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JASA ARTICLE

Self-reported occupational noise exposure and cardiovascular disease in Canada: Results from the Canadian Health Measures Survey

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ABSTRACT:

Self-reported occupational noise exposure has been associated with impaired hearing, but its relationship with extra-auditory affects remains uncertain. This research assessed the association between self-reported occupational noise exposure and cardiovascular outcomes. Participants (n = 6318, ~50% male) from the Canadian Health Measures Survey (2012–2015) aged 20–79 years were randomly recruited across Canada. An in-person household interview included basic demographics, perceived stress, diagnosed health conditions, and self-reported exposure to a noisy work environment. Direct physiological assessment in a mobile examination centre permitted the determination of biomarkers/risk factors related to cardiovascular function. Logistic or linear regression models explored the association between self-reported occupational noise exposure and several cardiovascular endpoints after adjusting for confounding variables. After adjustments, there was no evidence for an association between occupational noise and any of the evaluated endpoints, which included but were not limited to blood pressure, heart rate, blood glucose, insulin, lipids, diagnosed hypertension, medication for hypertension, myocardial infarction, stroke, or heart disease. There was no evidence that self-reported occupational noise exposure was associated with evaluated cardiovascular-related biomarkers, or cardiovascular diseases among Canadians aged 20–79 years. This study, and others like it, provides an important contribution to an evidence base that could inform policy related to occupational noise exposure.

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I. INTRODUCTION

The exposure and response to chronic stressors can lead to various illnesses broadly categorized as stress-related health effects (Anisman, 2015; Yusuf *et al.*, 2004). It is well-established that exposure to loud noise can cause an increase in stress reactions that include, but are not necessarily limited to, changes in cortisol, adrenaline, epinephrine, heart rate, and blood pressure (Basner *et al.*, 2014; Lusk *et al.*, 2004). What remains an open question is whether such changes are of a sufficient magnitude and duration that they would increase the risk of developing adverse health effects. The epidemiologic evidence for an association between environmental noise exposure and health, such as cardiovascular disease (CVD), often fails to pass many of the fundamental criteria for establishing causation (Hill, 1965). The evidence tends to be indirect, inconsistent, and the strength for the statistical contribution from noise is weak (van Kempen *et al.*, 2018; World Health Organization, 2018). More compelling evidence exists for occupational noise exposures (Davies *et al.*, 2005; Gan *et al.*, 2011, 2016; Gopinath *et al.*, 2011; Kerns *et al.*, 2018; Skogstad *et al.*, 2016; Zhou *et al.*, 2019) possibly owing to higher noise levels and more precisely defined exposures.

Based on their analysis of the data collected in the National Health and Nutrition Examination Survey (NHANES), Gan *et al.* (2011) reported increased odds of angina pectoris, coronary artery disease and isolated diastolic hypertension (IDH) among a nationally representative sample of workers that self-reported exposure to loud occupational noise. These associations remained after adjusting for several covariates and were more profound when the analysis was restricted to respondents below 50 years of age. Their results also pointed toward a "cumulative-response" insofar as the strength of association increased as self-reported exposure duration increased. Despite these observations, measured mean blood pressure, isolated systolic/general hypertension, heart rate, cholesterol, triglyceride concentrations, and inflammatory biomarkers were either not associated with noise or no longer statistically significant after adjusting for covariates.

In many respects, the Canadian Health Measures Survey (CHMS) mirrors the NHANES. Both are cross-sectional surveys on nationally representative samples, include an in-person household interview component and a

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physical examination on a sub-sample of participants in a mobile clinic. Like NHANES, CHMS (Cycles 3 and 4) included similar self-reported assessments of occupational noise exposure. This provided an opportunity to examine the associations between self-reported occupational noise exposure and the prevalence of several outcomes related to cardiovascular function and/or disease. As closely as possible, our assessment followed that of Gan *et al.* (2011). In the current analysis we report the association between self-reported occupational noise exposure and several outcomes including, but not limited to, measured blood pressure, heart rate, blood lipids, glucose, insulin, high sensitivity C-reactive protein (HS CRP), apolipoprotein (APO) A/B1, medically diagnosed history of myocardial infarction, heart disease, stroke, hypertension, and self-reported medication usage.

II. METHODS

Recruitment of CHMS participants took place for Cycle 3 (2012–2013) and Cycle 4 (2014–2015), across five regions of Canada: Atlantic, Quebec, Ontario, Prairies, and British Columbia. CHMS is an ongoing cross-sectional survey where one or two individuals per household are randomly selected. Each respondent has a sampling weight that accounts for the age and sex distribution of the population, non-response, and the survey sampling strategy. With this approach, CHMS data are representative of the Canadian population even though the sampling frame excludes approximately 4% of the population (i.e., First Nation reservations, other Aboriginal settlements, certain remote locations, full time Canadian Armed Forces, residents of institutions, and three territories). The survey entails an in-person household interview to gather demographic, socioeconomic, health and lifestyle information, and a subsequent visit to a mobile examination centre (MEC) for direct physical measures. Detailed information related to each CHMS cycle, including how to access the data, is accessible through Statistics Canada (Statistics Canada, 2014, 2016).

A. Study sample

A total of 6318 participants aged 20 to 79 years (excluding pregnant woman, $n = 39$) were included in the analysis, representing 25.3×10^6 Canadians (12.6×10^6 male, 12.7×10^6 female). The response rate for this age group in CHMS was 88.9% for the household questionnaire and 77.8% for the MEC component, which included audiometry and blood sample analysis. Participants that did not respond to the noise exposure assessment question ($n = 37$) were excluded from further analysis, reducing the available sample to 6281. Another 419 unique participants were excluded due to missing data on ethnic background ($n = 146$), smoking status ($n = 93$), perceived stress ($n = 2$), family history of heart disease, stroke or high blood pressure ($n = 92$), exposure time to leisure noise ($n = 5$), presence of diabetes ($n = 4$), waist circumference ($n = 38$) and ratio of total cholesterol (total-C) to high-density lipoprotein (HDL) ($n = 72$), leaving 5862 participants for the model fitting.

B. Occupational noise exposure assessment

Occupational noise exposure was assessed with the following question: "*At any time in your life have you worked in a noisy environment? By noisy, I mean so loud that you and your co-workers had to speak in a raised voice to be understood by or communicate with someone standing an arm's length away*." Reported duration was used to create the following exposure categories: (1) never exposed; (2) less than 10 years; and (3) 10 years or more. Categories are based on the finding that more than 10 years of exposure was associated with audiometric measures of hearing impairment in the same sample of participants, even after adjusting for established covariates (Feder *et al.*, 2017). Participants who reported having worked in a noisy environment were further asked "*Are you currently working in a noisy environment*?" This information along with the length of time of having worked in a noisy environment was used to create the following five exposure categories: (1) never exposed; (2) worked in a noisy environment for less than 10 years, but not currently; (3) worked in a noisy environment for 10 years or more, but not currently; (4) currently working in a noisy environment for less than 10 years; and (5) currently working in a noisy environment for 10 years or more. Both of these variable definitions: (1) duration of working in a noisy environment or (2) duration of working in a noisy environment and if currently working in a noisy environment, were used as self-reported exposure to a noisy work environment in modelling.

