



Impact of light rail transit on traffic-related pollution and stroke mortality

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Abstract

Objectives This paper evaluates the changes in vehicle exhaust and stroke mortality for the general public residing in the surrounding area of the light rail transit (LRT) in Houston, Texas, after its opening.

Methods The number of daily deaths due to stroke for 2002–2005 from the surrounding area of the original LRT line (exposure group) and the control groups was analyzed using an interrupted time-series analysis. Ambient concentrations of acetylene before and after the opening of LRT were also compared.

Results A statistically significant reduction in the average concentration of acetylene was observed for the exposure sites whereas the reduction was negligible at the control site. Poisson regression models applied to the stroke mortality data indicated a significant reduction in daily stroke mortality after the opening of LRT for the exposure group, while there was either an increase or a considerably smaller reduction for the control groups.

Conclusions The findings support the idea that LRT systems provide health benefits for the general public and that the reduction in motor-vehicle-related air pollution may have contributed to these health benefits.

Keywords Stroke mortality · Light rail transit · Intervention · Vehicle exhaust · Poisson regression models · Interrupted time-series analysis

Introduction

Interest in the effect of transportation-related air pollution on public health has increased dramatically in recent years. It is now generally accepted that automobile pollution poses significantly more harmful health impacts than previously realized. A comprehensive review of the health effects of transport-related air pollution edited by Krzyzanowski et al. (2005) concluded that transport-related air pollution contributes to an increased risk of death from cardiopulmonary causes and increases the risk of respiratory symptoms and diseases. Krzyzanowski et al. (2005) advocate the promotion of highly efficient, service-oriented, and clean public transport and improvements in the flow of traffic.

More recently, the effect of air pollution on stroke has received increasing attention in air pollution epidemiology. According to the Centers for Disease Control and Prevention, stroke is the fifth leading cause of death in the United States (Centers for Disease Control and Prevention 2015). There is growing evidence of the association between air pollution and stroke (Hong et al. 2002; Wellenius et al. 2012; Korek et al. 2015; Shah et al. 2015). Shah et al. (2015) conducted a systematic review and meta-analysis on short-term exposure to air pollution and stroke, and found that admission to the hospital for stroke or mortality from stroke was associated with an increase in the concentrations of carbon monoxide, sulfur dioxide, nitrogen dioxide, PM_{2.5}, and PM₁₀. A study by Wellenius et al. (2012) found that ischemic stroke risk was 34% higher (95% confidence

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interval, 13–58%; $P < 0.001$) on days with moderate $PM_{2.5}$ levels compared to days with low levels. These associations are most strongly associated with pollution from local or transported traffic emissions. A study by Hong et al. (2002) also demonstrated an association between exposure to air pollutants and stroke mortality, and showed that the elderly and women carry greater risks of stroke mortality due to the effect of particulate pollutants. Korek et al. (2015) investigated the risk of stroke from long-term exposure to air pollution from road traffic and found suggestive evidence of an association even at comparatively low levels of air pollution.

Despite extensive literature on the health effects of air pollution, relatively few studies have investigated the changes in health outcomes after interventions that led to improved ambient air quality (see, e.g., Friedman et al. 2001; Dockery et al. 2013; Johnston et al. 2013). A recent review by Henschel et al. (2012) concluded that there is consistent evidence that decreased air pollution levels following an intervention in general resulted in health benefits for the assessed population. Brauer et al. (2013) identified two short-term mitigation strategies for reducing population exposure to traffic-related air pollution, reducing vehicle emissions, and modifying existing infrastructure, and suggested that policies and regulations that target existing infrastructure and vehicles are likely to be most effective in reducing exposure because they operate at the population level. In this respect, public transportation has received increasing attention with the recognition of its widely agreed benefits at the intersection of health and transportation. While providing mobility solutions for transportation, new and expanded transit service improvements greatly help in reducing vehicle emissions and traffic congestion, and reaching out public health objectives.

