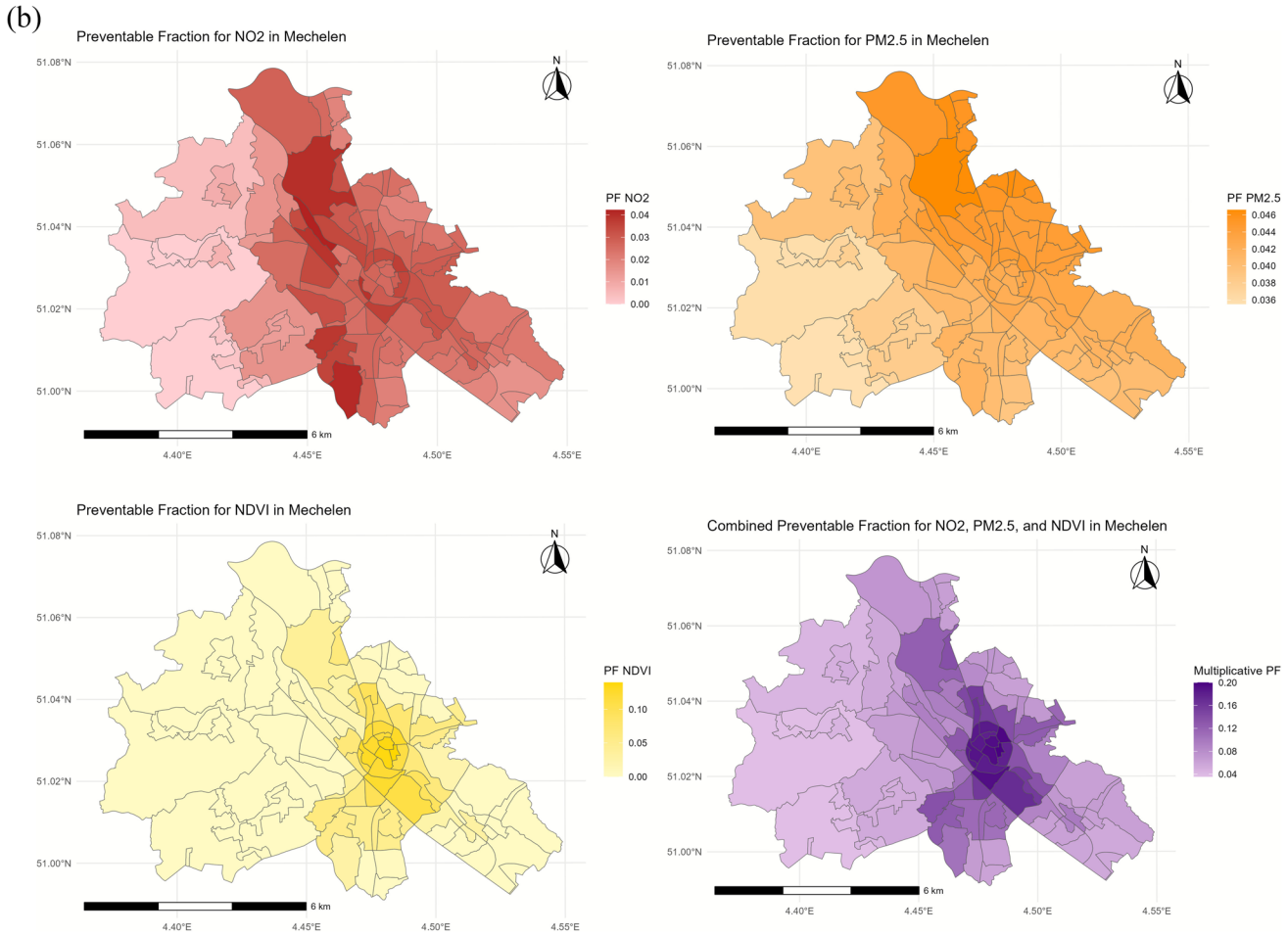


Fig. 2 (continued)

with a higher health burden (relative to $PM_{2.5}$) in Belgian cities (7.6–11.5% prevention of total mortality from reducing NO_2, Fig. 6), although the mitigation potential in reducing disease and death from reducing traffic-related $PM_{2.5}$ remains considerable (3.7–5.2% prevention of total mortality from reducing $PM_{2.5}$, Fig. 6). For total mortality, the multiplicative effects of traffic-related $PM_{2.5}$ and NO_2 are of a similar order of magnitude as the effect of green spaces, with a mean estimate between 11.2% in Liège and 16.1% in Brussels for traffic-related air pollution versus between 10.5% in Mechelen and 14.8% in Brussels for insufficient green space exposure through NDVI (Fig. 6). We observe that, for diabetes in particular, the multiplicative PF (considering both traffic-related air pollution and green spaces) of reduction is very strong, ranging from 30.8% in Liège to 40.3% in Brussels, with green spaces being a more important contributor to the multiplicative PF than traffic-related air pollution (Fig. 6).

Discussion

Summary and interpretation of the results

A strong correlation was found between air pollution, lack of green space, and adverse health outcomes in Mechelen, Liège, and Brussels. As is known from the literature review, cardiovascular mortality, diabetes, and asthma show varying degrees of correlation with environmental conditions. Population-weighted averages indicate that achieving WHO guidelines for air pollution and green space could significantly reduce diseases, including total mortality, cardiovascular mortality, diabetes, asthma, and depression.