C. Evaluated outcomes

Several measures considered risk factors for cardiovascular disease, or directly related to cardiovascular function were evaluated. Cardiovascular disease (CVD) encompassed the following medically diagnosed illnesses as reported by participants: heart disease, myocardial infarction, stroke, high blood pressure, and medication for high blood pressure. Medically diagnosed conditions were analyzed individually as well as collectively.

Participant's average systolic blood pressure (SBP), diastolic blood pressure (DBP), and resting heart rate (RHR) were calculated after excluding the initial measure from six automated oscillometric measurements separated by 1-min intervals. Measured blood pressure data defined the following hypertension subtypes: isolated systolic hypertension (ISH) ($SBP \geq 140$ and $DBP < 90$ mm Hg); IDH ($SBP < 140$ and $DBP \geq 90$ mm Hg); and systodiastolic hypertension (SDH) ($SBP \geq 140$ and $DBP \geq 90$ mm Hg). General hypertension was defined as $SBP \geq 140$ mm Hg, $DBP \geq 90$ mm Hg, or reporting to have received a medical diagnoses of high blood pressure.

Laboratory blood tests were performed on all participants for the following biomarkers: platelets, total-C, HDL, total-C/HDL (calculated), potassium, sodium, heavy metals

(cadmium, mercury, lead) and HS-CRP. The following endpoints were determined from a 10 h fasted sub-sample (non-diabetic) prior to their MEC appointment: low-density lipoprotein (LDL), APO A1, APO B, glucose, insulin, and triglycerides. A final subset of respondents aged 20 to 79 were randomly selected for methylmercury analysis.

D. Covariates

Covariates related to CVD and biomarkers were used to adjust the models based on the frequency of each distribution. Sex and age were adjusted for in all models. Annual household income was categorized into three groups (×$1000) (<50, ≥50, to<100, and ≥100). Education was grouped as: some post secondary or higher, and secondary graduation or less. Ethnicity was divided as Caucasian and non-Caucasian (which included Aboriginal Canadians). Small sample sizes did not permit a more refined classification of ethnicity.

Smoking status was defined using urinary cotinine levels (i.e., ≥50 ng/mL was defined as a smoker, <50 ng/mL were non-smokers) (SRNT Subcommittee on Biochemical Verification, 2002). Vigorous activity (a derived variable in CHMS) during leisure time over the past week was categorized as high (≥150 min/wk) and low (<150 min/wk) (Statistics Canada, 2014, 2016).

A single derived variable captured reported family history of heart disease, stroke, or high blood pressure. Perceived stress was assessed in two questions; one related to stress in one's life on most days, another concerned stress at work on most days. The response categories for both questions were *not at all stressful, not very stressful, a bit stressful, quite a bit stressful, and extremely stressful*. Quite a bit stressful or extremely stressful were grouped together as "high stress," and the three lower categories constituted the "low stress" group. The derived variable "perceived stress" reflects stress as reported in *either* question.

Alcohol consumption was based on the number of alcoholic drinks per week and grouped as follows: ≥7, 1–7, and never. Body mass index (BMI) was dichotomized as <25 (normal and underweight) and ≥25 (overweight and obese); waist circumference was not grouped.

Cumulative exposure to sources of loud leisure activities in the previous 12 months was considered a covariate in this analysis. Feder *et al.* (2019) provided a detailed description of these sources, including how their assigned average sound pressure levels and reported usage time were used to estimate noise exposure level (LEX) categories. Briefly, participants were assigned to the "high" category if their calculated weekly LEX was equivalent to (or above) the occupational limit (i.e., 85 dBA, LEX 40 h). The "low" category represents a noise exposure at 85 dBA LEX, <4 h, and the "medium" category was selected to be exclusive of the high and low categories. Participants not exposed to any loud non-occupational activities were included as the no exposed group.

The presence or absence of medically diagnosed diabetes along with a calculated total-C/HDL (<5 versus ≥5) were also considered as covariates. Finally, CHMS cycle was also included in the models to account for variations between cycles.

E. Statistical analysis

Population weighted frequencies and cross-tabulations were used to explore participant characteristics in the different self-reported occupational noise exposure groups and demographic variables. All estimates were weighted at the person level to represent the population (Statistics Canada, 2014, 2016).

The association between self-reported exposure to a noisy work environment (predictor variables) and the odds of cardiovascular outcomes (dependent variable) including hypertension subtypes were assessed with logistic regression. Linear regression models evaluated the association between loud occupational noise and mean differences in measured blood pressure and RHR. When considering occupational noise, the "never exposed" group was treated as the reference group. Models were fit in two stages. The first regression model adjusted for age, sex, and CHMS cycle. The second regression model further adjusted for ethnicity, income, waist circumference, family history of heart disease, stroke or high blood pressure, smoking status, alcohol consumption, perceived stress, vigorous activity, loud leisure noise exposure, diabetes, and total-C/HDL.

Linear regression models were also used to determine the association between self-reported occupational noise exposure and the mean difference of blood endpoints. These models were fit similar to the regression models above, except in the second stage model the total-C/HDL was not included.

Education and BMI were not included in further models as education was highly correlated with income and BMI was highly correlated with waist circumference. In order to avoid issues of multicollinearity, the variable most strongly associated with the outcome variable was used in the final models.

Analyses were conducted using SAS software ENTERPRISE GUIDE 7.15 for Windows (SAS Institute Inc., Cary, NC) and SUDAAN 11.0.0 software (Research Triangle Institute, Research Triangle Park, NC). To account for the complex survey design, p-values, 95% confidence intervals, and coefficients of variation (CVs) were estimated using the bootstrap technique with 22 degrees of freedom (Rao *et al.*, 1992; Rust and Rao, 1996). Statistical significance was specified as a p-value of less than 0.05. All comparisons were carried out using the Satterthwaite F test, and Bonferroni corrections were made where multiple pairwise comparisons were carried out. Estimates with a CV between 16.6% and 33.3% were designated "E" and are to be interpreted with caution due to the high sampling variability associated with them; CV estimates that exceeded 33.3%

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were designated "F" indicating that these data could not be released due to questionable validity.

III. RESULTS

Table I presents the sample characteristics of the population stratified by the number of years worked in a noisy environment. Some notable observations among respondents working in a noisy environment for 10 years or more included an advanced age, male, Caucasian, lower education, and elevated BMI. Smoking was also more prevalent, as was alcohol consumption, family history of heart disease, stroke, or high blood pressure. Perceived stress was highest among those who reported occupational noise exposures less than 10 years, but similar between the reference and longest exposed group. Exposure to high levels of loud leisure activities (i.e., 40 h or more per week) was more prevalent among those who worked in a noisy environment.

Unadjusted medically diagnosed conditions and biomarker data in relation to self-reported occupational noise exposure are also provided in Table I. Univariable statistical associations with self-reported occupational noise exposure should be interpreted with caution as age and sex has a strong influence on most of these outcomes. Among the medically diagnosed conditions considered in this analysis a higher prevalence of participants with hearing impairment, diabetes, high blood pressure, heart disease, myocardial infarction, and taking medication for high blood pressure was observed among those who worked in a noisy environment for 10 years or more (Table I). Further, general hypertension rates were significantly higher among the group who reported having worked in a noisy environment for 10 years or more, compared to the reference group. Similarly, measured DPB and SBP were higher among the subgroup of people who reported having worked in a noisy environment for 10 years or more, whereas levels were similar among those who were never exposed to a noisy work environment or worked in a noisy environment for less than 10 years (Table I). Table I also provides unadjusted lipid profiles. When compared to the reference group, triglycerides, APO B, and total-C/HDL were elevated among individuals that reported working in a noisy environment for 10 years or more. Finally, when compared to the reference group, glucose, insulin, dietary potassium, cadmium, and lead concentrations were higher in the group exposed 10 years or more, whereas mercury concentrations appeared to be lower in both exposure groups.