Light rail transit (LRT) is a major green transportation infrastructure. As shown by Puchalsky (2005), the emissions from LRT are considerably less than those from automobiles. Similar emission reductions from LRT were also confirmed in other studies (e.g., Chen and Whalley 2012; Porter et al. 2012). The potential health benefits that LRT may bring for its users have also been studied. For instance, MacDonald et al. (2010) found a significant association between the use of LRT systems and reductions in body mass index over time and meeting weekly recommended physical activity levels. On the other hand, quantification of the links between air pollution and public health are quite challenging since there are various uncertainties that might influence the process (Battelle and TTI 2014). While it is generally expected that LRT could bring air quality benefit for general public, to the best of our knowledge, no study has examined the potential health benefits that can be achieved through the reduction of vehicular emissions accompanied by the arrival of LRT.

The main objective of this study is to provide a first step in developing an evidence-based assessment of the health benefits of an LRT line after its opening for general public. This is achieved by evaluating the changes in stroke mortality and automobile exhaust emissions in the surrounding area of the Metropolitan Transit Authority of Harris County, Texas (Houston METRO) LRT line through an interrupted time-series design and analysis with a control group. It is important to note here that while automobile exhaust represents a complex mixture of various air pollutants including VOCs, PM, CO, NO_x, etc., this study focuses on the emission of acetylene to assess traffic-related pollution. This is because acetylene is emitted mainly by automobile exhaust, unlike other pollutants that are also emitted by other pollution sources (e.g., industrial sources), and the magnitude of the automobile emissions is proportional to the emission of acetylene. Small other source of acetylene, such as welding, certain chemical industries, and fires, might also exist, but are expected to be of little influence (Whitby and Altwicker 1978; Fortin et al. 2005; Warneke et al. 2007).

In addition to providing important insights on the relative risk of stroke mortality associated with the opening of LRT, this study contributes to the emerging evidence linking health and traffic-related pollution.

Methods

Study population

The study population consisted of the residents of Harris County, Texas. The area of interest was the surrounding area of the original Houston METRO LRT line. The original 7.5 mile Red Line (or North Line) took place on January 01, 2004 and ran from the Texas Medical Center to Downtown, with 16 stations along its route.

