Higher population density is associated with worse air quality and related health outcomes in Tāmaki Makaurau

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Abstract

Objectives: To explore associations between population density, air pollution concentrations, and related health outcomes in Tāmaki Makaurau Auckland, Aotearoa New Zealand.

Methods: Concentrations of nitrogen dioxide (NO_2) and fine particulate matter ($PM_{2.5}$) and associated health outcomes (premature mortality, cardiovascular and respiratory hospitalisations, and childhood asthma) were obtained from the Health and Air Pollution in New Zealand 2016 study. The possible mediating factors assessed were motor vehicle traffic, domestic fires, local industry, and green space.

Results: Higher population density was associated with higher concentrations of NO_2 (β: 0.18, $p < 0.01$) and $PM_{2.5}$ (β: 0.04, $p < 0.01$). Adverse health outcomes related to NO_2 and respiratory hospitalisations attributed to $PM_{2.5}$ exposure increased incrementally with the density of urban areas. The mediating factor with the strongest effect was motor vehicle traffic.

Conclusions: Higher population density in Auckland is associated with worse air quality and related health outcomes. Motor vehicle traffic is the most important source of pollution and is highest in the most densely populated parts of the city.

Implications for Public Health: Housing intensification in Auckland will likely increase ill health from air pollution unless steps are taken to reduce exposure to emissions from motor vehicles.

Key words: population density, air pollution, nitrogen dioxide, ambient particulate matter

Introduction

Ambient air pollution is the second largest risk factor for non-communicable diseases worldwide and is reported to be the greatest environmental threat to public health.[1] Compared to other countries, New Zealand has relatively good air quality.[2] However, long-term exposure to air pollution has adverse health effects, even at low levels.[3–5] The recent Health and Air Pollution in New Zealand 2016 (HAPINZ 3.0) study estimated that exposure to fine particulate matter ($PM_{2.5}$) and nitrogen dioxide (NO_2) caused over 3,300 deaths, 13,100 hospitalisations, 13,200 cases of childhood asthma, and 1.745 million restricted activity days in New Zealand in 2016.[2] Of these cases, 1118 deaths, 5,414 hospitalisations, 6134 cases of childhood asthma, and over 820,000 restricted activity days occurred in the Auckland region.

Urban development and transport planning decisions influence many urban exposures, including air pollution.[6] Over the past century, urban sprawl has been the dominant development pattern in cities worldwide.[7] Characterised by low-density housing and dependence on motor vehicles for transport, sprawl has led to many public health issues, ecological and air quality degradation, and has exacerbated global climate change.[7,8] Awareness of the negative effects of urban sprawl has led to calls for more compact development, which entails building more medium- to high-density housing within existing city limits.[9,10] If done well, compactness has many economic, environmental, and health benefits but may also pose some health risks unless steps are deliberately taken to create green, healthy cities.[10,11]

A review of urban form, air pollution, and health by Hankey and Marshall concluded that compact development has the potential to improve air quality but has mixed effects on air pollution-related health outcomes, as compactness can exacerbate concentration hotspots in densely populated areas.[12] Higher population density has been associated with poorer air quality in some locations, but not in others, suggesting that the relation between population density and

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air pollution exposure is context-dependent.[13] Sources of urban air pollution (e.g. motor vehicles) and factors that affect pollution exposure (e.g. the proximity of residential areas to major roads) mediate the association of interest.[13] The influence of these mediators differs between urban environments and forms and, in turn, affects the strength and direction of the relation between population density and air pollution exposure in different settings.[13] That being said, important and ubiquitous meditators, such as emissions from motor vehicles contribute to the fact that higher population density is associated with poorer air quality more commonly than not in the published literature to date.[13]

This study explores associations between population density, urban air pollution concentrations, and pollution-related health impacts in Tāmaki Makaurau Auckland (Auckland), Aotearoa New Zealand (NZ). We also examine variables that may explain the associations observed to shed light on actions to achieve healthy compact development in the future.

Methods

Study area

Tāmaki Makaurau Auckland is NZ's largest urban area. Officially the city includes an area of 4941 km^2 that contained a population of 1.78 million in June 2023.[14] However, the Auckland metropolitan area includes more than 95% of the regional population in a much smaller area (607 km^2).[15] The city has a monocentric urban form with high population density close to the city centre and, on the whole, suburban sprawl elsewhere.[15] The spatial unit applied throughout this study was the census area unit (CAU) from the 2013 NZ census. Area units in the Auckland region that contained offshore islands, marinas, and inlets were excluded from analyses as they typically had very small populations.