Unadjusted sample characteristics of the population stratified by duration and if currently working in a noisy environment are provided in Table S1 of the supplemental material.[1] Most of the results are similar to those above, but in some instances, currently working in a noisy environment appeared to impact variables that may be related to CVD. For instance, among respondent who reported working in a noisy environment for 10 years or more, smoking was slightly more prevalent among those who indicated they were currently working in this environment [32% (95% CI: 23–40.9)], when compared to those who were not [27.1% (95% CI: 20.5–33.7)]. Similarly, for the same history of exposure, high perceived stress was more prevalent among those who indicated they were currently working in this environment [48% (95% CI: 37.7–58.2)], when compared to those who were not [21.4% (95% CI: 15.5–27.3)]. Those currently working in a noisy environment, regardless of duration, were more likely to report high levels of leisure noise exposure (i.e., 40 h or more per week) (Table S1). Many of the hypertension subtypes and medically diagnosed conditions were unreportable due to small sample sizes and large variability in these groups.

Table II presents the odds ratios (ORs) of cardiovascular outcomes and medically diagnosed high blood pressure with respect to the number of years worked in a noisy environment. The reference group consisted of those who reported no history of working in a noisy environment. In all cases it was observed that the ORs for diagnosed heart disease, myocardial infarction, stroke, high blood pressure, medication for high blood pressure, and the collective variable, CVD, were similar between those exposed to a noisy work environment and those who were never exposed to a noisy work environment (i.e., the confidence interval for the ORs all include 1). These ORs are not to be interpreted as relative risks as the prevalence of these outcomes was greater than 10% (Davies *et al.*, 1998). Similarly, when self-reported occupational noise exposure including duration and if currently working in a noisy environment were considered as the predictive variable, no association was found between any of the above outcomes and exposure groups (Table S2).

Table III presents the modelled ORs of hypertension subtypes in relation to the number of years worked in a noisy environment. Although the ORs of hypertension subtypes were elevated among those who had the longest duration of exposure, none of these outcomes reached statistical significance. Again, the ORs of hypertension subtypes were elevated among those who reported to be currently working in a noisy environment, but none of these outcomes reached statistical significance (Table S3).

As shown in Table IV, modelling results did not provide evidence that the duration of working in a noisy environmental had an impact on average measured blood pressure and RHR. This was also the case when history of exposure was stratified by participants reporting to be currently or not currently working in a noisy environment (Table S4).

The association between serum biomarkers and self-reported exposure to a noisy work environment was investigated using linear regression models (Table V). In model 1 HDL, total mercury, and methylmercury concentrations were statistically higher among those who *never* worked in a noisy environment, whereas triglyceride and blood cadmium concentrations were significantly higher among those who worked in a noisy environment for 10 or more years. This was also the case when history of exposure was stratified by participants reporting to be currently or not currently working in a noisy environment (Table S5). In addition, it

TABLE I. Unadjusted sample characteristics as a function of reported duration of exposure to a noisy work environment.[a]

Variable	Overall (n = 6281)	Never worked in a noisy environment (n = 3572)	Worked in a noisy environment less than 10 years (n = 1653)	Worked in a noisy environment 10 years or more (n = 1056)
Demographic and Socioeconomic Variables				
Age (years), mean (CI)[b]	46.44 (46.16–46.73)	47.23 (46.42–48.03)	40.42 (39.13–41.72)	54.46 (53.3–55.63)
Sex % (CI)				
Male[b]	49.8 (49.6–50)	34.4 (31.7–37)	65.3 (60.6–70)	80.4 (76.3–84.6)
Female[b]	50.2 (50–50.4)	65.6 (63–68.3)	34.7 (30–39.4)	19.6 (15.4–23.7)
Ethnicity % (CI)				
Non-Caucasian/ Aboriginals[b]	21.3 (14.3–28.2)	27 (18.4–35.6)	16.8 (11.1–22.5)	7.5 (4.2–12.9)E
Caucasian[b]	78.7 (71.8–85.7)	73 (64.4–81.6)	83.2 (77.5–88.9)	92.5 (87.1–95.8)
Education % (CI)				
Some post secondary or higher[b]	78.1 (75–81.3)	79.4 (76.3–82.6)	79.5 (74.2–84.7)	70.6 (63.8–77.3)
Secondary graduation or less[b]	21.9 (18.7–25)	20.6 (17.4–23.7)	20.5 (15.3–25.8)	29.4 (22.7–36.2)
Household Income % (CI)				
$100 K or more[b]	32.3 (29.3–35.2)	32.4 (28.4–36.4)	33.1 (29.2–37)	30.4 (24.7–36.2)
$50 K–<$100 K[b]	34.7 (32.3–37.2)	33.7 (30.7–36.8)	36.1 (31.7–40.5)	36.1 (31.1–41.2)
<$50 K[b]	33 (29.8–36.1)	33.9 (30–37.8)	30.8 (26.3–35.3)	33.4 (27.1–39.7)
Vigorous Activity Level % (CI)				
≥150 min/wk[b]	17 (15.1–19)	13.3 (11.5–15.1)	26.4 (20.3–32.5)	14.1 (10.3–17.9)
<150 min/wk[b]	83 (81–84.9)	86.7 (84.9–88.5)	73.6 (67.5–79.7)	85.9 (82.1–89.7)
Smoker % (CI)				
Smoker (cotinine ≥50 ng/mL)[b]	22.4 (20.5–24.3)	19.3 (16.1–22.5)	25.1 (19.5–30.6)	29.4 (24.1–34.7)
Non-smoker (cotinine <50 ng/mL)[b]	77.6 (75.7–79.5)	80.7 (77.5–83.9)	74.9 (69.4–80.5)	70.6 (65.3–75.9)
Alcohol Consumption % (CI)				
≥7 alcoholic drinks/wk[b]	25 (22.2–27.9)	19.9 (16.9–22.9)	31.8 (25.7–38)	32.2 (27–37.3)
1–7 alcoholic drinks/wk[b]	31.1 (27.9–34.3)	33.4 (29.4–37.5)	28.3 (24.1–32.5)	27.6 (23.7–31.4)
No alcohol[b]	43.8 (40.4–47.2)	46.7 (43–50.3)	39.9 (32.6–47.1)	40.3 (35.6–45)
BMI % (CI)				
<25 (under/normal weight)[b]	35.9 (32.9–38.9)	38.8 (35.3–42.3)	37 (31.7–42.3)	22.6 (17–28.2)
≥25 (overweight/ obese)[b]	64.1 (61.1–67.1)	61.2 (57.7–64.7)	63 (57.7–68.3)	77.4 (71.8–83)
Waist Circumference (cm) mean (CI)[b]	95.07 (93.85–96.29)	93.41 (92.07–94.75)	94.86 (93.05–96.67)	101.77 (100.29–103.25)
Perceived stress[c] % (CI)				
High[b]	36.7 (34.3–39.1)	34.8 (32.1–37.6)	42 (37.4–46.6)	34.1 (28.8–39.4)
Low[b]	63.3 (60.9–65.7)	65.2 (62.4–67.9)	58 (53.4–62.6)	65.9 (60.6–71.2)
Leisure noise exposure % (CI)				
High[b,d]	22.6 (19.7–25.5)	12.1 (9.6–14.6)	36.5 (31.4–41.5)	37.3 (30.5–44.1)
Medium[b,e]	28.7 (26.5–30.8)	27.3 (24.3–30.4)	32.2 (28.8–35.5)	27.6 (20.4–34.8)
Low[b,f]	23.8 (21.5–26.2)	26.1 (23.1–29.1)	21.2 (16.9–25.5)	20.2 (16.6–23.7)
No leisure noise[b]	24.9 (22.1–27.6)	34.5 (31–38)	10.2 (7.7–12.8)	14.9 (11.8–18.1)
Family history of heart disease, stroke or high blood pressure % (CI)				
Yes[b]	68.3 (66–70.6)	69.4 (66.5–72.2)	61.4 (56.2–66.7)	76.7 (71–82.4)
No[b]	31.7 (29.4–34)	30.6 (27.8–33.5)	38.6 (33.3–43.8)	23.3 (17.6–29)
Hearing Loss[g] % (CI)				
Normal hearing[b]	63.7 (61.6–65.7)	64.5 (62–66.9)	73.4 (69.7–77.1)	41.5 (34.6–48.4)
Unilateral[b]	12.4 (10.9–14)	13.3 (11.6–14.9)	10.3 (7.3–13.3)	13.4 (7.9–18.9)E
Bilateral[b]	23.9 (22.4–25.4)	22.3 (19.8–24.7)	16.3 (13.7–18.9)	45.1 (38.4–51.8)
Either[b]	36.3 (34.3–38.4)	35.5 (33.1–38)	26.6 (22.9–30.3)	58.5 (51.6–65.4)
Blood pressure and defined subtypes of hypertension				
ISH (SBP ≥ 140 mmHg, DBP < 90 mmHg) % (CI)[b]	4.1 (3.3–5.1)	4.5 (3.4–6)	2.9 (1.5–5.4)E	4.9 (3.3–7.1)E
IDH (SBP < 140 mmHg, DBP ≥ 90 mmHg) % (CI)	1.6 (0.9–2.8)E	1.3 (0.7–2.5)E	1.9 (1–3.6)E	F
SDH (SBP ≥ 140 mmHg and DBP ≥ 90 mmHg) % (CI)	2.2 (1.7–2.8)	1.7 (1.1–2.5)E	2.6 (1.3–4.9)E	3.6 (2–6.2)E
General Hypertension (ISH, IDH, SDH or self-reported hypertension) % (CI)[b]	22.2 (20–24.4)	22.5 (19.2–25.7)	15.7 (12.3–19.1)	33 (27.3–38.8)
DBP (mmHg), mean (CI)[b]	72.07 (71.55–72.59)	71.35 (70.68–72.02)	72.23 (71.19–73.26)	74.55 (73.45–75.66)