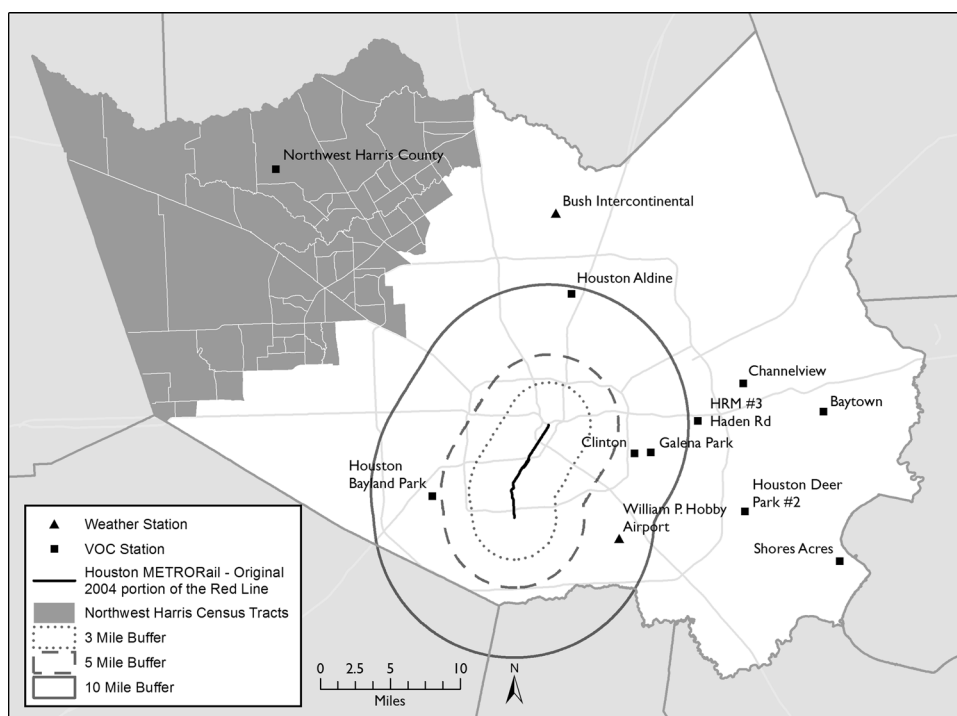
Data used

Health data

We obtained mortality data from the Texas Department of State Health Services Center for Health Statistics for 2002–2005. We extracted data concerning the number of daily deaths due to stroke [World Health Organization's International Classification of Diseases, 10th revision (ICD-10) code I60–I69].

Decedents whose residences at the time of death were near the LRT—whose information was extracted using the 3-, 5-, and 10-mile buffers surrounding the LRT—were selected as the LRT exposure group. Figure 1 shows

Fig. 1 Map of the Houston LRT Line and Air Pollution Monitoring Stations in Harris County, Texas, USA



the location of the LRT and the buffer areas in a 3-, 5-, and 10-mile radius.

For control groups unaffected by the intervention (the non-LRT exposure group), we considered two groups of population. The first group was those whose residences at the time of death were in a northwest region consisting of 67 census tracts (shaded in Fig. 1) that are distant from the LRT (control group 1). The second group was those whose residences at the time of death were outside the 10-mile buffer area falling within the Harris County (control group 2). The assignment of two control groups was arranged to ensure that the results are not very sensitive to the selection of a control group.

Weather data

We obtained meteorological data for 2002–2005 from the National Oceanic and Atmospheric Administration's National Climatic Data Center. Specifically, we used daily 24 h average temperature and dew point temperature data collected from the Automated Surface Observation System stations located at William P. Hobby Airport and George Bush Intercontinental Airport in Houston in the analysis. Figure 1 shows the location of two weather stations. Daily 24 h average temperature and dew point temperature for years 2002–2005 in Houston range from 34.5 to 89.3 (with mean 69.7 and standard deviation 12.5) and 9 to 77.5 (with mean 60.2 and standard deviation 14.1), respectively.

Traffic-related pollution data

We used the ambient concentrations of acetylene to estimate changes in automobile exhaust emissions. As aforementioned, it is well known that acetylene is emitted mainly by automobile exhaust and the magnitude of the automobile emissions is proportional to the emission of acetylene (see, e.g., Fortin et al. 2005; Warneke et al. 2007). As a result, acetylene has often been used as a tracer for automobile exhaust. Figure 1 shows ten monitoring sites in Harris County, Texas, collecting 24 hour canister volatile organic compound (VOC) data (acetylene is one of the measured species) every 6 days.

Out of the ten VOC monitoring sites shown in Fig. 1, the nine sites (excluding the Northwest Harris County monitoring site) are considered exposure sites in this study. These nine VOC monitoring sites are close to major highways and are likely affected by changes in automobile emissions accompanied by the opening of the LRT. Since the most predominant wind in the Houston area is from the southeast, automobile emissions from the VOC monitoring sites located in the east of the LRT buffer areas in addition to those belonging to the 10 mile buffer area in Fig. 1 may also affect the LRT buffer areas.

A distant monitoring site, the Northwest Harris County station, was set aside to be used as a control site for traffic-related pollution. The Northwest Harris County monitoring site is located farthest from the buffer areas and from major highways, and automobile emissions from that site are not

likely to affect the buffer areas considering that the most predominant wind in the Houston area is from the southeast.