Auckland has relatively good air quality compared to cities globally.[2] This has been attributed to low levels of industrial activity and geographical distance from other countries, preventing spillover effects. Despite this, in 2016, 91.3% and 59.4% of the Auckland population were exposed to $PM_{2.5}$ and NO_2 concentrations above the 2021 World Health Organization guidelines for annual exposure, respectively. The main sources of air pollution in the region are motor vehicles and domestic fires.[2]

Health and Air Pollution in New Zealand 2016 study

HAPINZ 3.0 is the most recent and comprehensive assessment of the health impact and social costs of exposure to air pollution in NZ.[2] It is based on a retrospective cohort study that estimated the burden of disease attributed to long-term exposure to $PM_{2.5}$ and NO_2 by linking census, hospitalisation, mortality, and fine-scale air pollution data for all NZ adults between 2013 and 2016.[3] NZ-specific exposure-response functions were obtained from the cohort study and were applied to estimate the health burden and social costs attributable to $PM_{2.5}$ and NO_2 exposure in 2016. The methods used in the study were peer-reviewed extensively by international subject matter experts.

Data sources

For this study, the estimated population of Auckland as of 30 June 2016 at the CAU level was provided by Statistics NZ.[16] Population density was calculated by dividing each CAU's population by its total area in km^2. All CAUs that contained areas defined as rural by the Auckland Unitary Plan were described as "rural".[17] All remaining units were designated as "urban" and allocated into one of four categories with an equal number of CAUs based on population density.

Annual average air pollution concentrations in 2016 were estimated at the CAU level. The nitrogen dioxide (NO_2) concentrations used in this analysis were extracted from the HAPINZ 3.0 model.[18] Concentrations were modelled at 50 m spatial resolution using a vehicle emissions modelling tool.[19] NO_2 concentration estimates in CAUs included a roadside and background component. The roadside component was population-weighted by multiplying the sum of the population by the sum of concentrations at dwellings within 200 m of major roads. The background component was calculated for the remaining area using the same equation. The elements were combined to give the population-weighted concentration in each CAU. Vehicle emissions were the only source of NO_2 considered, as HAPINZ 3.0 reported that vehicles account for more than 90% of NO_2 emissions in the Auckland region.[16]

Fine particulate matter ($PM_{2.5}$) measured or estimated concentrations from monitored sites in the Auckland region were extracted from the HAPINZ 3.0 model.[18] At four sites where the monitoring site was active between 2015 and 2017, the measured three-year average $PM_{2.5}$ concentration was taken to avoid fluctuations in concentrations due to weather conditions, etc.[16] At ten monitored sites that only measured PM_{10} levels, the three-year average PM_{10} concentration was taken, and an empirical model was used to estimate $PM_{2.5}/PM_{10}$ ratios from the percentage of PM_{10} concentration attributed to biomass burning.[16,20]

$$PM_{2.5}/PM_{10}\ \text{ratio} = \%PM_{10}\ \text{biomass} \times 0.77 + 0.3 \quad (1)$$

$PM_{2.5}$ concentrations were then estimated by multiplying the calculated $PM_{2.5}/PM_{10}$ ratio by the measured three-year average PM_{10} concentration from the site. The measured and estimated $PM_{2.5}$ concentrations from 14 sites were then extrapolated to cover the Auckland region at the CAU level using inverse distance weighted interpolation.

Air pollution-related health outcomes in 2016 were estimated at the CAU scale using exposure-response functions and CAU-specific pollutant concentrations, population, and health outcome data. A detailed description of the methods used to estimate the number of health outcome cases attributable to NO_2 and $PM_{2.5}$ at the CAU level is available in Kuschel et al., 2022.[16] Health outcomes included premature mortality in adults aged 30 years and older and respiratory and cardiovascular hospitalisation in all ages attributable to NO_2 and $PM_{2.5}$ exposure. As in HAPINZ 3.0, we also included asthma prevalence and hospitalisation in children aged 18 years and under attributable to NO_2.