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TABLE I. (*Continued*)

Variable	Overall (n = 6281)	Never worked in a noisy environment (n = 3572)	Worked in a noisy environment less than 10 years (n = 1653)	Worked in a noisy environment 10 years or more (n = 1056)
SBP (mmHg) mean (CI)[b]	113.22 (112.49–113.96)	112.79 (111.83–113.75)	111.8 (110.31–113.28)	117.49 (115.93–119.06)
RHR (beats per minute bpm) mean (CI)[h]	68.91 (68.33–69.49)	69.42 (68.61–70.22)	68.72 (67.67–69.78)	67.3 (65.98–68.62)
Medically Diagnosed Conditions % (CI)				
Diabetes[b]	6.5 (5.7–7.5)	6.8 (5.6–8.1)	3.6 (2.7–4.8)	11.2 (7.9–14.5)
Reported high blood pressure[b]	18.3 (16.4–20.1)	18.3 (15.8–20.9)	12.2 (9.2–15.3)	28.8 (22.8–34.9)
Medication for high blood pressure in previous month[b]	17.6 (15.7–19.5)	17.7 (14.9–20.5)	9.8 (7.6–12.5)	31.3 (25.9–36.7)
Heart disease[b]	4 (3.3–4.8)	3.4 (2.9–4.1)	3.1 (2.1–4.7)E	7.5 (4.7–11.6)E
Myocardial infarction[b]	2.6 (2–3.4)	2.4 (1.7–3.3)	1.7 (1.2–2.4)E	5.2 (3.1–8.5)E
Past stroke[h]	1 (0.7–1.4)E	0.9 (0.6–1.5)E	0.9 (0.4–1.7)E	F
Blood Biomarker (%<LOD, for overall sample) GM (CI)[i]				
Cadmium (nmol/L) (1.7% <LOD)[h]	0.4 (0.38–0.42)	0.39 (0.36–0.43)	0.36 (0.32–0.42)	0.51 (0.45–0.59)
Total mercury (nmol/L)(27.2%<LOD)[h]	0.8 (0.71–0.91)	0.88 (0.76–1.02)	0.7 (0.61–0.8)	0.74 (0.61–0.91)
Methyl mercury (nmol/L) (subsample) (18.4% <LOD), n, GM (CI)	2052, 0.59 (0.51–0.67)	1170, 0.64 (0.56–0.74)	553, 0.53 (0.42–0.66)	329, 0.51 (0.38–0.68)
Lead (μmol/L) (0.1% <LOD)[b]	11.45 (10.98–11.93)	11.09 (10.59–11.6)	10.55 (9.82–11.34)	14.97 (13.86–16.16)
Platelet count (109/L)[h]	224.12 (221.08–227.21)	226.44 (221.66–231.33)	224.13 (219.83–228.51)	215.58 (211–220.26)
Total-C/HDL[b]	3.62 (3.55–3.69)	3.5 (3.43–3.58)	3.65 (3.53–3.78)	4 (3.86–4.15)
Total-C/HDL ≥5, % (CI)[b]	17.5 (15.2–19.7)	14.9 (12.1–17.8)	20 (16.1–23.9)	22.5 (15.9–29.1)
Total-C (mmol/L)	4.73 (4.66–4.81)	4.74 (4.65–4.83)	4.7 (4.6–4.79)	4.77 (4.63–4.91)
HS CRP (mg/L) (4%<LOD)	1.32 (1.19–1.46)	1.33 (1.15–1.53)	1.16 (0.97–1.4)	1.61 (1.41–1.84)
HDL (mmol/L)[b]	1.31 (1.29–1.33)	1.35 (1.33–1.38)	1.28 (1.25–1.32)	1.19 (1.16–1.22)
Potassium (mmol/L)[h]	4.36 (4.33–4.39)	4.35 (4.32–4.38)	4.35 (4.33–4.38)	4.41 (4.36–4.47)
Sodium (mmol/L)	141.14 (140.64–141.64)	141.08 (140.55–141.62)	141.24 (140.69–141.79)	141.17 (140.66–141.69)
Morning fasted subsample (10 h), GM (CI)	Overall (n = 3185)	Never worked in a noisy environment (n = 1774)	Worked in a noisy environment less than 10 years (n = 856)	Worked in a noisy environment 10 years or more (n = 555)
LDL (mmol/L)	2.66 (2.6–2.72)	2.64 (2.56–2.73)	2.64 (2.53–2.76)	2.75 (2.62–2.88)
APO A1 (g/L)[b]	1.42 (1.39–1.46)	1.46 (1.42–1.5)	1.38 (1.33–1.42)	1.37 (1.32–1.42)
APO B (g/L) (0.2% <LOD)[b]	0.9 (0.88–0.92)	0.9 (0.87–0.92)	0.88 (0.84–0.92)	0.98 (0.94–1.01)
Glucose (mmol/L)[b]	5.19 (5.12–5.26)	5.13 (5.06–5.21)	5.1 (5.02–5.18)	5.56 (5.37–5.76)
Insulin (pmol/L)[b]	69.7 (66.07–73.54)	69.54 (65.6–73.73)	65.11 (59.81–70.89)	79.58 (73.27–86.43)
Triglycerides (mmol/L)[b]	1.24 (1.2–1.29)	1.21 (1.17–1.26)	1.16 (1.09–1.22)	1.54 (1.39–1.71)

