Highlights

1. Roadside and indoor exposures to traffic noise separately affected mortality risk
2. Consistent impact of indoor traffic noise when joining two exposures in a model
3. UHI, light pollution, and greenness modified the impacts of roadside noise

Indoor and roadside exposures to traffic noise and cardiovascular mortality and the role of urban environmental stressors across a high-rise, high-density environment: a case study in Hong Kong

Pin Wang[1], Haipei Xiang[2], Mengdi Guo[3], Yuan Shi[4], Ka Chun Chong[5], Jianxiang Huang[6], Hung Chak Ho[7]

[1] Department of Environmental Health Sciences, Yale School of Public Health, New Haven, Connecticut, USA

[2] Graham School of Continuing Liberal and Professional Studies, The University of Chicago, Illinois, USA

[3] School of Architecture, Tianjin University, Tianjin, China

[4] Department of Geography and Planning, University of Liverpool, Liverpool, United Kingdom

[5] Jockey Club School of Public Health and Primary Care, The Chinese University of Hong Kong, Hong Kong SAR, China

[6] Department of Urban Planning and Design, The University of Hong Kong, Hong Kong SAR, China

[7] Department of Anaesthesiology, School of Clinical Medicine, The University of Hong Kong, Hong Kong SAR, China

Corresponding author:

Name: Hung Chak Ho

Email: hcho21@hku.hk

Phone: (852)51146540

Abstract

Traffic noise is a common factor associated with elevated cardiovascular risks. Compact environment and building morphology in a high-rise, high-density city can magnify traffic noise in various locations (e.g., roadside and indoor environments). However, no studies have investigated how indoor and roadside traffic noise separately and jointly influenced cardiovascular risks across a compact environment. Thus, this study applied negative binomial generalized linear mixed models to estimate associations between roadside/indoor exposures to traffic noise and cardiovascular mortality (2006–2015). Stratified analyses were applied to evaluate effect modifications from canopy-layer and surface-layer urban heat island (UHI), night-time light, and greenness. Our results showed that each 1 dB increase in roadside and indoor exposures to traffic noise was positively associated with 1.183 (95% confidence interval (CI) 1.068–1.311) and 1.046 (95% CI 1.012–1.081) times the risk of all cardiovascular deaths, respectively. When both types of noise were included in the models, the associations of roadside exposure were attenuated and became non-significant whereas the associations of indoor exposure remained consistent. Stronger and significant associations between roadside exposure and cardiovascular mortality were observed in areas with more intensive UHI, more severe light pollution, and lower average greenness. Robust impacts regarding indoor exposure to traffic noise were found after roadside exposure was accounted for. In conclusion, urban environment stressors could act synergistically on their adverse effects and enlarge noise impacts on cardiovascular outcomes. Built environment interventions should be applied to minimize indoor exposure to traffic noise to reduce cardiovascular risks, despite complex urban morphology.

Keywords: noise exposure; traffic noise; cardiovascular mortality; high-rise environment; high-density city; urban environmental stressors

1 Introduction

Compact environment and building morphology in a high-rise, high-density city (e.g., Hong Kong) can magnify traffic noise in various locations (e.g., roadside and indoor environment). For example, compared to Europe, Hong Kong has a similar proportion of the population exposed to a high level of day-evening-night noise level (>70 dB), a much higher proportion exposed to a medium level of 60–64 dB, and a much lower proportion to a lower level (<55 dB).[1] Building morphology in the high-rise, high-density environment across Hong Kong has also resulted in a high level of indoor exposure to traffic noise, even though many flats are on high floors. Particularly, narrow urban corridors and compact environmental settings may result in a greater noise level on higher floors. At the same time, dense road networks, urban corridors, and high-rise buildings can induce problems from other urban environmental stressors. For example, a high-rise, high-density environment can reduce urban ventilation resulting in a high level of heat and air pollution across the urbanized area. Urban corridors can also trap air pollutants. Furthermore, extensive coverage of high-rise buildings across a dense neighborhood can lead to environmental inequality, such as a lack of green space and clusters of night-time light sources. These unfavorable environmental stressors have been found to be risk factors of multiple health indicators. However, despite the fact that a high-rise, high-density environment can be a problematic urban morphology affecting traffic noise and health, no studies have investigated how traffic noise in various locations separately influenced cardiovascular risks across such a compact environment. Furthermore, no studies investigated whether urban environmental stressors co-affect the noise influences on cardiovascular risks. As cardiovascular risks are key factors affecting the lifespan and healthy living of urban citizens, understanding the co-impacts of these environmental determinants is thus necessary. The findings can be useful for modifying guidelines and protocols of environmental management and urban planning in order to maintain the livability and sustainability of a neighborhood based on better environmental hygiene with manageable health impacts.

1.1. Noise and cardiovascular risk

Recent years have seen a growing body of evidence associating environmental noise, a ubiquitous exposure globally, with adverse impacts on both physical and mental health.[2-7] It has been estimated by the World Health Organization (WHO) that 1.0–1.6 million disability-adjusted life years (an indicator that combines both morbidity and mortality) have been lost every year from traffic-related noise in western European countries, among which sleep disturbance and annoyance have constituted most of the burden.[8] It has also been estimated by the WHO that about 40% of the European population is exposed to ambient road traffic noise over 55 A-weighted decibels (dB(A)); 20% is exposed to levels exceeding 65 dB(A) during the daytime; and over 30% is exposed to levels over 55 dB(A) at night.[9] Both associated with motor vehicle traffic, noise and air pollution can play an interactive and synergistic role on human health.[10]

Extensive studies have been conducted to explore how different sources of traffic noise were associated with various cardiovascular outcomes.[3,11-17] Using all types of transportation noise, including road, rail, and aircraft, Viennau reported a pooled relative risk (RR) for ischemic heart disease (IHD) of 1.06 per 10 dB increase in noise exposure, starting at 50 dB.[12] Similarly, Babisch found a pooled estimated risk for IHD of 1.08 associated with a 10 dB(A) increase in weighted day-night road traffic noise level within the exposure range of 52–77 dB(A).[13] Significant associations have also been found for other cardiovascular outcomes such as hypertension,[14] myocardial infarction,[15] and stroke.[16] However, substantial discrepancies were found across not only individual studies but also pooled studies, [12-17] primarily driven by varied locations, study designs, noise type, and disease outcomes. The link between noise exposure and negative cardiovascular impacts has been demonstrated not only in adults but also among children and infants.[4,18,19]

1.3. Indoor exposure to traffic noise and health risk

Previously outdoor exposure to traffic noise has been primarily examined and few studies have employed indoor exposure to traffic noise as the indicator to investigate its health impact. A study in Germany reported that the associations of cognitive function with indoor exposure to traffic noise were stronger than those with outdoor exposure.[20] Long-term exposure to indoor noise from road traffic has been found to be significantly associated

with elevated systolic blood pressure.[21] Moreover, stronger associations between traffic noise and both systolic and diastolic blood pressure have been observed among those who reported higher indoor noise annoyance,[22] suggesting an interaction between outdoor and indoor exposures to traffic noise. However, evidence on how different indoor exposure levels from traffic noise influence cardiovascular health other than blood pressure is lacking.