Spatial analyses of the PF under current conditions reveal notable disparities within cities, with urban centres bearing a higher disease burden. Socio-economic factors, assessed through the BIMD, strongly correlate with health impacts,

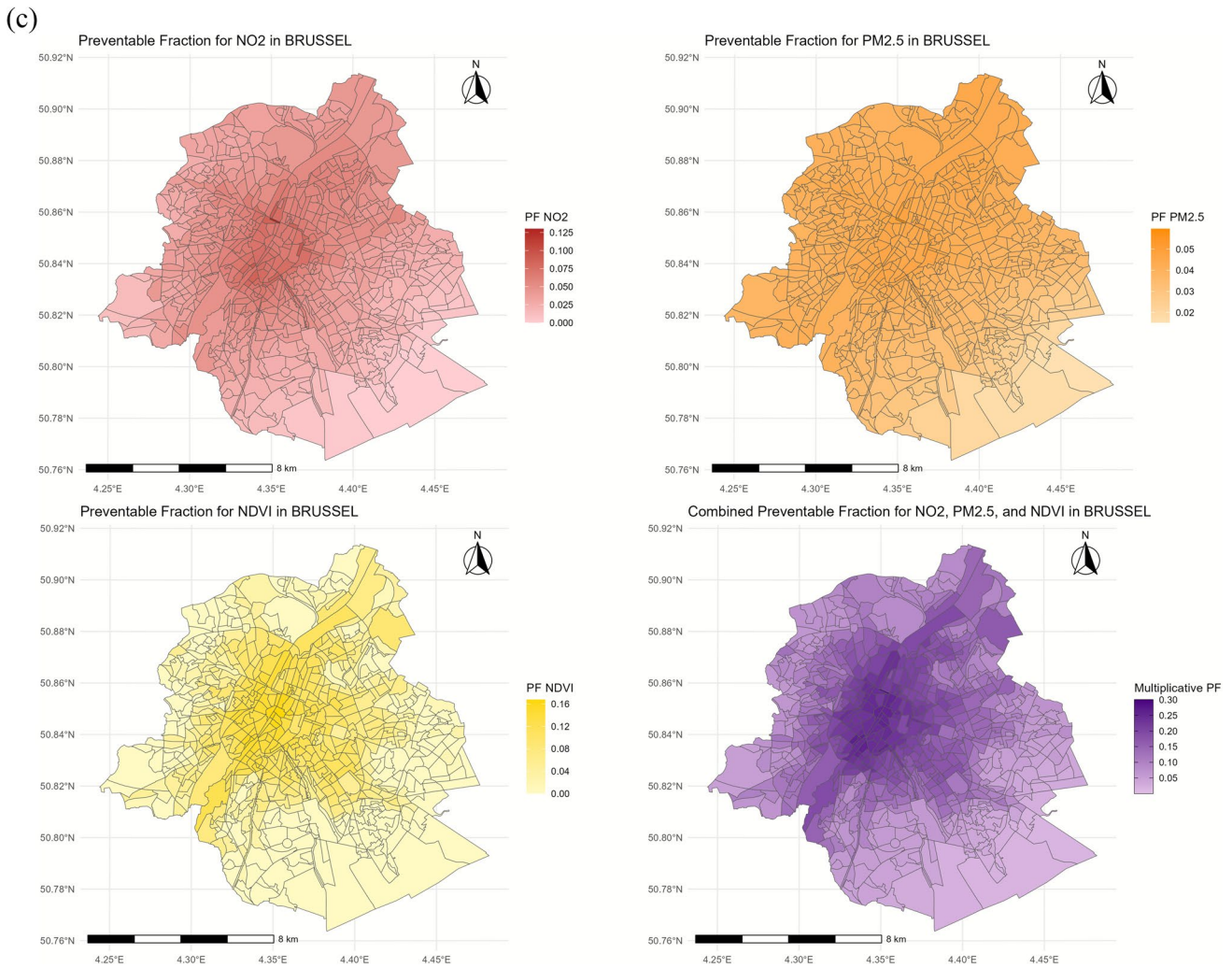


Fig. 2 (continued)

emphasising the complex interaction between environmental and social determinants of health.

The analysis of air pollution at measurement stations in Mechelen, Liège, and Brussels shows that relying solely on background concentrations, as implemented in the commonly used SHERPA tool, can lead to substantial underestimations of NO_2 levels from traffic, sometimes by over 50%. This could result in policy analyses underestimating the health burden and the mitigation potential of urban and transport planning-related interventions. In contrast, $PM_{2.5}$ concentrations from traffic are lower, with only modest underestimations at very busy traffic locations. However, for $PM_{2.5}$, not applying traffic-specific dose–response functions may be a source of underestimation of the traffic-related disease burden.

Our research, even from a purely mathematical perspective, highlights the impracticality of achieving WHO air pollution targets (5 µg/m^3 for $PM_{2.5}$ and 10 µg/m^3 for NO_2) in urban contexts without substantially reducing traffic-related emissions. Furthermore, the prevalent allocation of urban spaces to vehicles, often at the cost of green areas, exemplifies poor urban and transport planning from a health standpoint. This lack of green spaces negatively affects health outcomes. Mechanisms involved here are potential direct effects of green spaces such as mitigating mental stress, improving social interactions, and enhancing microbial diversity. In addition, a higher availability of green spaces increases physical activity and reduces urban heat islands (Yang et al. 2021). Insufficient green space not only contributes to the development of urban heat island effects but also adversely affects the physical activity levels of urban residents (Choi et al. 2022; Iungman et al. 2023; Wang et al. 2021).