Statistical analysis

Several analyses were performed to examine the changes in automobile exhaust emissions and stroke mortality before and after the opening of LRT. To assess the change in exposure to vehicle exhaust for the LRT exposure group, we first calculated the spatial average of acetylene levels across the nine monitoring sites (excluding the control site of Northwest Harris County). We then compared the average concentrations of acetylene and the log of acetylene for the 2-year before and after the intervention periods for the exposure and control sites. We defined the 2-year before period to be January 1, 2002, through December 31, 2003, and the after period to be January 1, 2004, through December 31, 2005. We also computed the average number of daily deaths due to stroke for before and after the intervention periods for the LRT exposure and control groups. To investigate whether the effect of LRT on stroke mortality changes with shorter before and after periods, the 3-month before period (October 2003–December 2003) and the 3-month after period (January 2004–March 2004) were also considered in the analysis.

To further assess the effect of the intervention on stroke mortality, we performed interrupted time-series analysis (see, e.g., Campbell and Stanley 1966; Campbell and Russo 1999) for each LRT exposure group defined by 3-, 5-, and 10-mile buffers surrounding the LRT based on daily mortality time-series data from 2002 to 2005. We employed Poisson regression models to estimate the percent change in the relative risk for stroke mortality associated with the opening of LRT while controlling for seasonal and long-term trends, day of the week (weekday versus weekend), and the effects of meteorology. The periods before and after the intervention were included as an indicator variable, smoothing terms for calendar time, temperature, and dew point temperature using natural spline smoothers. Indicator variables for weekday (coded as 1 for weekdays and 0 for weekend) and extreme temperature (coded as 1 for values less than the 5th percentile or greater than the 95th percentile, and 0 for all other temperature values) were also included in the model. Specifically, we considered the following model:

$$\begin{aligned}
 y_t &\sim \text{Poisson}(\lambda_t), \\
 \log(\lambda_t) &= \beta_0 + \beta_1 \times \text{Intervention}_t + \beta_2 \times \text{WorkDay}_t \\
 &\quad + \beta_3 \times \text{ExtremeTemp}_t \\
 &\quad + \beta_4 \times ns(\text{DATE}_t, df_{\text{DATE}}) + \beta_5 \\
 &\quad \times ns(\text{TEMP}_t, df_{\text{TEMP}}) + \beta_6 \\
 &\quad \times ns(\text{DEWP}_t, df_{\text{DEWP}}),
 \end{aligned}$$

Table 1 Average acetylene concentrations for each year during the study period (2002–2005) in Harris County, Texas, USA

Area	Period	Year	Average acetylene level (in parts per billion carbon-ppbC)
Exposure sites	Before	2002	1.49
		2003	1.50
	After	2004	1.41
		2005	1.17
	Percent change (average)		
Control site	Before	2002	0.79
		2003	0.83
	After	2004	0.82
		2005	0.78
	Percent change (average)		

Percent changes are computed using the averages for each time period (i.e., before and after periods), i.e., Percent change = 100 × (average after – average before)/average before

where y_t is the stroke mortality count for day t , $ns(\text{DATE}_t, df_{\text{DATE}})$, $ns(\text{TEMP}_t, df_{\text{TEMP}})$, and $ns(\text{DEWP}_t, df_{\text{DEWP}})$ are natural spline smoothers with degrees of freedom, df_{DATE} , df_{TEMP} , and df_{DEWP} , respectively, and β 's are model parameters.

The degrees of freedom for the natural splines were selected to minimize the Akaike information criterion (AIC) values. We explored 2–4 degrees of freedom (df_{time}) per year for the smoothing of calendar time and selected the one minimizing the AIC. For the smoothing of temperature and dew point temperature, we explored a range of alternative lags and degrees of freedom, but there was no significant difference in AIC or in the estimated coefficient for the intervention. We used 4 df for the smoothing of temperature and 3 df for dew point temperature with 0 lag days for both temperature and dew point temperature. We conducted an identical statistical analysis for control groups.

We also examined the sensitivity of our results to the study period by applying a similar Poisson regression model to the subset of mortality time-series data with the 3-month before period and the 3-month after period. The same covariates except for the smoothing terms for calendar time were included. Seasonality and long-term trends were not included in this model due to the short study period.