1.4. Potential impacts of urban environmental stressors

In addition to traffic noise, urban heat, greenness, night light pollution, and air pollution are also influenced by urbanization, and these environmental exposures are collectively and interactively responsible for urban discomfort caused by anthropogenic actions to a certain degree. For example, researchers have found that excess heat,[23] insufficient access to urban green space,[24] and outdoor light at night[25] were all associated with cardiovascular illnesses. Moreover, green space could alleviate urban heat island (UHI)[26] and reduce annoyance to road traffic noise.[27] an experimental biological study has shown the interactive influences between light and noise pollution on species abundance.[28] Additionally, increase in urban greenness has also been found to be associated with a lower indoor noise level, although the association was not statistically significant.[29] However, current knowledge on whether the impact of traffic noise on cardiovascular diseases could be modified by these environmental factors is largely unknown.

1.5. Objectives

This study aimed to quantify the impact of both roadside and indoor exposures to traffic noise on cardiovascular mortality and its attributable burden in Hong Kong, a highly densely populated metropolis, and to investigate whether these associations were modified by sociodemographic and other environmental parameters including UHI, urban greenness, night light, and air pollution.

2 Data and methods

2.1 Data

2.1.1 Mortality data

Datasets including all known deaths in Hong Kong during 2006–2015 were obtained from the Hong Kong Census and Statistics Department (HKCSD). We retrieved the information on the cause of death (ICD-10 coded), sex, age, and tertiary planning unit (TPU, a geographic reference system demarcated for the purpose of town planning) of residence for each decedent. The analysis was only performed among those aged 15 years or older. The following cardiovascular deaths of common causes were extracted: all cardiovascular deaths (ICD-10: I00–I99), deaths due to IHD (ICD-10: I20–I25), acute myocardial infarction (AMI) (ICD-10: I21), heart failure (ICD-10: I50), and cerebrovascular disease (ICD-10: I60–I69). We next calculated the death count for each stratum of TPU, sex, and 5-year age group (15–19, 20–24, …, 85+) combination during the same study period. Other TPU-level demographic and socioeconomic information, including population from the census in 2011, percentage of the population over 15 years with tertiary education attainment, percentage of the population over 15 who were unmarried, and median monthly household income, was also acquired from the HKCSD.

2.1.2 Traffic noise

Two noise indicators were used in this study: roadside exposure and indoor exposure. Particularly, roadside exposure to traffic noise represented the outdoor exposure directly caused by noise from traffic during the day and night, whereas indoor exposure to traffic noise represents indoor exposure in a high-density city that is mostly affected by the indoor-outdoor exchange of noise due to road traffic. Previous studies have also indicated that indoor and roadside exposures to traffic noise were not directly correlated due to the complex mechanism of indoor-outdoor exchange,[30] especially in a high-rise, high-density urban environment. Therefore, indoor exposure to traffic noise cannot be estimated based on linear regression, and it usually requires an engineering-based modeling software to simulate the noise level in different conditions and scenarios for better accuracy.

Roadside exposure to traffic noise was estimated based on the road network in Hong Kong for the year 2011. Particularly, from the iB1000 digital topographic map from Hong Kong Lands Department[31] we first obtained the center lines of all roads, based on which we then calculated the annual averages of road traffic noise level for the daytime period (7:00–23:00) and night-time period (23:00–7:00) using an established algorithm.[32] Day-night-weighted noise exposure was estimated based on daytime and night-time noise exposure.[33] The input variables for calculation include: 1) annual average hourly road traffic volume and the breakdown of heavy vehicles, calculated from the hourly total traffic volume and heavy vehicle volumes of counting stations from the annual average daily traffic data of the two years,[34] using Kriging method;[35] 2) average traffic speed of the roads obtained from Google Maps Platform (https://cloud.google.com/maps-platform) and traffic speed map of Hong Kong (https://data.gov.hk/en-data/dataset/hk-td-sm_1-traffic-speed-map); 3) gradient of the road was calculated from the topography data of terrain.[31]

Indoor exposure to traffic noise was estimated based on CadnaA, a common software that has been widely used to simulate indoor noise influenced by road traffic in high-rise, high-density cities such as Wuhan, China.[36] The window locations of representative urban households from the FAMILY Cohort[37] and Birth Cohort,[38] two comprehensive public health datasets that cover a majority of Hong Kong's urban neighborhoods, were first estimated. Particularly, a sum of 8,158 3D geo-coordinates of the window of the living room or bedroom was retrieved, of which 6,358 were from apartments of FAMILY Cohort participants and 1,800 from Birth Cohort participants. Then the indoor noise exposure of each household was simulated with input of road traffic noise levels of the year 2009 and 2011, household window locations, and 3D built environment information such as building, topography, and road centerlines. Particularly, CadnaA simulated the noise based on 3D coordinates of receiver points which were located 1m away from all identified window locations in the perpendicular direction to the building façade, with the use of territory-wide building data, topographical information, as well as the road centerlines with annual averaged road traffic noise levels noted above. For modeling with CadnaA, Hong Kong was re-grouped to 237 urban units to improve the calculation capacity. We also assembled a 300m outer buffer for the built environment and traffic data for noise simulation within each urban unit. Detailed methods of estimation have been noted in Guo *et al*.[38]

2.1.3 Urban heat island

This study used two types of UHI indicators: canopy-layer UHI and surface-layer UHI.

