Environmental Research 157 (2017) 153–159

Contents lists available at ScienceDirect

Environmental Research

journal homepage: www.elsevier.com/locate/envres

The role of traffic noise on the association between air pollution and children's lung function

Meredith Franklin*, Scott Fruin

Department of Preventive Medicine, University of Southern California, Los Angeles, CA, USA

ARTICLE INFO

Keywords:
Noise
Air pollution
Traffic
Children's respiratory health
Lung function
Negative confounding

ABSTRACT

Although it has been shown that traffic-related air pollution adversely affects children's lung function, few studies have examined the influence of traffic noise on this association, despite both sharing a common source.

Estimates of noise exposure ($L_{dn,}$ dB), and freeway and non-freeway emission concentrations of oxides of nitrogen (NO_x, ppb) were spatially assigned to children in Southern California who were tested for forced vital capacity (FVC, n = 1345), forced expiratory volume in 1 s, ($FEV_{1,}$ n = 1332), and asthma. The associations between traffic-related NO_x and these outcomes, with and without adjustment for noise, were examined using mixed effects models.

Adjustment for noise strengthened the association between NO_x and reduced lung function. A 14.5 mL (95% CI −40.0, 11.0 mL) decrease in FVC per interquartile range (13.6 ppb) in freeway NO_x was strengthened to a 34.6 mL decrease after including a non-linear function of noise (95% CI −66.3, −2.78 mL). Similarly, a 6.54 mL decrease in FEV_1 (95% CI −28.3, 15.3 mL) was strengthened to a 21.1 mL decrease (95% CI −47.6, 5.51) per interquartile range in freeway NO_x.

Our results indicate that where possible, noise should be included in epidemiological studies of the association between traffic-related air pollution on lung function. Without taking noise into account, the detrimental effects of traffic-related pollution may be underestimated.

1. Introduction

Numerous studies have examined the association between exposure to traffic-related air pollution and children's respiratory health (e.g., Gauderman et al., 2007; McConnell et al., 2010; Rice et al., 2016; Urman et al., 2014). Long-term exposure to nitrogen oxides (NO, NO_2, NO_x) leads to a reduction in lung development in children (Gauderman et al., 2004, 2007). Fortunately, decreases in air pollution in Southern California over the past 17 years have led to significant reductions in these detrimental effects (Gauderman et al., 2015).

Traffic is also a source of noise, but the joint effects of noise and air pollution on children's respiratory health have not been studied in the U.S. despite high noise exposures. Noise levels in U.S. urban areas generally exceed the World Health Organization (WHO) community noise guideline (WHO, 1999) of 55 dB (dB) for a day-evening noise average that includes a 10 dB evening and night penalty ("L_{dn}"). In downtown Los Angeles during the daytime (9 am-5 pm with no 10 dB penalty), the measured mean noise level at 26 locations was 66.4 dB, exceeding the WHO guideline by more than 10 dB (Lee et al., 2014). At the most extreme, an estimated 90% of New York City residents were exposed to noise levels greater than 70 dB (Neitzel et al., 2012).

In Europe, traffic noise ranks second, behind fine particles, as the environmental risk factor with the highest health impact (Hänninen et al., 2014). Studies of noise as an environmental stressor have shown associations with a variety of health outcomes including annoyance, sleep deprivation, cardiovascular disease prevalence, and premature mortality (Stansfeld, 2015). In children, noise has deleterious effects on behavioral (Tiesler et al., 2013), mental (Dreger et al., 2015), cardiovascular (Belojevic et al., 2008; Bilenko et al., 2013; Liu et al., 2014), and respiratory health (Ising et al., 2003, 2004; Niemann et al., 2006; Linares et al., 2006). In Madrid, noise (> 80% of which is attributed to traffic sources) was found to be the variable most strongly associated with child hospitalization for respiratory causes in general, ahead of cold weather, and for pneumonia, ahead of pollen. These models included NO_x, ozone and PM_{10} (Linares et al., 2006). In the Harz Mountain region of Northern Germany, a study of 400 children found that those with self-reported exposures to heavy lorry and motorcar traffic due to bedrooms facing traffic had high risk ratios for chronic bronchitis 10.8 (95% CI 5.2, 22.4) (Ising et al., 2003). While air pollution and noise were not measured in that study, follow-up

* Correspondence to: 2001 N. Soto St. SSB-202A, Los Angeles, CA 90089, USA.
E-mail address: meredith.franklin@usc.edu (M. Franklin).

http://dx.doi.org/10.1016/j.envres.2017.05.024
Received 9 February 2017; Received in revised form 21 April 2017; Accepted 22 May 2017

measurements of NO_2 and noise outside of child bedroom windows (10 pm – 6 am), along with salivary cortisol in a sub-study of 68 children (Ising et al., 2004) verified that noise levels above 54 dB and NO_2 above ~21 ppb were associated with increased morning cortisol and increased doctor visits for bronchitis. Morning cortisol was a stronger predictor than NO_2, indicating the stressful aspect of traffic noise may have been the driving mechanism of illness.

