# TOWARD A BETTER UNDERSTANDING OF THE ROLE OF TRANSPORTATION NOISE IN CHRONIC DISEASE: THE VANCOUVER STUDIES

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## Résumé

Le bruit est un problème majeur de santé publique. Un programme de recherche de l'Université de la Colombie-Britannique a examiné le rôle du bruit, de façon indépendante et combiné à la pollution de l'air, dans la causalité des maladies chroniques. Depuis 2000, une série d'études a modélisé le bruit environnemental dû au transport routier, ferroviaire et aérien, étudié les corrélations entre les polluants atmosphériques et sonores et examiné l'association entre le bruit et l'incidence d'un certain nombre de maladies chroniques telles que les maladies coronariennes, le diabète et l'arthrite. Nous avons également examiné l'association du bruit avec des issues défavorables de naissance. Les conclusions de ces études ont été largement citées et incorporées dans des revues systématiques à l'appui des principes directeurs de l'OMS pour bruit communautaire. Cet article résume les méthodes et les résultats de ce programme de recherché.

**Mots clefs :** Le bruit de la circulation et de modélisation du bruit, les maladies chroniques, les résultats à la naissance.

## Abstract

Noise is a major public health problem. A research program at the University of British Columbia has been examining the role of noise, independently and combined with air pollution, in chronic disease causation. Since 2000, a series of studies has modeled environmental noise due to road, rail and air transportation, investigated correlations between air and noise pollutants, and examined the association of noise and incidence of a number of chronic diseases such as coronary heart disease, diabetes and arthritis. We also examined the association of noise with adverse birth outcomes. The findings of these studies have been widely cited and incorporated into reviews supporting WHO community noise guidelines. This paper summarizes the methods and results of this program of research.

**Keywords:** Traffic Noise, Noise Modelling, Chronic Disease, Birth Outcomes.

## 1 Introduction

Noise has been the source of annoyance and sleep disturbance complaints for millennia; through the ages there are accounts of cities trying to reduce noise from tradespeople and from traffic – all well before the advent of the automobile. In the middle of the 20th century, however, the impact of noise on human health began to be more widely studied in a scientific manner. Early studies showed some intriguing results – for example, associations with increased blood pressure - but the early body of evidence was criticized as being contradictory, of generally low quality, and showing "slow evolution in sophistication" [1]. The science however improved and broadened in scope and in 2000, Passchier-Vermeer & Passchier published a review of noise exposure and public health that concluded "…there is sufficient scientific evidence that noise exposure can induce hearing impairment, hypertension and ischemic heart disease, annoyance, sleep disturbance, and decreased school performance…for other effects such as changes in the immune system and birth defects, the evidence is limited" [2]. The quality of studies had improved greatly, with larger cohort studies of noise and cardiovascular disease (CVD) [3] and noise and school performance [4], and a plausible biological model of effects was proposed [5]. Still, there remained uncertainty about the role of noise in traffic-related chronic disease, as noise was considered to be potentially confounded - perhaps highly - by exposure to traffic-related air pollution, a field with a great deal of ongoing research in the early 2000's [6].

At this time at the University of British Columbia, Brauer was undertaking a large-scale traffic-related air pollution study (the Border Air Quality Study, or BAQS) combining exposure data from land-use regression modelling of major air pollutants [7] with "linked" health insurance database resources [8]. The linked-health databases covered the entire population of BC and allowed researchers to follow individuals over long time periods, tracking health events through hospital discharge records, cancer registries, physician billing records, pharmacy usage, as well as data submitted to the BC perinatal registry, and birth and death certificates. BAQS study subjects were assigned exposures via their residential postal code which had been geocoded, and linked to various modeled air-pollutant exposure "surfaces". At the same time, Davies was completing studies on the role of occupational noise in BC sawmills on cardio-vascular disease outcomes [9, 10]. It seemed an obvious next step to combine these programs of inquiry at UBC to examine these two major traffic-related pollutants – air and noise – and the nature of their co-exposure on effects on human health. It would provide one of the first opportunities to examine the

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pollutants simultaneously, and thus examine the interaction of the exposures, in a very strong study setting.

This paper, summarizes the corresponding programme of research at UBC on the combined effects of noise and traffic-related air pollution on human health. It does not include new analyses of the data but does direct the reader to recent systematic reviews on environmental noise and health that incorporate the BC study findings.

