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Long-term effects of traffic exposures on mortality in a Chinese cohort

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ABSTRACT

A large proportion of the population living in close proximity to roads are continuously exposed to vehicle traffic and vulnerable to its ill effect in everyday life. However, only a limited number of epidemiological studies have examined effects of long-term road vehicle traffic exposures on mortality. We investigated long-term associations between road vehicle traffic exposures measured by vehicle-kilometres travelled (VKT) and mortality in a cohort of Chinese elderly in Hong Kong, where road vehicle traffic is generally high. The Elderly Health Service cohort consisted of 66,820 adults aged 65 years or older enrolled at 18 Elderly Health Centres of the Department of Health from 1998 to 2001. Deaths were followed-up until 2011. All cohort members had medical, socio-demographic, lifestyle, anthropometric data and place of residence recorded at baseline. The residential addresses of cohort members were geo-coded and aggregated by geographic areas, Tertiary Planning Units (TPU). The annual total VKT was used as traffic exposure at the TPU level. We used Cox proportional hazards regression to estimate hazard ratios (HR) for one million km increase in the annual total VKT for natural-cause and cardiopulmonary mortality, adjusted for individual demographic and socio-economic variables, lifestyle, and medication, as well as environmental covariates. There were 17,422 deaths in 197 TPUs, with the median age at death of 75.3 years. The mean annual total VKT was 26 million kilometres. A 1 million increase in the annual total VKT was associated with excess risk [100 × (HR - 1)] of 0.45% (95% confidence interval [CI]: 0.17, 0.74) for natural-cause mortality and 0.81% (95% CI: 0.30, 1.32) for cardiovascular mortality. No significant association was found for respiratory mortality. These findings suggest that vehicle traffic exposure is associated with raised risks of natural-cause and cardiovascular mortality in the older population.

Abbreviations: AADT, annual average daily traffic; BMI, body mass index; CI, confidence interval; COPD, chronic obstructive pulmonary disease; EHC, Elderly Health Centres; ER, excess risk; HR, hazard ratio; ICD, International Classification of Diseases; IHD, ischaemic heart diseases; M, million; PM, particulate matter; $PM_{2.5}$, particulate matter with aerodynamic diameter $\leq 2.5\,\mu m$; TPU, Tertiary Planning Unit; VKT, vehicle-kilometres travelled; WHO, World Health Organization

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1. Introduction

Hong Kong is one of the world's most densely populated cities, with about 7.3 million people in 2016 residing in a small land area of 1,106 km^2 (Census and Statistics Department, 2016). Roadside concentrations of pollutants are extremely high and will remain so for the foreseeable future (Hedley Environmental Inde, 2017). Sixty one percent of the population live in close proximity to roads and are continuously exposed to vehicle traffic pollution (Lai et al., 2011). Furthermore, traffic congestion in the midst of densely populated zones of multi-storied buildings amplifies pollutant concentrations because of street canyon effects that prevent free circulation of air and trap pollutants (Lee et al., 2017). This level of exposure has enormous public health implications; in particular, for the more sensitive individuals, such as the elderly and children who are particularly susceptible to cardiopulmonary diseases and premature deaths from air pollution. The ever-increasing traffic volumes and congestion levels will have predictable ill effects on public health.

A number of cross-sectional studies reported increasing prevalence of respiratory symptoms in individuals living in close proximity to roads with high traffic flows (Rosenlund et al., 2009; Skrzypek et al., 2013; Venn et al., 2005). A study in Vancouver reported that adults living near a major road experienced a 29% increase in the risk of death from coronary heart diseases (Gan et al., 2011). Although these reported studies have found associations with indicators of traffic exposure, there are a number of other risk factors associated with residents living close to roads. Roadside residents may experience not only vehicular emissions but also traffic noise, road dust, and other stress effects associated with their residential locations. Therefore, it is difficult to quantify which of these potential factors may be causal agents of health effects or surrogate indicators for other agents (Lipfert et al., 2008). For example, people exposed to higher levels of air pollution may also be exposed to excessive road traffic noise (Davies et al., 2009a; Foraster et al., 2011a). Because both exposures are strongly associated with road vehicle traffic, it is plausible that the observed associations between air pollution and adverse health outcomes could be modified or confounded by traffic noise. In addition, these co-existing environmental pollutants might interact with each other to amplify the associations with health outcomes (Davies et al., 2009a; Foraster et al., 2011a). While it is challenging to disentangle the effects of traffic-related air pollution and noise, some studies have supported the existence of a relationship between road traffic noise and cardiovascular mortality (Davies et al., 2009a; Foraster et al., 2011a; Allen et al., 2009a; Gan et al., 2012). These reported observations suggest that a geographic approach, with spatial measurements that take into account several aspects of an urban environment, can conceivably improve the assessment of traffic exposures and health impacts. Given that much of the epidemiological evidence on the effects of traffic-related pollution has focused on specific pollutants by using different exposure metrics, including residential proximity to major roads or circular buffer gradients in the pollutant concentrations (Venn et al., 2005; Dorans et al., 2017; Su et al., 2009), these metrics may have captured traffic-related pollution levels only near the place of residence.