[a]Unweighted sample size (n) and population weighted percentage or means/GM and CI to account for multistage probability sampling design. Column sample sizes represent the maximum number of observations included in the group, but variations may occur as not all participants answered all questions, or had valid biomarker tests.
[b]$p < 0.01$ for the comparison between the years of exposure to occupational noise and the never exposed group.
[c]Perceived stress encompasses both perceived stress in personal life and work life.
[d]40 h or more per week at LEX(8) 85 dBA.
[e]Between 4 h and 40 h per week at LEX(8) 85 dBA.
[f]Up to 4 h per week at LEX(8) 85 dBA.
[g]Average hearing threshold ≥ 25 dB at 3, 4, and 6 kHz in one ear (unilateral), both ears (bilateral), and either one or both ears (either). Those with conductive hearing loss are removed from the analysis.
[h]$p < 0.05$ for the comparison between the years of exposure to occupational noise and the never exposed group.
[i]Biomarkers with greater than 40% of data below the LOD in any of the subgroups are indicated, otherwise the percent detected is above 60%. Observations of biomarkers with less than 40% of the data below the LOD were replaced with the LOD/2. If greater than 40% of the data were below the LOD, then the biomarker was not included in further analysis [15–16]. CI 95% confidence interval; GM geometric mean; LOD limit of detection; Total-C total cholesterol; HDL high density lipoprotein; LDL low density lipoprotein; APO apolipoprotein; HS CRP high sensitivity C-reactive protein; E coefficient of variation between 16.6% and 33.3%, interpret estimates with caution; F coefficient of variation greater than or equal to 33.3%, estimate could not be reported due to large variation in the estimate.

was observed that those who were currently working in a noisy environment for 10 years or more had higher lead concentrations compared to those who never worked in a noisy environment (Table S5).

IV. DISCUSSION

It is unequivocal that unprotected exposure to excessive noise can result in irreversible damage to hearing and

TABLE II. Modelled odds ratios (95% CI) for medically diagnosed cardiovascular outcomes as a function of duration of exposure to a noisy work environment. The "never exposed" group was the reference category.

	Length of time worked in a noisy environment		
	Any duration	Less than 10 years	10 years or more
Heart disease			
Number of cases[a]	123	58	65
Model 1	1.1 (0.7–1.8)	1.2 (0.7–2.1)	1.1 (0.6–2.1)
Model 2	1.1 (0.6–1.9)	1.3 (0.7–2.4)	0.9 (0.4–2)
Myocardial infarction			
Number of cases	73	31	42
Model 1	0.9 (0.5–1.7)	0.8 (0.4–1.5)	0.9 (0.4–2.1)
Model 2	0.8 (0.3–1.8)	0.8 (0.4–1.7)	0.8 (0.3–2.3)
Stroke			
Number of cases	27	13	14
Model 1	1 (0.4–2.5)	1.1 (0.2–6)	0.9 (0.3–2.5)
Model 2	1 (0.4–2.4)	1.2 (0.2–6.1)	0.8 (0.3–2.1)
High blood pressure			
Number of cases	498	214	284
Model 1	1.2 (0.9–1.6)	1.1 (0.7–1.6)	1.3 (0.8–2.1)
Model 2	1.1 (0.9–1.5)	1.1 (0.8–1.6)	1.2 (0.7–1.9)
Medication for high blood pressure			
Number of cases	464	187	277
Model 1	1.1 (0.8–1.6)	0.8 (0.6–1.2)	1.5 (1–2.3)
Model 2	1 (0.8–1.4)	0.8 (0.6–1.2)	1.3 (0.9–1.9)
CVD[b]			
Number of cases	605	264	341
Model 1	1.2 (0.9–1.6)	1.1 (0.8–1.6)	1.3 (0.9–2)
Model 2	1.2 (0.9–1.5)	1.2 (0.9–1.6)	1.2 (0.8–1.7)

[a]Number of cases are unweighted.
[b]CVD includes diagnosed heart disease, heart attack, stroke, high blood pressure, medication for high blood pressure; model 1: adjusted for age, sex and CHMS cycle; model 2: adjusted for age, sex, ethnicity, family history of heart disease, stroke or high blood pressure, diabetes, smoking, alcohol consumption, level of vigorous activity, income, waist circumference, perceived stress, exposure to loud non-occupational noise, and total-C/HDL.

TABLE III. Modelled odds ratios (95% CI) for hypertension subtypes as a function of duration of exposure to a noisy work environment. The "never exposed" group was the reference category.

	Length of time worked in a noisy environment		
	Any duration	Less than 10 years	10 years or more
ISH (SBP>=140 mm Hg and DBP < 90 mm Hg)			
Number of cases[a]	88	36	52
Model 1	1.3 (0.8–2.1)	1.6 (0.7–3.6)	1.1 (0.6–1.9)
Model 2	1.2 (0.8–1.9)	1.6 (0.7–3.4)	1 (0.6–1.7)
IDH (SBP < 140 mm Hg and DBP>=90 mm Hg)			
Number of cases	57	33	24
Model 1	1 (0.5–2.1)	1.1 (0.5–2.8)	0.9 (0.4–2)
Model 2	1.3 (0.5–3)	1.3 (0.5–3.5)	1.2 (0.4–3.4)
SDH (SBP>=140 mm Hg and DBP>=90 mm Hg)			
Number of cases	56	30	26
Model 1	1.5 (0.7–3.1)	1.5 (0.6–3.8)	1.5 (0.7–3.6)
Model 2	1.6 (0.7–3.5)	1.6 (0.6–4.3)	1.6 (0.6–3.8)
General hypertension (SBP>=140 mm Hg or DBP>=90 mm Hg or medically diagnosed hypertension)			
Number of cases	598	265	333
Model 1	1.1 (0.9–1.5)	1.1 (0.7–1.5)	1.2 (0.8–1.8)
Model 2	1.1 (0.8–1.5)	1.1 (0.8–1.6)	1.1 (0.7–1.7)

[a]Number of cases are unweighted; model 1: adjusted for age, sex and CHMS cycle; model 2: adjusted for age, sex, ethnicity, family history of heart disease, stroke or high blood pressure, diabetes, smoking, alcohol consumption, level of vigorous activity, income, waist circumference, perceived stress, exposure to loud non-occupational noise, and total-C/HDL.

