

Impacts of Transportation Emissions on the Risk of Mortality: Findings from the Literature and Policy Implications

Center for Transportation, Environment, and Community Health
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by
Razieh Nadafianshahamabadi, Mohammad Tayarani, H.Oliver Gao

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7. Author(s) Razieh Nadafianshahamabadi (0000-0001-6885-1010) Mohammad Tayarani (0000-0002-3405-3800) Oliver.H Gao (0000-0002-7861-9634)		8. Performing Organization Report No.	
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16. Abstract Background: Exposure to vehicle emissions have been considered a cause of several negative health outcomes including mortality. The existing findings are too inconsistent to drive a well-founded exposure-response function to be fully exploited to curb the negative impacts of transportation systems on public health. In this study, we investigate the association between exposure to air pollution and mortality. We then evaluate how using different air quality methods may result in detecting different health outcomes. Methods: We conduct an analysis of reviewing a representative sample of main published studies that specifically focused on the association between vehicle air pollution and mortality. Results: Our study finds that vehicle air pollution may increase the risk of mortality through a high association. The risk of overall mortality increases by 5% per 10 µg/m ³ increase in NO ₂ concentration, 2% per unit of traffic intensity on the road, and 7% per unit of distance closer to the road. Conclusion: The findings imply the role of exposure to vehicle emissions in increasing risk of mortality. The method used to detect the health outcomes can alter the health finding from positive to null or vice versa and even extensively affect the analysis outcomes. The results suggest the need for establishing indicators to benchmark the performance of air quality methods and emphasize the necessity to integrate public health measures into urban and transportation planning process.			
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1. Introduction

Vehicle traffic is responsible for a large portion of toxic air pollutant emissions in urban areas such as particular matter, ozone, carbon monoxide, and nitrogen oxide. Epidemiological studies found an association between exposure to vehicle emissions and adverse health outcomes (Allen et al., 2009; Brugge et al., 2007; Franco Suglia et al., 2008; Gan et al., 2010a; Garshick et al., 2003; Gauderman et al., 2007; HEI, 2010; McConnell et al., 2006; Peters et al., 2004; Wilhelm and Ritz, 2003), such as chronic respiratory and heart diseases. Epidemiological studies first estimate exposure to vehicle emissions and then use a cohort or a case-control approach to evaluate the association between vehicle pollution and adverse health outcomes.

While the research in recent decades shows the adverse effects of air pollution on health, but it is still too inconsistent to introduce a well-founded concentration-response function to quantify the relationship between traffic-related air pollution and health outcomes. Methods of quantifying air pollution which model how air pollutants disperse over the surrounding terrain range from simple surrogate models to more complex models that provide higher temporal resolutions for air pollutant concentration (Jerrett et al., 2005; Özkaynak et al., 2013). The main approaches used in most epidemiologic studies are proximity, intensity, land use regression (LUR), and dispersion models. Using proximity models is the most basic approach, which considers distance to the source of pollution, such as distance to a major roadway, to estimate exposure to vehicle emissions (Jerrett et al., 2005). Intensity approach considers the intensity of the source of pollution, such as traffic volume. LUR measures the relationship between pollutant concentrations and relevant environmental variables (Briggs et al., 2000). Dispersion model estimates air pollution concentrations using input data on emissions and numerical formulations such as Gaussian plume equations (Jerrett et al., 2005; Pan et al., 2017).

The models of quantifying the adverse effects of traffic-related air pollution on health perform differently and there has been little research, if any, on evaluating the performance of different models on epidemiological findings. There is some evidence that the more sophisticated dispersion models perform better than the simple dispersion models, but there has been little quantitative research investigating how different models affect the results of epidemiological studies. A study by Molitor et al. (Molitor et al., 2007) compared the association between exposure to NO₂ and lung function using CALINE4, an air dispersion model, and a monitoring method. They report that the monitoring method overestimates the association by 15% compared to the dispersion model. Wu et al. (2007) compared the performance of the CALINE4, a LUR model, a traffic intensity model, and concentration measured at the nearest central site monitoring station. They found that the highest difference in the association between exposure to vehicle emissions and adverse pregnancy outcomes is 17%, observed for the monitored PM_{2.5} concentration versus the concentration modeled with CALINE4. Zou et al. (2009) reviewed a large number of studies to compare the performance of the proximity and hybrid models and found that the results from the proximity methods are questionable. However, most prior research has been limited to small study samples. Moreover, prior research mostly accounted for ambient air pollution and not specifically for vehicle air pollution. This distinction is crucial because the rapid decline in vehicle air pollution concentration from the edge of the road and the chemical composition of traffic emissions are different from ambient air pollution.

In this study, we aimed to understand how different air pollution exposure methods may result in different outcomes by conducting an analysis of reviewing a representative sample of main published studies that

specifically focused on the association between vehicle air pollution and mortality. The contribution of this study is to find whether using different exposure approaches cause differences in reported health outcomes and whether sophisticated approaches have also resulted in more accurate epidemiological inferences. The outcome of this study contributes to discussions of investing in expensive sophisticated air pollution methods and allows for the evaluation of benefits from the change in adverse health outcomes due to vehicle emissions and the determination of whether the investments are justifiable.