Results

Table 1 presents the annual average concentrations of acetylene for the exposure sites (computed as the yearly mean of the spatial averages across the nine sites) and the control site (Northwest Harris County site) during the study period. The results show a noticeable reduction (13%) in the average acetylene level from the before to the after period at exposure sites, while the reduction was negligible (1%) at the control site.

Table 2 summarizes the results of *t* tests that compared average concentrations of acetylene and concentrations of ‘log of acetylene’ for the periods before and after the intervention for each of the exposure and control sites. Supporting the results of Table 1, the statistically significant reduction in the average concentration of acetylene was observed only for the exposure sites.

Table 3 contains the average daily mortality for stroke mortality for each of the before and after periods for the LRT exposure groups and the control groups. The average daily mortality or stroke mortality showed a significant reduction from the before to the after period within the buffer areas (the LRT exposure group), while the average mortality for a distant northwest region from the LRT (control group 1) or outside the 10-mile buffer area (control

group 2) showed either an increase or a considerably less reduction. The reduction in average daily stroke mortality was also found to be larger for the shorter (3-month) before and after periods.

Table 4 shows the results of the Poisson regression models applied to the mortality time-series data with the 2-year window before and after the opening of LRT. We found that the percent reductions for stroke mortality were notably larger for the population belonging to the buffer areas surrounding the LRT (the exposure groups) compared to those for the control groups. In general, as the buffer area surrounding the LRT gets smaller, the percent reduction gets larger although the reduction was not statistically significant for the smallest (3-mile) buffer area possibly due to the limited sample size. For both the 5-mile buffer area and the 10-mile buffer area, the reduction for the selected model (based on the AIC) was statistically significant. For the control groups, there is an increase rather than a reduction (for northwest region) or the reduction is considerably smaller (for outside the 10-mile buffer).

Finally, the results of Poisson regression models with the 3-month window before and after the opening of the LRT are presented in Table 5. Again, the percent reductions for stroke mortality are noticeably larger for the population belonging to the buffer areas surrounding the LRT (the

Table 2 Comparison of average concentrations of acetylene and the log of acetylene for the 2-year before and after the intervention periods (2002–2005) for the control site and exposure sites in Harris County, Texas, USA

	Site	Average difference (after–before)	Standard error	95% CI
Acetylene	Exposure sites	−0.194	0.094	−0.380 to −0.009
	Control site	−0.008	0.062	−0.131 to 0.115
Log (acetylene)	Exposure sites	−0.126	0.055	−0.234 to −0.019
	Control site	−0.033	0.073	−0.177 to 0.112

Average difference = Average for after period – Average for before period

Statistically significant results at the 95% confidence level are denoted in bold

Table 3 Average daily stroke mortality for each period during the study period (2002–2005) and buffer area with a varying radius in Harris County, Texas, USA

Study period	Study group	Before	After	Percent change
4 years (before: 2 years, after: 2 years)	Inside 3-mile buffer	0.52	0.45	−14
	Inside 5-mile buffer	1.01	0.89	−12
	Inside 10-mile buffer	2.17	1.91	−12
	Northwest region (control group 1)	0.34	0.36	5
	Outside 10-mile buffer (control group 2)	1.54	1.52	−1
6 months (before: 3 months after: 3 months)	Inside 3-mile buffer	0.52	0.37	−28
	Inside 5-mile buffer	1.17	0.79	−33
	Inside 10-mile buffer	2.46	1.66	−30
	Northwest region (control group 1)	0.29	0.35	20
	Outside 10-mile buffer (control group 2)	1.42	1.47	3

Percent change = 100 × (after – before)/before

Table 4 Percent changes in stroke mortality estimated for the 2-year window before and after the Opening of the light rail transit on January 1, 2004, in Harris County, Texas, USA