Canopy-layer UHI is a weather phenomenon related to spatial variability of air temperature at the canopy level (2m above ground), which is formed due to the disparity of microclimate patterns and urban ventilation.[39] Particularly, air temperature at the canopy level is the temperature that a person is usually exposed to at the ground level. Because of the diurnal temperature range caused by the diffusion of longwave radiation, the spatial pattern of daytime and night-time canopy-layer UHI can be entirely different.[40] Previous studies have found that daytime and night-time canopy-layer UHI can both affect health risks.[41] We estimated air temperatures at the canopy level during the summer (May–August) from a land use regression model based on 224 urban morphometric predictors and local weather information during 2013–2016. The model performance (R^2 of 0.6–0.7) showed that the estimation of air temperature was accurate. The methodological details have been noted in our previous study[42] and this estimated dataset has also been used in other local environmental studies.[43]

Surface-layer UHI was represented by spatial variations of land surface temperature (LST) caused by land and material emissivity, which also shows a strong impact on health risk.[44] Spatial variation of LST across Hong Kong was estimated from Landsat Thematic Mapper TM 5 on March 25, 2010 using an improved urban emissivity model based on the sky view factor.[45]

2.1.4 Night-time light

Night-time light pollution is an environmental phenomenon caused by excessive light emissivity from urban areas. Previous studies have found that light pollution increases the risk of IHD.[46] In this study, spatial variability of light emissivity for measuring night-time light pollution across Hong Kong was estimated based on a radiance map retrieved from a cloud-free Visible Infrared Imaging Radiometer Suite (VIIRS) image (750m resolution).

2.1.5 Greenness

Greenness was evaluated by the normalized difference vegetation index (NDVI). NDVI is a spectral index derived by the infrared and red bands with a range between -1 and 1, with a higher value representing a higher level of the greenness of a pixel. We first created an NDVI map based on a resampled IKONOS multispectral image with 15m resolution. Both the mean and standard deviation (SD) of NDVI were used in this study. Particularly, mean NDVI represents the average greenness across a TPU whereas the SD of NDVI represents the variability of greenness over a TPU which is also related to the mixed level of greenery and impervious surface.

Data on canopy-layer and surface-layer UHI, night-time light, and mean and variability of greenness were all aggregated at the TPU level for further analysis.

2.1.6 Air pollution

Particulate matter ($PM_{2.5}$) data were estimated based on land use regression, a common method for modeling spatial variation of air pollution across the urban area.[47] For the Hong Kong's model, it was a hybrid result of land use regression and field measurements operated by the Hong Kong Environmental Protection Department with 365 potential geospatial predictors from previous studies.[47] The data were originally in 10m spatial resolution and were spatially averaged based on the TPU boundary to obtain the measures relevant to this current study.

2.2 Main models

To test whether there was spatial autocorrelation among deaths across TPUs, we first calculated global Moran's I by extracting the cardiovascular mortality count and coordinates for each TPU. The observed Moran's I was 0.03 (p<0.01), indicating that the spatial distribution of all cardiovascular deaths was almost completely random. Therefore, we proceeded without consideration of spatial models.

We initially explored the separate association of roadside and indoor exposures to traffic noise with all cardiovascular deaths using Poisson generalized additive mixed models

adjusting for overdispersion. We found a linear relationship with the outcome for both variables, and thus we subsequently regressed mortality counts for all five cardiovascular diseases for each TPU, sex, and age group on TPU-level sociodemographic and environmental predictors using negative binomial generalized linear mixed models with a random intercept by TPU:

$$Ln\left(E\left(D_{ijk}\right)\right) = \beta_0 + P_i + Q_i + S_i + \text{factor}(sex) + \text{factor}(age\ group) + Pop_{ijk}$$

where D_{ijk} and Pop_{ijk} are the death count and population, respectively, for the *i*th TPU, *j*th sex, and *k*th age group during 2006–2015; P_i represents the index for TPU-level roadside or indoor noise, Q_i represents other environmental predictors including daytime and night-time canopy-layer UHI, surface-layer UHI, averaged NDVI, SD of NDVI, night-time light, or $PM_{2.5}$; S_i,, which was included in all models, denotes the combination of TPU-level sociodemographic factors, including population density, percentage of the unmarried population, percentage of the population that attained tertiary education, and median monthly household income. TPU-level population was incorporated in the models as an offset. All results are reported as an RR of per 1 dB increase in the roadside or indoor exposure to traffic noise with a corresponding 95% confidence interval (CI). We first conducted a univariate analysis using only roadside or indoor traffic noise and sociodemographic variables to assess the standalone association of different sources of noise with cardiovascular outcomes. Bivariate models were subsequently constructed including both roadside and indoor noise to examine possible added effects of indoor traffic noise exposure. We further controlled for potential confounding effects by UHI, greenness, night-time light, and $PM_{2.5}$ after checking the Pearson correlations between these environmental factors.

We next calculated the yearly number of cardiovascular deaths in Hong Kong that can be avoided per each 1 dB decrease in the noise of both sources using the following equations:

$$RR_i = e^{\beta_i}$$

$$D_{attr} = \frac{RR_i - 1}{RR_i} \times Population \times ASMR_{CVD}$$

where RR_i is the RR for an increase of 1 dB in the noise level; β_i represents the estimated exposure-response function from the univariate models; D_{attr} stands for the yearly number of avoidable deaths in Hong Kong; $ASMR_{CVD}$ is the age-standardized mortality rate due to cardiovascular diseases. We used the 2021 mid-year population[48] and calculated the sum of avoidable mortality for diseases of the heart and cerebrovascular diseases classified by the Hong Kong Department of Health.[49]

2.3 Effect modification

We fit separate stratified models by sex, age group (<65 and ≥65 years), TPU-level monthly household income (<50th (~3,000 USD) and ≥50th percentile, similarly hereinafter), daytime canopy-layer UHI, night-time canopy-layer UHI, surface-layer UHI, averaged NDVI, SD of NDVI, night-time light, and $PM_{2.5}$ to test if there was effect modification of the association between traffic noise and cardiovascular deaths by these parameters. The statistical significance of the differences between estimates of different groups was tested by calculating the 95% CI as $(\hat{Q}_1 - \hat{Q}_2) \pm 1.96\sqrt{(\widehat{SE}_1)^2 + (\widehat{SE}_2)^2}$, where $\hat{Q}_1$ and $\hat{Q}_2$ are the estimates for different groups, and $\widehat{SE}_1$ and $\widehat{SE}_2$ are their respective standard errors.[50] All stratified analyses were conducted in bivariate models with all cardiovascular deaths as the outcome and no environmental variables other than traffic noise exposures were included.

2.4 Sensitivity analysis

To examine model robustness, we substituted day-night weighted roadside exposure to traffic noise with daytime and night-time roadside exposures to traffic noise separately to assess whether day-night traffic noise fluctuation modified the observed associations. We ran this test for both the main models and the effect modification analysis.

All analyses were performed using R software version 4.0.2, with the *ape* package for spatial autocorrelation test and *glmmTMB* package for regression analysis.