2. Method

Cases of mortality attributed to transportation-related air pollution are derived from the health outcomes itemized in table 1. These studies are included because they met the following criteria: are human-based epidemiologic studies written in English; provide a quantitative value, such as relative risk (RR), hazard ratio (HR), or odds ratio (OR) and their confidence intervals (CI) or standard errors (SE), for change in mortality associated with vehicle air pollution; and clearly estimate the contribution to pollution concentration from road traffic instead of ambient or background air pollution. Vehicle air pollution is recognized as different from ambient air pollution if a study uses one of the following approaches to measure pollution exposure: proximity models, which measure the distance to traffic sources, such as roads/highways; intensity models, which define traffic intensity, such as traffic volume; land use regression models which measure NO₂; and dispersion models, which are based on the road and mobile sources. If there were multiple studies that considered the same or overlapping cohorts, we included the study that had the longest follow-up period or had a greater sample of the population under study. Excluded studies either do not investigate association between vehicle emission and mortality or they did investigate impacts of ambient air pollution and not traffic-related air pollution. We also exclude those studies that specifically investigated the association between mortality and vehicle emission in a particular subgroup of the population such cases with lung transplant, myocardial survivors, survival of heart failure, and post-stroke cases.

Table 1 shows the characteristics of the total of twenty studies included in our study. The studies are published between 2000-2017, ten of which cover North America. A total of 31,943,248 adults who were followed up from the 1980s to 2013 were included in this study.

Table 1. Characteristics of the reviewed studies

Author	Measure	Country	Year	Age	Study type
Beelen et al., 2008 (Beelen et al., 2008)	RR	Netherland	1987-1996	55-69	Prospective Cohort
Cesaroni et al., 2013 (Cesaroni et al., 2013)	HR	Rome	2001-2010	≥ 30	Cohort
Chen et al., 2013 (Chen et al., 2013)	RR	Canada	1982-2004	35-85	Cohort
Finkelstein et al., 2004 (Finkelstein et al., 2004)	RR	Canada	1992-2001	≥40	Cohort
Finkelstein et al., 2005 (Finkelstein et al., 2005)	RR	Canada	1985-1999	≥40	Cohort
Gehring et al., 2006 (Gehring et al., 2006)	RR	Germany	1980s-1990s	50-59	Cohort
Hoek, 2002 (Hoek et al., 2002)	RR	Netherland	1986-1994	55-69	Cohort
Jerrett et al., 2009 (Jerrett et al., 2009)	RR	Canada	1992-2002	NA	Cohort
Jerrett et al., 2005 (Jerrett et al., 2005)	RR	USA	1982-2000	NA	Cohort
Maheswaran & Elliott, 2003 (Maheswaran and Elliott, 2003)	RR	UK	1990-1992	≥45	Ecological
Rosenlund et al., 2009 (Rosenlund et al., 2009)	OR	Sweden	1985-1996	15-79	Case-Control
Gan et al., 2010 (Gan et al., 2010b)	RR	Canada	1994-1998	45-85	Cohort
Yorifuji et al., 2013 (Yorifuji et al., 2013)	HR	Japan	1999-2009	NA	Cohort
Turner et al., 2017 (Turner et al., 2017)	HR	USA	1982-2004	≥30	Cohort
Crouse et al., 2015 (Crouse et al., 2015)	HR	Canada	1991-2006	25-89	Cohort
Thurston et al., 2016 (Thurston et al., 2016)	HR	USA	1982-2004	≥30	Cohort
Raaschou-Nielsen et al., 2012 (Raaschou-Nielsen et al., 2012)	MRR	Denmark	1993-2009	50-64	Cohort
Pedde et al., 2017 (Pedde et al., 2017)	OR	USA	2009-2013	NA	Case-Crossover
Bidoli et al., 2016 (Bidoli et al., 2016)	RR	Italy	1990-2010	NA	Ecological
Halonan et al., 2016 (Halonan et al., 2016)	RR	UK	2003-2010	≥25	-

RR: relative risk; HR: hazard ratio; OR: odds ratio; MRR: mortality rate ratio; NA: not available.

To synthesize the data, the effect estimates related to NO₂, PM_{2.5}, CO, and PM₁₀ were all converted to a standard increment, 10 µg/m³, in pollution concentration. Parts per billion (ppb) units of concentration were converted into micrograms per cubic meter using a factor of 1.88 for NO₂, and 1.145 for CO. The following formula was then used to convert the other values in µg/m³ to 10 µg/m³.

$$RR_{\text{Standard}} = \exp(\ln(RR_{\text{Origin}}) / \text{Increment}_{\text{Origin}} \times \text{Increment}_{\text{Standard}}) \quad (1)$$

The hazard ratio, odds ratio, and relative risk of mortality were considered the measure of association between air quality and mortality across studies. While each study might report different effect sizes using different approaches, we extracted the effect estimates that were adjusted for confounding factors and

discussed as the main results by the authors. One of the following equations is used to calculate the standard error of the effect.