Earlier studies focusing on Barcelona and Vienna found that 20% and 8% of mortality, respectively, could be prevented through urban and transport planning considering air pollution, green space availability, physical activity levels,

noise, and urban heat islands, with air pollution and physical activity being the most important factors (Khomenko et al. 2020; Mueller et al. 2017). However, these studies were based on now outdated recommendations of 40 µg/m^3 NO_2 and 10 µg/m^3 $PM_{2.5}$ exposure limits, and thus underestimate the mitigation impact under the new WHO guidelines of 10 µg/m^3 NO_2 and 5 µg/m^3 $PM_{2.5}$ exposure.

Strengths and limitations

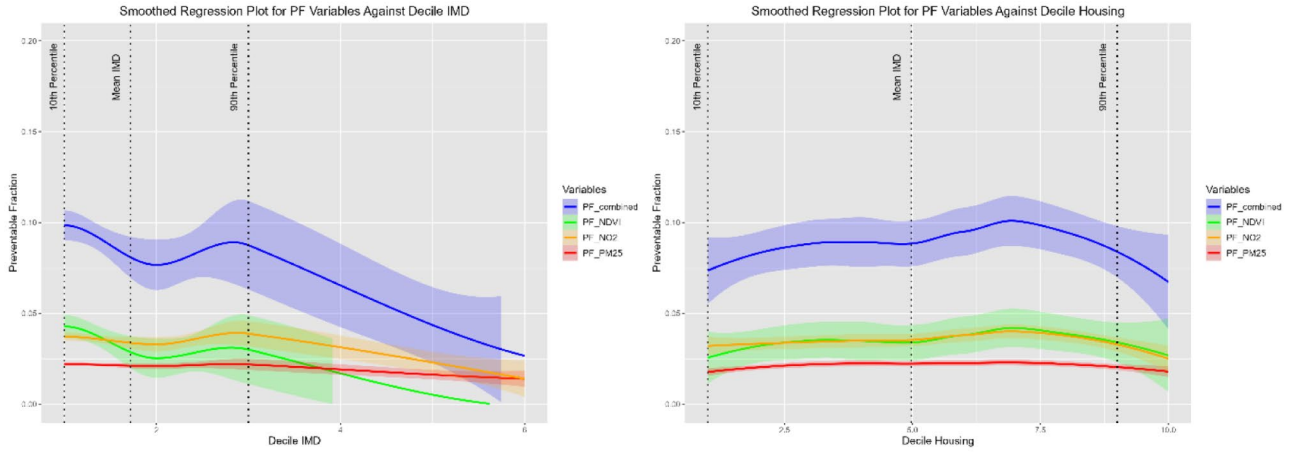
Our study has several strengths. The results align with the research objectives, offering clear insights into the (preventable) disease burden linked to urban and transport planning practices. We identified important gaps in air pollution source allocation methods that could enhance mitigation strategies. The underestimation of NO_2 levels from traffic highlights a crucial issue in current methodologies, which must be addressed to improve the accuracy of HIAs. Future research should focus on developing and validating techniques that more accurately quantify traffic-related pollution. Another strength of our study is its focus on the health burden of specific diseases, such as cardiovascular mortality, asthma, diabetes, and depression, rather than only assessing total mortality. A profound understanding of the impact on individual diseases is essential for effective mitigation.

Beyond these elements, this study advances previous HIA work in several important ways. It applies the framework to small and mid-sized Belgian cities such as Liège and Mechelen, which are rarely analysed in European research dominated by large metropolitan case studies. It also uses a high-spatial-resolution approach that enables neighbourhood-level assessment of preventable disease burden, providing more granular insights into intra-urban differences. By linking environmental exposures with socio-economic deprivation indicators, the study highlights inequalities in environmental health risks that remain invisible in aggregate analyses. Together, these advances provide a more policy-relevant perspective, helping to identify where and for whom urban and transport interventions could yield the largest health benefits.