A territory-wide exposure measure that is unrelated to air quality could be adopted in examining such relationships. Traffic exposure is potentially a surrogate for all environmental impacts of vehicular traffic emissions that include noise, road dust, and stress. As such, it is considered as a more robust surrogate compared with any single measure of ambient air quality, community noise or traffic proximity (Lipfert et al., 2006). To this end, we set out to investigate long-term associations between road vehicle traffic exposure, defined as the annual total vehicle-kilometres of travel (VKT) in different geographical areas, and mortality in an exposure cohort of Chinese elderly in Hong Kong, where the road vehicle traffic is generally high.

2. Materials and methods

2.1. Participants

The Hong Kong Department of Health manages 18 Elderly Health Centres (EHC) that provide primary health care services to residents aged 65 years or older. This cohort included 66,820 Chinese elders enrolled in the EHC from July 1998 to December 2001. All participants were recruited through their voluntary visits to EHC. Personal characteristics of the participants including demographic attributes, socio-economic position, lifestyle factors and health conditions were recorded at the baseline period (Sun. et al., 2009; Wong et al., 2015; Wong et al., 2016). Vital data about status, date and cause of death of each participant were obtained from the death registration database. We obtained ethical approval from the Institutional Review Board of the University of Hong Kong, Hong Kong Hospital Authority West Cluster, and the Department of Health of Hong Kong SAR.

2.2. Mortality outcomes

We obtained mortality outcomes using deaths coded according to the International Classification of Diseases, Ninth Revision before 2001 and Tenth Revision since 2001. The following codes were used: natural causes (ICD-9: 1–799; ICD-10: A00-R99), cardiovascular disease (ICD-9: 390–459; ICD-10: I00–I99) with sub-categories cerebrovascular (ICD-9: 430–438; ICD-10: I60–I69) and ischaemic heart diseases [IHD] (ICD-9: 410–414; ICD-10: I20–I25); and respiratory diseases (ICD-9: 460–519; ICD-10: J00-J99) with sub-category chronic obstructive pulmonary disease [COPD] (ICD-9: 490–496; ICD-10: J40-J47). The agreement between these two mortality ICD coding systems was over 90% in Hong Kong (Kong Department of Health, 2005).

2.3. Estimation of vehicle-kilometres travelled (VKT)

The Tertiary Planning Unit (TPU) comprises geographic subdivisions of Hong Kong devised by the Hong Kong Planning

Department for town planning purposes. At the baseline year of our cohort in 2001, the land area of Hong Kong was subdivided into 282 TPUs with areas ranging from 0.06 to 32 km^2, and a median value of 1.83 km^2. Our study also contained demographic and socio-economic data at the TPU level from the 2001 Hong Kong census. Further aggregation of census data for TPUs with small populations yielded 197 TPUs for subsequent analyses.

Annual traffic census over the past 50 years is also available for Hong Kong. The 2001 traffic census (TD, 2001) surveyed traffic volumes covering 87% of trafficable roads. The remaining 13% involved restricted or low-volume trafficable roads situated in rural areas and not covered by the survey. These traffic flow data are available from the Transport Department with scaling and growth factors estimated to yield hourly, daily and monthly variations in the annual average daily traffic [AADT]. We estimated the annual total VKT for each TPU by summing the product of AADTs for each road type and the respective road lengths. That is,

$$VKT_i = \sum_{\{\text{All road links in jth TPU}\}} AADT_{ij} \times L_{ij};\ \ i = 1,\ 2,\ 3,\ ...,\ 282$$

where VKT_i is the annual vehicle kilometre traffic travelled (vehicles × km/year) of the ith TPU, $AADT_{ij}$ is the average annual daily traffic of the jth road link (km) in the ith TPU, and L_{ij} is the length of the jth road link in the ith TPU. We found that for those large TPU in rural areas with no roads and scant populations, the number of AADT stations is small compared to those of similar geographical size TPUs in urbanized areas. Since TPU area may not be a valid indicator for estimating traffic density (defined as VKT per TPU area) in Hong Kong, we adjusted for TPUs areas in the model.