3 Results

In the current study 209 TPUs covering the entire Hong Kong territory were included. There were in total 101,939 deaths due to cardiovascular diseases during 2006–2015, in which 53,062 (52.1%) were male, 87,050 (85.4%) were those aged 65 years or above. Regarding the cause of deaths, 41,856 (41.1%) were IHD (among which 18,337 (18.0%) were AMI), 21,081 (20.1%) were cerebrovascular disease, and 8,014 (7.9%) were heart failure. The descriptive summary for all environmental factors included in this study is shown in Table 1, and the Pearson correlation coefficients between these variables are shown in Table 2. There was a negligible correlation between the roadside and indoor noise variables. Daytime and night-time canopy-layer UHI, surface-layer UHI, and average NDVI were strongly correlated with each other, while night-time light and SD of NDVI were moderately correlated with other variables. Therefore, we included all environmental variables other than noise separately in the following regression analysis to avoid multi-collinearity.

The observed associations are shown with univariate (only roadside or indoor exposure to traffic noise was included) and bivariate (both roadside and indoor exposures to traffic noise were simultaneously included) models with sociodemographic variables controlled for (Figure 1). In the unadjusted univariate models (without other environmental covariates), a 1 dB increase in roadside exposure to traffic noise was found to be significantly and positively associated with all cardiovascular outcomes, with an increased RR of 1.183 (95% CI 1.068–1.311), 1.182 (95% CI 1.064–1.313), 1.234 (95% CI 1.112–1.369), 1.248(95% CI 1.128–1.381), and 1.220 (95% CI 1.094–1.360) for all cardiovascular deaths, and deaths due to IHD, AMI, heart failure, and cerebrovascular diseases, respectively. After the inclusion of five different types of environmental predictors, constantly significant associations with roadside exposure to traffic noise were observed for all types of cardiovascular deaths. Indoor exposure to traffic noise alone was significantly and positively associated with all outcomes except for heart failure. Each 1 dB increase in indoor traffic noise was found to be associated with an RR of 1.046 (95% CI 1.011–1.081), 1.044 (95% CI 1.010–1.078), 1.037 (95% CI 1.006–1.070), and 1.042 (95% CI 1.008–1.077) for all cardiovascular mortality, deaths due to IHD, AMI, and cerebrovascular diseases, respectively.

When roadside and indoor exposures to traffic noise were incorporated in the models at the same time (bivariate models), the associations between roadside exposure to traffic noise and all outcomes were considerably attenuated and became non-significant or borderline significant. However, despite the adjustment of roadside exposure to traffic noise, only negligible changes were observed for both the magnitude and significance of all associations with indoor traffic noise exposure.

The age-standardized mortality rates for diseases of the heart and cerebrovascular diseases in 2020 in Hong Kong were 34.0 and 16.6 per 100,000 population, respectively.[49] Accordingly, we estimated that a yearly total of 580 (95% CI 240–890) and 163 (95% CI 43–280) deaths due to cardiovascular diseases could be avoided with each 1 dB decrease within the range of 71.7–80.4 dB and 45.5–78.3 dB in the roadside and indoor exposure level, respectively.

We then stratified our analysis (with both roadside and indoor exposures to traffic noise included in the models) by sociodemographic and other environmental parameters (Figure 2). A significantly stronger association for indoor noise was observed among those aged younger than 65 than among those aged 65 or above. Monthly household income showed contradictory effect modification of the associations for roadside and indoor exposures to traffic noise, although the differences did not reach statistical significance. In addition, although non-significant associations were found between all cardiovascular deaths and roadside exposure to traffic noise in the main analysis (bivariate models), a stronger and statistically significant association was found for people living in TPUs with higher daytime and night-time canopy-layer UHI, higher surface-layer UHI, brighter night-time light, and lower average NDVI.

The results of sensitivity analysis and their comparisons with the results of the main models are presented in Figures S1–S5 in the Supplementary Information. The associations of all types of cardiovascular deaths with daytime and night-time noise were found to be largely consistent with the associations with day-night weighted noise (Figure S1). Using different types of roadside traffic noise exposure showed trivial influences on the effect modifications by all investigated factors (Figures S2–S5).

4 Discussion

4.1 Interpretations of results

The current study found significantly positive standalone associations between roadside/indoor exposures to traffic noise and cardiovascular mortality. However, in the models with roadside and indoor exposures both included, the associations with roadside exposure to traffic noise became non-significant or borderline significant, whereas the associations with indoor exposure to traffic noise remained robust. Stronger and significant associations between cardiovascular mortality and roadside exposure to traffic noise were observed for people living in areas with higher canopy-layer and surface-layer UHI, more severe night-time light pollution, and lower average NDVI.

Various pathophysiological pathways have been established for the cardiovascular damage induced by noise,[7,51] among which sleep disturbance has been considered to be the most influential non-auditory effect.[7,8] Nocturnal autonomic arousals induced by noise could cause reduced sleep quality or sleep of insufficient length through changes in sleep structure,[52] and may compromise cardiovascular performance during the following wake period.[7] On the other hand, chronic noise-annoyance-associated increased release of stress hormones, including cortisol and catecholamines, may result in impaired arterial endothelial function.[53] The vascular dysfunction may subsequently introduce a series of presentations, including increases in blood pressure, heart rate, and blood lipids, and could eventually raise the risk of exacerbated cardiovascular diseases.[54]

The solid link between environmental noise and cardiovascular diseases has been ascertained by a large body of epidemiological evidence,[3,10,51,55] among which the majority contributed significantly to the associations for noise of traffic sources including road, railway, and aircraft,[12-16,55] although existing meta-analyses have shown significant heterogeneity across studies.[12-15] We found a remarkably stronger relationship between roadside traffic noise and cardiovascular mortality than most previous studies did.[12-16] However, the reported traffic noise exposure in most literature included in these meta-analyses ranged from 40 to 70 dB,[13,14] notably lower than that in our study (Table 1), although our noise estimates were largely consistent with those published by previous analysis in Hong Kong.[56] A study in Taiwan also reported a strong effect of 2.23 (odds

ratio) for all cardiovascular diseases per 5 dB increase in traffic noise.[57] In addition, most previous studies used either a modeled threshold or a range of exposure and suggested a non-linear association between traffic noise and cardiovascular outcomes. This relationship has also been explicitly indicated by a meta-analysis, although there were significant discrepancies between individual studies.[12] Another study in Sweden also found a considerably stronger effect when the association was estimated for the highest noise category.[58] The strong association with noise exposure between 72 and 80 dB in our study indicates the steep increase in the risk above the usual noise range seen in other studies.