Despite its strengths, however, our study has limitations, primarily due to data gaps and the lack of robust source allocation methods. This restricted our analysis to measuring the disease burden from traffic-related air pollution only at specific reference stations, rather than across entire cities, making it challenging to convert attributable factors to common indicators like disability-adjusted life years (DALYs). Key aspects such as the impact of physical inactivity and the urban environment on food consumption, safety, and general well-being are still missing for a complete understanding. Achieving zero-exposure concentrations is unrealistic, particularly due to natural particulates like soil dust and sea salt (in coastal areas), and traffic emissions contributing to secondary particulate matter. Therefore, we selected WHO target values for air pollution as the counterfactual scenario instead of zero exposure. However, the limit values of 5 µg/m^3, and particularly 10 µg/m^3, are not natural thresholds beyond which no progress can be made, as lower concentrations are observed in regions like Scandinavia. In 2015, the lowest observed annual values in Europe were 3.7 µg/m^3 for $PM_{2.5}$ and 3.5 µg/m^3 for NO_2 (Khomenko et al. 2021). In addition, the use of a two-pollutant model adjusted for $PM_{2.5}$ may attenuate the estimated NO_2 effect, as both pollutants share substantial variance in traffic-related exposures, including black carbon as a key fraction of $PM_{2.5}$. This approach provides a conservative estimate of the potential health gains from achieving the WHO NO_2 guideline, since part of the shared mixture effect is removed when both pollutants are modelled simultaneously. When both NO_2 and $PM_{2.5}$ are mutually adjusted, the resulting ERFs may therefore not fully capture the total traffic-related burden, while unadjusted models could overestimate it. In contrast, adjustment of $PM_{2.5}$ for NO_2 generally has a smaller influence, as $PM_{2.5}$ originates from a multitude of sources beyond traffic, including residential heating, industry, and agriculture. Overall, the reported PFs should be interpreted as conservative and prudent lower-bound estimates. Sensitivity analyses using the unadjusted NO_2 ERF are presented in Appendix IV, confirming that higher PFs are obtained when the $PM_{2.5}$ adjustment is omitted. The ERFs for both mortality and morbidity were derived from large European multi-cohort studies that almost always included comparable adjustments for key individual and socio-demographic factors such as age, smoking, body mass index, and neighbourhood income. These ERFs therefore represent population-weighted, broadly generalisable estimates for the European adult population. When applied at the city level, modest deviations may occur due to local demographic or health characteristics, but they provide a robust basis for comparing preventable mortality and morbidity attributable to air pollution across cities.

Further, we assume a sub-additive effect through the multiplicative PF, while some studies note the possibility of synergistic effects of unfavourable outcomes from combined exposure to suboptimal environmental conditions (Vandeninden et al. 2024). Lastly, The NDVI data used in this study correspond to the year 2016, whereas air pollution data were obtained for 2022, which introduces a temporal mismatch between the datasets. However, comparisons of other consistent land-cover datasets (e.g. CORINE Land Cover 2012 vs. 2018) indicate that land-use changes in Brussels, Liège, and Mechelen have been minimal over this period. It is therefore reasonable to assume that overall green space patterns remained largely stable between 2016 and 2022. While small local greening projects may not be captured,

(a)


Smoothed Regression Plot for PF Variables Against Decile IMD
Smoothed Regression Plot for PF Variables Against Decile Housing
10th Percentile
Mean IMD
90th Percentile
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Preventable Fraction
Decile IMD
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Variables
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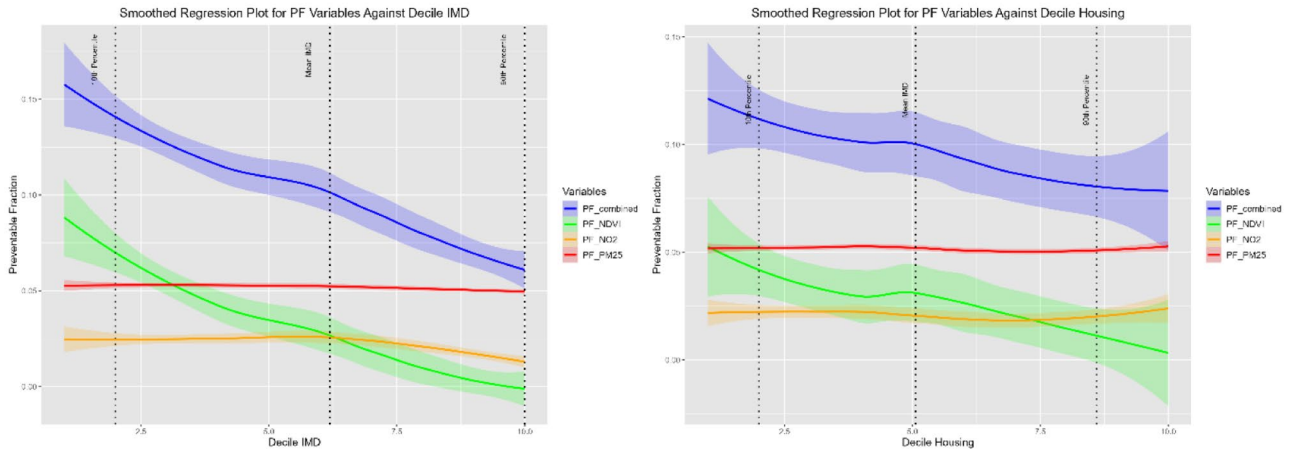
(b)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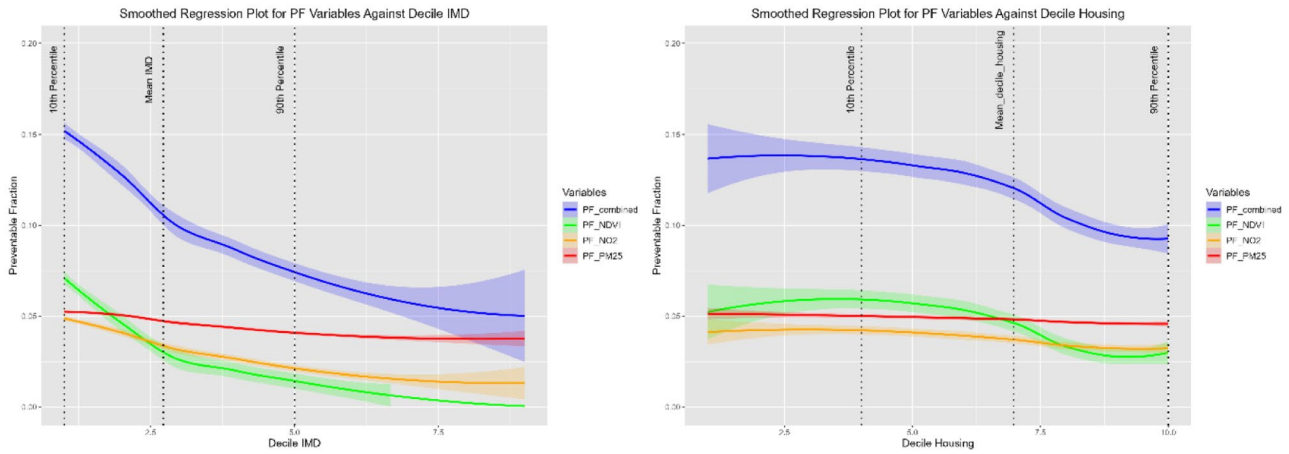
(c)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90th Percentile
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Preventable Fraction
Decile IMD
Decile Housing
Variables
PF_combined
PF_NDVI
PF_NO2
PF_PM25