# 2 Methods

## 2.1 Exposure assessment – community noise

### Road and rail

We utilized noise-propagation modeling software to create a "noise map" of the 22 municipalities that comprised the Greater Vancouver Regional District (GVRD, now Metro Vancouver). GVRD had a population of 2.1 million at the time; the modeled area was approximately 2,200 $Km^2$. Noise-prediction models estimate community noise exposure through quantitative analysis of the generation (e.g. road traffic), propagation (e.g. topographical effects, atmospheric absorption), and attenuation (e.g. screening effects and building reflections) of various noise sources [11]. We used CadnaA (DataKustik GmbH; Greifenberg, Germany), a model-based computer program, to estimate annual average community noise levels for the year 2003. Model input data included road traffic volumes (from a transportation planning model EMME/2 (INRO Consultants, Montreal, Canada); road-width data (the distance between the center lines of the outer most lanes); and road type (based on the provincial Digital Road Atlas [12], divided into three categories freeway: highway, arterial, or collector and local. Each road type was assigned a specific percentage of truck traffic. In addition, the model took into account the influence of road speed limits, intersections, road gradients, road surface material, elevated sections (bridges), building height, footprint, reflection/absorption characteristics, and land topography. We used GIS Innovations (Tallahassee, Florida, USA) and other available data sources including Google Earth and geographic information systems (GIS; ArcGIS; ESRI, Redlands, CA, USA) to retrieve these road traffic data.

In the study region, the urban rapid transit system (''Skytrain'') is the major railway noise source. Rail noise exposure assessment was based on railway operation data including length of trains, velocity, percentage of disk brakes, and number of each type of train by day, evening, and night. Road and rail noise prediction used the RLS-90 and Schall-03 (edition 1990) models respectively.

### Air

Vancouver International Airport was presumed the dominant source of aircraft noise in the study region. The Airport Authority produces aircraft noise exposure forecast (NEF) contours to describe the noise exposure levels in the area surrounding the airport [13]. The noise contours are created using noise prediction models that take into account aircraft operation information including flightpath, frequency, aircraft types, and local meteorology. We used the noise exposure forecast contours for 2003 to estimate the contribution of aircraft to community noise in the study area.

### Modeling

Based on the above input data, annual day-evening-night A-weighted equivalent continuous noise levels ($L_{DEN}$ dB(A)) were calculated for a 10 X 10 m grid (at 4 m above the ground). The $L_{DEN}$ metric integrates noise levels during the day ($L_{DAY}$, 06:00–18:00), the evening (18:00–22:00), and the night ($L_{NIGHT}$, 22:00–06:00); it reflects increased sensitivity of residents to community noise during evening and night by adding a 5 dB penalty to evening noise levels and a 10 dB penalty to night noise levels. Based on the estimated noise levels for these grids, we calculated annual average noise levels for each 6-character postal code area in the study region by energetically averaging the noise levels of the grids contained in a postal code area (using the Statistics Canada Postal Code Conversion File [14]). Noise levels were calculated for road traffic only and from all sources combined. In the study region, postal code areas vary greatly in size depending on the density of population: in urban areas, a postal code typically represents one side of a city block or a high-rise building; however, in rural areas a postal code may represent a larger area. Because metropolitan Vancouver is a highly urbanized region, the vast majority of the postal codes represent small geographical areas; on average, a residential postal code may include about 35 individuals.

### Air pollution and other exposures

We used high-spatial-resolution land-use regression models to estimate participants' residential exposures to traffic-related air pollutants, including black carbon, $PM_{2.5}$, nitrogen dioxide, and nitric oxide, for the year 2003. Furthermore, these estimates were combined with air quality-monitoring data to calculate monthly concentrations and average concentrations during the 5-year exposure period for each pollutant in each postal code area [7].

## 2.2 Cohort studies

In British Columbia, the mandatory health insurance program provided health care coverage for nearly all residents [8]. We used linked administrative health datasets to assemble two population-based cohorts. Each had a 5-year exposure period (January 1994–December 1998) and a 4-year follow-up period (January 1999–December 2002). In the "adult cohort" all GVRD residents who had registered with the provincial health insurance plan, had resided in the study region during the 5-year exposure period, and were 45–85 years of age were included. The "birth cohort" comprised all live-births in the follow up period in the greater GVRD. We excluded multiple births, children who were missing key information such as birthweight or parity, maternal age at delivery, or who were identified to be of First-Nations descent.

