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Original Articles

Motor vehicle traffic and cardiovascular mortality in male adults

ABSTRACT

OBJECTIVE: To assess the association between indicators of exposure to motor vehicle-related air pollution and cardiovascular mortality in male adults.

METHODS: Information on roads and traffic volume for the year 2007 in the city of São Paulo, Southeastern Brazil, was obtained from the local Traffic Engineering Division. Data on mortality from cardiovascular diseases among men aged ≥40 years in 2005 were obtained from the mortality database of the city of São Paulo. Socioeconomic data from the 2000 Population Census and information on location of health care units were also collected. Exposure was assessed by road density and traffic volume for each geographic unit (administrative districts). Spatial regression (α= 5%) between these indicators of exposure and standardized mortality rates from cardiovascular diseases were estimated. The models were adjusted for socioeconomic variables, number of health care units in the districts and spatial autocorrelation.

RESULTS: It was found a modest correlation between road density and traffic volume ($r^2 = 0.28$). The central districts had the highest road densities. The spatial regression model of road density showed an association with mortality from cardiovascular diseases ($p = 0.017$). No association was found in the model of traffic volume. The socioeconomic variable was statistically significant in both models of road and traffic volume.

CONCLUSIONS: The association between mortality from cardiovascular diseases and road density is consistent with literature data. Further individual-level epidemiological studies should be performed using more accurate methods for the assessment of exposure.

DESCRIPTORS: Cardiovascular Diseases, mortality. Air Pollution. Vehicle Emissions. Air Pollutants, adverse effects.

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INTRODUCTION

Air pollution is one of the top environmental risks with greater impact on health due to its ubiquitous and pervasive nature. A recent study estimated the impact on overall mortality of exposure to ozone (O_3) and particulate matter ≤2.5 µg/m³ ($PM_{2.5}$) from human-related (anthropogenic) sources of pollution.[1] Exposure to O_3 was associated with estimated 0.7 (standard deviation [SD] = 0.3) million deaths from respiratory causes and 6.3 (SD = 3.0) million years of life life lost annually, while exposure to $PM_{2.5}$ was associated with 3.5 (SD = 0.9) million cardiorespiratory deaths and 220,000 deaths (SD = 80,000) from lung cancer (30 [SD = 7.6] million years of life lost) annually.

Children, elderly and individuals with cardiopulmonary conditions are the most vulnerable to air pollution. Every year approximately 5% of deaths from

respiratory causes among children (≤5 years of age) and elderly (≥65 years of age) can be attributed to particulate matter (PM_{10}) pollution in seven Brazilian cities: Belo Horizonte (Minas Gerais State), Curitiba (Paraná State), Fortaleza (Ceará State), Porto Alegre (Rio Grande do Sul State), Rio de Janeiro (Rio de Janeiro State), São Paulo (São Paulo State) and Vitória (Espírito Santo State).[8]

Studies have shown positive associations between exposure to air pollutants and increased risk of cardiovascular diseases.[5-7,10-12] though the adverse effects of different pollutants on the cardiovascular system are not yet clear.

Air pollution may cause oxidative stress in the respiratory epithelium when pollutants (PM or O_3) have direct contact with alveolar epithelial cells that are not covered by the epithelial lining fluid or with other cells such as macrophages. Oxidative stress induces cell apoptosis and triggers inflammation. This inflammation have cardiovascular effects including blood hypercoagulation, atherosclerosis progression and increased platelet breakdown[13] leading to acute atherosclerotic and ischemic cardiovascular complications.[2,3] Air pollution also affects the autonomic system causing cardiac arrhythmias.[13]

Motor vehicle traffic is a major contributing factor to air pollution in the city of São Paulo with a fleet of 7,012,795 motor vehicles reported as of March 2011.[a] These vehicles account for 97% of emissions of carbon monoxide (CO), 97% of hydrocarbons, 96% of nitrogen oxides, 40% of PM and 32% of sulfur oxides, as well as resuspension of soil dust and formation of secondary aerosols in the particulate matter.[b]

Motor vehicles are the main cause of air pollution and higher concentrations of pollutants are seen near busy road with high traffic and they gradually decline with distance.[14] Thus, many studies have used indirect methods to assess exposure based on road and traffic data, which allows to measure the impacts of vehicle emissions on health of the exposed population and can provide input for developing emission control measures.