Study group	$df_{time}/year$	Percent change	95% CI	Akaike information criterion (AIC)
Inside 3-mile buffer	2	-46.9*	-72.0 to 0.6	2687.0
	3	-44.2	-74.7 to 23.0	2688.4
	4	-43.5	-78.2 to 46.3	2692.8
Inside 5-mile buffer	2	-39.3*	-60.4 to -6.8	3763.1
	3	-32.5	-60.1 to 14.1	3765.6
	4	-16.5	-54.7 to 53.7	3769.6
Inside 10-mile buffer	2	-33.3*	-50.6 to -10.0	5079.9
	3	-40.3	-58.6 to -13.9	5080.8
	4	-27.8	-52.8 to 10.5	5080.3
Northwest region (control group 1)	2	21.3*	-40.7 to 148.3	2233.4
	3	9.5	-54.3 to 162.3	2240.0
	4	24.8	-53.7 to 236.5	2243.8
Outside 10-mile buffer (control group 2)	2	-4.1*	-31.9 to 35.1	4509.4
	3	-9.7	-40.4 to 36.7	4514.3
	4	-7.0	-42.3 to 49.9	4522.6

$df_{time}/year$ denotes the degrees of freedom per year used in the natural splines for calendar time
 Percent change was computed by $100 \times (e^\beta - 1)$ where β is the estimated regression coefficient for intervention
 Statistically significant changes at the 95% confidence level are denoted in bold
 *The selected model ($df_{time}/year$) for each group based on Akaike information criterion (AIC)

Table 5 Percent changes in stroke mortality estimated for the 3-month window before and after the opening of the light rail transit on January 1, 2004, in Harris County, Texas, USA

Study group	Percent change	95% CI
Inside 3-mile buffer	-30.8	-57.2 to 11.9
Inside 5-mile buffer	-34.1	-52.4 to -8.8
Inside 10-mile buffer	-30.4	-44.6 to -12.7
Northwest region (control group 1)	-9.6	-48.0 to 57.2
Outside 10-mile buffer (control group 2)	-4.9	-27.0 to 23.9

Percent change was computed by $100 \times (e^\beta - 1)$ where β is the estimated regression coefficient for intervention
 Statistically significant changes at the 95% confidence level are denoted in bold

exposure groups) than the reductions for the control groups. The estimated percent reductions for the 5- and 10-mile buffer areas were comparable to those for the longer study period, and their statistical significance remained.

Discussion

We evaluated changes in stroke mortality that are relevant to the general public residing in the surrounding area of the Houston METRO LRT system after its opening in January 2004. To the best of our knowledge, this study was the

first evaluation of the potential health benefits of LRT on stroke mortality accompanied by reduction in automobile-related air pollution. The interrupted time-series design and analysis with control groups were used to determine the impact of the opening of the LRT. The results indicate that the operation of the LRT is associated with reductions in daily stroke mortality and pollution from automobile emissions in Houston. We estimated that daily stroke mortality in the area surrounding the LRT was reduced by more than 30%, and automobile exhaust emissions were reduced by 13% (estimated by reduction in acetylene level). On the other hand, the reductions for stroke mortality for control groups were less than 10% at most (for the short study period) and automobile exhaust emissions at the control site was reduced only by 1%. We analyzed the ambient concentrations of acetylene to estimate changes in automobile exhaust emissions. It is well known that acetylene is emitted mainly by automobile exhaust. Although automobiles emit many other pollutants (e.g., benzene, PM, CO, NOx, etc.), those are also emitted by other pollution sources (such as industrial sources) not just by automobiles. By focusing on acetylene, which is emitted mainly by automobile exhaust, we aimed at reducing the chance of confounding of the effect of LRT on vehicle exhaust with the effects of other sources. On the other hand, future studies would benefit from additional assessment of the effect of LRT on multiple pollutants from automobile exhaust in addition to acetylene in order to obtain a more comprehensive

and comparative picture of the effects of LRT on those pollutants.