$$\text{Standard Error (SE)} = (\ln RR - \ln \text{Lower CI}) / 1.96 \quad (2)$$

$$\text{Standard Error (SE)} = (\ln \text{Upper CI} - \ln RR) / 1.96 \quad (3)$$

To combine the outcomes of studies and calculate the pooled effect estimates, a random effects model technique was performed, which accounts for the risk of heterogeneity in the effect size reported, unlike the fixed-effect estimation. Two-sided tests with a significance level of 0.05 were conducted. Q and I^2 were both used to evaluate the heterogeneity within the studies. The null hypothesis that the studies were homogeneous was rejected if the p-value was less than 0.10 or the I^2 was greater than 50% (Phan et al., 2015). Subgroup analyses were also performed on the cause of mortality (all-cause mortality, cardiovascular mortality, and respiratory mortality) and air pollutants (NO_2 , $\text{PM}_{2.5}$, CO, traffic intensity, and distance to the road). Forest plots were created to provide visual representations of the distribution of studies (i.e., gray squares), subtotal effects (i.e., purple diamonds) and overall effects (i.e., red diamonds). The plots help to interpret the results where for each individual study the wider the confidence interval means less reliability and the overall effects are significant if diamonds do not cross the dashed red line.

3. Results

3.1. All-Cause Mortality

The pooled estimate of all-cause mortality risk due to exposure to NO_2 concentration, traffic intensity, and distance to highway contains fifteen studies (Table 2 and Figure 1). Among the modeling methods, Jerret et al. (2009) reported the highest association, with an 18% increase in the risk of all-cause mortality associated with a $10 \mu\text{g}/\text{m}^3$ increase in exposure to NO_2 concentration, modeled by a LUR model. While the highest association reported by the intensity method was 1.04 (95% CI=1.03-1.06) by Cesaroni et al. (Cesaroni et al., 2013), the proximity method resulted in a 1.41 (95% CI=0.94-2.12) risk reported by Hoek et al. (Hoek et al., 2002). The estimated overall risk ratio of 1.04 indicates that exposure to vehicle pollution can significantly increase all-cause mortality by 4% (95% CI=1.02-1.06). Exposure to pollution, measured by the distance from the place of residence to major roads, has the highest but non-significant association with the all-cause mortality, 1.07 (95% CI=0.98-1.15). The overall effects of the exposure to NO_2 and traffic intensity both have positive and significant effects on mortality, by a 5% and 2% increase in risk, respectively. The overall 88% I^2 shows that high heterogeneity existed among these studies and indicates that the variability across studies is due to genuine differences rather than chance.

TABLE 1. Pooled Estimate of Relative Risks of All-Cause Mortality from Exposure to Traffic-Related Emission

Study	RR (95% CI)	Metric	Exposure
Raaschou-Nielsen et al., 2012	1.08 (1.01, 1.15)	NO ₂ (10 µg/m ³)	Dispersion
Halonen et al., 2016	0.99 (0.97, 1.01)	NO ₂ (10 µg/m ³)	Dispersion
Cesaroni et al., 2013	1.03 (1.02, 1.04)	NO ₂ (10 µg/m ³)	LUR
Jerrett et al., 2009	1.18 (0.95, 1.45)	NO ₂ (10 µg/m ³)	LUR
Yorifuji et al., 2013	1.12 (1.07, 1.17)	NO ₂ (10 µg/m ³)	LUR
Crouse et al., 2015	1.03 (1.02, 1.04)	NO ₂ (10 µg/m ³)	LUR
Subtotal effect I² = 96.18%, p < 0.0001	1.05 (1.00, 1.10)		
Beelen et al., 2008	1.02 (0.97, 1.12)	Traffic intensity in 100 m	Intensity
Cesaroni et al., 2013	1.04 (1.03, 1.06)	Traffic intensity in 150 m	Intensity
Beelen et al., 2008	1.03 (1.00, 1.08)	Traffic intensity on nearest road	Intensity
Cesaroni et al., 2013	1.01 (1.00, 1.02)	Traffic intensity on the road 10,000< vehicles per day	Intensity
Subtotal effect I² = 69.48%, p = 0.0087	1.02 (1.00, 1.04)		
Jerrett et al., 2005	0.98 (0.89, 1.06)	Distance to highway (1000 m)	Proximity
Gehring et al., 2006	1.29 (0.93, 1.78)	Distance to major road (50 m)	Proximity
Finkelstein et al., 2004	1.18 (1.02, 1.38)	Distance to major road (50 m) and highway (100 m)	Proximity
Hoek et al., 2002	1.41 (0.94, 2.12)	Distance to major road (50 m) and highway (100 m)	Proximity
Beelen et al., 2008	1.05 (0.97, 1.12)	Distance to major road	Proximity
Subtotal effect I² = 57.63%, p = 0.045	1.07 (0.98, 1.15)		
Overall effect I² = 88.67%, p < 0.0001	1.04 (1.02, 1.06)		

Note: RR=relative risk; CI= confidence interval; µg/m³ = microgram per cubic meter; m= meter; LUR: land-use regression; null hypothesis that studies are homogeneous is rejected if p < 0.10 or I² > 50%.