The current study aimed to assess the association between indicators of exposure to motor vehicle-related air pollution and cardiovascular mortality in male adults.

METHODS

The study was conducted in São Paulo (Southeastern Brazil), a city with a population of 11,324,102 inhabitants, high population density (≈7,500 inhabitants per km²), an area of 1,509 km² and 98.9% urbanization. The city is divided into 96 administrative districts grouped into west, north, east, south and central areas.

Mortality information was obtained from the Program for Improvement of Mortality Data of the City of São Paulo (PROAIM) database. This database includes information from death certificates such as age, gender, and area of residence of individuals at the time of their death, in addition to their primary cause of death coded according to International Classification of Diseases and Related Health Problems, Tenth Revision (ICD-10). Deaths in men ≥40 years of age that occurred in 2005 where the underlying cause was diseases of the circulatory system (ICD-10 Chapter I00 to I99) were selected. Mortality rates from cardiovascular diseases for each administrative district were estimated (per 1,000 inhabitants). The mortality rate was standardized by age group (standardized mortality rate, SMR) using direct standardization with mortality rates in the city as reference.

Population and socioeconomic data obtained from the 2000 Population Census conducted by the Brazilian Institute of Geography and Statistics including total population by gender and age group and average monthly income of the head of the household were grouped by administrative districts.

It was assumed that increased access to health services could have an impact on mortality rates from cardiovascular diseases in the population. Data on health services was collected from the Digital Map of the City of São Paulo database for the construction of the variable inhabitants per health service (HS). HS were public or private facilities including primary care units, outpatient care units, general emergency rooms, health centers, hospitals, general hospitals, single or and multispecialty medical centers.

Data on road system including traffic count and simulation of the main roads (collector and arterial roads and rapid transit corridors) for 2007 were provided by the Traffic Engineering Company of São Paulo. Charts with road networks, average daily number of vehicles, and traffic type (light/heavy vehicle) were also provided.

Two indirect indicators of exposure to motor vehicle-related pollutants were constructed for each administrative district using MapInfo Professional geographic information system (GIS) software (version 8.5, MapInfo Corporation, New York, NY, USA):

- Road density: sum (km) of arterial and collector roads and rapid transit corridors divided by the area (km²) of an administrative district.

$$\frac{\sum roads}{area}$$

- Traffic volume (subdivided by light and heavy vehicles): sum of traffic flow divided by the length of arterial and collector roads and rapid transit corridors (in km) of an administrative district.

$$\frac{\sum traffic}{\sum roads}$$

Since this information was grouped by administrative districts any spatial autocorrelation between districts could affect the model's explanatory power. Thus, the estimates included the spatial structure in the potential association between SMR and other independent variables. Spatial regression was performed (α = 5%).

Spatial regression analysis takes into account spatial dependence by adding a new term to the regression model in the form of spatial relationship for the dependent variable,[c] expressed as:

$$Y = \rho WY + X\beta + \varepsilon$$

where Y is the dependent variable, ρ is the spatial autoregressive coefficient, W is the neighborhood matrix and the WY product expresses the spatial dependence in Y. The null hypothesis for non-spatial autocorrelation is $\rho = 0$.

The neighborhood matrix W (n x n), a component of the model, estimated spatial variability of data from a set of n districts $\{D_1,..,D_n\}$, where each element w_{ij} represents a measure of proximity between D_i e D_j. This measure of proximity was calculated using the following criteria:

$w_{ij} = 1$, if D_i shares a common border with D_j, otherwise $w_{ij} = 0$.