Although a previous field study reported that indoor noise level increased with the outdoor noise level in children's bedroom and main room particularly during the daytime,[59] we did not observe a similar pattern between the noise of these two sources (Table 2), neither did another study.[21] We found a weaker yet more robust association between indoor exposure to traffic noise and cardiovascular mortality except for deaths due to heart failure. In addition, the inclusion of indoor noise in the models substantially dampened the association for roadside exposure to traffic noise whereas the inclusion of roadside noise barely changed the magnitude of the association for indoor exposure to traffic noise. This result suggests a consistent and independent effect of indoor exposure to traffic noise on cardiovascular deaths even after the impacts of roadside noise were accounted for. This may be a particular scenario in high-rise, high-density cities, considering previous studies regarding effects from indoor exposure to traffic noise were conducted in cities with comparably low-rise, low-density settings.

There has been some evidence of the positive association between indoor exposure to traffic noise and blood pressure, with a stronger and more apparent association with systolic blood pressure.[21,22,60] Currently the relationship between indoor exposure of traffic noise and other cardiovascular outcomes is largely unknown. Two other studies investigating the impact of both outdoor and indoor exposures to noise on cognitive function and diabetes mellitus reported that indoor noise was found to be significantly associated with the outcomes, and the estimates using indoor traffic noise exposure were broadly more precise.[20,36] Traffic noise in and around the home during night-time could be a major determinant for worse sleep quality and a higher stress level, particularly for those who sleep with open windows or whose bedroom faces the main road, and thus its related

pathophysiological responses advocate our largely consistent findings across model specifications. Furthermore, studies have shown that noise had a more pronounced adverse impact on endothelial function for subjects who had previously been exposed to noise.[53] This further elucidates an elevated cardiovascular risk for most of the population who were first exposed to mixed types of traffic noise during the daytime and then to residential noise during the night.

Despite the attenuated and non-significant association of roadside traffic noise after the inclusion of indoor traffic noise observed in the main analysis, our study revealed that unfavorable urban characteristics, including more intensive UHI, a higher level of night-time light, and lower average NDVI, were noticeable factors that significantly associated a higher level of outdoor traffic noise with a higher risk of cardiovascular deaths. This is the first comprehensive study evaluating the interactive effects of common urban environmental variables on cardiovascular health. The independent impacts of these stressors on cardiovascular outcomes have been reported elsewhere.[24,25,61] Furthermore, synchronous interrelationships between these factors[26-29] would complicate their associations with health and challenge the multi-dimensional mitigation of these coupling parameters.[62] A recent study in Hong Kong incorporated night-time traffic noise when estimating the impacts of night light on coronary heart diseases.[25] Although no interaction assessment was administered, the authors found the synergetic presence of higher levels of light and noise.[25] Our findings urge further assessment of health risks to consider the possible coalescent effects of urban environmental indicators.

4.2 Planning recommendations

Based on the results above, several planning recommendations are suggested.

1) Further built environment interventions to reduce indoor exposure to traffic noise are needed. Specifically, as both traffic noise and UHI can be influenced by air ventilation, how to improve urban ventilation corridors across compact environments is essential. One question of future planning guidelines is the balance between the volume of urban corridors and high-rise building complexes to maintain both air ventilation and the number of housing units across the neighborhood. For future studies, simulation of built environment

based on computational fluid dynamics (CFD) with generative urban design protocols is recommended as it can identify the best solutions generically.

2) As our results indicated that lower average NDVI could modify noise-related health issue and greenness has commonly been identified to be a key determinant to mitigate noise level, further improvement of urban greenery is thus necessary. To target the built environment characteristics of a high-rise, high-density city, instead of only improving greenery along the roadside, vertical greening may also be important as it can increase total green percentage across the neighborhood environment to reduce noise exposure. Specifically, terrace gardens could be an option because ground-level land use, especially in Hong Kong, is often restricted by existing building complex and road network. Unless large-scale urban renewal, it is almost impossible to considerably increase the percentage of greenery across urbanized areas. Furthermore, as mixed land use across a high-rise, high-density environment is the key determinant to maintaining urban vibrancy. Balance between urbanized areas and green space is always another factor counter-influencing environmental mitigation. Thus, one alternative is to well use the potential of the volumetric design of a high-rise, high-density environment. To further improve planning protocols, future tests should focus on the comparison of vegetation characteristics (e.g., roughness, size, types) in order to simulate the best composition that can mitigate noise across large high-density building complexes at the same time while not creating heavy load on high-rise building facades.

3) Urban design should cooperate with environmental strategies that can reduce night-time brightness, as night-time light can induce interactive effects on cardiovascular mortality. Based on these facts, future urban design across a high-rise, high-density environment should be focused on mixed-use development, as this planning strategy 1) will reduce urban-rural disparity across the entire city, 2) can increase the proportion of public spaces with more greenery and better ventilation across neighborhoods, and 3) can minimize the total brightness of a neighborhood caused by a high concentration particular land uses associated with high-rise buildings (e.g., commercial land, residential land).

4.3 Limitations

Potential limitations of the present study need to be acknowledged. First, it was unachievable to conduct individual exposure assessment for this ecological study using aggregated data, resulting in possible misclassification bias. Specifically, our environmental predictors had varied spatial resolutions and were all averaged at the TPU level. However, given the compact territory of Hong Kong and the use of small-area geographically referenced units, this bias could be reduced significantly. Second, the environmental indicators in this study were not estimated for the same period due to data availability. The assumption of unchanged exposure level at the TPU level during the study period could introduce risk misestimation. Third, individual data on previous health conditions were not available, and thus there could be uncontrolled residual confounding of the reported associations due to the exclusion of this information.

5 Conclusions

Strong and positive associations were observed between day-night weighted roadside traffic noise of 72-80 dB and cardiovascular deaths. The impact of indoor traffic noise was found to be more robust and consistent. Disadvantageous urban environmental conditions, including intensive UHI, night light pollution, and insufficient access to green space, were associated with a significant and higher risk of cardiovascular mortality due to excess roadside traffic noise. More intelligent urban design relaxing the cooperative impacts of multiple environmental stressors, particularly in a highly densely populated city with a compact high-rise environment, is desired to promote cardiovascular health.

Contributors

H.C.H. and P.W. contributed to the conceptualization. P.W. contributed to the literature search. P.W., H.C.H., H.X., M.G., and Y.S. contributed to the data curation, P.W., H.C.H., H.X., K.C.C., and J.H. contributed to the methodology, P.W. contributed to the investigation, visualization, and writing original draft. H.C.H. contributed to the administration and supervision. H.C.H., H.X., M.G., Y.S., K.C.C., and J.H. contributed to the writing review and editing.

Declaration of interests

The authors declare that there is no conflict of interest.

Acknowledgements

This work was supported by Natural Science Foundation of Guangdong Province [grant number: 2021A1515012571]. I want to thank my mentor, the late Prof. William B. Goggins, without whom this project would never have been possible. I wholeheartedly appreciate your guidance along my academic journey all these years, and I sincerely hope I have made you proud.