The estimated VKT for each TPU was used as an indicator for traffic exposure for its residents, which averaged about 24,800 vehicles travelling on the roads per day. We geocoded addresses for all participants into x- and y-coordinates onto the Hong Kong area map with demarcation of areas of TPU using the ArcGIS software (ESRI, Redlands, California). The cohort members were assigned the estimated VKT based on the corresponding TPU of their residential addresses.

2.4. Statistical analysis

Cox proportional hazards regression modelling was used to assess relationships between mortality and VKT adjusted for demographic attributes (gender and age), socio-economic position (education attainment and monthly expenditure), lifestyle factors (smoking status, alcohol consumption and exercise frequency), health status (BMI and medication), housing density, floor number and TPU area (km^2). Hazard ratios and the associated 95% confidence intervals (95% CI) were obtained for one million km increase in VKT. We presented results as excess risk (ER) in percent [100% × (HR - 1)]. We examined the proportional hazards assumption by adding an interaction term between VKT and time.

2.5. Sensitivity analysis

To account for possible clustering effects of the TPU, a random effect was incorporated into the model. We examined relationships between VKT and mortality using different mathematical transformations for the exposure variable to determine the shapes of the dose-response relationships: (i) logarithm of VKT, (ii) further adjustment for VKT^2 term, and (iii) categorization of VKT into tertile.

To assess how the effect estimate of VKT changes in the presence of particulate matter with aerodynamic diameter $\leq 2.5\,\mu m$ ($PM_{2.5}$) concentrations, we further adjusted for baseline $PM_{2.5}$ at resolution 1 km × 1 km grid in the model (Wong et al., 2014).

All statistical analyses were performed using SAS 9.4 (SAS Institute, Inc., Cary, North Carolina).

3. Results

3.1. Cohort participants, characteristic and traffic exposure conditions

5,352 participants with unknown date of death, missing residential address or individual-level covariates were excluded from the Elderly Cohort database. The remaining 61,468 participants were geo-coded by their residential addresses and assigned a traffic exposure of VKT value based on their corresponding TPU of residential addresses. Table 1 shows the study had 40,528 (66%) women and 20,940 (34%) men, with a mean age of 72 years. Most participants were non-smokers (71%), non-alcoholic drinkers (96%), had primary education or below (83%), had moderate monthly expense (USD $128 - $385, 69%), and exercised every day (72%). Around 47% of the participants took regular medications.

The mean annual total VKT among TPUs was 26 million km in the baseline year. Twenty nine of 197 TPUs did not have AADT data. Participants (3.5%) living in these TPUs without AADT were further excluded from this study. Fig. 1 shows VKT by TPUs and reveals areas with exceptionally high VKT in red (more than 2.5 standard deviations from the mean). These areas are important logistic hubs for Hong Kong, where Kwai Tsing is a shipping container terminal and the other three areas are major storage and distribution centres (Kwun Tong, Shatin, and Wong Tai Sin). TPUs with low VKT (≤0.5 standard deviation from the mean) are shaded in pale yellow and green and they are mostly under-developed or in rural areas at the baseline year of the study. This geographic mapping approach enables differentiation of TPUs based on traffic exposure inferred by annual total VKT. For the elderly cohort with limited mobility, the VKT by TPU of residence represents a more comprehensive measure of their exposures.

Table 1
Baseline characteristics of study participants in Chinese elderly cohort 1998–2001, by tertile of vehicle-kilometres travelled (VKT).

Parameter		Overall	1st tertile	2nd tertile	3rd tertile
Number of death (%)		28.3	27.3	28.4	28.8
Sex (%)	Male	34.1	34.7	34.6	33.0
	Female	65.9	65.3	65.4	67.0
Age (year)		72.0 ± 5.6	72.0 ± 5.6	72.1 ± 5.5	72.1 ± 5.7
BMI (kg/m^2) (%)	Q1 (< 21.6)	23.1	23.1	22.8	23.4
	Q2 - Q3 (21.6–26.3)	50.7	51.1	50.8	50.2
	Q4 (> 26.3)	26.2	25.8	26.4	26.4
Education (%)	Secondary or above	17.2	19.0	15.8	17.1
	Primary	36.9	34.5	38.0	38.6
	Below primary	46.0	46.5	46.2	44.3
Expense/month (USD $) (%)	< 128	14.9	15.3	17.2	17.1
	128–385	68.6	69.1	68.6	15.7
	≥385	16.5	15.6	14.2	67.2
Smoking status (%)	Never smoker	71.1	71.3	70.9	70.8
	Quitted	19.3	19.2	19.3	19.6
	Current	9.6	9.5	9.8	9.6
Exercise (days/week) (%)	0	15.3	15.2	14.9	15.9
	1–3	7.0	6.9	6.9	7.2
	4–6	5.7	5.7	5.5	5.9
	7	72.0	72.3	72.7	71.0
Alcohol consumption (%)	Drinker	4.0	4.1	3.8	3.9
	Non-drinker	96.0	95.9	96.2	96.1
Medication taken (%)	Yes	46.9	52.9	53.5	47.0
	No	53.1	47.1	46.5	53.0