### Data linkage and statistical analysis

During the 5-year exposure period, subjects' exposures to community noise and traffic-related air pollutants were estimated based on averages weighted by time spent at each person's residence (through their residential postal code). For subjects who changed their residences, we calculated equivalent noise levels and average air pollution levels during the exposure period. Among adults, subjects who had partially missing data because of changes in residences (moving from or to areas outside the exposure assessment domain) for more than a total of 15 months, or in more than 3 consecutive months during the exposure period were excluded.

Appropriate statistical modeling analyses were applied in each study to examine relative risks (e.g. cox proportional hazard, logistic regression) and to examine the exposure/response relations (e.g. smoothed splines). Potential confounders were examined at the individual level if available (e.g. age, sex) or at the neighbourhood level if not (e.g. socio-economic status, ethnicity). In the birth cohort, additional individual level co-variates such as maternal age at birth, smoking status, parity etc. were also available and used.

Studies were approved by the institutional review board of The University of British Columbia (Behavioural Research Ethics Board certificate #H08-00185).

## 3 Results

### 3.1 Noise exposure estimation

The resulting noise map for GVRD estimated annual averages of $L_{DEN}$, $L_{Day}$ and $L_{Night}$ for road only, as well as for road, air and light rail [15]. An excerpted area is shown Figure 1 as an illustration. The model did not include freight and passenger trains, industrial sources, or float plane traffic.

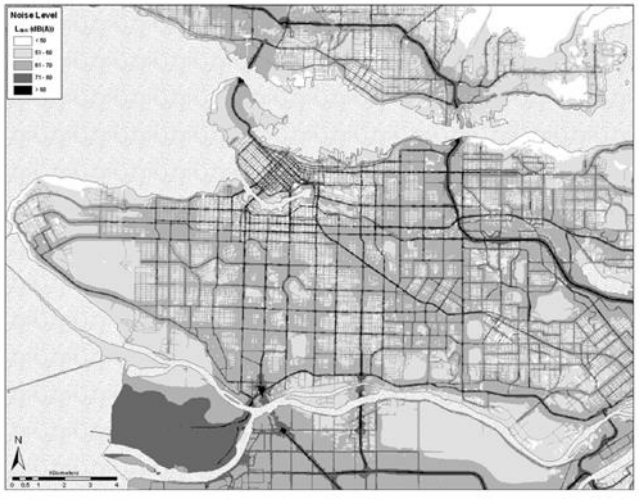


**Figure 1:** An excerpt of the noise map produced for Metro Vancouver, for the year 2003 (from [14])

Summary noise exposure statistics are shown in Table 1. One of the key findings from this study was that the correlations between concentrations levels of air pollutants and noise were quite low, with the exception of carbon black. The correlation coefficient of modelled noise levels with black carbon concentrations was 0.48 (95% CI, 0.47–0.48), but only r = 0.18 (95% CI, 0.17–0.18) for PM2.5. These findings were consistent with correlations between measured noise and pollution data from earlier work done at UBC [16], that countered earlier assumptions of potential confounding; along with other concurrent studies [17] it suggested that it would be reasonable to attempt to examine the independent health effects of noise and air-pollution.

**Table 1:** Summary noise and air pollution model statistics (from[14]).

| Pollutant | Mean ± standard deviation | Median | Interquartile range |
|---|---|---|---|
| Noise | | | |
| $L_{den}$ (dB(A)) | 63.8 ± 6.0 | 62.6 | 59.8–67.6 |
| $L_{day}$ (dB(A)) | 64.3 ± 5.9 | 63.1 | 60.3–68.1 |
| $L_{night}$ (dB(A)) | 53.8 ± 5.9 | 52.6 | 49.9–57.5 |
| Road traffic, $L_{den}$ (dB(A)) | 63.1 ± 5.9 | 61.9 | 59.2–66.8 |
| Aircraft, $L_{den}$ (dB(A)) | 32.1 ± 16.2 | 34.8 | 22.3–43.9 |
| Air pollutant | | | |
| BC ($10^{-5}$/m)[a] | 1.71 ± 1.35 | 1.01 | 0.85–2.15 |
| $PM_{2.5}$ (μg/m$^3$) | 4.06 ± 1.73 | 4.01 | 3.14–4.78 |
| $NO_2$ (μg/m$^3$) | 31.4 ± 8.3 | 29.5 | 26.0–34.0 |
| NO (μg/m$^3$) | 32.8 ± 15.6 | 28.5 | 23.3–37.4 |

BC: black carbon, $L_{day}$ dB(A): annual daytime A-weighted equivalent continuous noise level, $L_{den}$ dB(A): annual day-evening-night A-weighted equivalent continuous noise level, $L_{night}$ dB(A): annual nighttime A-weighted equivalent continuous noise level.