Figure 1 provides a summary of the studies by examining the change in all-cause mortality risk associated with traffic exposure. While most studies expectedly indicated an increased relative risk of mortality due to exposure to vehicle pollution, some studies did not provide enough evidence of a significant association. Four studies found a significant relationship between all-cause mortality and a 10 µg/m³ increase in NO₂, a well-known traffic marker (Cesaroni et al., 2013; Crouse et al., 2015; Raaschou-Nielsen et al., 2012; Yorifuji et al., 2013). Two studies that used traffic intensity as a measure found a significant but weak association between change in mortality and vehicle pollution (Beelen et al., 2008; Cesaroni et al., 2013). Only one study found a significant association between distance to the road and risk of mortality (Finkelstein et al., 2004), but the rest mostly found a strong but insignificant association (Beelen et al., 2008; Gehring et al., 2006; Hoek et al., 2002).

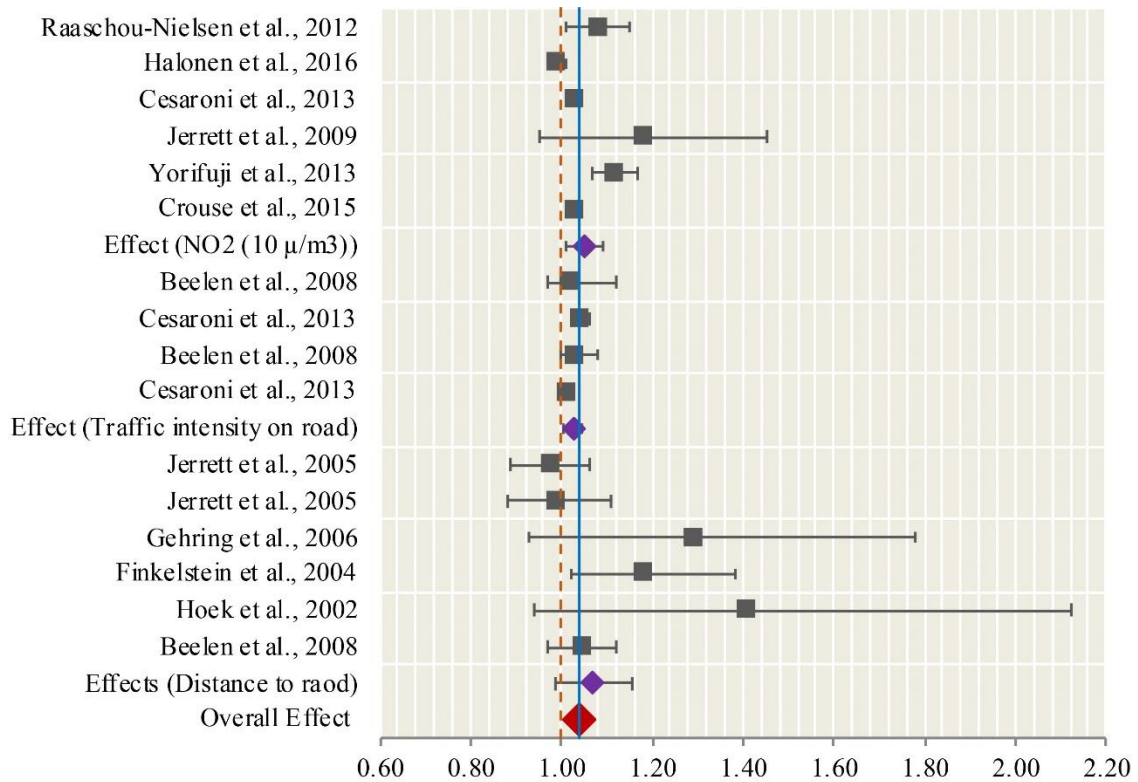


FIGURE 1. Forest Plot of Relative Risk (RR) Of All-Cause Mortalities Associated with Traffic-Related Air Pollution

3.2. Cardiovascular Mortality

We estimate 1.07 as a pooled relative risk of cardiovascular mortality due to exposure to traffic-related emission, which is higher than the overall risk of all-cause mortality. Table 3 shows the estimates from individual studies together with pooled estimates of the relative risk of the cardiovascular mortality associated with traffic-related air pollution. Figure 2 presents a forest plot to summarize the findings of studies that examine the change in cardiovascular mortality associated with traffic-related air pollution. Overall relative risk from sub-groups ranges between 1.04 for the traffic intensity method to 1.17 for the proximity method. Those who live within 50 meters of major roads have a higher risk of cardiovascular mortality. As expected, the results also show that a 10 µg/m³ increase in exposure to PM_{2.5} is associated with a 1.08 (1.04-1.11) risk of mortality, which is higher than the cardiovascular mortality risk of exposure to NO₂ where 10 µg/m³ increase in exposure is associated with 1.06 (1.02-1.10) risk of mortality. Similar to all-cause mortality, the intensity measures found a weaker association, 1.04 (1.02-1.05), between exposure to vehicle emissions and cardiovascular mortality.