One of the critical assumptions in our study is that no other abrupt changes that could possibly affect vehicle exhaust emissions or stroke mortality occurred around the same time as the opening of the LRT. Otherwise, the observed effects of the LRT would not be separated from those of other confounding factors. It did not seem that there were other changes in a major type of transportation infrastructure around January 1, 2004, in Houston. To control for the changes in extraneous factors, we also estimated the changes in stroke mortality and vehicle exhaust emissions for the control group (site). The reductions for the control group (site) for stroke mortality and vehicle exhaust emissions from the before to the after periods were considerably smaller than those for the exposure group (sites), which support that the observed reductions for the exposure group (site) might be attributed to the opening of the LRT. We tested the sensitivity of our results to the use of different control group and the length of the study period. The results were not sensitive to the use of different control groups or the length of study period.

The reduction in stroke mortality that might have been related to a reduced traffic/congestion may also come from the reduction in traffic noise and not just from the reduction in automobile emissions. For instance, a recent study published in 2015 (Halonen et al. 2015) suggests a link between long-term exposure to road traffic noise and deaths, as well as a greater risk of stroke, particularly in the elderly. The study found that significant nighttime noise from road traffic, ranging from 55 to 60 decibels, was linked to a 5% increased stroke risk but only among the elderly. The health benefit of LRT on stroke mortality may also come from increased physical activity (i.e., walking to and from the LRT station) in addition to the reduction in automobile-related air pollution.

Given the current data, it is not possible to separate the effect of LRT on mortality into the benefit due to the reduction in air pollution and the benefits due to increased physical activity or decreased traffic noise. Considering that stroke mortality changes are for the entire population within proximity of the LRT system, not just for LRT users, the portion of the effect of LRT on stroke mortality due to increased physical activity (that applies to LRT users only) is deemed small compared to the health benefit obtained from the decreased automobile-related air pollution (that applies to all residents within proximity of the LRT system). Likewise, the portion of the effect of LRT on stroke mortality due to the reduction in traffic noise is expected to be small because it would mainly apply to the people residing very close to the roadways.

In practice, assessing the overall health benefit of LRT on stroke mortality for the general public—whether it is

from the reduction of traffic-related air pollution, reduction of traffic noise, or increased physical activity—is of interest to both transportation and health fields. Our results provide a first evidence of health benefits of LRT on stroke mortality for the general public, while complemented by traffic-related pollution analysis. Future studies would benefit from examining whether associations between LRT and stroke mortality change by different ethnic group, gender, age group, or socio-economic status, as well as by different land use and built environment characteristics.

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Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

Ethical approval This article does not contain any studies with human participants or animals performed by any of the authors.

References

- Battelle and Texas A&M Transportation Institute (2014) MAP-21 air quality and congestion mitigation measure outcomes assessment study: Project performed in cooperation with the U.S. Department of Transportation, Federal Highway Administration, and the Environmental Protection Agency. Technical report. FHWA-HEP-15-002. September 2014. http://www.fhwa.dot.gov/environment/air_quality/cmaq/research/outcomes_assessment/index.cfm. Accessed 1 May 2016
- Brauer M, Reynolds C, Hystad P (2013) Traffic-related air pollution and health in Canada. *Can Med Assoc J* 185(18):1557–1558
- Campbell DT, Russo MJ (1999). *Social experimentation*. Sage, Thousand Oaks
- Campbell DT, Stanley JC (1966) *Experimental and quasi-experimental designs for research*. Rand McNally, Chicago
- Centers for Disease Control and Prevention (2015) Stroke facts. <http://www.cdc.gov/stroke/facts.htm>. Accessed 24 Mar 2015
- Chen Y, Whalley A (2012) Green Infrastructure: the effects of urban rail transit on air quality. *Am Econ J Econ Policy* 3(4):58–97
- Dockery DW, Rich DQ, Goodman PG, Clancy L, Ohman-Strickland P, George P, Kotlov T (2013) Effect of air pollution control on mortality and hospital admissions in Ireland. Research report 176. Health Effects Institute, Boston
- Fortin TJ, Howard BJ, Parrish DD, Goldan PD, Kuster WC, Atlas EL, Harley RA (2005) Temporal changes in US benzene emissions inferred from atmospheric measurements. *Environ Sci Technol* 39(6):1403–1408