◂**Fig. 3** The relationship between the preventable fraction of cardiovascular mortality (for green space, NO_2, $PM_{2.5}$, and the multiplicative PF) and the Belgian Index of Multiple Deprivation (BIMD) and the housing decile of the BIMD in **a** Liège, **b** Mechelen, and **c** Brussels through smoothed localised regression plots. Shaded areas surrounding the lines show 95% confidence intervals (CI)

this limitation is unlikely to affect the overall magnitude or direction of the results.

Implications for research and policy

The findings of this study have implications for both future research and policymaking in the realm of urban and transport planning. Firstly, the clear linkage between air pollution, lack of green space, and various health outcomes necessitates a re-evaluation of urban planning priorities. Policies aimed at reducing air pollution, particularly NO_2 and $PM_{2.5}$, and increasing green spaces in urban areas could significantly diminish the disease burden from asthma, diabetes, depression, and cardiovascular diseases. This is especially pertinent in densely populated urban centres, and adjacent suburbs, where the study identified the highest health risks, and where a mitigation of a lack of green space and reducing NO_2—the traffic-related component of air pollution—can result in strong health gains for the residing population. A recent meta-analysis (Kuss and Nicholas 2022) investigated the effect of mitigation measures on car use in cities and found that congestion charging, parking and traffic control, zones with limited traffic, investments in public transport and making public transport more affordable or free, and apps for sustainable mobility all result in reductions in car use of more than 10% each.

Considering the broader context of integrated urban and transport planning, the adoption of electric vehicles, while beneficial in certain aspects, including reducing NO_2 exposure, falls short in addressing key urban environmental challenges. In 2020, 50% of $PM_{2.5}$ in the EU originated from non-exhaust emissions such as brakes and tyres, with an increasing trend of $PM_{2.5}$ non-exhaust emissions and a decreasing trend of $PM_{2.5}$ exhaust emissions (European Topic Centre on Air pollution, Transport, Noise and Industrial Pollution, 2020). Transitioning to electric vehicles does not curtail the emission of non-tailpipe $PM_{2.5}$ particles, nor does it mitigate the extensive spatial demands of urban transportation infrastructure. This continued space consumption hinders the expansion of green areas, which is crucial for reducing urban heat island effects and fostering increased physical activity among city dwellers (Dávalos et al. 2016; Nieuwenhuijsen 2023).

This study underscores the need for advanced source allocation methods in environmental health research, emphasising that current approaches may underestimate NO_2 levels from traffic. Future research should prioritise the refinement of these techniques to more precisely measure traffic-related pollution and improve HIAs. Policymakers and urban planners can utilise these insights to develop more effective strategies for urban design and transport management, ultimately leading to healthier cities and communities.

More detailed discussion of policy relevance and effectiveness of selected urban and transport measures

Low emission zones (LEZs) remain among the most widely discussed urban traffic measures. Across European cities, LEZs result in varying reductions in NO_2 and PM concentrations. In some countries and LEZ zones, such as in the Netherlands, significant differences were not observed, while in other areas such as Germany, the LEZ typically reduced NO_2 and PM concentrations by a few percent. In London, the ULEZ reduced PM_{10} by 27% and NO_2 by 12.4% (Broster et al. 2025; Holman et al. (2015). In London, introduction of the ULEZ is estimated to result in a reduction in the incidence of childhood asthma of up to 8% in some neighbourhoods such as Camden and Kenningston and a 6% reduction in coronary heart disease, 5% reduction in low birth weight, and 2% reduction in diabetes in the same neighbourhoods (Holman et al. (2015). In Brussels, the LEZ reduced NO_2 by 30% in the highest-traffic routes, with projections suggesting an additional 24% decline by 2035 (Pelgrims et al. 2024). LEZs also tend to yield larger benefits in more deprived neighbourhoods, thus contributing to environmental equity (Pelgrims et al. 2024). However, LEZs primarily affect air pollution and traffic emissions and do not directly increase green space availability.