[a] Equivalent to approximately 1.19 ± 0.88 μg/m$^3$ elemental carbon ($10^{-5}$/m black carbon ≈ 0.8 μg/m$^3$ elemental carbon).

### 3.2 Chronic disease studies

#### Coronary Heart Disease (CHD)

We began by examining the joint effects of transportation noise and air pollution in heart disease mortality [18]. We defined the outcome as death from CHD during the 4-year follow-up period, defined as having International Classification of Diseases (ICD) codes 410–414 and 429.2 (9th revision) or I20–I25 (10th revision) listed as the cause of death on the death certificate.

Our adult cohort had a total of 445,868 subjects who met the inclusion criteria, of whom 33,448 (7.5%) were lost to follow-up (the greatest percentage of these because they left the Province). During the follow-up period, 3,095 subjects died of CHD (mortality rate = 1.83 per 1,000 person-years).

Residential noise exposure was associated with CHD mortality: A 10-dB(A) elevation in noise levels was associated with a 26% (95% CI: 17, 35) increase in the risk of CHD mortality. Adjustment for age, sex, preexisting comorbid conditions, and neighborhood socio-economic status (SES) halved the effect estimate, whereas further adjusting for PM2.5 and nitrogen dioxide concentrations had little influence. Additional adjustment for back carbon levels (black carbon is a marker for diesel engine exhaust) had a greater influence on the effect estimate, but a 10-dB(A) elevation in noise levels was still associated with a 9% (95% CI: 1, 18) increase in the risk of death from CHD in the final model. Both noise and black carbon exposure were independently associated with death from CHD (Figure 2). Elevations equal to the interquartile range in noise (6.6

dB(A)) and black carbon (0.97 10-5/m) were associated with 6% (95% CI: 1, 11) and 4% (95% CI: 1, 8) increases in CHD mortality respectively. In our study, aircraft noise was less correlated with traffic-related air pollutants than was road traffic noise and there was no significant increase in the risk of death from CHD associated with exposure to aircraft noise.

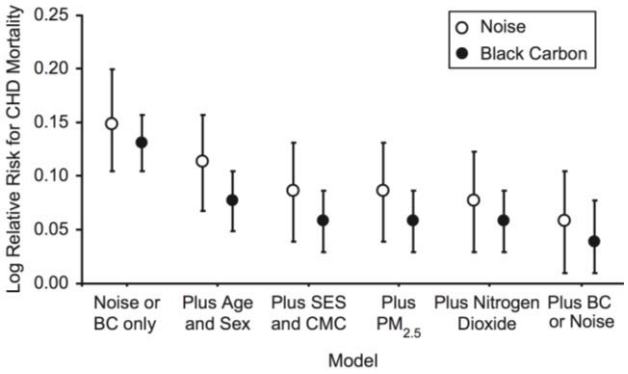


**Figure 2:** Log relative risks for coronary heart disease (CHD) mortality associated with an elevation equal to the interquartile range in noise levels (6.6 dB(A)) or black carbon (BC) concentrations (0.97 10-5 /m). Models were successively adjusted for the indicated variables (CMC=comorbid condition). The final black carbon model was adjusted for noise, and the final noise model was adjusted for black carbon. Bars are 95% confidence intervals (from [18]).

### Diabetes

Given the link between noise and coronary heart disease observed in our study and others, and the hypothesized biological mechanisms, we anticipated associations between noise exposure and metabolic risk factors. A few studies had examined the influence of transportation noise exposure on metabolic risk factors for cardiovascular health such as body mass index, waist circumference, central obesity. Diabetes is another metabolic risk factor that places an enormous burden on the Canadian population but had received only limited study. We undertook to study the association between noise and diabetes in the Vancouver adult cohort [19], using a case definition of a hospitalization or physician visits with codes for diabetes in the follow-up period (ICD-9 code 250, with ICD-10 coding back-translated to ICD-9 coding). We excluded subjects who had prior hospital or doctor visits for diabetes during the exposure period so as to identify only new (incident) cases. The cohort comprised 380,738 individuals. Of these, 3.4% were identified as incident diabetes cases during the follow-up period. Noise exposure was associated with incidence of diabetes after adjustment for age, gender, and area-level household income; there was an 8% increase in the incidence of diabetes with an IQR increase in noise exposure (~ 7 dBA LDEN). The relationships were found to be linear for a wide range of the data with the exception of extreme exposure values (Figure 3). By the time of this study, we were also able to look at not only the possible joint effects of traffic related air pollution, but also potential protective effects of neighbourhood walkability and greenness, following up on the work of UBC colleagues Frank [20] and Hystadt [21] respectively. We showed that greenness showed a protective association with the incidence of diabetes, with an interquartile range (IQR) increase in greenness being associated with a 10% decrease in odds for the incidence of diabetes in the fully adjusted models, while neighborhood walkability showed a 5% decrease in odds for the incidence of diabetes in the fully adjusted models with an interquartile increase in walk-ability score.