Regression analyses between the dependent variable (SMR) and independent variables (per capita monthly income of people living in the districts and inhabitants per HS) were performed in the univariate models. The variables with p≤0.2 were retained in the multivariate models including road density and traffic volume (overall, light and heavy traffic). The analyses were performed in GEO Data Analysis.

This study was approved by the Research Ethics Committee of Clínicas Hospital at Universidade de São Paulo Medical School (number 0662/09; July 16, 2009).

RESULTS

The correlation between traffic volume and road density was modest but statistically significant (p<0.001), mainly for light vehicle traffic. The correlation between road density and heavy traffic volume was weak and not statistically significant (Figure 1). The highest road density was found in central districts (República, Bela Vista, Sé, Santa Cecília, Bom Retiro and Consolação). The southernmost districts of the city showed the lowest road density (Marsilac, Parelheiros, Grajaú) and the lowest traffic volume as well. The heaviest traffic volumes were not seen in central districts but rather in those near major highways of the city (Figure 2).

A total of 31,476 deaths were reported in men ≥40 years in 2005, 35.2% from cardiovascular diseases. There were excluded 11.5% of the records due to missing information on district of residence. The SMR ranged from 1.37 deaths per 1,000 in the district of Marsilac (south of the city) to 8.64 deaths per 1,000 in Pari (central area). Per capita income decreased with distance from the city center (Figure 2).

The single variable that was significantly associated with SMR in the univariate spatial regression was average per capita income. Road density and traffic volume were not statistically significant (Table 1).

Monthly per capita income and inhabitants per HS were included in the multiple spatial regression analysis. Only monthly per capita income remained statistically significant and was retained in the final model including road density and traffic volume (overall, light and heavy). After adjustment for monthly per capita income the strength of association between road density and mortality from cardiovascular diseases increased (p=0.017), showing that a 10-km increase of road/km² was associated with an increase in SMR of the district by about 1 death/1,000 inhabitants. The multivariate models including overall, light and heavy traffic volumes were not statistically significant even after adjustment (Table 2).

DISCUSSION

The current study showed an association between road density in the district of residence and SMR for cardiovascular diseases in male adults in the city of São Paulo. This result is in agreement with the literature which suggests a relationship between motor vehicle-related air pollution and increased morbidity and mortality rates from cardiovascular disease.

Kan et al[6] assessed coronary artery disease in four U.S. communities. The adjusted hazard ratio (HR) of the