occupational hearing conservation programs are a testament to this. However, extra-auditory health effects of noise are presented as occurring at *lower* exposure levels (Basner *et al.*, 2014). It is therefore reasonable to hypothesize that self-reported exposure to a noisy work environment, which we have previously reported to be associated with impaired hearing (Feder *et al.*, 2017), would likewise predict extra-auditory effects. Indeed, self-reported exposure to loud occupational noise has been associated with hypertension and elevated mean blood pressure in a sizable cross-sectional study that targeted steel workers (Zhou *et al.*, 2019). In a large prospective cohort study from Australia, after adjusting for confounding variables, self-reported exposure to workplace noise was associated with angina, stroke, CVD, increased mortality from CVD, but was not associated with measured changes in blood pressure, hypertension, or medication usage for hypertension (Gopinath *et al.*, 2011). A nationally representative study from Bulgaria recently reported an association between self-reported exposure to loud occupational noise and CVD among woman but not men, although the working definition of CVD was defined as an affirmative response to the question "does your work affect your health: heart disease?" (Dzhambov and Dimitrova, 2016). Several of these aforementioned studies have methodological differences from CHMS that make comparisons tenuous, including how self-report reflected occupational noise exposure. We selected our approach based on the degree of vocal effort and distance between speakers that would suggest an exposure to sound levels above 80 dBA (Health Safety Executive, 2012; Palmer *et al.*, 2002; Tak *et al.*, 2009. Dzhambov and Dimitrova (2016) used vocal effort, but not proximity between speakers, in their study definition: "*Are you exposed at work to noise so loud that you would have to raise your voice to talk to people*?" In Zhou *et al.* (2019) "loud" noise exposure at work was not qualified with vocal effort, or distance between speakers and could be interpreted differently between respondents. Gopinath *et al.* (2011) assessed the duration of exposure to a "noisy" industry or farm environment and followed up with those who said "yes" with a question to rate the noise as either "mostly quiet," "tolerable but able to hear speech," or "unable to hear anyone speaking." For their overall design, a more analogous comparison to the current analysis is Gan *et al.* (2011) and this extends to the similarities in how they defined "loud occupational noise" as: "*Thinking of all the jobs you have ever had, have you ever been exposed to loud noise at work for at least three months? By loud noise I mean noise was so loud that you had to speak in a raised voice to be heard.*"

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TABLE IV. Adjusted mean (95% CI) blood pressure and resting heart rate as a function of duration of exposure to a noisy work environment.

	Length of time worked in a noisy environment			
	Never	Any duration	Less than 10 years	10 years or more
DPB (mm Hg)				
Number of cases[a]	3325	2530	1539	991
Model 1	71.86 (71.12–72.59)	72.24 (71.52–72.97)	72.1 (71.17–73.03)	72.52 (71.3–73.75)
Model 2	71.97 (71.19–72.74)	72.09 (71.36–72.83)	72.04 (71.19–72.88)	72.21 (70.93–73.48)
SBP (mm Hg)				
Number of cases	3325	2530	1539	991
Model 1	112.79 (112–113.57)	113.5 (112.45–114.56)	114 (112.52–115.48)	112.55 (110.95–114.15)
Model 2	113.11 (112.19–114.03)	113.09 (112.17–114.01)	113.8 (112.67–114.93)	111.68 (110.26–113.1)
RHR (bpm)				
Number of cases	3325	2530	1539	991
Model 1	69.01 (68.19–69.84)	68.63 (67.75–69.51)	68.4 (67.25–69.56)	69.06 (67.85–70.28)
Model 2	69.16 (68.26–70.06)	68.44 (67.57–69.32)	68.47 (67.44–69.51)	68.39 (67.09–69.69)

[a]Number of cases are unweighted; model 1: adjusted for age, sex and CHMS cycle; model 2: adjusted for age, sex, ethnicity, family history of heart disease, stroke or high blood pressure, diabetes, smoking, alcohol consumption, level of vigorous activity, income, waist circumference, perceived stress, exposure to leisure noise, and total-C/HDL. DBP-diastolic blood pressure; SBP-systolic blood pressure; RHR-resting heart rate.

TABLE V. Modelled biomarker profiles as a function of duration of exposure to a noisy work environment.

	Length of time worked in a noisy environment			
	Never exposed	Any duration	Less than 10 years	10 years or more
Platelet count (109/L)		Adjusted geometric mean (95% CI)		
Number of cases[a]	3324	2537	1545	992
Model 1	222.33 (217.33–227.43)	225.34 (221.67–229.07)	225.49 (220.98–230.09)	225.06 (219.98–230.25)
Model 2	223.61 (218.4–228.95)	223.66 (220.03–227.36)	224.87 (220.39–229.45)	221.28 (215.99–226.69)
Total-C (mmol/L)				
Number of cases	3330	2533	1542	991
Model 1	4.72 (4.63–4.81)	4.75 (4.64–4.85)	4.78 (4.68–4.87)	4.68 (4.52–4.86)
Model 2	4.74 (4.66–4.82)	4.72 (4.62–4.82)	4.75 (4.66–4.84)	4.66 (4.51–4.82)
HDL (mmol/L)				
Number of cases	3330	2532	1541	991
Model 1	1.31 (1.28–1.33)	1.31 (1.28–1.34)	1.34 (1.31–1.37)	1.26 (1.22–1.3)[b]
Model 2	1.31 (1.28–1.34)	1.31 (1.28–1.34)	1.32 (1.29–1.36)	1.28 (1.24–1.32)
LDL (mmol/L)[c]				
Number of cases	1663	1337	813	524
Model 1	2.68 (2.61–2.77)	2.64 (2.53–2.75)	2.65 (2.54–2.77)	2.62 (2.47–2.77)
Model 2	2.7 (2.63–2.77)	2.62 (2.52–2.72)	2.63 (2.52–2.74)	2.6 (2.46–2.74)
APO A1 (g/L)[c]				
Number of cases	1672	1345	819	526
Model 1	1.42 (1.4–1.46)	1.42 (1.38–1.47)	1.42 (1.38–1.47)	1.41 (1.36–1.47)
Model 2	1.43 (1.4–1.46)	1.42 (1.38–1.46)	1.42 (1.37–1.46)	1.42 (1.37–1.47)
APO B (g/L)[c]				
Number of cases	1672	1344	818	526
Model 1	0.91 (0.89–0.93)	0.9 (0.86–0.94)	0.88 (0.84–0.93)	0.92 (0.87–0.97)
Model 2	0.91 (0.89–0.93)	0.89 (0.85–0.93)	0.88 (0.84–0.93)	0.9 (0.86–0.95)
Glucose (mmol/L)[c]				
Number of cases	1680	1353	822	531
Model 1	5.18 (5.11–5.26)	5.19 (5.08–5.31)	5.14 (5.03–5.26)	5.28 (5.09–5.48)
Model 2	5.18 (5.11–5.26)	5.19 (5.1–5.28)	5.18 (5.08–5.28)	5.2 (5.08–5.33)
Insulin (pmol/L)[c]				
Number of cases	1677	1353	822	531
Model 1	70.6 (65.84–75.7)	68.2 (62.32–74.64)	64.79 (58.61–71.63)	75.26 (68.02–83.27)
Model 2	71.33 (66.94–76.01)	67.36 (63.5–71.45)	66.84 (62.21–71.83)	68.39 (63.89–73.22)
Triglycerides (mmol/L)[c]				
Number of cases	1680	1353	822	531
Model 1	1.24 (1.2–1.29)	1.22 (1.15–1.29)	1.16 (1.1–1.21)[b]	1.36 (1.23–1.5)[b]

