

Occupational noise exposure and its association with incident hyperglycemia: a retrospective cohort study

Ta-Yuan Chang (✉ tychang@mail.cmu.edu.tw)

China Medical University <https://orcid.org/0000-0001-5664-4559>

Tzu-Yi Yu

China Medical University

Chiu-Shong Liu

China Medical University Hospital

Li-Hao Young

China Medical University

Bo-Ying Bao

China Medical University

Research

Keywords: Incidence, hyperglycemia, occupational noise, octave-band analysis, retrospective cohort study

Posted Date: November 9th, 2019

DOI: <https://doi.org/10.21203/rs.2.17051/v1>

License: © ⓘ This work is licensed under a Creative Commons Attribution 4.0 International License. [Read Full License](#)

Abstract

Background: Epidemiological studies have demonstrated the association between noise exposure and diabetes, but few studies have reported the relationship between noise frequency components and fasting blood glucose. This study investigated the associations between noise levels, frequency characteristics, and the incident hyperglycemia.

Methods: An industry-based cohort of 905 volunteers was enrolled and followed-up from the data of first employment to 2012. Personal noise levels and octave-band frequencies of environmental noise were measured systematically in 2012 to classify subjects' exposure retrospectively. Cox regression models were applied to calculate the relative risk (RR) of hyperglycemia by continuous and categorical noise-exposure and frequency-component levels, adjusting for potential confounders.

Results: Subjects exposed to ≥ 80 A-weighted decibels (dBA) had an increased RR for hyperglycemia of 1.78 (95% confidence interval [CI]: 1.11, 2.84) compared with those exposed to < 70 dBA. The high-exposure groups at frequencies of 31.5 (≥ 33 decibel [dB]), 63 (≥ 44 dB), 125 (≥ 52 dB), 250 (≥ 59 dB), 500 (≥ 65 dB), 1000 (≥ 68 dB), and 2000 (≥ 68 dB) Hz had a significantly higher risk of hyperglycemia (all p values < 0.05) than did the low-exposure groups, and those exposed at 31.5 Hz had the highest risk (Adjusted RR=1.97, 95% CI: 1.23, 3.16). Per 5-dB increase in noise frequencies at 31.5, 63, 125, 250, 500 Hz, and 1000 Hz were associated with an elevated incidence of hyperglycemia (all p < 0.05), with the highest risk of 1.27 (95% CI: 1.10, 1.47) at 31.5 Hz (p = 0.001).

Conclusions: Exposure to occupational noise may be associated with an increased incidence of hyperglycemia, with the highest risk observed at 31.5 Hz, providing a possible link between noise exposure and cardio-metabolic disease.

Background

Growing numbers of field studies have suggested that chronic exposure to occupational noise is associated with cardiovascular disease morbidity and mortality, including ischemic heart disease [1], coronary heart disease [2–4], acute myocardial infarction [5–7], and hypertension [8–10]. A hypothesized mechanism suggests that noise exposure as an environmental stressor causes adverse health effects via both direct (i.e., sleep disturbance) and indirect (i.e., annoyance) pathways; this leads to a physiological acute response, producing elevated levels of stress hormones (such as cortisol, catecholamine, adrenalin, and noradrenalin) through activation of the hypothalamus-pituitary-adrenal axis and the sympathetic-adrenal-medulla axis. The repeated and chronic stimulus may cause overproduction of stress hormones (e.g. cortisol) to restore homeostasis partly by increasing energy supply in the form of fatty acids and glucose. In addition, catecholamines also boost energy supply by breaking down triacylglycerol. Such increases in stress hormones may lead to pathophysiological alterations in the risk factors of cardiovascular disease (such as blood pressure, blood lipids, blood viscosity, and blood glucose), which promotes the development of hypertension, arteriosclerosis, ischemic heart disease, and stroke [11–13].

Because the overproduction of cortisol may cause inhibition of pancreatic insulin secretion and decreased insulin sensitivity in the liver, skeletal muscle, and adipose tissue [13], it is plausible that long-term noise exposure may produce adverse changes in blood glucose. Many animal and environmental epidemiological studies have determined the association between noise exposure and diabetes; however, few studies have reported the relationship between noise frequency characteristics. Two animal studies have observed the association between noise-induced hearing loss and diabetes in male Wistar rats [14, 15]. The effects of chronic noise exposure on diabetes onset have been found from alterations in gut microbiota composition and intestinal inflammation in rats [16] and from the exacerbation of insulin resistance to promote the manifestations in mice [17]. A cross-sectional study also found that impaired fasting glucose was a risk factor of noise-induced hearing loss among automobile manufacturing workers [18]. Environmental epidemiological studies have demonstrated the association between traffic noise exposure and the incidence [19] and mortality [20] of diabetes. A cohort study in Europe reported that an inter-quartile range (IQR) higher noise (4.2 A-weighted decibel, [dBA]) was associated with a 0.2% (95% confidence interval [CI]: 0.1–0.3%) increase in fasting glucose [21]. In a systematic literature review, a 6.0% (95% CI: 3.0–9.0%) increase in the risk of

diabetes was associated with the 5-dB increase in noise exposure, mainly related to air and road traffic noise [22]. To the best of our knowledge, no studies have been conducted to investigate the relationship between occupational noise exposure and incident hyperglycemia. Furthermore, little is known about the association between noise frequency components. Therefore, this retrospective study aimed to elucidate the relationship between exposure to occupational noise and incident hyperglycemia. We also determined whether there were differences in associations between hyperglycemia and different noise frequency components.