Abbreviations: BMI, Body mass index; Q1 - Q4, 1st quartile to 4th quartile; VKT, vehicle-kilometres travelled.

3.2. Mortality effects of VKT

The excess risks (ER) by different levels of adjustment are summarized in Table 2. Using a hierarchical approach, the first Cox proportional hazards regression, which included a linear term (VKT) produced a ER of 0.53% (95% CI: 0.27, 0.80) per one million km increase in the annual total VKT for natural-cause mortality (M1). After adjusting for sex, age, socio-economic position, lifestyle factors, health status, residential floor level, TPU area, and housing density, and coastline factor, the ER remains stable at 0.45% (95% CI: 0.17, 0.74) for natural-cause mortality (M10). The adjusted ER for cardiovascular mortality was higher (0.81, 95% CI: 0.30, 1.32) than that of natural causes and was positive but not significant ($p > 0.05$) for respiratory mortality. The ERs showed reduced risks for natural cause, cardiovascular, and respiratory mortality, and remained not significant for respiratory mortality in the model that adjusted for all covariates (M10).

A summary of the ERs for mortality due to specific causes according to ICD-9 and ICD-10 classifications is shown in Table 3. The ERs were 0.45% (95% CI: 0.17, 0.74) for natural causes, 0.81% (95% CI: 0.30, 1.32) for cardiovascular mortality and −0.01% (95% CI: 0.67, 0.66) for respiratory mortality. For sub-categories of the cardiovascular diseases, one million km increase in the annual total VKT was associated with mortality for ischaemic heart diseases (0.84%, 95% CI: 0.02, 1.67), but not for cerebrovascular disease (0.55%, 95% CI: 0.33, 1.45). Associations with respiratory mortality and COPD were positive, but not statistically significant ($p > 0.05$). We found no evidence that the proportionality hazards assumption was violated.

3.3. Sensitivity analyses

There was an increase in the effect estimates after including TPU as random effects into the model: the ERs were 0.67% (95% CI: 0.14, 1.20) for natural causes, 1.20% (95% CI: 0.14, 2.27) for cardiovascular mortality, and 0.04% (95% CI: 0.71, 0.80) for respiratory mortality (Table 4). Interaction terms between VKT and each of the individual covariates were included in the model and none of them were found to be statistically significant (data not shown). There was a small reduction in the effect estimates after including $PM_{2.5}$ into the model, with ER of 0.38% (95% CI: 0.09, 0.66). The ER per 10 μg/m^3 increase in $PM_{2.5}$ alone was 2.25% (95% CI: 1.55, 2.95).

We examined relationship between VKT and mortality by different mathematical transformations of the exposure variable: (1) natural logarithm of VKT, (2) further adjustment of VKT^2 and (3) categorization of VKT into tertiles to detect threshold of VKT. The model relationships for tertiles (Q1: 0–1.67 M km, Q2: 1.68 M–4.08 M km, Q3: 4.09 M–30.74 M km) of exposure are shown in Fig. 2.

4. Discussions

We found that an increase in the annual total VKT was strongly associated with increase in mortality risks due to natural-cause and cardiovascular diseases for those aged 65 or older, but not in respiratory diseases. The number of deaths attributable to VKT was