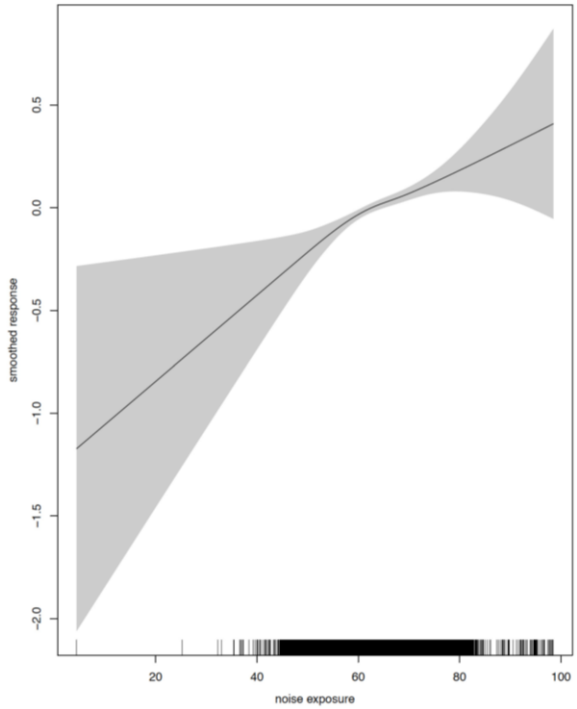


**Figure 3:** Functional relationship between noise exposure (LDEN) and incident diabetes cases; adjusted for age, gender and area-level income. Line is the log odds of diabetes incidence (log base 10); gray area is 95% CI (from [19]).

### Rheumatoid Arthritis

The risk of rheumatoid arthritis (RA) has been associated with living near traffic, and prior evidence suggesting that air pollution may not be responsible for this association [22]. Noise, another traffic-related potential hazard, had not been studied as a risk factor for RA and so we undertook to examine the possible relation [23]. RA cases are harder to define than CHD or diabetes. We used three different case definitions that included (i) multiple contacts with health system with a relevant ICD-9 code (714.0–714.9) listed for an out-patient or in-patient visit, (ii) use of anti-rheumatic drugs, and (iii) visits to specialists such as internists or rheumatologists.

We observed an increased risk of RA with residential proximity to traffic as anticipated; the largest risk increases were seen in association with road types of higher traffic volume and with greater proximity, suggesting a dose–response pattern. In that way, our findings were similar to those from the US Nurse's Health Study [22]. However, we did not find an association of RA incidence with noise, nor

**Table 2:** Risk of incident Rheumatoid Arthritis in relationship to Noise and air pollution during 5-years before diagnosis (odd's ratios with 95% CI, per interquartile range increase in exposure) (from[23]).