TABLE V. (*Continued*)

	Length of time worked in a noisy environment			
	Never exposed	Any duration	Less than 10 years	10 years or more
Model 2	1.25 (1.2–1.31)	1.21 (1.14–1.28)	1.18 (1.12–1.24)	1.27 (1.15–1.41)
Cadmium (nmol/L)				
Number of cases	3316	2534	1543	991
Model 1	0.37 (0.33–0.4)	0.43 (0.39–0.47)[d]	0.4 (0.35–0.46)	0.5 (0.43–0.58)[d]
Model 2	0.38 (0.35–0.4)	0.42 (0.39–0.44)[d]	0.4 (0.38–0.43)	0.44 (0.41–0.48)[d]
Mercury (nmol/L)				
Number of cases	3316	2534	1543	991
Model 1	0.9 (0.77–1.06)	0.71 (0.63–0.8)[b]	0.72 (0.62–0.84)[d]	0.69 (0.56–0.85)[d]
Model 2	0.85 (0.76–0.95)	0.77 (0.7–0.85)	0.76 (0.69–0.85)	0.79 (0.65–0.97)
Methylmercury (nmol/L)[e]				
Number of cases	1105	836	525	311
Model 1	0.65 (0.56–0.76)	0.54 (0.44–0.66)	0.6 (0.47–0.76)	0.42 (0.31–0.57)[d]
Model 2	0.63 (0.55–0.72)	0.56 (0.48–0.67)	0.62 (0.51–0.74)	0.46 (0.34–0.62)
Lead (μmol/L)				
Number of cases	3317	2534	1543	991
Model 1	11.35 (10.88–11.83)	11.6 (10.98–12.25)	11.25 (10.56–11.99)	12.29 (11.31–13.35)
Model 2	11.32 (10.9–11.77)	11.62 (11.05–12.22)	11.3 (10.65–11.99)	12.28 (11.35–13.29)
HS-CRP (mg/L)				
Number of cases	3265	2472	1508	964
Model 1	1.25 (1.09–1.43)	1.37 (1.19–1.59)	1.28 (1.03–1.59)	1.57 (1.33–1.85)
Model 2	1.32 (1.2–1.45)	1.28 (1.12–1.45)	1.24 (1.04–1.47)	1.36 (1.18–1.57)

[a]Number of cases are unweighted.
[b]$p < 0.01$ adjusted geometric mean was significantly different from the reference group "Never worked in a noisy environment."
[c]Based on a morning fasting sample.
[d]$p < 0.05$, adjusted geometric mean was significantly different from the reference group "Never worked in a noisy environment."
[e]Sub-sample; model 1: adjusted for age, sex and CHMS cycle; model 2: adjusted for age, sex, ethnicity, family history of heart disease, stroke or high blood pressure, diabetes, smoking, alcohol consumption, level of vigorous activity, income, waist circumference, perceived stress, and exposure to loud non-occupational noise; total-C, total cholesterol; HDL, high density lipoprotein; LDL, low density lipoprotein; APO, apolipoprotein; HS-CRP, high sensitivity C-reactive protein. Models for dietary potassium and sodium could not be conducted due to non-convergence (singularity).

Gan *et al.* (2011) reported population-based associations between self-reported exposure to loud occupational noise and angina pectoris, coronary artery disease (which included angina pectoris), and IDH, after adjusting for several confounding variables. The US NHANES and CHMS are similar in both design and scope and both countries have comparable laws governing occupational noise. Their proximity to one another and shared population demographics also lead to natural comparisons between Canada and the United States. An exception to their similarities is that CHMS does not have data related to angina pectoris.

As expected, respondents reporting to have the longest duration of exposure to loud occupational noise were of an advanced age and tended to be male. Other variables were also more common in this group, which could have increased their risk of CVD and masked contributions from noise. Some of the more notable differences in the group that reported to have worked in loud occupational noise for 10 years or more were smoking, increased weekly alcohol consumption, higher BMI, increased prevalence of diabetes, and higher blood lipid profiles. Despite these differences, after adjusting for only age and sex, we found no evidence that self-reported exposure to a noisy work environment increased the odds of exceeding the clinical guidelines for systolic, diastolic, or systodiastolic hypertension. Similarly, no group differences were observed in mean blood pressure or use of hypertensive medication. None of the many other outcomes evaluated showed any apparent pattern suggesting that exposure to a noisy work environment contributed to adverse cardiovascular outcomes. Although we can only speculate, it is possible that the disparities between Gan *et al.* (2011) and the current analysis are, at least in part, owing to slight differences in the approach to characterizing loud occupational noise exposure. In the current study, the analysis was based on recalled length of time in years. The NHANES reported recalled duration of time exposed in a typical day, and when combined with duration in months, estimating an accumulated exposure was feasible. It is also notable that several of the effects reported by Gan *et al.* (2011) in their model 1 (unadjusted) were also found in the current analysis, but we caution against interpreting these as evidence for noise effects as most of them are highly influenced by age and/or sex, and none of them remained significant after adjustment. In Gan *et al.* (2011) the reported association with IDH was observed in their fully adjusted model (i.e., model 3), but not model 2. Several of the variables in their fully adjusted model appear to be correlated, e.g., BMI and waist circumference, education and income,

and multiple variables related to cigarette smoking. Having highly correlated covariates in a model could cause issues with multicollinearity and lead to spurious results.

The current study has the strength of using a large nationally representative sample and a reliance on objectively measured biomarkers related to cardiovascular risk and/or outcomes. The analysis also considered a wide range of potentially confounding variables, including heavy metals. Elevated heavy metal concentrations, observed among participants reporting 10 or more years of loud occupational noise exposure, have been associated with cardiovascular abnormalities (Chowdhury *et al.*, 2018). The list of evaluated outcomes was extensive, but not complete. A limitation of the current study was an inability to extend the analysis to additional biomarkers of stress. This area of research should always incorporate established stress biomarkers, including catecholamines and cortisol (Anisman, 2015) insofar as the proposed mechanism of action is thought to be one mediated through stress responses marked by changes in both (Basner *et al.*, 2014). Furthermore, for some of the evaluated outcomes (e.g., medically diagnosed stroke and IDH among the most exposed group) there were too few participants to conduct a meaningful statistical analysis. In fact, when we attempted to stratify the analysis by age group for males and females, many of the models would not converge and several cell counts were below 10 and could not be reported (data not shown). None of the stratified results changed the results in any meaningful way. Similarly, when we considered if the respondent currently worked in a noisy environment, much of the data were unreportable and that which could be did not affect the overall results (see Tables S2–S5). Another study limitation is the questionnaire was not designed to determine a typical daily duration of exposure to a noisy work environment. This means a respondent only briefly exposed to a noisy work environment (e.g., while moving about the workplace) could not be distinguished from one who worked their entire day in such an environment. Furthermore, our exposure assessment was based on self-report, unadjusted for the use of personal hearing protection. Although this afforded us the ability to directly compare with the NHANES analysis (Gan *et al.*, 2011), we know from previous studies that a high prevalence of the current respondents reported to use personal hearing protection (Feder *et al.*, 2017). This would not affect the exposure groups in the current analysis, but it would contribute to the variance in the cumulative exposure of noise at the eardrum and any dose-dependent stress reactions that might ensue. Noise dosimetry is a tenable option (Li *et al.*, 2019), but the dose at the eardrum is still influenced by personal hearing protection no matter the degree of compliance with manufacturer specifications. Support for this comes from observations by Gopinath *et al.* (2011), where the associations reported between unprotected occupational noise exposure and prevalent CVD and angina were not found among their study participants who self-reported wearing personal hearing protection when exposed to loud occupational noise. One viable approach to circumvent this source of uncertainty would be to substitute self-reported exposure with severity of noise-induced hearing loss as recently reported elsewhere (Gan *et al.*, 2016; Zhou *et al.*, 2019).