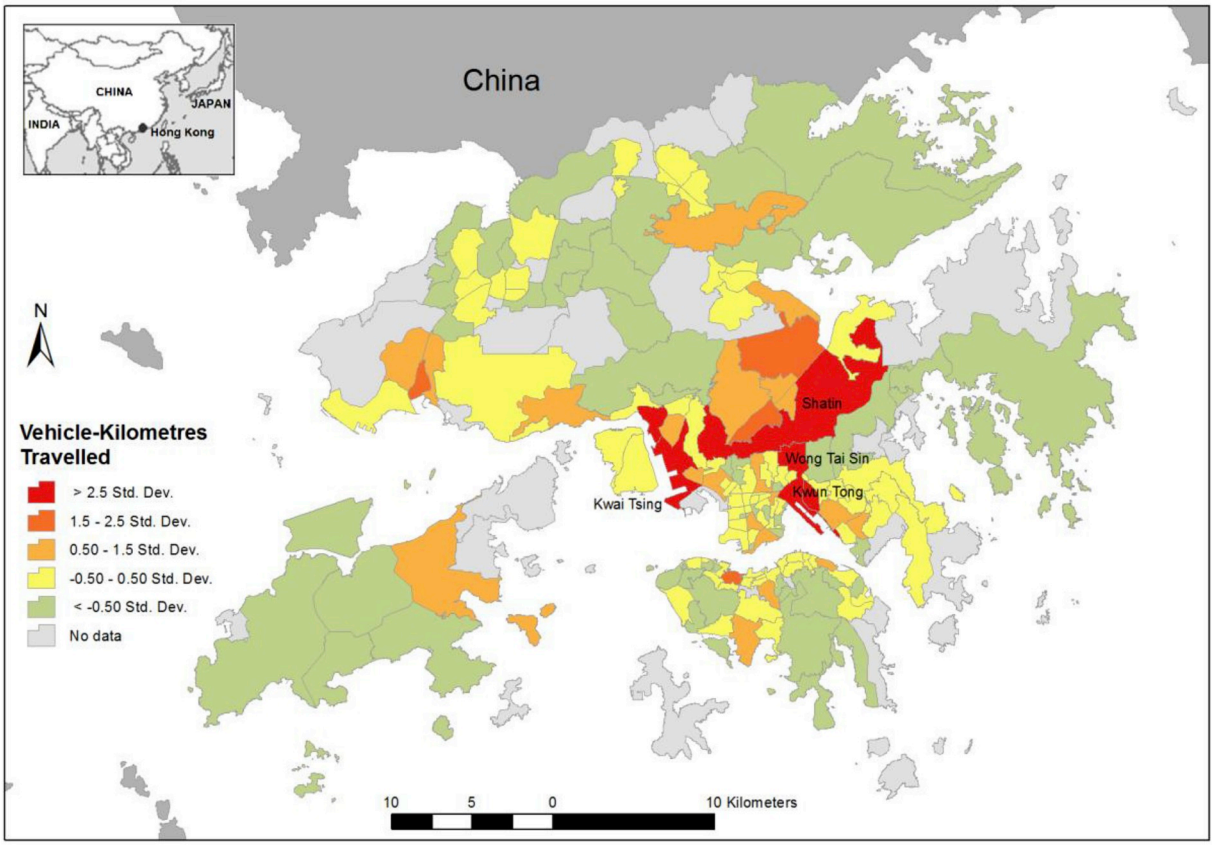

Fig. 1. Annual total vehicle-kilometres travelled (VKT) by Tertiary Planning Unit (TPU) of Hong Kong (2001).
The above map is produced by the ArcGIS (ESRI, Redlands, California) mapping utility software that calculates the mean and standard deviation of Vehicle-Kilometres Travelled (VKT). The standard deviation classification method shows how much the VKT value of a TPU varies from the mean. Class breaks are created with equal value ranges by proportions of the standard deviation. The example shows classes at intervals of one standard deviation; with a neutral class (shaded in pale yellow) starting at mean value and one-half standard deviations from either side of the mean.

Table 2
Hierarchical excess risks in percent of mortality (95% CI) of the annual total vehicle-kilometres travelled (VKT) per one million km by causes of death.

Model	Natural causes	Cardiovascular	Respiratory
M1: VKT	0.53 (0.27, 0.80)***	0.89 (0.43, 1.36)***	0.16 (−0.45, 0.78)
M2: + sex, age	0.60 (0.33, 0.86)***	0.94 (0.48, 1.41)***	0.23 (−0.38, 0.85)
M3: + BMI (kg/m^2)	0.60 (0.33, 0.86)***	0.94 (0.47, 1.41)***	0.28 (−0.34, 0.90)
M4: + education, income	0.59 (0.32, 0.85)***	0.93 (0.47, 1.40)***	0.28 (−0.34, 0.89)
M5: + smoking, alcohol, exercise	0.52 (0.26, 0.78)***	0.89 (0.43, 1.36)***	0.18 (−0.43, 0.79)
M6: + health status	0.51 (0.25, 0.78)***	0.87 (0.41, 1.34)***	0.19 (−0.42, 0.80)
M7: + TPU area	0.51 (0.25, 0.78)***	0.85 (0.37, 1.32)***	0.14 (−0.48, 0.77)
M8: + floor level	0.40 (0.13, 0.68)**	0.69 (0.19, 1.18)**	−0.03 (−0.68, 0.64)
M9: + housing density	0.40 (0.12, 0.68)**	0.68 (0.18, 1.18)**	−0.03 (−0.69, 0.63)
M10: + coastline TPU	0.45 (0.17, 0.74)**	0.81 (0.30, 1.32)**	−0.01 (−0.67, 0.66)

Abbreviation: BMI, body mass index; CI, confidence interval; TPU, Tertiary Planning Unit; VKT, vehicle-kilometres travelled.
*** significant at $p \leq 0.001$; ** significant at $p \leq 0.01$.