Annual average temperature, wind speed, and total annual rainfall data for 2016 were extracted from the National Institute of Water and Atmospheric Research's National Climate Database for NZ. Fourteen monitoring stations had data available for the Auckland region in 2016, but not all sites measured the variables of interest.[21] The raw data for each variable were spatially weighted across CAUs in the Auckland region using inverse distance weighted interpolation in QGIS version 3.28. The mean value of raster pixels within the boundaries was taken as the annual average value in each CAU. The distance to Auckland Central Business District (CBD) was calculated as the distance in kilometres from the centroid of each CAU.

Potential mediators were included in regression models to investigate their influence on the association between population density and air pollution concentrations. These included measures of motor vehicle traffic (road density, bus stop density, and traffic level) and green space (the proportion of total CAU area containing green areas and normalised difference vegetation index (NDVI)). Domestic fires and local industrial activity were also assessed for the association between population density and $PM_{2.5}$. The methods used to create these variables are outlined in the supplementary material.

Analysis methods

All statistical analyses were conducted using R version 4.3.0 software. The olsrr package was used to perform the ordinary least squares (OLS) regressions. The significance level was set at 5%, but statistical significance at the 1% level was also reported. The epitools package was used to calculate risk ratios of air pollution exposure-related health outcomes by density level.

We used OLS regression to investigate associations between population density and air pollution concentrations. Population density (PD) and NO_2 and $PM_{2.5}$ concentrations (pollutant) were converted to a log scale as the relationship between density and air pollution levels was non-linear in exploratory analyses. Temperature (TM), wind speed (WS), rainfall (RF), and distance to CBD (CBD) were included in all regressions as controls for the influence of meteorological factors and rurality, respectively. Equation 2 is the model used for the Auckland region regressions.

$$\log\text{pollutant} = \alpha + \log \beta_{PD} + \beta_{TM} + \beta_{WS} + \beta_{RF} + \beta_{CBD} \quad (2)$$

The regional analysis was conducted using all CAUs in the Auckland region. We altered the city boundaries to exclude rural areas as an additional check. This was done to investigate to what extent the urban-rural divide explained the association between population density and air pollution. Equation 3 is the model used for the Auckland urban regressions.

$$\log\text{pollutant} = \alpha + \log \beta_{PD} + \beta_{TM} + \beta_{WS} + \beta_{RF} \quad (3)$$

The analysis methods used for the mediator regressions are outlined in the supplementary material. Pearson's correlation coefficient was used to assess the strength and direction of associations between the proposed mediators and population density.

Results

Population density and air pollution concentrations

Neighbourhoods with higher population density generally had higher population-weighted NO_2 and ambient $PM_{2.5}$ concentrations. Density appeared to have a positive log-linear relationship with NO_2 concentrations, while the association with ambient $PM_{2.5}$ levels included a more obvious non-linear element (Figure 1).

Table 1 shows the coefficient of the variable of interest, log population density, hereafter referred to as the density coefficient, and the coefficients for the control variables: temperature, wind speed, rainfall, and distance to CBD. Estimates of density coefficients for NO_2 are substantially larger than those for $PM_{2.5}$, indicating that population density has a stronger association with NO_2 than $PM_{2.5}$ in Auckland. This can be attributed to the fact that local sources are more important in determining NO_2 concentrations due to rapid chemical conversion, while $PM_{2.5}$ is more persistent in ambient air, the multiplicity of sources contributing to urban $PM_{2.5}$ concentrations, or due to differences in measurement methods between pollutants.

The associations between population density and NO_2 are similar in urban areas of Auckland (β: 0.18, $p < 0.01$) and the Auckland region as a whole (β: 0.17, $p < 0.01$). This suggests that rurality does not significantly influence the association between population density and NO_2. In contrast, for $PM_{2.5}$, the regression coefficient for urban areas was less than half that for the Auckland region, which implies that other factors associated with rurality modified the association between population density and $PM_{2.5}$ concentrations.

Population density and air pollution-related health outcomes

The association between population density and health outcomes attributed to NO_2 and $PM_{2.5}$ exposure is shown in Table 2. As expected from the pattern of exposures, there was a continuous

Figure 1: Scatter plots of population-weighted NO_2 (left plot) and ambient $PM_{2.5}$ (right plot) concentrations by the population density of the CAU with loess smooth trendline fits. CAU: census area unit.