Similarly, congestion charging schemes achieve 10–20% reductions in traffic volumes and accompanied decreases in air pollution levels. It is estimated that in the inner city of Stockholm, there are 20–25 fewer premature deaths annually due to the congestion charging policy introduced in 2005. Public support for the policy rose sharply after it was implemented compared to 2005 when the policy was introduced. Further, congestion charging generates public revenue to reinvest in sustainable transport such as public transport, which could further result in indirect positive effects on health (Eliasson 2014). However, although effective for emission reduction, congestion charging has limited direct effects on urban greening, unless revenues are explicitly allocated to public space redesign.

Investments in public transport and active mobility deliver broader and longer-term benefits. Scenario analyses for the city of Barcelona show that replacing 40% of car trips by cycling and public transport could prevent around 76 premature deaths annually through combined effects of reduced air pollution and increased physical activity (Rojas-Rueda

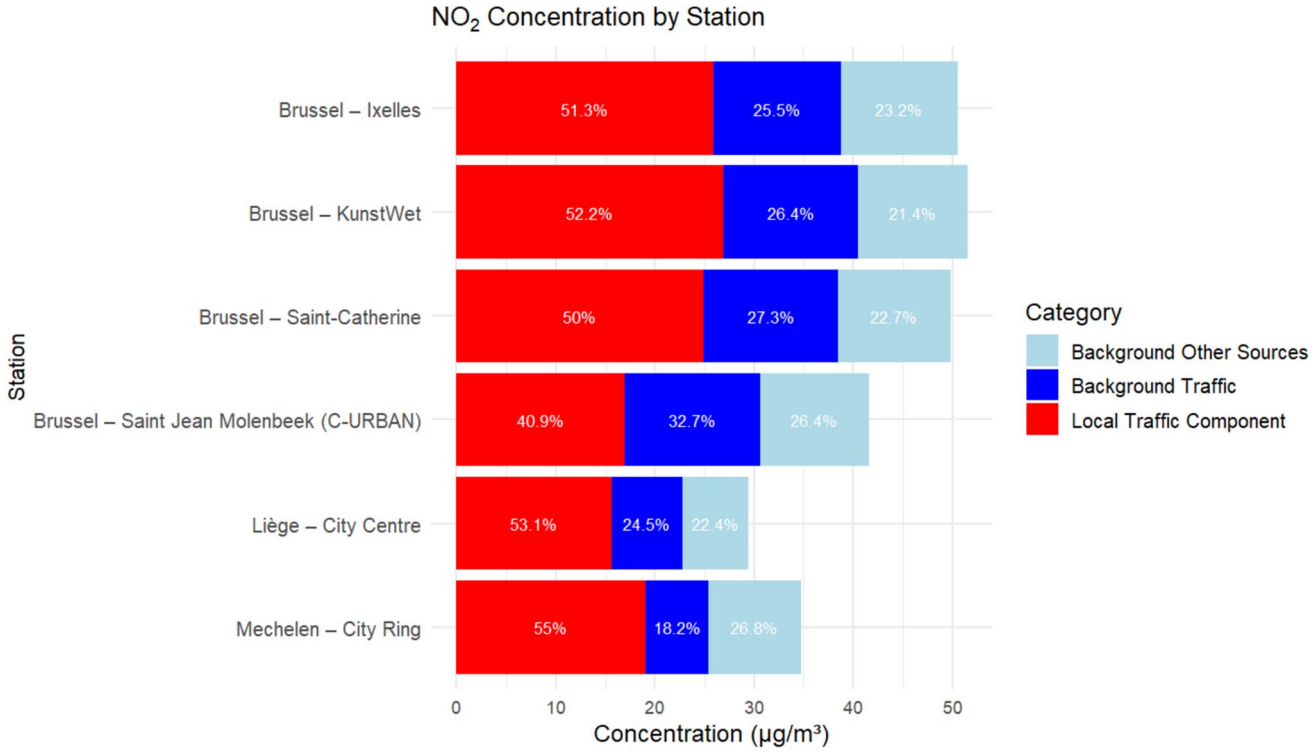

Fig. 4 Decomposition of total air pollution concentrations for NO_2 in three components: background other sources, background traffic, and local traffic component