4,936 in our cohort, which accounted for 31.3% of deaths for natural causes. The findings are consistent with those of our previous work that used traffic density, road density and vehicle density as measures of traffic exposure (Ma, 2014). They are also consistent with findings of a study on associations between traffic density and mortality of a Veteran cohort in the United States (Lipfert et al., 2006). The Nurses' Health Study found a 5% increased risk in all-cause mortality for women living near to traffic exposure compared with those women living far away (Hart et al., 2013).

Furthermore, VKT was found to be associated with mortality due to ischaemic heart diseases. The effect estimates are comparable with those in a case-control analysis, for which an increase in cumulative traffic near the participants' home was associated with a 4%

Table 3
Excess risks in percent (95% CI) per one million km increase in vehicle-kilometres travelled (VKT) by cause-specific diseases (N = 57,234).

ICD-9	ICD-10	Diseases	n	ER%
1–799	A00-R99	Natural causes	15,781	0.45 (0.17, 0.74)**
390–459	I00–I99	Cardiovascular	4525	0.81 (0.30, 1.32)**
410–414	I20–I25	IHD	1774	0.84 (0.02, 1.67)*
430–438	I60–I69	Cerebrovascular	1571	0.55 (−0.33, 1.45)
460–519	J00-J99	Respiratory	3026	−0.01 (−0.67, 0.66)
490–496	J40-J47	COPD	915	0.15 (−1.03, 1.35)
800–999	S00-T99	Accidental cause	405	0.75 (−0.93, 2.46)

Abbreviations: CI, confidence interval; COPD, chronic obstructive pulmonary disease, ER, excess risk; ICD, International Classification of Diseases; IHD, ischaemic heart diseases; VKT, vehicle-kilometres travelled.
** significant at $p \leq 0.01$; * significant at $p \leq 0.05$.

Table 4
Excess risks in percent (95% CI) by causes of death based modelling approaches of VKT.

Model	Variable referred	Natural causes	Cardiovascular	Respiratory
M10	VKT	0.45 (0.17, 0.74)**	0.81 (0.30, 1.32)**	−0.01 (−0.67, 0.66)
M10 + random effects	VKT	0.67 (0.14, 1.20)*	1.20 (0.14, 2.27)*	0.04 (−0.71, 0.80)
M10 + $PM_{2.5}$	VKT	0.38 (0.09, 0.66)**	0.67 (0.16, 1.18)**	−0.07 (−0.73, 0.61)
M10 + VKT^2	VKT	1.21 (0.47, 1.97)**	1.73 (0.39, 3.10)*	1.05 (−0.67, 2.80)
	VKT^2	−0.03 (-0.06, 0.00)*	−0.04 (−0.09, 0.01)	−0.04 (−0.11, 0.02)
VKT replaced with natural log of VKT in M10	log (VKT)	2.47 (1.15, 3.81)***	4.13 (1.71, 6.61)***	1.33 (−1.61, 4.37)
VKT replaced with $PM_{2.5}$ in M10	$PM_{2.5}$	2.25 (1.55, 2.95)***	4.47 (3.19, 5.77)***	0.63 (−0.94, 2.23)

Abbreviations: CI, confidence interval; $PM_{2.5}$, particulate matter with aerodynamic diameter ≤ 2.5 μm; VKT, vehicle-kilometres travelled.
*** significant at $p \leq 0.001$; ** significant at $p \leq 0.01$; * significant at $p \leq 0.05$.

increase in the odds of acute myocardial infarction (sub-category of ischaemic heart diseases) per interquartile range (Tonne et al., 2007). The same study also found that living near a major roadway was associated with a 5% increase in the odds of acute myocardial infarction per kilometre of road.