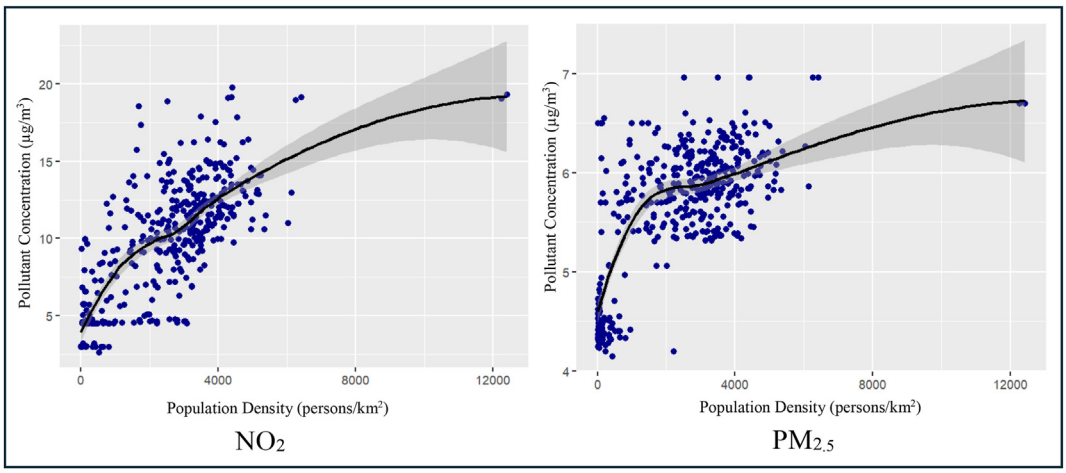

Table 1: OLS regressions assessing the association between population density and population-weighted NO_2 and ambient $PM_{2.5}$ concentrations in the Auckland region and urban areas of Auckland.

	Log NO_2		Log $PM_{2.5}$	
	(1)	(2)	(3)	(4)
	Auckland region	Auckland urban	Auckland region	Auckland urban
Log population density	0.168 (0.011)**	0.179 (0.017)**	0.035 (0.003)**	0.017 (0.004)**
Temperature	-0.036 (0.020)	-0.096 (0.021)**	-0.005 (0.005)	-0.012 (0.005)*
Wind speed	-0.046 (0.012)**	-0.087 (0.012)**	-0.002 (0.003)	-0.005 (0.003)
Rainfall	-0.072 (0.010)**	-0.108 (0.009)**	-0.023 (0.002)**	-0.028 (0.002)**
Distance to CBD	-0.004 (0.001)**		-0.001 (0.000)**	
R^2	0.750	0.530	0.729	0.451
F-statistic	238.037**	94.870**	212.548**	68.989**
CAU included	403	344	403	344

Values are presented as β-coefficient (standard error), rounded to 3 d.p. ** $p < 0.01$, * $p < 0.05$.

OLS: ordinary least squares; CBD: Central Business District; CAU: census area unit.

relationship between NO_2-related health outcomes and density, with the risk of developing disease increasing with the neighbourhood's population density. This effect was more pronounced for respiratory outcomes than cardiovascular hospitalisation or premature mortality. On the other hand, outcomes attributed to $PM_{2.5}$ exposure, except respiratory hospitalisation did not show a relationship with population density within urban areas. Rural areas had substantially less NO_2 and $PM_{2.5}$ exposure-related disease than the least dense urban areas.

Influence of potential mediators

Seeking to explain the mechanisms that lead to worse air quality and more air pollution-related disease in densely populated urban areas of Auckland, we investigated the influence of several mediator variables using OLS regressions of population density and air pollution concentrations (Supplementary Tables S1 and S2).

Both measures of green space, NDVI and the proportion of total CAU area containing green areas were inversely associated with population density, indicating that denser areas of the Auckland region had less green space (r: -0.59 and -0.84, respectively). When added to the regression model, both measures of green space were inversely associated with NO_2 and $PM_{2.5}$ concentrations and reduced the size of coefficients for population density.

Higher population density was associated with increased density of roads and public bus stops (r: 0.92 and 0.82, respectively). Based on this, we estimate that motor vehicle traffic increases accordingly with population density. Higher road and bus stop density were also associated with higher NO_2 and $PM_{2.5}$ concentrations in the regression outputs. For both pollutants, the coefficients were larger for road density than bus stop density, implying that motor vehicle traffic density had a greater influence on air pollution levels than bus stop density.