[c] Câmara G, Carvalho MS, Cruz OG, Correa V. Análise espacial de áreas. São José dos Campos: Instituto Nacional de Pesquisas Espaciais; 2002.
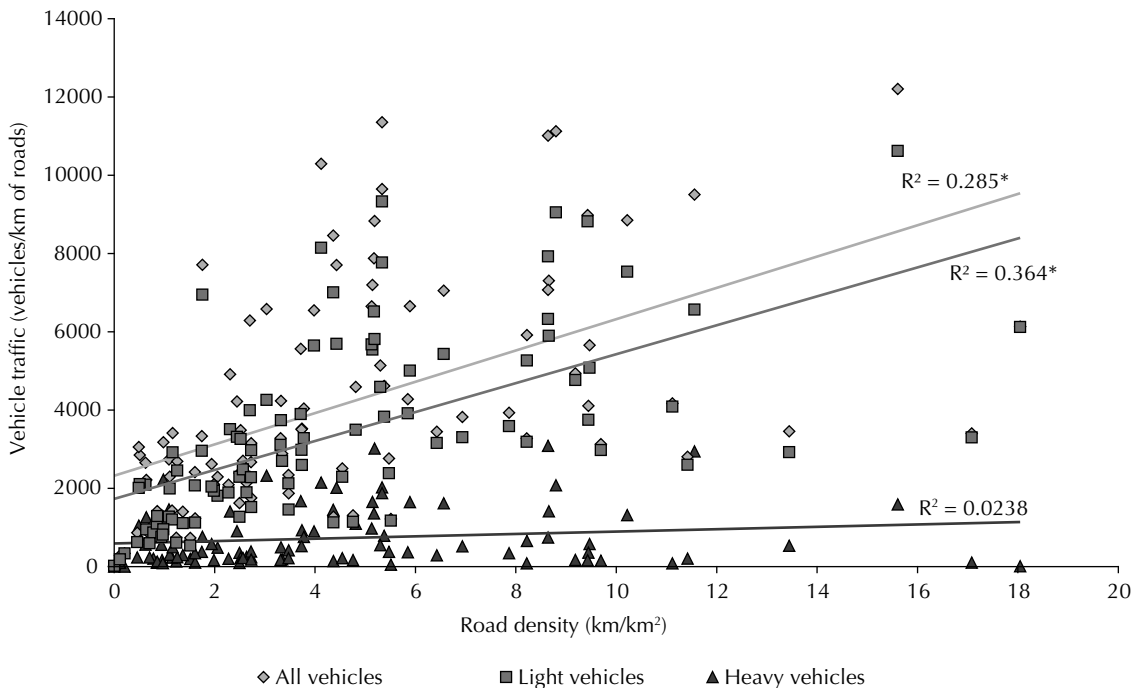
Figure 1. Scatter plot for overall, light and heavy traffic volume and road density. São Paulo, Southeastern Brazil, 2007.

highest quartile of exposure to road traffic up to 300 m from the residence was 32% higher (95%CI: 6;65) than that found in the lowest quartile.

Maheswaran and Elliott[7] investigated mortality from stroke in the UK and found an relative risk (RR) of 1.7 (95%CI: 1.4;1.9) of death from stroke in men living within 200 m of a major road compared with those living at a distance of more than 1,000 m.

Medina-Ramon et al[10] investigated mortality from heart failure in Worcester, US. The interquartile increase in traffic volume up to a distance of 100 m from residence was associated with a HR of 1.15 (95%CI: 1.05;1.25) and up to a distance of 300 m was 1.09 (95%CI: 1.01;1.19). The mortality risk decreased with increasing distance to bus routes (HR = 0.88 [95%CI: 0.81;0.96]) and was higher for those who lived up to 100 m from a highway or 50 m from a bus route (HR = 1.30 [95%CI: 1.13;1.49]).

Rosenlund et al[11] found increased motor vehicle-related exposure to air pollution and risk of coronary disease in Rome. The RR was 1.03 (95%CI: 1.00;1.07) for every increment of 10 μg/m³ in NO_2. Stronger associations were found in fatal (RR = 1.07 [95%CI: 1.02;1.12]) and non-hospital cases (RR = 1.08 [95%CI: 1.02;1.13]).

In Germany there was found an association between coronary heart disease and exposure to vehicle emissions in people living up to 150 m from a main road compared to those living >150 m (OR = 1.85 [95%CI: 1.21;2.84]).[5] But no association was found with $PM_{2.5}$ measured by monitoring stations, which stresses the importance of using indirect measures to assess motor vehicle-related air pollution.

In a case-control study conducted in Boston, U.S.,[12] the interquartile increase in traffic near the residence was associated with OR of 1.4 (95%CI: 1.02;1.07) for acute myocardial infarction. For this outcome, living near highways showed an OR of 1.05 (95%CI: 1.03; 1.06).

One of the limitations of the current study was not analyzing data about behaviors that might affect the development of cardiovascular diseases such as smoking. However, in their study, Kan et al[6] adjusted for risk factors (smoking, obesity, cholesterol, hypertension) but it did not significantly change their results.