TABLE 2. Pooled Estimate of Relative Risks of Cardiovascular Mortality from Exposure to Traffic-Related Emission

Study	RR (95% CI)	Metric	Exposure
Jerrett et al., 2005	1.05 (0.89, 1.24)	Distance to highway (1000 m)	Proximity
Beelen et al., 2008	1.05 (0.93, 1.18)	Distance to major road	Proximity
Finkelstein et al., 2005	1.38 (1.07, 1.78)	Distance to major road (50 m) and highway (100 m)	Proximity
Hoek et al., 2002	1.95 (1.09, 3.52)	Distance to major road (50 m) and highway (100 m)	Proximity
Gan et al., 2010	1.29 (1.18, 1.41)	Distance to major road (50 m) and highway (150 m)	Proximity
Chen et al., 2013	1.04 (1.00, 1.08)	Distance to major road (50 meter)	Proximity
Gehring et al., 2006	1.70 (1.02, 2.81)	Distance to major road (50 meter)	Proximity
Subtotal effect I² = 92.91%, p <0.0001	1.17 (1.03, 1.31)		
Rosenlund et al., 2009	1.02 (0.98, 1.05)	CO (10 µg/m ³)	Dispersion
Cesaroni et al., 2013	1.03 (1.02, 1.04)	NO ₂ (10 µg/m ³)	LUR
Chen et al., 2013	1.09 (1.05, 1.12)	NO ₂ (10 µg/m ³)	LUR
Rosenlund et al., 2009	1.03 (1.00, 1.07)	NO ₂ (10 µg/m ³)	Dispersion
Yorifuji et al., 2013	1.22 (1.15, 1.30)	NO ₂ (10 µg/m ³)	LUR
Crouse et al., 2015	1.03 (1.02, 1.03)	NO ₂ (10 µg/m ³)	LUR
Raaschou-Nielsen et al., 2012	1.08 (0.89, 1.30)	NO ₂ (10 µg/m ³)	Dispersion
Halonen et al., 2016	0.93 (0.86, 0.98)	NO ₂ (10 µg/m ³)	Dispersion
Subtotal effect I² = 99.12%, p <0.0001	1.06 (1.02, 1.10)		
Rosenlund et al., 2009	1.20 (1.04, 1.40)	PM ₁₀ (10 µg/m ³)	Dispersion
Cesaroni et al., 2013	1.06 (1.04, 1.08)	PM _{2.5} (10 µg/m ³)	Dispersion
Thurston et al., 2016	1.13 (0.96, 1.33)	PM _{2.5} (10 µg/m ³)	Monitors
Subtotal effect I² = 62.45%, p=0.0833	1.08 (1.04, 1.11)		
Beelen et al., 2008	1.00 (0.92, 1.08)	Traffic intensity in 100 m	Intensity
Cesaroni et al., 2013	1.05 (1.02, 1.07)	Traffic intensity in 150 m	Intensity
Beelen et al., 2008	0.96 (0.88, 1.03)	Traffic intensity on the road with 1,255 to 10,000 vehicles per day	Intensity
Beelen et al., 2008	1.11 (0.99, 1.25)	Traffic intensity on the road with 10,000< vehicles per day	Intensity
Cesaroni et al., 2013	1.03 (1.01, 1.05)	Traffic intensity on the road with 10,000< vehicles per day	Intensity
Subtotal effect I² = 2.23%, p=0.2309	1.04 (1.02, 1.05)		
Overall effects I² = 97.49%, p <0.0001	1.07 (1.04, 1.09)		

Note: RR=relative risk; CI= confidence interval; µg/m³= microgram per cubic meter; m= meter; LUR: land-use regression; null hypothesis that studies are homogeneous is rejected if p < 0.10 or I² > 50%.