References

1. Brown AL, Lam KC, van Kamp I. Quantification of the exposure and effects of road traffic noise in a dense Asian city: a comparison with western cities. *Environ Health* 2015; **14**: 22.

2. Recio A, Linares C, Banegas JR, Diaz J. Road traffic noise effects on cardiovascular, respiratory, and metabolic health: An integrative model of biological mechanisms. *Environ Res* 2016; **146**: 359-70.

3. Kempen EV, Casas M, Pershagen G, Foraster M. WHO environmental noise guidelines for the European region: A systematic review on environmental noise and cardiovascular and metabolic effects: A summary. *Int J Environ Res Public Health* 2018; **15**(2): 379.

4. Stansfeld S, Clark C. Health effects of noise exposure in children. *Curr Environ Health Rep* 2015; **2**(2): 171-8.

5. Hegewald J, Schubert M, Freiberg A, et al. Traffic noise and mental health: A systematic review and meta-analysis. *Int J Environ Res Public Health* 2020; **17**(17): 6175.

6. Paul KC, Haan M, Mayeda ER, Ritz BR. Ambient air pollution, noise, and late-life cognitive decline and dementia risk. *Annu Rev Public Health* 2019; **40**: 203-20.

7. Munzel T, Gori T, Babisch W, Basner M. Cardiovascular effects of environmental noise exposure. *Eur Heart J* 2014; **35**(13): 829-36.

8. WHO Regional Office for Europe. Burden of disease from environmental noise - Quantification of healthy life years lost in Europe. Copenhagen, Denmark, 2011.

9. World Health Organization Regional Office for Europe. Data and statistics. https://www.euro.who.int/en/health-topics/environment-and-health/noise/data-and-statistics (accessed 20 Mar 2021).

10. Munzel T, Sorensen M, Gori T, et al. Environmental stressors and cardio-metabolic disease: part I-epidemiologic evidence supporting a role for noise and air pollution and effects of mitigation strategies. *Eur Heart J* 2017; **38**(8): 550-6.

11. Halonen JI, Hansell AL, Gulliver J, et al. Road traffic noise is associated with increased cardiovascular morbidity and mortality and all-cause mortality in London. *Eur Heart J* 2015; **36**(39): 2653-61.

12. Vienneau D, Schindler C, Perez L, Probst-Hensch N, Roosli M. The relationship between transportation noise exposure and ischemic heart disease: A meta-analysis. *Environ Res* 2015; **138**: 372-80.
13. Babisch W. Updated exposure-response relationship between road traffic noise and coronary heart diseases: A meta-analysis. *Noise Health* 2014; **16**(68): 1-9.
14. Dzhambov AM, Dimitrova DD. Residential road traffic noise as a risk factor for hypertension in adults: Systematic review and meta-analysis of analytic studies published in the period 2011-2017. *Environ Pollut* 2018; **240**: 306-18.
15. Khosravipour M, Khanlari P. The association between road traffic noise and myocardial infarction: A systematic review and meta-analysis. *Sci Total Environ* 2020; **731**: 139226.
16. Dzhambov AM, Dimitrova DD. Exposure-response relationship between traffic noise and the risk of stroke: a systematic review with meta-analysis. *Arh Hig Rada Toksikol* 2016; **67**(2): 136-51.
17. Rabiei H, Ramezanifar S, Hassanipour S, Gharari N. Investigating the effects of occupational and environmental noise on cardiovascular diseases: a systematic review and meta-analysis. *Environ Sci Pollut Res Int* 2021; **28**(44): 62012-29.
18. van Kempen E, van Kamp I, Fischer P, et al. Noise exposure and children's blood pressure and heart rate: the RANCH project. *Occup Environ Med* 2006; **63**(9): 632-9.
19. Wachman EM, Lahav A. The effects of noise on preterm infants in the NICU. *Arch Dis Child Fetal Neonatal Ed* 2011; **96**(4): F305-9.
20. Tzivian L, Soppa V, Winkler A, et al. The role of depressive symptoms within the association of long-term exposure to indoor and outdoor traffic noise and cognitive function - Results from the Heinz Nixdorf Recall study. *Int J Hyg Environ Health* 2020; **230**: 113570.
21. Foraster M, Kunzli N, Aguilera I, et al. High blood pressure and long-term exposure to indoor noise and air pollution from road traffic. *Environ Health Perspect* 2014; **122**(11): 1193-200.
22. Lee PJ, Park SH, Jeong JH, Choung T, Kim KY. Association between transportation noise and blood pressure in adults living in multi-storey residential buildings. *Environ Int* 2019; **132**: 105101.

598 23. Sun Z, Chen C, Xu D, Li T. Effects of ambient temperature on myocardial infarction:
599 A systematic review and meta-analysis. *Environ Pollut* 2018; **241**: 1106-14.
600 24. Chen H, Burnett RT, Bai L, et al. Residential greenness and cardiovascular disease
601 incidence, readmission, and mortality. *Environ Health Perspect* 2020; **128**(8): 87005.
602 25. Sun S, Cao W, Ge Y, et al. Outdoor light at night and risk of coronary heart disease
603 among older adults: a prospective cohort study. *Eur Heart J* 2021; **42**(8): 822-30.
604 26. Zhang XX, Wu PF, Chen B. Relationship between vegetation greenness and urban heat
605 island effect in Beijing City of China. Procedia Environ Sci; 2010. p. 1438-50.
606 27. Schaffer B, Brink M, Schlatter F, Vienneau D, Wunderli JM. Residential green is
607 associated with reduced annoyance to road traffic and railway noise but increased
608 annoyance to aircraft noise exposure. *Environ Int* 2020; **143**: 105885.
609 28. McMahon TA, Rohr JR, Bernal XE. Light and noise pollution interact to disrupt
610 interspecific interactions. *Ecology* 2017; **98**(5): 1290-9.
611 29. Mueller W, Steinle S, Parkka J, et al. Urban greenspace and the indoor environment:
612 Pathways to health via indoor particulate matter, noise, and road noise annoyance. *Environ*
613 *Res* 2020; **180**: 108850.
614 30. Locher B, Piquerez A, Habermacher M, et al. Differences between outdoor and indoor
615 sound levels for open, tilted, and closed windows. *Int J Env Res Pub He* 2018; **15**(1): 149.
616 31. Lands Department of the Government of the Hong Kong Special Administrative
617 Region. Digital map. 2021. https://www.landsd.gov.hk/en/survey-
618 mapping/mapping/multi-scale-topographic-mapping/digital-map.html (accessed 10 Jul
619 2021).
620 32. Environmental Protection Department of the Government of the Hong Kong Special
621 Administrative Region. Road traffic noise impact assessment under the environmental
622 impact assessment ordinance. Hong Kong; 2010.
623 33. World Health Organization Regional Office for Europe. Environmental noise
624 guidelines for the European region. Copenhagen; 2018. p. 160.
625 34. Transport Department of the Government of the Hong Kong Special Administrative
626 Region. The annual traffic census 2011. Hong Kong; 2012.