It is well-documented that air pollution is detrimental to health and probably increases mortality risk. Fine particulate matter concentrations are high in Hong Kong, with an annual level of 37.6 μg/m^3 (data not shown) in 2001, exceeding the annual World Health Organization (WHO) Air Quality Guidelines (10 μg/m^3) and it has been found to be associated with mortality in our previous study using this same cohort of elderly adults (Wong et al., 2015, 2016). After adjusting for $PM_{2.5}$ our analysis showed little change in the magnitude of the hazard ratio of natural-cause mortality (0.45% *vs* 0.38%), suggesting that VKT is a robust indicator of traffic-related air pollution.

In our study, a continuous exposure measure of VKT was used to assess the risk of mortality in a Chinese elderly cohort. In general, continuous measurements of exposure would have greater statistical power than would categorical measurements of exposure (e.g. in proximity studies for comparing between major and minor roads). However, continuous measurements are likely to produce perceived lower risk estimates (Lipfert and Wyzga, 2008). Proximity studies show risks (e.g. near highways) that may affect only a small fraction of the population in a given radius (Patil et al., 2015), while our exposure metric affects the entire population. Moreover, to abate the effects of proximity to traffic would require geographic relocation. By contrast, abating traffic density merely requires reducing the numbers of vehicles.

Our study that made use of a large territorial-wide cohort of older population aggregated at the TPU level has important methodological implications. With some incomplete information in detailed residential address up to the floor level of the de-identified data, address matching at the TPU level can reduce misclassification. The availability of a wide range of individual-level potential confounders from the cohort data can combine with TPU or group-level indicators to gain in data efficiency and analytical power. In addition, we were able to adjust for floor level of residents' flats in the model, which is a particularly important factor for assessments of air pollution exposure in Hong Kong – a city crowded with multi-storied buildings.

It is difficult to ascertain accurate measurements of traffic-related air pollution level, including traffic noise and to disentangle these effects separately. Studies have supported the existence of a relationship between traffic noise and cardiovascular mortality (Gan et al., 2012; Recio et al., 2016), and between air pollution and a range of mortality outcomes (Wong et al., 2015, 2016). We developed a VKT surrogate exposure at the TPU-level that is suitable for cohort and population-based epidemiological studies.

The noise exposure could also be related to cardiovascular mortality. Accumulating evidence has suggested that community noise from road and air traffic is associated with an increased risk of CHD, especially myocardial infarction (Babisch et al., 2005; Selander et al., 2009; Huss et al., 2010; Babisch, 2011; Davies et al., 2009b). In metropolitan areas, road traffic is a major contributor to ambient air pollution and the dominant source of community noise.

(Allen et al., 2009b; Brauer et al., 2003; Zhu et al., 2002; Foraster et al., 2011b). Persons exposed to higher levels of air pollution might also be exposed to excessive traffic noise (Davies et al., 2009b; Allen et al., 2009b; Foraster et al., 2011b). Therefore, it is possible that the observed associations between air pollution and adverse cardiovascular outcomes could be confounded by

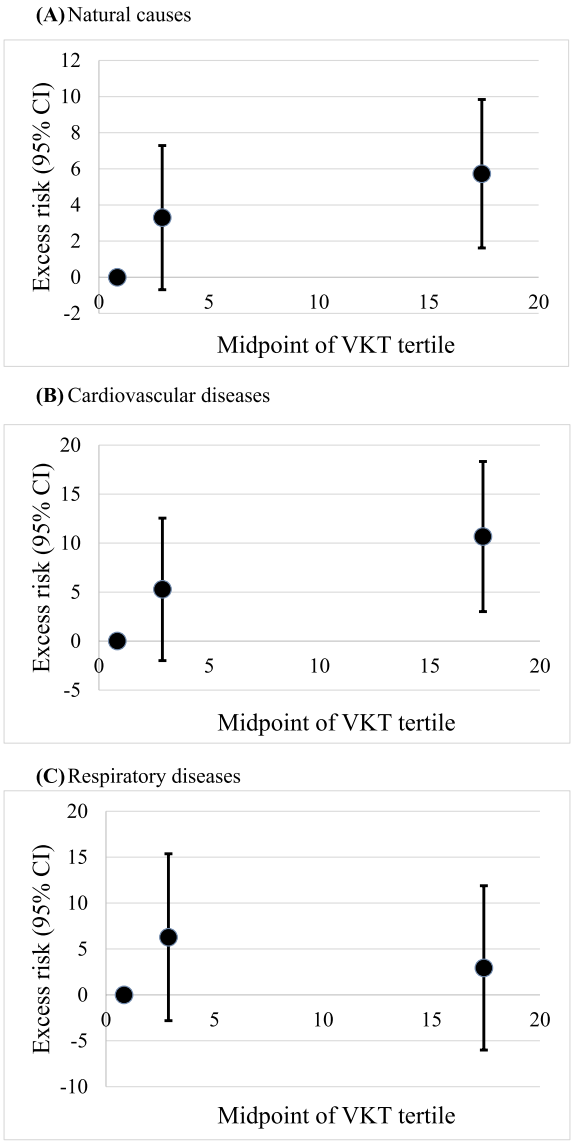

Fig. 2. Excess risks (%) of mortality by VKT tertile.
Abbreviations: VKT, vehicle-kilometres travelled.

community noise and vice versa (Davies et al., 2009b; Allen et al., 2009b). Furthermore, these coexistent environmental pollutants might interact with each other in association with coronary mortality (Allen et al., 2009b).