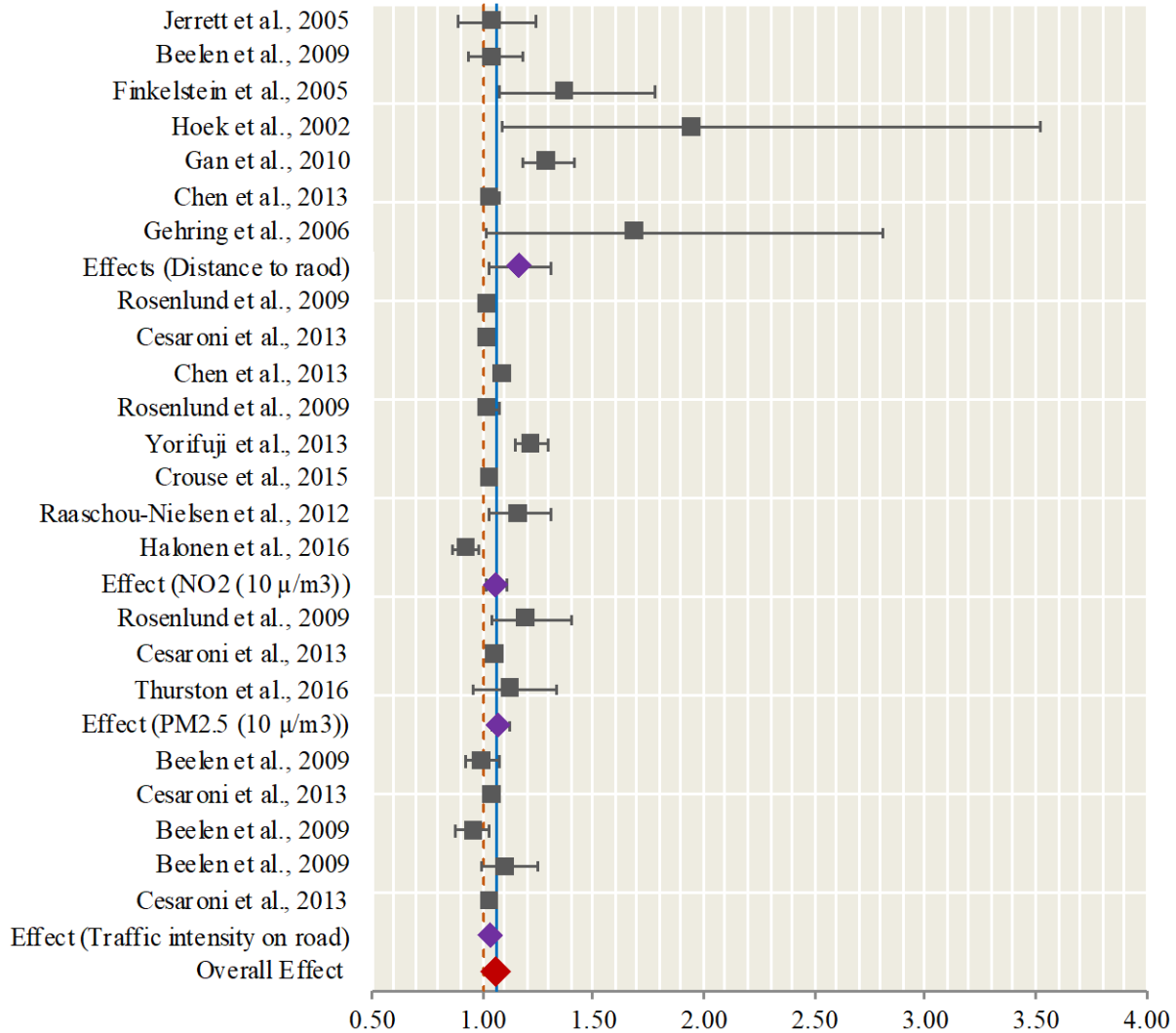


FIGURE 2. Forest Plot of Relative Risk (RR) of Cardiovascular Mortalities Associated with Traffic-Related Air Pollution

3.3. Respiratory Mortality

Unlike what the literature has reported on the high association between respiratory diseases (HEI, 2010), such as asthma and COPD, a weak association was found between respiratory mortality and exposure to vehicle emissions, with the exception of a study by Jerrett et al. (2005) that reports a 44% increase in the risk of respiratory mortality for those living within 500 meters of major roadways. We find that no matter what method is used, all resulted in a weak association (Figure 3). The overall results were also affected by low heterogeneity ($I^2 = 18.37\%$) showing that less inconsistency exists among these studies. Aside from proximity to roads, at the highest level, there were 20% increases in the risk of mortality due to respiratory diseases reported by Beelen et al. (2008) and Yorifuji et al. (2013) in which the latter used the proximity method and the former used a $10 \mu\text{g}/\text{m}^3$ increase in the exposure to NO_2 (Table 4).

TABLE 3. Pooled Estimate of Relative Risks of Respiratory Mortality from Exposure to Traffic-Related Emission

Study	RR (95% CI)	Metric	Exposure
Jerrett et al., 2005	1.44 (0.94, 2.21)	Distance to highway (500 m)	Proximity
Beelen et al., 2008 ^a	1.19 (0.91, 1.56)	Distance to major road	Proximity
Beelen et al., 2008 ^b	1.20 (0.98, 1.47)	Distance to major road	Proximity
Finkelstein et al., 2005 ^a	0.96 (0.72, 1.27)	Distance to major road (50 m) and highway (100 m)	Proximity
Pedde et al., 2017 ^a	1.06 (0.76, 1.5)	Distance to road (150 m)	Proximity
Pedde et al., 2017 ^a	1.02 (0.95, 1.10)	Distance to road (150-300 m)	Proximity
Bidoli et al., 2016 ^b	1.04 (0.92, 1.23)	Distance to road (100 m)	Proximity
Bidoli et al., 2016 ^b	1.05 (0.97, 1.12)	Distance to road (100-500 m)	Proximity
Subtotal Effect I² = 0, p = 0.3186	1.04 (1.01, 1.08)		
Cesaroni et al., 2013 ^a	1.03 (1.00, 1.06)	NO ₂ (10 µg/m ³)	LUR
Cesaroni et al., 2013 ^b	1.04 (1.02, 1.07)	NO ₂ (10 µg/m ³)	LUR
Yorifuji et al., 2013 ^b	1.20 (1.03, 1.40)	NO ₂ (10 µg/m ³)	LUR
Crouse et al., 2015 ^c	1.05 (1.03, 1.06)	NO ₂ (10 µg/m ³)	LUR
Crouse et al., 2015 ^d	1.02 (1.01, 1.04)	NO ₂ (10 µg/m ³)	LUR
Subtotal Effect I² = 55.43%, p = 0.0118	1.03 (1.02, 1.05)		
Cesaroni et al., 2013 ^a	1.03 (0.97, 1.08)	PM _{2.5} (10 µg/m ³)	Dispersion
Cesaroni et al., 2013 ^b	1.05 (1.01, 1.10)	PM _{2.5} (10 µg/m ³)	Dispersion
Subtotal Effect I² = 0, p = 0.5677	1.04 (1.00, 1.07)		
Beelen et al., 2008 ^a	1.21 (1.02, 1.44)	Traffic intensity in 100 m	Intensity
Beelen et al., 2008 ^b	1.07 (0.93, 1.23)	Traffic intensity in 100 m	Intensity
Cesaroni et al., 2013 ^a	1.08 (1.00, 1.15)	Traffic intensity in 150 m	Intensity
Cesaroni et al., 2013 ^b	1.03 (0.97, 1.09)	Traffic intensity in 150 m	Intensity
Beelen et al., 2008 ^a	1.10 (0.95, 1.26)	Traffic intensity on nearest road	Intensity
Beelen et al., 2008 ^b	1.07 (0.96, 1.19)	Traffic intensity on nearest road	Intensity
Cesaroni et al., 2013 ^a	1.01 (0.95, 1.08)	Traffic intensity on the road with 10,000< vehicles per day	Intensity
Cesaroni et al., 2013 ^b	0.99 (0.94, 1.05)	Traffic intensity on the road with 10,000< vehicles per day	Intensity
Subtotal Effect I² = 27.56%, p = 0.1795	1.04 (1.00, 1.08)		
Overall effects I² = 18.37%, p = 0.0832	1.03 (1.02, 1.05)		