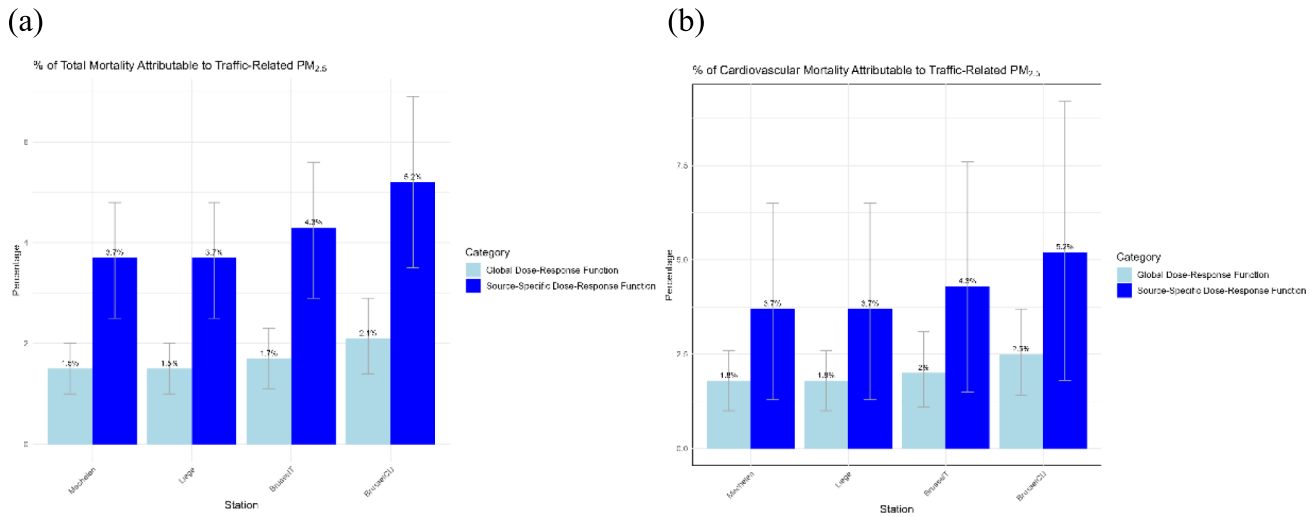

Fig. 5 Fraction of **a** total mortality and **b** cardiovascular mortality that is attributable to $PM_{2.5}$, using the global ERF versus using source-specific ERF for $PM_{2.5}$

et al. 2013). In cities such as Granada and Ljubljana, the implementation of integrated low-traffic and public transport improvement measures, including the closure of streets to private vehicles, renewal of bus fleets, and reorganisation of public transport routes, resulted in substantial air quality benefits, with reductions of up to 72% in local black carbon (BC) and 30% in PM_{10} concentrations (Pelgrims et al. 2024).

School Streets interventions consistently demonstrate localised air quality benefits and associated health improvements: specifically, a 23% decrease in NO_2 peak exposure

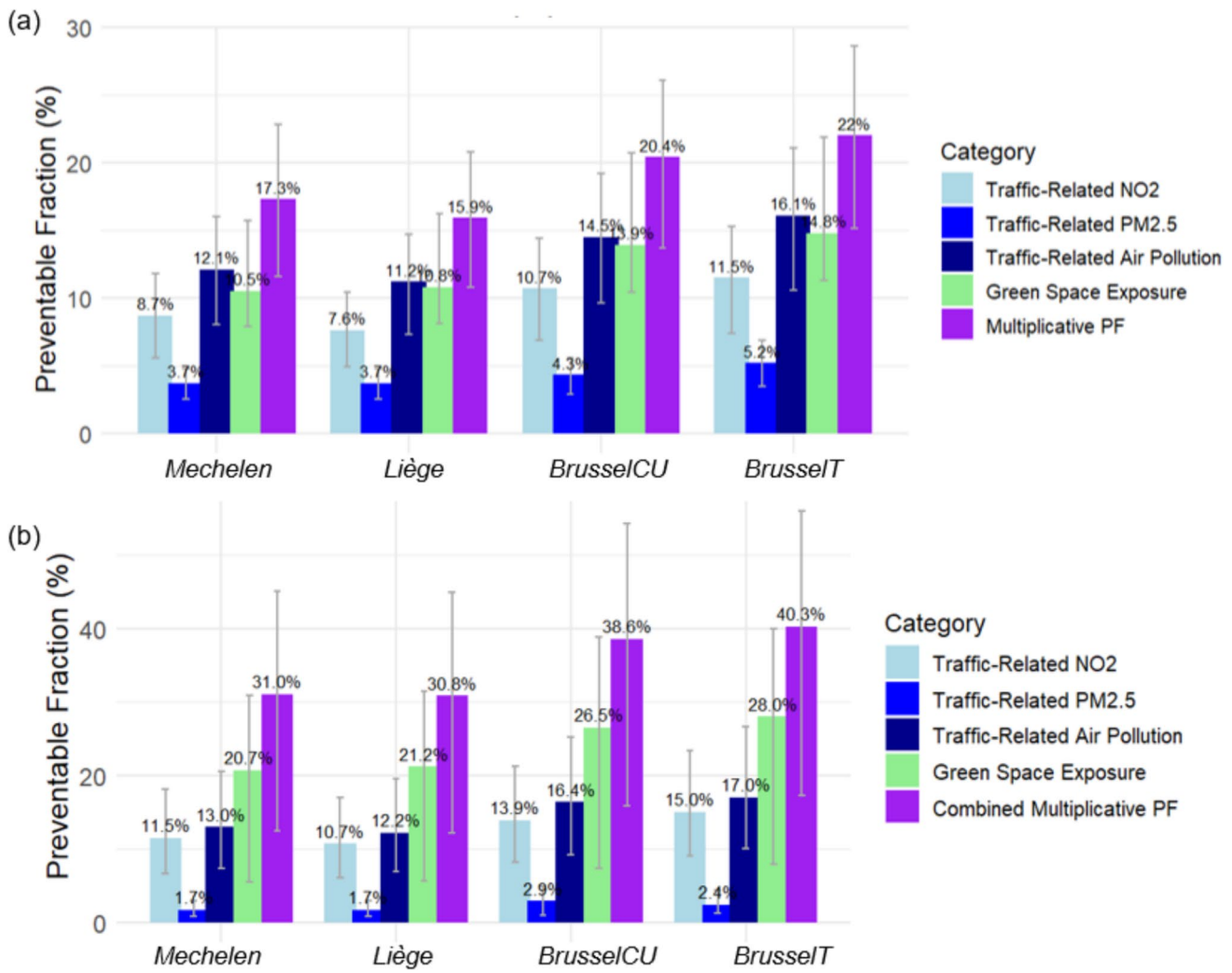

Fig. 6 Integrated assessment of preventable fraction (PF) of **a** total mortality and **b** diabetes prevalence from traffic-related air pollution and insufficient green space exposure in the analysed air pollution measurement stations