627 35. Harman BI, Koseoglu H, Yigit CO. Performance evaluation of IDW, Kriging and
628 multiquadric interpolation methods in producing noise mapping: A case study at the city
629 of Isparta, Turkey. *Appl Acoust* 2016; **112**: 147-57.
630 36. Ohlwein S, Hennig F, Lucht S, et al. Indoor and outdoor road traffic noise and incident
631 diabetes mellitus: Results from a longitudinal German cohort study. *Environ Epidemiol*
632 2019; **3**(1): e037.
633 37. Leung GM, Ni MY, Wong PT, et al. Cohort profile: FAMILY cohort. *Int J Epidemiol*
634 2017; **46**(2): e1.
635 38. Guo M, Huang J, Yuxuan Ni M. 3D built-environment attributes and household road
636 traffic noise exposure in Hong Kong. *IOP Conf Ser Earth Environ Sci* 2019; **329**: 012012.
637 39. Williamson T, Erell E. The implications for building ventilation of the spatial and
638 temporal variability of air temperature in the urban canopy layer. *Int J Vent* 2008; **7**(1): 23-
639 35.
640 40. Wang Y, Li YG, Xue Y, Martilli A, Shen J, Chan PW. City-scale morphological
641 influence on diurnal urban air temperature. *Build Environ* 2020; **169**: 106527.
642 41. Ho HC, Knudby A, Walker BB, Henderson SB. Delineation of spatial variability in the
643 temperature-mortality relationship on extremely hot days in greater Vancouver, Canada.
644 *Environ Health Perspect* 2017; **125**(1): 66-75.
645 42. Shi Y, Katzschner L, Ng E. Modelling the fine-scale spatiotemporal pattern of urban
646 heat island effect using land use regression approach in a megacity. *Sci Total Environ* 2018;
647 **618**: 891-904.
648 43. Ho HC, Fong KNK, Chan TC, Shi Y. The associations between social, built and
649 geophysical environment and age-specific dementia mortality among older adults in a high-
650 density Asian city. *Int J Health Geogr* 2020; **19**(1): 53.
651 44. Wong MS, Peng F, Zou B, Shi WZ, Wilson GJ. Spatially analyzing the inequity of the
652 Hong Kong urban heat island by sociodemographic characteristics. *Int J Environ Res*
653 *Public Health* 2016; **13**(3): 317.
654 45. Yang JX, Wong MS, Menenti M, et al. Development of an improved urban emissivity
655 model based on sky view factor for retrieving effective emissivity and surface temperature
656 over urban areas. *ISPRS J Photogramm Remote Sens* 2016; **122**: 30-40.

46. Munzel T, Hahad O, Daiber A. The dark side of nocturnal light pollution. Outdoor light at night increases risk of coronary heart disease. *Eur Heart J* 2021; **42**(8): 831-4.
47. Lee M, Brauer M, Wong PLN, et al. Land use regression modelling of air pollution in high density high rise cities: A case study in Hong Kong. *Science of the Total Environment* 2017; **592**: 306-15.
48. Census and Statistics Department. Population estimates. 28 Apr 2022. https://www.censtatd.gov.hk/en/scode150.html (accessed 20 May 2022).
49. Centre for Health Protection. Age-standardised death rates by leading causes of death, 2001-2020. 14 Sep 2021. https://www.chp.gov.hk/en/statistics/data/10/27/339.html (accessed 20 May 2022).
50. Zeka A, Zanobetti A, Schwartz J. Individual-level modifiers of the effects of particulate matter on daily mortality. *Am J Epidemiol* 2006; **163**(9): 849-59.
51. Guzik TJ, Channon KM. Linking noise to cardiovascular disease pathogenesis. *Eur Heart J* 2017; **38**(37): 2850-2.
52. Basner M, Muller U, Elmenhorst EM. Single and combined effects of air, road, and rail traffic noise on sleep and recuperation. *Sleep* 2011; **34**(1): 11-23.
53. Schmidt FP, Basner M, Kroger G, et al. Effect of night-time aircraft noise exposure on endothelial function and stress hormone release in healthy adults. *Eur Heart J* 2013; **34**(45): 3508-14a.
54. Munzel T, Sorensen M. Noise pollution and arterial hypertension. *Eur Cardiol* 2017; **12**(1): 26-9.
55. Heritier H, Vienneau D, Foraster M, et al. A systematic analysis of mutual effects of transportation noise and air pollution exposure on myocardial infarction mortality: a nationwide cohort study in Switzerland. *Eur Heart J* 2019; **40**(7): 598-603.
56. Cai C, Mak CM, He X. Analysis of urban road traffic noise exposure of residential buildings in hong kong over the past decade. *Noise Health* 2019; **21**(101): 142-54.
57. Yang WT, Wang VS, Chang LT, et al. Road traffic noise, air pollutants, and the prevalence of cardiovascular disease in Taichung, Taiwan. *Int J Environ Res Public Health* 2018; **15**(8): 1707.
58. Barregard L, Bonde E, Ohrstrom E. Risk of hypertension from exposure to road traffic noise in a population-based sample. *Occup Environ Med* 2009; **66**(6): 410-5.

688 59. Pujol S, Berthillier M, Defrance J, et al. Indoor noise exposure at home: a field study
689 in the family of urban schoolchildren. *Indoor Air* 2014; **24**(5): 511-20.
690 60. Li S, Fong DYT, Wong JYH, et al. Indoor nocturnal noise is associated with body mass
691 index and blood pressure: a cross-sectional study. *BMC Public Health* 2021; **21**(1): 815.
692 61. Urban A, Davidkovova H, Kysely J. Heat- and cold-stress effects on cardiovascular
693 mortality and morbidity among urban and rural populations in the Czech Republic. *Int J*
694 *Biometeorol* 2014; **58**(6): 1057-68.
695 62. Kousis I, Pisello AL. For the mitigation of urban heat island and urban noise island:
696 two simultaneous sides of urban discomfort. *Environ Res Lett* 2020; **15**(10): 103004.

697

698

Captions of Figures

Figure 1 The associations between roadside and indoor noise and cardiovascular mortality (univariate model: only roadside or indoor noise was included; bivariate model: both roadside and indoor noise exposure were included).