| Exposure | RA-ICD-9 | | | RA-prescription | | | RA-specialist | | |
|---|---|---|---|---|---|---|---|---|---|
| | Cases (*n*) | Controls (*n*) | OR (95% CI) [IQR] | Cases (*n*) | Controls (*n*) | OR (95% CI) [IQR] | Cases (*n*) | Controls (*n*) | OR (95% CI) [IQR] |
| NO-LUR ($\mu g/m^3$) | 3,280 | 33,234 | 0.99 (0.95, 1.02) [10.6] | 2,659 | 26,846 | 0.96 (0.92, 1.00) [10.8] | 1,883 | 19,059 | 0.96 (0.91, 1.00) [10.6] |
| $NO_2$-LUR ($\mu g/m^3$) | 3,278 | 33,229 | 0.95 (0.90, 0.99) [6.3] | 2,657 | 26,842 | 0.89 (0.84, 0.94) [6.3] | 1,881 | 19,059 | 0.90 (0.85, 0.96) [6.3] |
| Black carbon-LUR ($\mu g/m^3$) | 3,138 | 32,159 | 0.97 (0.93, 1.01) [0.62] | 2,553 | 25,935 | 0.94 (0.90, 0.98) [0.61] | 1,818 | 18,420 | 0.92 (0.87, 0.97) [0.62] |
| $PM_{2.5}$-LUR ($\mu g/m^3$) | 3,175 | 32,304 | 0.96 (0.91, 1.00) [2.7] | 2,567 | 26,144 | 0.93 (0.88, 0.98) [2.7] | 1,819 | 18,518 | 0.92 (0.87, 0.98) [2.7] |
| $PM_{10}$-IDW ($\mu g/m^3$) | 2,712 | 27,208 | 0.91 (0.88, 0.95) [0.87] | 2,135 | 21,850 | 0.90 (0.86, 0.94) [0.87] | 1,653 | 15,709 | 0.91 (0.86, 0.96) [0.87] |
| $O_3$-IDW ($\mu g/m^3$) | 3,055 | 30,698 | 1.15 (1.08, 1.23) [8.4] | 2,454 | 24,791 | 1.26 (1.18, 1.36) [8.6] | 1,724 | 17,636 | 1.07 (0.98, 1.16) [8.4] |
| CO-IDW ($\mu g/m^3$) | 2,826 | 28,269 | 0.87 (0.82, 0.91) [169] | 2,249 | 22,807 | 0.83 (0.78, 0.88) [169] | 1,633 | 16,274 | 0.86 (0.80, 0.92) [169] |
| $SO_2$-IDW ($\mu g/m^3$) | 3,082 | 30,963 | 0.90 (0.86, 0.94) [3.1] | 2,477 | 25,011 | 0.84 (0.79, 0.89) [3.1] | 1,733 | 17,761 | 0.88 (0.82, 0.93) [3.1] |
| Noise [dB(A)] | 2,188 | 22,734 | 1.03 (0.97, 1.09) [6.9] | 1,711 | 18,346 | 1.00 (0.93, 1.07) [7.0] | 1,315 | 13,173 | 0.96 (0.88, 1.04) [6.9] |

ORs were adjusted for age, sex, and neighborhood SES.

was RA not associated with other traffic-related air pollutants such as NO2 or PM2.5 in our study.

### 3.3 Birth outcomes

We examined birth outcomes among noise-exposed mothers in the Vancouver birth cohort [24]. The cohort was constructed by extracting data from the same linked administration files as the adult cohort but with added data from the BC Perinatal Database (Perinatal Services British Columbia). These data comprised all live births in 1999 through 2002 in the GVRD. Vital statistics records were identified for 81,347 children born in the study area during the study period; 73,387 had mothers with verified complete residential history within the study area during pregnancy. Following exclusions, 68,238 births remained for study.

Among the subjects, the primary outcomes studied were preterm births (<37 weeks of gestation), moderately preterm (30–36 weeks) and very preterm (<30 weeks of gestation) births versus term (≥37 weeks of gestation) births; term birth weight (in grams); term low birth weight (<2,500 g); and fetal growth defined as small size for gestational age (birth weight below the 10th percentile of the cohort, stratified by sex, for each week of gestation).

As estimated noise levels across the various pregnancy periods (entire pregnancy and trimesters) were highly correlated in all subjects ($r > 0.96$), as well as for those who changed residence during pregnancy ($r > 0.89$), only analyses of average exposure during the entire pregnancy were reported.

The associations of noise and the primary outcomes are shown in Figure 4. Our findings suggested an effect of residential exposure to traffic noise on being small for gesta tional age, term low birth weight, and term birth weight, but not on preterm birth. The association of noise exposure and term birth weight showed a mean reduction of - 19 g per 6 dB(A) [95% CI – 23 to - 15]. In joint air pollution-noise

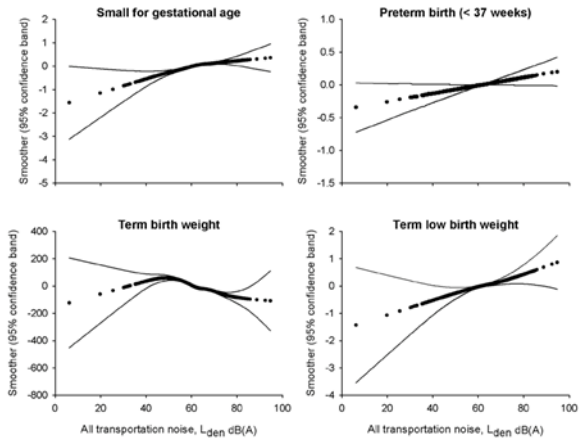


**Figure 4:** Smoothing splines with pointwise ± 1.96 standard error bands for the associations of all transportation noise exposure with the logits of small for gestational age, preterm birth, low weight at term and with birth weight at term (from [24]).

models, associations between noise and term birth weight remained largely unchanged, whereas associations decreased for all air pollutants (Figure 5).