A potential confounder controlled for in the analysis was socioeconomic condition. Lower socioeconomic condition has been associated with higher prevalence of smoking,[11] obesity and lower schooling and, consequently, less prevention and lower access to health care.[7] This may explain the statistically significant association between average monthly per capita income in the districts and mortality rates from cardiovascular diseases.

The use of place of residence as an indicator of exposure to motor vehicle-related air pollution is another limitation of the study since it may not be the place where people spent most of their time and therefore does not reflect the actual exposure scenario,[4] and may result in exposure misclassification.

The estimated risk of death from cardiovascular diseases did not take into consideration residential mobility as PROAIM death certificates provide information only on the last place of residence of an individual. However, a study of intra-urban residential mobility in the metropolitan area of São Paulo showed an average time of residence of 14 years,[d] suggesting a significant exposure length.

Another potential limitation of this study is that we used mortality data from 2005 and motor vehicle traffic information from 2007. Traffic counts and simulation
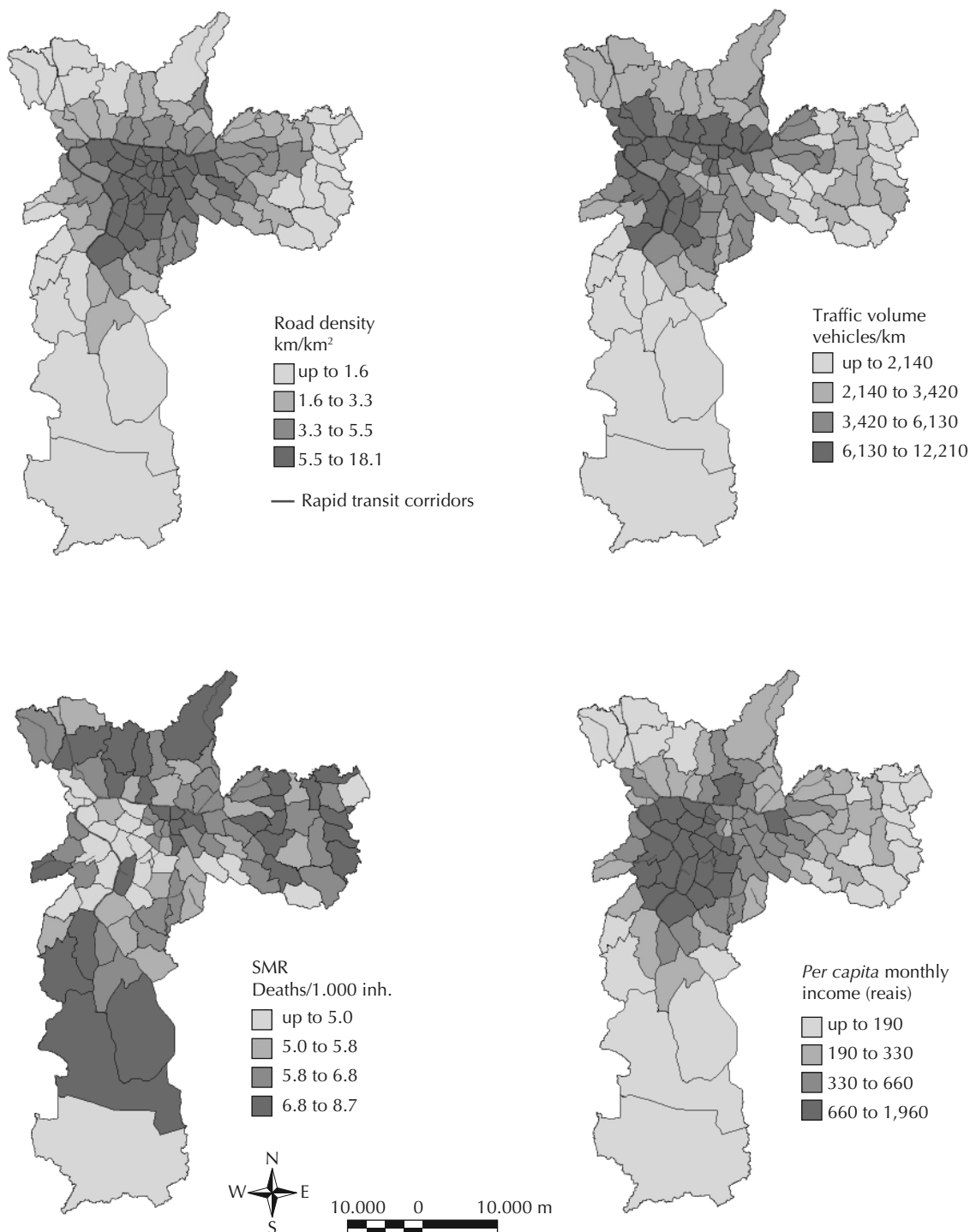