- Friedman MS, Powell KE, Hutwagner L, Graham LM, Teague GW (2001) Impact of changes in transportation and commuting behaviors during the 1996 Summer Olympic Games in Atlanta on air quality and childhood asthma. *JAMA* 285(7):897–905
- Halonon JI, Hansell AL, Gulliver J, Morley D, Blangiardo M, Fecht D, Toledano MB, Beevers SD, Anderson HR, Kelly FJ, Tonne C (2015) Road traffic noise is associated with increased cardiovascular morbidity and mortality and all-cause mortality in London. *Eur Heart J* 36(39):2653–2661
- Henschel S, Atkinson R, Zeka A, Le Tertre A, Analitis A, Katsouyanni K, Chanel O, Pascal M, Forsberg B, Medina S, Goodman PG (2012) Air pollution interventions and their impact on public health. *Int J Public Health* 57(5):757–768
- Hong YC, Lee JT, Kim H, Ha EH, Schwartz J, Christiani DC (2002) Effects of air pollutants on acute stroke mortality. *Environ Health Perspect* 110(2):187–191
- Johnston FH, Hanigan IC, Henderson SB, Morgan GG (2013) Evaluation of interventions to reduce air pollution from biomass smoke on mortality in Launceston, Australia: retrospective analysis of daily mortality 1994–2007. *BMJ* 346:e8446
- Korek MJ, Bellander TD, Lind T, Bottai M, Eneroth KM, Caracciolo B, de Faire UH, Fratiglioni L, Hilding A, Leander K, Magnusson PK, Pedersen NL, Östenson CG, Pershagen G, Penell JC (2015) Traffic-related air pollution exposure and incidence of stroke in four cohorts from Stockholm. *J Expo Sci Environ Epidemiol* 25:517–523
- Krzyzanowski M, Kuna-Dibbert B, Schneider J (2005) Health effects of transport-related air pollution. World Health Organization Regional Office Europe, Copenhagen
- MacDonald JM, Stokes RJ, Cohen DA, Kofner A, Ridgeway GK (2010) The effect of light rail transit on body mass index and physical activity. *Am J Prev Med* 39:105–112
- Porter C, Hussey L, Kall D, Dempster J, Noland R, Hanson C, Zemba S, Armstrong S, Tosta N, Boese J, Cavanaugh M, Hewett D (2012) Assessing and comparing environmental performance of major transit investments. Final report for TCRP Project H-41. January 2012. http://onlinepubs.trb.org/onlinepubs/tcrp/tcrp_w55.pdf. Accessed 1 May 2016
- Puchalsky CM (2005) Comparison of emissions from light rail transit and bus rapid transit. *Transport Res Rec* 1927:31–37
- Shah ASV, Lee KK, McAllister DA., Hunter A, Nair H, Whiteley W, Langrish JP, Newby DE, Mills NL (2015) Short term exposure to air pollution and stroke: A systematic review and meta-analysis. *BMJ* 350:h1295
- Warneke C, McKeen SA, De Gouw JA, Goldan PD, Kuster WC, Holloway JS, Williams EJ, Lerner BM, Parrish DD, Trainer M, Fehsenfeld FC, Kato S, Atlas EL, Baker A, Blake DR (2007) Determination of urban volatile organic compound emission ratios and comparison with an emissions database. *J Geophys Res Atmos* 112:D10S47
- Wellenius GA, Burger MR, Coull BA, Schwartz J, Suh HH, Koutrakis P, Schlaug G, Gold DR, Mittlemen MA (2012) Ambient air pollution and the risk of acute ischemic stroke. *Arch Intern Med* 172(3):229–234
- Whitby RA, Altwicker ER (1978) Acetylene in atmosphere—sources, representative ambient concentrations and ratios to other hydrocarbons. *Atmos Environ* 12:1289–1296