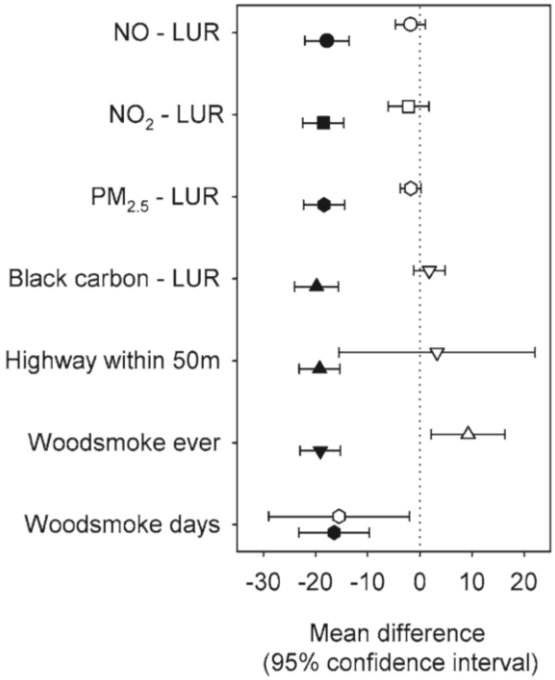


**Figure 5:** Joint associations of all transportation noise (black symbols) and land use regression estimates of air pollution, road proximity, and wood-smoke exposure (white symbols) with term birthweight. Associations are presented as mean difference (in grams) with 95% CI. Increments are 6 dBA noise, 10 ug/m3 NO, NO2, 1 ug/m3 PM2.5; Black Carbon 10-5/m, 40 wood-smoke days (from [24]).

## 4 Conclusion

This program of research at UBC has contributed to the better understanding of the role of noise in chronic disease in adults, and its effect on birth outcomes among mothers exposed to noise during pregnancy. While no association was observed between exposure to noise and rheumatoid arthritis, positive associations were observed between noise and increased risk for coronary heart disease, noise and diabetes, and negatively between noise and term birthweight. In all cases, the effects of noise were found to be independent of other exposures related to traffic and urban form, most notably, air pollution. It is worth noting that although the relative risks or effect sizes observed were often small, the outcomes (such as diabetes or coronary heart disease) are highly prevalent in the Canadian population, meaning these differences still carry potentially large public health significance. The actual burden of disease related to community noise exposure has been estimated in some countries (e.g. such as Ericksson's work [25]) but not yet in Canada. The findings of the Vancouver studies have cited over 750 times, and have been incorporated into more recent systematic reviews such as those by the WHO on CVD and metabolic disorders [26] and birth effects [27] which form the basis for the European Noise Guidelines [28].

The strengths of the research program include the very large cohort size (almost half a million in the adult cohort and 70,000 in the birth cohort) as well as the quality of the outcome data (access to health insurance data and vital statistics via linked health records, see Population Data BC at https://www.popdata.bc.ca) and co-variate data, as well as the scale and quality of the exposure data, and the ability to create residential histories. The creation of concurrent noise and air pollution exposure "surfaces" allowed for increased understanding of the covariance of noise and air pollution, and for some of the first joint-exposure studies to be undertaken; now it is standard practice for major epidemiological studies in this field to include both (as well as other measures of urban form) to better adjust for potential confounding and for better understanding of potential interactions.