was observed outside of schools in London, alongside a 25% decrease in NO_2 concentration in Paris between 2021 and 2023, with this reduction in peak exposure in Flanders being associated with a decrease in airway inflammation among children near the school environment (Pelgrims et al. 2024).

Car-Free Sundays, as a form of local traffic restriction, have demonstrated significant public health benefits, achieving substantial localised NO_2 reductions ranging from 63% to 83% in parts of Brussels and 27% to 56% in Paris. This was associated with an estimated reduction in paediatric asthma incidence up to 34% in heavily trafficked areas of Brussels, if those local traffic restrictions were implemented permanently.

Further, there is considerable potential in finding space for nature and green areas in cities by converting redundant car parking:for example, reducing the number of parking spaces in a city or converting parking spaces to underground parking spaces (Croeser et al. 2022). This is associated with a range of physical and mental health benefits including reducing loneliness and obesity (Croeser et al. 2022). Urban green infrastructure, such as street trees, parks, and green roofs, provides benefits in both air quality and well-being (World Health Organization Regional Office for Europe 2017).

In the city of Paris, it was demonstrated that reducing lane capacity (for cars) by percentages up to 50% resulted in a modal shift towards active mobility without an overall increase in congestion (Natterer et al. 2025). This is presumably resulting in a wide range of health benefits, from both reduced pollution and increased physical activity. Reducing lane capacity also allows for the implementation of more urban green spaces.

The increasing number of cities that have successfully implemented these measures indicates that these strategies

are feasible and politically achievable under a range of local conditions. While implementation costs can vary, many interventions—such as LEZs, congestion charging, and School Streets—rely primarily on regulatory and organisational changes rather than large infrastructure investments. Moreover, measures that generate revenues (e.g., congestion charges) or co-benefits across multiple policy domains such as investments in public transport and active mobility (e.g., health, climate, equity, economy) tend to be cost-effective in the long term.

Conclusion

This study offers compelling evidence of the substantial impact that air pollution and lack of green space have on public health in urban settings. We have quantified the preventable fraction of the disease burden for asthma, diabetes, depression, and cardiovascular diseases that could be avoided by meeting WHO guidelines, directly linked to the environmental disease burden associated with urban and transport planning practices. Notably, the underestimation of NO_2 exposure attributable to traffic in current source allocation methods underscores a crucial area for methodological improvement. These findings serve as a clear call for the integration of health impact assessments in urban and transport planning, emphasising the need for enhanced green spaces and reduced air pollution to mitigate disease burden in urban populations. The study not only enriches the existing body of environmental health research but also provides actionable insights for policymakers and urban planners, stressing the urgency of adopting health-informed strategies in urban development to foster healthier, more sustainable communities.

Supplementary Information The online version contains supplementary material available at https://doi.org/10.1007/s10389-025-02660-5.

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Authorship contribution B.V., C.B., E.(M.)D. and B.D. were involved in the conceptualisation of the research and in developing the methodology. B.V. conducted literature research for writing the introduction and discussion section of the manuscript. B.V. conducted all analysis related to the research and also wrote the initial version of the main manuscript. Figure 1 was created by E.(M.).D. All other figures and tables, both in the main manuscript and in the supplementary materials, were created by B.V. All co-authors were involved in reviewing the manuscript and approved the final version of the manuscript.

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Data availability Most of the input data used for this research, including air pollution data from both models and measurement stations, MODIS satellite data for NDVI, socio-economic data from the Index of Multiple Deprivation, and exposure–response functions (ERFs) derived from existing literature and meta-analysis, are available as open-source or public data. The air pollution model data can be accessed at http://ftp.irceline.be/atmostreet/, while data from air pollution measurement stations are available at https://www.irceline.be/en/air-quality/measurements/monitoring-stations. MODIS satellite data for NDVI can be found at https://modis.gsfc.nasa.gov/data/dataprod/mod13.php, and information on the Belgian Index of Multiple Deprivation is available in its GitHub repository at https://github.com/bimd-project/bimd. Details on exposure–response functions (ERFs) can be obtained through the references cited in the scientific papers included in the manuscript.

Code availability R-code available upon request. Specific formulas used during the analysis are included in the main manuscript text.

Declarations

Ethics approval This study did not require ethical approval as it did not involve any of the following: direct or indirect interaction with human participants (via computer/internet, in clinical or other settings), the use of personal data of identifiable individuals, or the use of animals. All data used in this study were obtained from existing datasets, including environmental data and exposure–response functions (ERFs) from existing literature. No new data were generated, and there was no human participation in the study.

Consent to participate Not applicable.

Consent for publication Not applicable.

Conflict of interest The authors declare no competing interests.

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