Note: RR=relative risk; CI= confidence interval; µg/m³= microgram per cubic meter; m= meter; LUR: land-use regression; null hypothesis that studies are homogeneous is rejected if p < 0.10 or I² > 50%.

^a Respiratory disease;

^b Lung Cancer;

^c Trachea, bronchus, and lung cancers;

^d Diseases of the respiratory system;

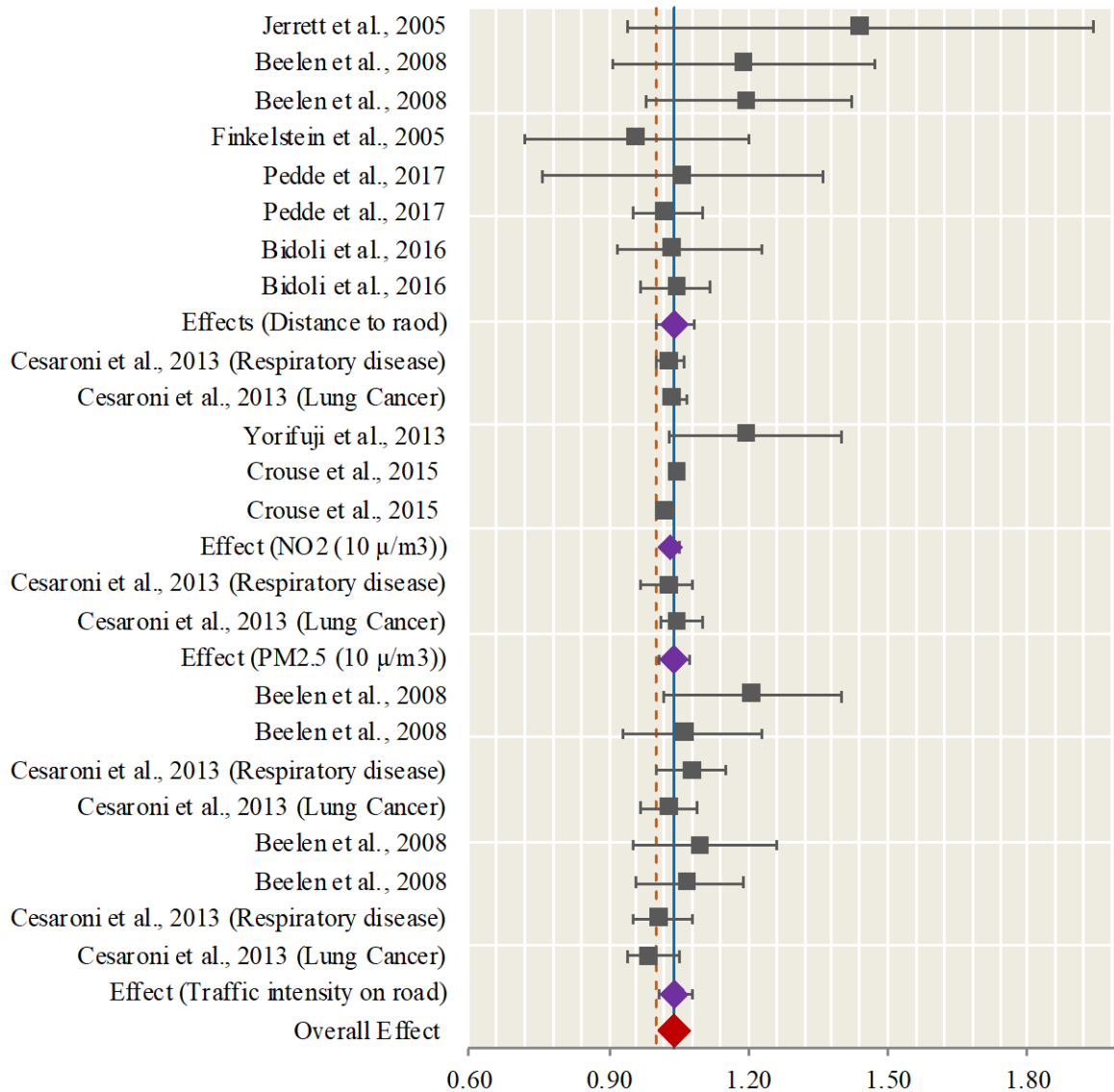


FIGURE 3. Forest Plot of Relative Risk (RR) of Respiratory Mortalities Associated with Traffic-Related Air Pollution