Figure 2. Interquartile ranges of road density, traffic volume, monthly *per capita* income and standardized mortality rate for cardiovascular diseases. São Paulo, Southeastern Brazil, 2007.

Table 1. Means and standard deviations of independent variables, r² and coefficients obtained in the spatial regression analysis. São Paulo, Southeastern Brazil, 2007.

Variable	Mean (SD)	Constant	β	r^2	p-value
Average monthly *per capita* income (reais)	479.11 (421.52)	6.61	−0.0015	0.204	<0.001
Inhabitants per health service	16,299 (9,009)	3.63	0.000028	0.107	0.06
Light traffic volume (vehicles/km)	3,396.5 (2,345.9)	4.62	−0.0000822	0.086	0.16
Heavy traffic volume (vehicles/km)	729.9 (748.8)	4.05	0.000065	0.075	0.72
Traffic volume (vehicles/km)	4,126.36 (2,865.6)	4.41	−0.0000488	0.080	0.31
Road density (km/km²)	4.51 (3.8)	4.22	−0.017	0.074	0.64

Table 2. Multiple spatial regression models. São Paulo, Southeastern Brazil, 2007.

Model[a]	Variables of exposure	Constant	β	r^2	p-value
1	Road density	6.63	0.096	0.25	0.017
2	Light traffic volume	6.56	0.0000237	0.21	0.703
3	Heavy traffic volume	6.55	0.0000821	0.20	0.636
4	Traffic volume	6.55	0.0000212	0.21	0.663

[a] Adjusted for *per capita* income

are conducted by the Traffic Engineering Company every 10 years and the most current and complete information at the time of the study was from 2007. However, we believe that there have been no significant changes in traffic distribution in the last two years, and a possible increase in vehicle flow during this period was likely consistent in the city.

No association between traffic volume and SMR was seen, even after stratifying by traffic type (light/heavy). This can be partly attributed to limited accuracy and timeliness of traffic count and simulation, which are estimated as annual averages disregarding potential daily and annual variations in traffic flow. Another explanation could be low speed of vehicles in more congested, narrower central roads with emission of high volume of pollutants despite low vehicle flow.

Traffic estimates were stratified by vehicle type (light and heavy) and thus allowed to identify the type of fuel used, as heavy vehicles use diesel while light vehicles generally use gasoline or ethanol in Brazil. This was a limitation pointed out by Kan et al[6] as their traffic data did not have this level of detail.

The association between living near major roads and mortality from cardiovascular diseases can be attributed to traffic noise as both exposures (traffic noise and pollution) are concurrent.[9] It is likely that they may have an effect at the same time, or one is confounding the other in cardiovascular disease-related outcomess.[4] The measures used do not reflect weather conditions that may influence the interaction between air pollutants and their entrainment.

Although no association between traffic volume and SMR for cardiovascular diseases was found, the study results obtained from road density data in the districts support the use of this information to construct indirect measures of exposure. These measures can be especially useful in sites where there is no air quality monitoring while investigating potential negative impacts of motor vehicle traffic on people's health.