However, no method can measure true exposure, and sources of error in estimated exposure included not modeling all salient features of the local environment (such as not including float plane traffic; Coal harbor – CYHC – handled over 54,000 movements in 2009, the 35th busiest airport in Canada) and not taking into account individual factors, such as room orientation, window opening behaviour, and time-activity patterns. Nevertheless, many of these exposure misclassification errors are likely to be non-differential in nature (the direction of the error is not associated with outcome status); this typically results in an underestimation of the true exposure-response estimate, not an overestimation [29]. The use of administrative health databases meant that we were unable to obtain all relevant individual-level confounder data, such as socioeconomic and various risk factor data (ethnicity, education level, income, BMI, smoking history, diet, etc.) into account. Such factors may confound findings observed between noise and the incidence of chronic disease. However, we were able to partially adjust for some of these factors by using neighborhood-level socioeconomic status (SES) measures, and ethnicity characteristics. This study was reliant on administrative records for diagnosis, which will have missed undiagnosed cases and residents who

do not attend health care providers, although registration in the BC universal health care system is very high (nearly 100%).

The elucidation of noise from transportation sources as an independent risk factor for chronic health disease has far reaching implications not only for future research (such that major studies of the effects of air pollution on health now also consider noise) but also on policy, and on urban planning. Measures to mitigate air pollution levels from traffic sources may not always be consistent with reducing noise levels [30]. For example, electric vehicles may eliminate tail-pipe emissions, but they do not reduce noise at higher speeds where noise from the road/tire interface is more dominant. Currently, policy around noise mitigation in Canada is still largely regulated at the municipal level, and driven by annoyance and economic outcomes. Despite the growing evidence linking noise to chronic disease such as those described in this paper, Canada lags far behind Europe in terms of formulating true health-based regulations to govern noise exposures.

Moving forward, our research group is currently a participant in the CIHR-funded Canadian Urban Environmental Health Research Consortium or "CANUE" [31]. As part of the CANUE Noise Data Team, we are working to further develop noise exposure data resources for all major urban centers in Canada, using noise measurement, data propagation techniques as well as land-use regression modelling, in a common harmonized manner consistent with the CANUE mission [32]. New research projects are under way. We are currently we are examining the role of noise in neurological disease; preliminary results indicate a possible increase in incident multiple sclerosis with increasing transportation noise exposure (unpublished data). We will also begin assessing the role of noise in early child development through a recently CIHR-funded study 'Born to be Wise: Impact of Modifiable Early-life Environmental Exposures on the Health and Development of Children', that has a primary goal of addressing the research gap between the effect of environmental exposures on early development, and the influence of interactions between harmful exposures and beneficial exposures, as well as studying relations between various environmental exposures and children's early development and health [33].

If you wish to learn more about these studies or our ongoing program of noise and health research, please contact the author.

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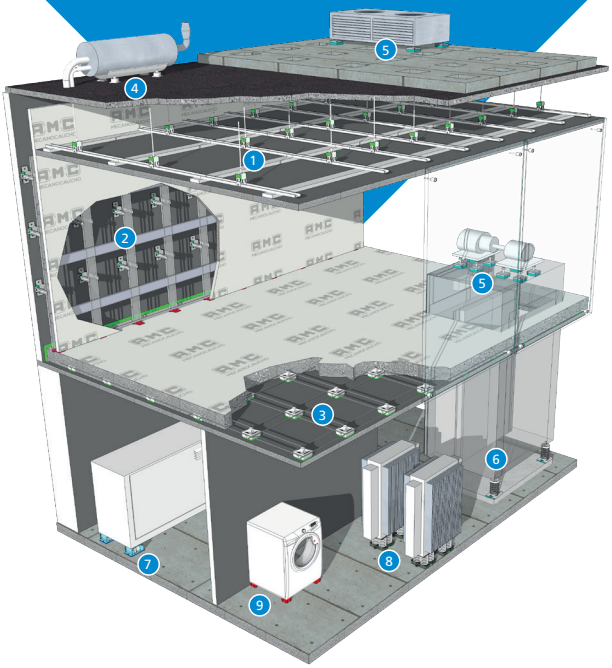

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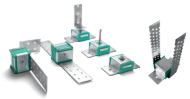

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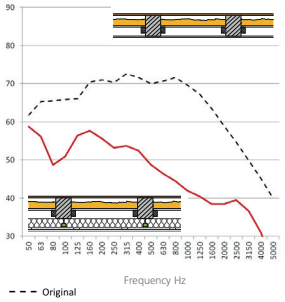


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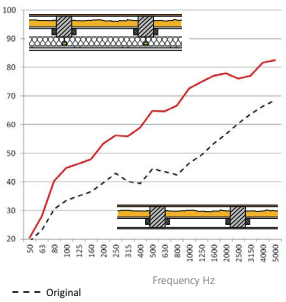


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