Figure 2 Stratified relative risks of cardiovascular deaths by sociodemographic and environmental factors (significant differences at the significance level of 0.05 are indicated with an asterisk. UHI: urban heat island; NDVI: normalized difference vegetation index; PM: particulate matter).

Table 1 Descriptive summary of environmental variables

	Mean (SD)	Min	Median	Max	IQR
Daytime roadside noise	73.4 (1.7)	69.1	73.4	77.9	72.3–74.6
Night-time roadside noise	67.4 (2.2)	53.2	67.5	71.8	66.2–68.7
Weighted roadside noise	76.1 (1.7)	71.7	76.1	80.4	75.0–77.3
Average indoor noise	64.2 (6.4)	45.5	64.6	78.3	60.4–68.5
Daytime canopy-layer UHI	23.4 (0.6)	20.6	23.6	24.5	23.1–23.8
Night-time canopy-layer UHI	21.7 (0.6)	19.2	21.8	22.7	21.4–22.2
Surface-layer UHI	22.9 (2.0)	18.0	23.0	27.1	21.4–24.5
Average NDVI	0.4 (0.2)	0.1	0.4	0.8	0.3–0.6
Variability of NDVI	0.2 (0.1)	0.1	0.2	0.4	0.2–0.3
Night-time light	14.2 (10.9)	0.5	10.8	62.3	6.1–19.0
Particulate matter 2.5	31.1 (3.3)	23.3	31.5	40.8	28.7–33.3

SD: standard deviation; IQR: interquartile range; UHI: urban heat island; NDVI: normalized difference vegetation index.

Table 2 Pearson correlation coefficients between environmental variables.

	Average indoor noise	Daytime canopy-layer UHI	Night-time canopy-layer UHI	Surface-layer UHI	Average NDVI	Variability of NDVI	Night-time light	$PM_{2.5}$
Average roadside noise	**0.07**	**0.13**	0.01	**0.18**	**0.10**	**0.15**	**0.14**	**-0.04**
Average indoor noise		**0.36**	**0.43**	**0.30**	**-0.40**	**-0.41**	**0.29**	**0.16**
Daytime canopy-layer UHI			**0.86**	**0.83**	**-0.79**	**-0.47**	**0.30**	**0.51**
Night-time canopy-layer UHI				**0.76**	**-0.86**	**-0.56**	**0.35**	**0.39**
Surface-layer UHI					**-0.85**	**-0.53**	**0.44**	**0.47**
Average NDVI						**0.73**	**-0.42**	**-0.43**
Variability of NDVI							**-0.37**	**-0.33**
Night-time light								**0.10**

Remarks: significant correlations at the significance level of 0.05 are indicated in bold. UHI: urban heat island; NDVI: normalized difference vegetation index; PM: particulate matter.

Figure 1

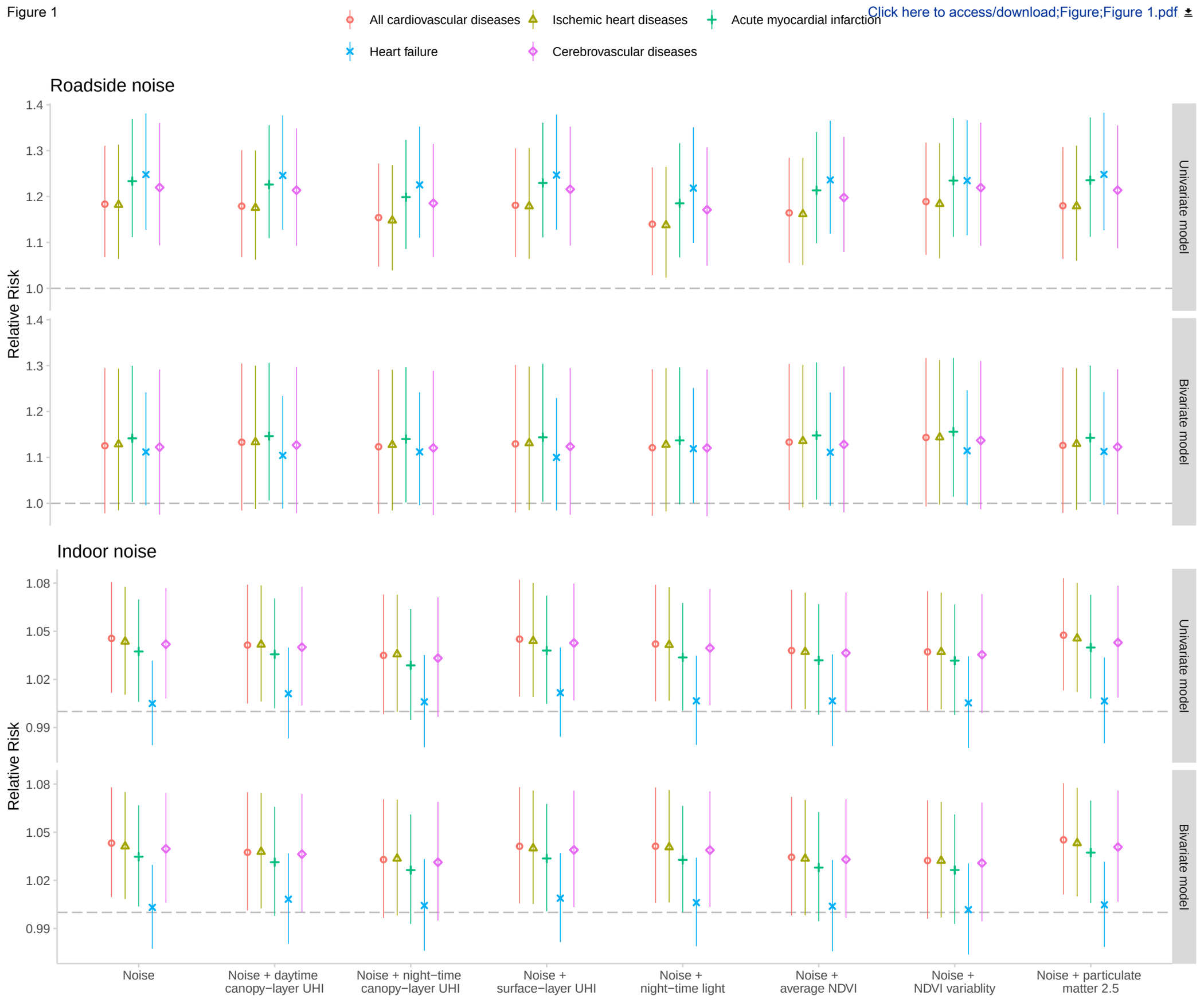

Figure 2