In conclusion, the data from the current study do not support the hypothesis that self-reported exposure to a noisy work environment is associated with adverse cardiovascular outcomes, or any of the evaluated risk factors that would promote CVD among Canadians.

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[1]See supplementary material at https://www.scitation.org/doi/suppl/10.1121/10.0005588 for the univariate analysis and additional modelling results.

Anisman, H. (**2015**). *Stress and Your Health: From Vulnerability to Resilience* (Wiley, West Sussex).

Basner, M., Babisch, W., Davis, A., Brink, M., Clark, C., Janssen, S., and Stansfeld, S. (**2014**). "Auditory and non-auditory effects of noise on health," Lancet **383**(9925), 1325–1332.

Chowdhury, R., Ramond, A., O'Keeffe, L. M., Shahzad, S., Kunutsor, S. K., Muka, T., Gregson, J., Willeit, P., Warnakula, S., Khan, H., Chowdhury, S., Gobin, R., Franco, O. H., and Di Angelantonio, E. (**2018**). "Environmental toxic metal contaminants and risk of cardiovascular disease: Systematic review and meta-analysis," BMJ **362**, k3310.

Davies, H. T. O., Crombie, I. K., and Tavakoli, M. (**1998**). "When can odds ratios mislead?," BMJ **316**, 989–991.

Davies, H. W., Teschke, K., Kennedy, S. M., Hodgson, M. R., Hertzman, C., and Demers, P. A. (**2005**). "Occupational exposure to noise and mortality from acute myocardial infarction," Epidemiology **16**, 25–32.

Dzhambov, A., and Dimitrova, D. D. (**2016**). "Heart disease attributed to occupational noise, vibration and other co-exposure: Self-reported population-based survey among Bulgarian workers," Med. Pro. **67**(4), 435–445.

Feder, K. P., Marro, L., McNamee, J., and Michaud, D. (**2019**). "Prevalence of loud leisure noise activities among a representative sample of Canadians aged 6–79 years," J. Acoust. Soc. Am. **146**(5), 3934–3946.

Feder, K., Michaud, D., McNamee, J., Fitzpatrick, E., Davies, H., and Leroux, T. (**2017**). "Prevalence of hazardous occupational noise exposure, hearing loss, and hearing protection usage among a representative sample of working Canadians," JOEM **59**, 92–113.

Gan, W. Q., Davies, H. W., and Demers, P. A. (**2011**). "Exposure to occupational noise and cardiovascular disease in the United States: The National Health and Nutrition Examination Survey 1999–2004," Occup. Environ. Med. **68**, 183–190.

Gan, W. Q., Moline, J., Kim, H., and Mannino, D. M. (**2016**). "Exposure to loud noise, bilateral high-frequency hearing loss and coronary heart disease," Occup. Envrion. Med. **73**, 34–41.

Gopinath, B., Thiagalingam, A., Teber, E., and Mitchell, P. (**2011**). "Exposure to workplace noise and the risk of cardiovascular disease events and mortality among older adults," Prev. Med. **53**, 390–394.

Health Safety Executive (**2012**). "Noise at Work, A brief guide to controlling the risks," London, UK, http://www.hse.gov.uk/pubns/indg362.htm (Last viewed January 7, 2021).

Hill, A. B. (**1965**). "The environment and disease: Association or causation?," Proc. R. Soc. Med. **58**(5), 295–300.

Kerns, E., Masterson, E. A., Themann, C. L., and Calvert, G. M. (**2018**). "Cardiovascular conditions, hearing difficulty, and occupational noise exposure within US industries and occupations," Am. J. Ind. Med. **61**(61), 477–491.

Li, X., Dong, Q., Wang, B., Song, H., Wang, S., and Zhu, B. (**2019**). "The influence of occupational noise exposure on cardiovascular and hearing conditions among industrial workers," Sci. Rep. **9**, 11524.
Lusk, S. L., Gillespie, B., Hagerty, B. M., and Ziemba, R. A. (**2004**). "Acute effects of noise on blood pressure and heart rate," Arch. Environ. Health **59**(8), 392–399.
Palmer, K. T., Griffin, M. J., Syddall, H. E., Davis, A., Pannett, B., and Coggon, D. (**2002**). "Occupational exposure to noise and the attributable burden of hearing difficulties in Great Britain," Occup. Environ. Med. **59**, 634–639.
Rao, J. N. K., Wu, C. F. J., and Yue, K. (**1992**). "Some recent work on resampling methods for complex surveys," Survey Methodol. **18**(2), 209–217.
Rust, K. F., and Rao, J. N. K. (**1996**). "Variance estimation for complex surveys using replication techniques," Stat. Meth. Med. Res. **5**, 283–310.
Skogstad, M., Johannessen, H. A., and Tynes, T. (**2016**). "Systematic review of cardiovascular effects of occupational noise," Occup. Med. **66**, 10–16.
SRNT Subcommittee on Biochemical Verification (**2002**). "Biochemical verification of tobacco use and cessation," Nicotine Tob. Res. **4**, 149–159.
Statistics Canada (**2014**). "Canadian Health Measures Survey (CHMS) Detailed information for January 2012 to December 2013 (Cycle 3)," https://www23.statcan.gc.ca/imdb/p2SV.pl?Function=getSurvey&Id=136652 (Last viewed April 7, 2021).
Statistics Canada (**2016**). "Canadian Health Measures Survey (CHMS) Detailed information for January 2014 to December 2015 (Cycle 4)," https://www23.statcan.gc.ca/imdb/p2SV.pl?Function=getSurvey&Id=148760 (Last viewed April 7, 2021).
Tak, S. W., Davis, R. R., and Calvert, G. M. (**2009**). "Exposure to hazardous workplace noise and the use of hearing protection devices among US workers: NHANES, 1999–2004," Am. J. Indust. Med. **52**, 358–371.
van Kempen, E., Casa, M., Pershagen, G., and Foraster, M. (**2018**). "WHO environmental noise guidelines for the European Region: A systematic review on environmental noise and cardiovascular and metabolic effects: A summary," Int. J. Environ. Res. Public Health **15**(2), 379.
World Health Organization (**2018**). *Environmental Noise Guidelines for the European Region* (Regional Office for Europe, Copenhagen).
Yusuf, S., Hawken, S., Ounpuu, S., Dans, T., Avezum, A., Lanas, F., McQueen, M., Budaj, A., Pais, P., Varigos, J., and Lisheng, L. (**2004**). "Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): Case-control study," Lancet **364**, 937–952.
Zhou, F., Shrestha, A., Mai, S., Tao, Z., Li, J., Wang, Z., and Meng, X. (**2019**). "Relationship between occupational noise exposure and hypertension: A cross-sectional study in steel factories," Am. J. Ind. Med. **62**, 961–968.