The level of traffic within a CAU was strongly associated with NO_2 concentrations. Area units within 200 m of a state highway or intersected by a major road tended to have higher levels of NO_2, while urban areas with low traffic levels differed little from rural areas. This suggests that the NO_2 concentration within a CAU is closely related to the proximity of major roads and state highways, not minor roads. It should be noted that this result is, to some extent, determined by the approach taken to estimate NO_2 exposure, which was based in part on traffic modelling.[2] Traffic level had a weaker influence on $PM_{2.5}$ concentrations. Although the coefficients incrementally increased with traffic level, these increases were marginal. This suggests the result could largely be explained by urban areas having higher background $PM_{2.5}$ concentrations than rural areas.

Controlling for road density, bus stop density, and traffic level caused the density coefficients to drastically reduce in size for both pollutants compared to the baseline regressions. For NO_2, the coefficient remained positive but decreased from 0.17 to 0.06, while the $PM_{2.5}$ coefficient turned negative. This indicates that motor vehicle traffic substantially contributes to higher concentrations of air pollutants in areas of higher population density.

Table 2: Relative risk of health outcomes attributed to NO_2 and $PM_{2.5}$ exposure by population density level of the CAU in the Auckland region. Urban 1 is used as the comparison for all relative risk calculations.

	Urban 4 (most dense 25% of urban CAUs)	Urban 3	Urban 2	Urban 1 (least dense 25% of urban CAUs)	Rural areas
NO_2 exposure—related health outcomes					
Premature mortality (30+ years)	1.14 (0.91—1.43)	**1.32 (1.06—1.64)**	1.05 (0.84—1.33)	1.00	**0.35 (0.22—0.56)**
Cardiovascular hospitalisation (all ages)	1.21 (0.98—1.50)	1.20 (0.97—1.49)	1.12 (0.90—1.39)	1.00	**0.43 (0.28—0.66)**
Respiratory hospitalisation (all ages)	**1.70 (1.52—1.90)**	**1.31 (1.17—1.48)**	1.10 (0.97—1.24)	1.00	**0.33 (0.25—0.43)**
Asthma prevalence (0—18 years)	**1.46 (1.36—1.58)**	**1.35 (1.25—1.45)**	**1.18 (1.09—1.28)**	1.00	**0.43 (0.37—0.51)**
Asthma hospitalisation (0—18 years)	**2.00 (1.50—2.68)**	**1.61 (1.19—2.18)**	1.19 (0.86—1.65)	1.00	**0.24 (0.10—0.55)**
$PM_{2.5}$ exposure—related health outcomes					
Premature mortality (30+ years)	0.78 (0.59—1.04)	0.97 (0.75—1.26)	0.88 (0.67—1.16)	1.00	**0.54 (0.35—0.83)**
Cardiovascular hospitalisation (all ages)	0.83 (0.69—1.01)	0.90 (0.75—1.07)	0.96 (0.81—1.15)	1.00	**0.69 (0.52—0.90)**
Respiratory hospitalisation (all ages)	**1.25 (1.04—1.51)**	1.04 (0.85—1.27)	0.97 (0.79—1.19)	1.00	**0.54 (0.38—0.77)**

Values are presented as relative risk (95% confidence intervals), rounded to 2 d.p. Risk ratios that were statistically significant at the 5% level are in bold text.

CAU: census area unit.

Population density was inversely associated with the proportion of $PM_{2.5}$ concentration attributed to domestic fires, suggesting that domestic fires become a less significant source of $PM_{2.5}$ as density increases (r: -0.41). Domestic fires were also inversely associated with $PM_{2.5}$ levels, indicating that other sources are dominant contributors at high concentrations. Similarly, on average, CAUs with local industrial activity had substantially lower population densities than CAUs without. When added to the regression model, local industrial activity was positively associated with $PM_{2.5}$, suggesting that industrial activity drives local concentration increases. Controlling for local industry and domestic fires increased the regression coefficients for population density from 0.35 to 0.39 for $PM_{2.5,}$ indicating that fireplaces and local industry, to a small extent, confound the association between population density and air pollution.

Discussion

We found that neighbourhoods with higher population densities in the Auckland region tend to experience poorer air quality and more air pollution-related disease. The relationship between population density and NO_2 was stronger and more consistent than that observed between density and $PM_{2.5}$. The difference in $PM_{2.5}$ levels between rural and urban neighbourhoods is more marked than the differences between urban neighbourhoods. Health outcomes related to NO_2 increased incrementally with the density of urban areas; this was also true for respiratory hospitalisations attributed to $PM_{2.5}$.