4. Discussion

In this study, we evaluated the association between exposure to vehicle emissions and mortality and analyzed how using different air quality models may result in different results. While the adverse health outcomes of exposure to vehicle emissions are investigated by many researchers, it is still unclear what exposure-response function can fully measure the impacts of pollution from transportation on public health.

We chose mortality over the range of adverse health outcomes due to a larger body of available literature. We found an overall weak but significant association between exposure to vehicle emissions and mortality. Our analysis of twenty epidemiological studies, finds that the risk of mortality increases by exposure to vehicle emissions; 10 µg/m³ increase in NO₂ concentrations increase the risk of overall mortality, cardiovascular mortality, and respiratory mortality by 5%, 6%, and 3% respectively. The risk of

overall mortality, cardiovascular mortality, and respiratory mortality increases by 2%, 4%, and 4%, per unit of traffic intensity and increases by 7%, 17%, and 4% per unit of distance to the road. 10 $\mu\text{g}/\text{m}^3$ increase in $\text{PM}_{2.5}$ concentrations increase the risk of cardiovascular mortality, and respiratory mortality by 8% and 4%, respectively.

Despite these evidences, federally mandated air quality analysis related to the transportation sector provides very little information regarding exposure to air pollutants in vehicle exhaust. One example is the air quality analysis, the inventory analysis, conducted by planning agencies such as Metropolitan Planning Organizations (MPOs). Estimating emission inventory, however, is not an appropriate method to study the negative effects of vehicle emissions on human health because of the high gradient of variation in air pollutant concentration (Tayarani et al., 2018). If MPOs fail to formulate the best plans and projects that address air quality problems, they waste large sums of money (roughly US\$350 billion each year) while also failing to address major issues pertaining to factors such as public health and environmental equity, among other contemporary challenges (Poorfakhraei et al., 2017). Therefore, a more detailed air quality analysis is required not only to evaluate exposure to transportation-related air pollution but also to select transportation projects that reduce the risk of adverse health effects. The findings along with previous findings (Hankey et al., 2012) implied the necessity of integrated transportation, land use, and health planning so not only to save on urban infrastructure sectors' cost but also to promote preventive medicine and save on public health costs.

In this study, we also evaluated the potential driver of variation in health outcomes, the methods used to measure the exposure to vehicle emission. The results show that different exposure approaches can substantially affect analytical health outcomes. The results indicate that some surrogate models, such as proximity, tend to show a higher association, but traffic intensity, finds a lower association between exposure to vehicle emission and mortality. It seems that for overall mortality and cardiovascular mortality, using proximity methods such as distance to major roads and highways shows higher risk than other methods like LUR and dispersion. For respiratory mortality, the risks taken from all four methods including LUR, dispersion, proximity, and intensity are almost equal. This finding was expected since the literature has shown the variability of air pollution within urban environment (Marshall et al., 2008) and in particular a rapid decline in pollution concentration from the edge of the roads (Karner et al., 2010); thus, traffic intensity measured in a buffer around the roads may be unable to capture the rapid decline in pollution concentration. Based on the results, proximity methods are more reliable than what had been previously described in the literature, since the proximity methods more strongly agree with the mathematical modeling methods, such as air dispersion modeling. However, the uneven spatial distribution of vehicle emission exposure along with the population movement pattern during the daily activity make the study of exposure to vehicle emissions very complex. The health and transportation sectors should consider the tradeoff between the simplicity of using the surrogate models against the accuracy of the mathematical air quality modeling and spatially detailed exposure analysis. Sophisticated models can be extremely complex and data hungry. The relatively large staffing, computational, and data requirements increase costs, while increased complexity limits transparency and increases the risk of unseen modeling errors.

Although the increase in mortality risk due to exposure to vehicle emissions may seem small, one should consider that the majority of available studies have been conducted in the developed countries. These countries have significantly tackled their air pollution challenges through establishing standards such as

the Clean Air Act for the USA. Conducting rigorous epidemiology studies in the areas with high vehicle pollution concentration around the world such as India, Mexico, and Iran we may further underlie the negative role of vehicle emissions on public health.

Although we aimed to cover all available studies, there might be studies that were omitted. While grouping studies together might also affect the calculated overall association, comparability of grouped studies is controlled for gender, age, income, and race. Grouping based on exposure metric methods may have also affected the overall association because every study has defined its own specific exposure metric, which might be slightly different from that of the others. There is also high possibility of publication bias due to the low and insignificant risk of association between exposure to traffic-related emission and mortality.

5. References

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