Greenery could be a potential confounder in the association between traffic exposure and mortality. We have adjusted for housing density in our model - which is negatively correlated with greenery. That is to say, TPUs with higher housing density tend to have lower greenery. So due to multicollinearity problem, adjusting for either one in the model is adequate.

In our study the unit of analysis is the number of TPUs, which consists of 276 TPU. So, residents living in the same TPU are assumed to be exposed to the same traffic exposure. It is possible that traffic exposure varies within block-level in the TPU and we did not have data on traffic density to account for this variation. However, the inferences of effect estimates are still valid at the TPU level.

4.1. Strengths and limitations

The modelling method used in this study follows a well-established approach adopted in the assessment of long-term effects of traffic exposure on mortality in a cohort study (Lipfert et al., 2008). A salient feature of this method is that the likelihood of confounding effects of VKT is minimized by adjusting for other covariates, which may be associated with variations in mortality at the same time. This modelling approach provides a systematic and structured way of investigating the relationship between VKT and

mortality in the presence of possible confounding covariates.

Some limitations were associated with our study. First, we could not distinguish vehicle types, such as lorries or cars, from the AADT data. The exposure effects from diesel and gasoline vehicles could be different for the same value of VKT. Second, traffic congestion is another potential factor that was not accounted for in the study. A recent study suggested that for each 10 min-kilometre increase in upwind congestion, the odds of cerebrovascular and respiratory mortality were higher even though evidence for associations was not found for natural-cause and cardiovascular mortality (Pedde et al., 2017). It should be noted that most of the minor roads were not monitored by the AADT stations and 15% of the TPUs lacked AADT stations, which may lead to underestimation of the annual total VKT. However, we presumed that TPUs with no AADT data or very few AADT stations should have relatively low traffic. Third, some of the residential addresses in the cohort database were incomplete. For example, participants provided only street names or estate names, but without block numbers. In this instance, geo-coding by centroid method (i.e., middle of a street or an estate) was employed. This method may not be able to capture the exact X–Y coordinates of residential addresses of the cohort members, but it should be valid at the TPU level. Moreover, we did not have a complete residential history of the cohort participants. It is possible that some participants might have moved into a particular TPU recently and so their past exposures may contribute more to their mortality outcomes. Additionally, we did not have information on personal level of exposure outside of residential area, such as at work or when commuting on different modes of transport as there may be significant differences between indoor and outdoor exposures of pollution levels such as the use of different cooking methods and air purifiers. This is another confounding factor of the study which can only be addressed through real-time sensing of personal exposures. Our study used ecological measure of exposure but not individual exposure. Indeed, few studies have considered spatio-temporal activity patterns (including home, work, and commute) into the exposure assessment, which could enhance personal level exposure estimates (Chau et al., 2002; Dias and Tchepel, 2014; Kwan, 2012, 2018a, 2018b). Our exposure assessment may not have covered the time window that is most relevant to the time of death. However, members of this cohort are of older-aged group who tend to have stable residential histories and a vast majority of them are retired and with low mobility. Fourth, the results of this cohort may not be generalized to younger sub-populations, or to cohort members with frequent changes of residence. Fifth, we recognize that the same VKT in large TPUs areas would not be comparable to the same VKT in small TPUs. It is noted that large TPUs are situated in rural areas and the number of AADTs is limited, resulting in a small VKT; while geographically small TPU in urbanized area with high number of AADT would yield a high VKT. So, given an uneven distribution of AADT, we believe that VKT is a robust measure for traffic-related exposures in Hong Kong. Last, this study should be replicated in different settings because of different counting systems used for measuring traffic counts from core and coverage counting stations.

5. Conclusions

Our study presents the first assessment of mortality effects linking long-term exposure to traffic-related pollution. The outcomes of this study provide health effect estimates which are directly relevant to public health, environmental policy and the support needed for evidence-based strategies to mitigate the damage caused to population health by traffic exposures.

Declarations of interest

None.

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