In this study, we investigated the associations between traffic-related air pollution and noise on children's respiratory health by taking advantage of a large, pre-existing cohort of children in Southern California. While the association between air pollution and respiratory health has been studied extensively using this cohort, the role of noise has not. Our investigation of the dynamics between NO_x, noise, and lung function begins with the *a priori* and scientifically substantiated hypothesis that there is an association between respiratory outcomes (Y) and air pollution (X) (Gauderman et al., 2004, 2007, 2015), and then consider the role of a third variable, noise (Z) on this relationship. We also hypothesized that while X has a direct effect on Y, Z plays an indirect role in that its inclusion in the regression allows for a more concise estimate of the association between X and Y. We specifically tested the associations (marginal and joint) between traffic NO_x exposure, traffic noise, and children's lung function measurements of forced expiratory volume in one second (FEV_1, mL), forced vital capacity (FVC, mL), and asthma.

2. Material and methods

2.1. Study Population

Since its inception in the early 1990s, the Southern California Children's Health Study (CHS) has enrolled over 11,000 children in a series of five cohorts. In this study, we focused on the most recent cohort enrolled in 2002–3 at ages 5–7 years and examined in 2011–12 when they were 14–17 years old. There was a total of 5000 children, and those receiving lung function tests (approximately 1400 children) resided and went to school in eight communities in the greater Los Angeles, California area: Anaheim, Glendora, Long Beach, Mira Loma, Riverside, Santa Barbara, San Dimas, and Upland (Fig. 1). The CHS was designed to capture gradients of traffic emissions, with some communities (e.g. Anaheim) having many study subjects' residential locations close to freeways to ensure that a portion of the cohort would have high freeway emission exposures as well as high noise exposures. Additional details of CHS community and subject selection have been previously reported (Peters et al., 1999; McConnell et al., 2006).

2.2. Health outcomes

Pulmonary function tests were conducted on each child by trained respiratory staff. FEV_1 and FVC were measured using pressure transducer-based spirometers (Screenstar Spirometers, Morgan Scientific, Haverhill, Massachusetts, USA). Asthma status was based on physician diagnoses, confirmed by a written questionnaire that also obtained information including age, sex, self-identified race and ethnic background, parental education, occurrences of acute respiratory illness, exercise, tobacco-smoke exposure (personal smoking or environmental), and house characteristics (air conditioning, age of house, presence of mildew, pets in the home). Ethnic background in the CHS specifically relates to Hispanic ancestry, identifying Caucasian subjects with Hispanic and non-Hispanic ethnicity.

2.3. Environmental exposures

2.3.1. Air pollution

We applied the CALINE4 line source dispersion model (Benson, 1992) to estimate annual average ambient concentration of NO_x from local traffic at each subject's residence for the calendar year preceding each child's lung function test. The CALINE4 dispersion model uses residential locations, roadway geometry, vehicle traffic volume and emission rate by roadway link, and meteorological conditions as inputs. The estimated pollutant exposures included both freeway and non-freeway sources separately, and are regarded as indicators of incremental increases in air pollution over background ambient levels due to primary emissions from local vehicular traffic. CALINE4-estimated freeway NO_x has been shown to explain much of the local-scale spatial variation in annual average ambient NO_2 and NO_x concentrations in Southern California (Franklin et al., 2012). A separate variable for distance from each study subject's residential location to the nearest freeway was also examined.

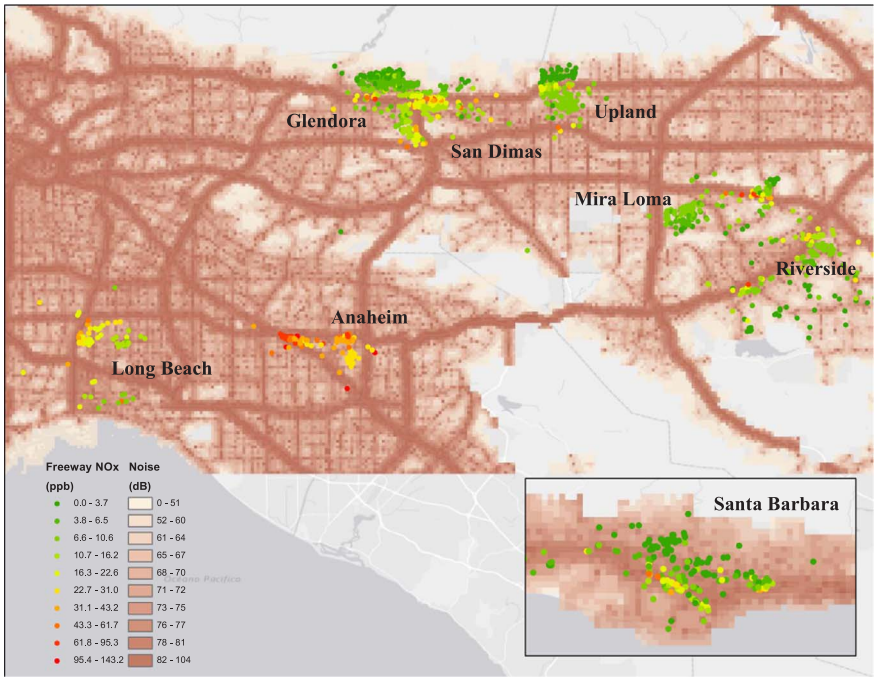

Fig. 1. Map of study area with noise estimates (L_{dn}, dB) in the Southern California region and freeway NO_x concentrations (ppb) at locations of subjects in the 8 CHS communities.