[d] Barbon AL. Mobilidade residencial intra-urbana em grandes centros: região metropolitana de São Paulo - estudo de caso [master's dissertation]. Campinas: Pontifícia Universidade Católica de Campinas; 2003.

REFERENCES

1. Anenberg SC, Horowitz LW, Tong DQ, West JJ. An estimate of the global burden of anthropogenic ozone and fine particulate matter on premature human mortality using atmospheric modeling. *Environ Health Perspect*. 2010;118(9):1189-95. DOI:10.1289/ehp.0901220

2. Brook RD, Franklin B, Cascio W, Hong Y, Howard G, Lipsett M, et al. Air pollution and cardiovascular disease: a statement for healthcare professionals from the Expert Panel on Population and Prevention Science of the American Heart Association. *Circulation*. 2004;109(21):2655-71. DOI:10.1161/01.CIR.0000128587.30041.C8

3. Davies HW, Vlaanderen JJ, Henderson SB, Brauer M. Correlation between co-exposures to noise and air pollution from traffic sources. *Occup Environ Med*. 2009; 66(5):347-50. DOI:10.1136/oem.2008.041764

4. Hoek G, Brunekreef B, Goldbohm S, Fischer P, Van Den Brandt PA. Association between mortality and indicators of traffic-related air pollution in the Netherlands: a cohort study. *Lancet*. 2002;360(9341):1203-9. DOI:10.1016/S0140-6736(02)11280-3

5. Hoffmann B, Moebus S, Stang A, Beck EM, Dragano N, Mohlenkamp S, et al. Residence close to high traffic and prevalence of coronary heart disease. *Eur Heart J*. 2006;27(22):2696-702. DOI:10.1093/eurheartj/ehl278

6. Kan H, Heiss G, Rose KM, Whitsel EA, Lurmann F, London SJ. Prospective analysis of traffic exposure as a risk factor for incident coronary heart disease: the atherosclerosis risk in communities (ARIC) study. *Environ Health Perspect*. 2008;116(11):1463-8. DOI:10.1289/ehp.11290

7. Maheswaran R, Elliott P. Stroke mortality associated with living near main roads in England and Wales: a geographical study. *Stroke*. 2003;34(12):2776-80. DOI:10.1161/01.STR.0000101750.77547.11

8. Marcilio I, Gouveia N. Quantifying the impact of air pollution on the urban population of Brazil. *Cadernos Saude Publica*. 2007;23 Sup l 4:S529-36. DOI:10.1590/S0102-311X2007001600013

9. Mead MN. Noise pollution: the sound behind heart effects. *Environ Health Perspect*. 2007;115(11):536-57. DOI:10.1289/ehp.115-a536b

10. Medina-Ramón M, Goldberg R, Melly S, Mittleman MA, Schwartz J. Residential exposure to traffic-related air pollution and survival after heart failure. *Environ Health Perspect*. 2008;116(4):481-5.

11. Rosenlund M, Picciotto S, Forastiere F, Stafoggia M, Perucci CA. Traffic-related air pollution in relation to incidence and prognosis of coronary heart disease. *Epidemiology*. 2008;19(1):121-8. DOI:10.1097/EDE.0b013e31815c1921

12. Tonne C, Melly S, Mittleman M, Coull B, Goldberg R, Schwartz J. A case-control analysis of exposure to traffic and acute myocardial infarction. *Environ Health Perspect*. 2007;115(1):53-7. DOI:10.1289/ehp.9587

13. World Health Organization. Who Air Quality Guidelines Global Update 2005. Report on a Working Group Meeting Bonn, Germany 23-24 October 2006; Geneva; 2006.

14. Zhu Y, Hinds WC, Kim S, Sioutas C. Concentration and size distribution of ultrafine particles near a major highway. *J Air Waste Manag Assoc*. 2002;52(9):1032-42.