We conclude that motor vehicle traffic is the predominant factor contributing to poorer air quality in densely populated areas in Auckland. Green space may mediate the association between population density and air pollution as vegetation removes pollutants from ambient air, improving air quality. However, the effect of green space may be overstated here, as the measures used in this study are likely confounded by land use. It appears local industrial activity and domestic fires account for elevations in $PM_{2.5}$ concentration that are not related to population density.

Comparison with previous research

We are not aware of previous studies investigating the association between population density and NO_2 concentrations at the neighbourhood level. However, city comparisons have reported that higher population density is associated with higher NO_2 concentrations,[22–25] consistent with the findings of this study. The results for the association between population density and $PM_{2.5}$ concentration in Auckland are comparable to those of three similar studies at the neighbourhood level in Shanghai and Wuhan, China, and the United States.[26–28] The main factor contributing to the association observed between population density and air pollution concentration in these studies was the concentration of motor vehicle traffic in densely populated areas. It is worth noting that there is evidence of a population scale effect on the association between population density and air pollution,[22,28] with initial evidence suggesting this is primarily driven by motor vehicle use by the commuting population. This affects the comparison of results between cities of different sizes.

A systematic review by Carnegie et al. (2022) found that higher population density was associated with higher disease incidence and mortality rates for various cancers and cardiovascular and respiratory diseases. Higher density levels were also associated with a higher incidence of childhood asthma.[29] The authors proposed that this was mediated at least in part by air pollution exposure.

Strengths and limitations

To our knowledge, this analysis is the first to investigate associations between population density and air pollution in NZ at the level of CAUs, which is particularly relevant for policymakers. Our study uncovered some of the mechanisms contributing to the density effect on pollution. Further research should seek more robust and finer-grained measures of exposure to pollution, motor vehicle traffic, and green space.

The analysis utilises an air pollution concentration dataset from a longitudinal study investigating the health effects of long-term exposure to pollution in NZ. NO_2 concentrations were modelled using a peer-reviewed method based on vehicle emissions data. $PM_{2.5}$ concentrations were obtained from a small number of monitoring stations dispersed across the Auckland region, resulting in low spatial coverage of $PM_{2.5}$ concentrations. We applied inverse distance weighted interpolation to increase the spread of $PM_{2.5}$ concentration across the region. Future studies could directly measure $PM_{2.5}$ concentration at finer scales using methods such as satellite estimation.

In this analysis, population density and air pollution levels are based on residential location, which assumes that people stay in their neighbourhood and do not travel for employment, education, or other purposes. However, census data show that most people leave their immediate neighbourhood area daily for work or education in the Auckland region.[30] Therefore, the population density of areas within cities changes throughout the day, and individuals are exposed to different pollution levels at various locations they visit. To address the fact that people are not only exposed to air pollution at home, some recent studies have used smartphone data to track changes in the population density of areas of cities throughout the day due to commuting and assigned exposure data to individuals based on the areas in which individuals spend time.[27,31] Further studies should consider such measures of exposure to address the aforementioned limitation.

Implications for public health

Although air quality is generally improving across NZ, steep population growth, a similar projected increase in motor vehicle use, slow uptake of electric cars, and the rise of light trucks and other heavy passenger vehicles will tend to increase traffic-related air pollution exposure in Auckland in the future.

There is ample reason to believe that greater urban density benefits health and the environment. Compact cities contribute to climate change mitigation by reducing greenhouse gas emissions from household energy use and transport and aid climate adaptation by reducing the amount of developed land in areas prone to climate risk.[10] Density can also have well-being benefits, such as increased neighbourhood satisfaction, improved access to amenities and social infrastructure, and increased frequency of physical activity compared to sprawling neighbourhoods.[11,32,33] However, these benefits will not be fully realised unless steps are taken to disrupt the association that now exists between housing intensification and exposure to motor vehicle traffic. This may be achieved through a combination of transport reforms to provide alternatives to private motor vehicles, penalties for pollution, traffic restrictions, and street design.

Conclusion

Higher population density is associated with higher NO_2 and $PM_{2.5}$ concentrations and higher air pollution-related disease in the Auckland region. This is due mainly to the concentration of motor vehicle use in densely populated areas. To enhance health, compact development must be accompanied by policies to reduce motor vehicle emissions, particularly in densely populated areas.

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Ethics approval

Ethical approval is not applicable for this article.

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Conflicts of interest

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

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Appendix A Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.anzjph.2024.100213.