Methods

Study population

The detailed procedures to invite cooperating companies were mentioned in a previous study [10]. Briefly, we recruited 1028 volunteers in four machinery and equipment manufacturing companies in 2012. Among them, 2 subjects with a history of diabetes before employment, and 121 subjects followed-up for less than one year were excluded. Finally, we enrolled 905 participants as study subjects in this industry-based cohort. High noise levels were identified among workers in the processes of metal cutting, pressing, grinding, sand blasting, polishing, and gear washing. No subjects have the shift work.

The Institutional Review Board of the School of Public Health, China Medical University reviewed and approve this study. Written informed consent was acquired from each participant.

Hyperglycemia cases

We required all participants to fast overnight before blood sampling during the annual health examinations in 2012. Venous blood samples collected by trained nurses were used to perform blood glucose measurements by a standard glucose oxidase method. Hyperglycemia was defined as a positive response to the following questions: “have you been diagnosed with a diabetes by a physician?” or “do you use antidiabetic drugs, and was the treatment initiated after your employment start date with the current company?”, or if upon assessment in 2012 the fasting blood glucose (FBG) level was ≥ 100 mg/dl [23]. In addition, height, body weight, systolic blood pressure (SBP), diastolic blood pressure (DBP), total cholesterol level, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, and triglyceride levels were measured for all subjects. A trained nurse applied an automated sphygmomanometer (Ostar model P2; Ostar Meditech Corp., Taipei, Taiwan) to measure each subject’s bilateral blood pressure in sitting position, and the mean of 2 measurements was represented an individual’s blood pressure. We defined hypertensive cases as he or she had one or more of the following criteria: a diagnosed hypertension by a physician; taking antihypertensive medicine; a SBP of ≥ 140 mmHg; a DBP of ≥ 90 mmHg.

Potential risk factors related to hyperglycemia or diabetes were recognized by using a self-administered questionnaire. Demographic characteristics, lifestyle habits, a family history of diabetes, working activity, and the use of hearing-protection devices were collected and defined specifically to avoid information bias [10, 24]. Working activity was considered each subject’s time of sitting, walking, lifting heavy objects during working periods and the distance walked between the workplace the home that was further categorized into high and low levels based on the cut-off point of 10 in a scoring system [25].

Subjects’ follow-up

We applied employment personnel records from the four companies to obtain the date of first employment for each subject, and assigned this date retrospectively as the starting time to be followed up. The end of the follow-up time was established as either the date to be diagnosed as a diabetes case by a physician, the date of antidiabetic medication initiation, or the date on which blood glucose was measured in December 2012.

Occupational noise exposure evaluation and frequency component analyses

The processes of noise exposure evaluation and frequency component analyses were described in detailed in a previous study [10]. Briefly, we conducted a walk-through survey and combined the workplace information to identify different numbers of similar exposure groups (SEGs) for each participating company. Workers assigned to the same SEGs showed similarities in types and frequency of tasks, agents and processes involved, and in the way of performing tasks [26].

We used a personal noise dosimeter (Logging Noise Dose Meter Type 4443, Brüel & Kjær, Nærum, Denmark) to automatically report 5-minute continuous equivalent sound levels (Leq) with the unit of A-weighted decibel (dBA) during working periods (0800–17:00). The total amount of 96 values in 5-minute Leq (except from 12:00–13:00 pm) was used to calculate one value of 8-h time-weighted average noise levels for each SEG. Before conducting noise measurements, we calibrated this dosimeter with a sound-level calibrator (Type 4231, Brüel & Kjær, Nærum, Denmark) and setup its determining range between 50–120 dBA for all SEGs.

In addition, an octave-band analyzer (TES–1358, TES Electronic Corp., Taipei, Taiwan) was used to record 5-minute continuous Leq with the unit of decibel (dB) at frequencies of 31.5, 63, 125, 250, 500, 1000, 2000, 4000, and 8000 Hz during the monitoring periods. We applied the total amount of 96 values in 5-minute Leq at each frequency to calculate one value of 8-h time-weighted average noise levels for a specific frequency component. The analyzer was calibrated by a sound-level calibrator (TES–1356, TES Electronic Corp., Taipei, Taiwan) before noise measurements. The 8-h time-weighted average Leq and its octave-band frequencies were collected by two occupational hygienists to allocate specific levels of environmental noise (dBA) and octave-band frequencies (dB) for each SEG in the four companies.

Because the regulatory workplace monitoring from these companies showed no significant difference in noise levels within the last 10 years (82.89.1 dBA vs. 82.08.3 dBA), we assumed personal noise levels and the frequency spectrum of occupational noise to be equal over the employment duration. Subjects were divided into high-exposure, medium-exposure, and low-exposure groups based on noise exposure assessment. The cut-off value of 80 dBA was selected to classify field workers into the high-exposure (≥ 80 dBA) and medium-exposure (< 80 dBA) groups because this was the median value in the distribution of personal noise exposure. Office workers were chosen as the low-exposure group in the present study. We used the same approach to divide participants into the high-exposure (field workers exposed to \geq the median) and medium-exposure (field workers exposed to $<$ the median) groups to compare with the low-exposure (office workers) group because of the observation of large variations in median noise levels and exposure ranges at nine frequencies as shown in the Supplemental Figure S1. Additionally, we used per 5-dBA increase in personal noise exposure and per 5-dB increase in octave-band analyses of environmental noise to investigate associations with hyperglycemia.

Statistical analysis

The Shapiro-Wilk test was used to determine the normality of continuous variables. The Kruskal-Wallis test was used to perform multiple comparisons of continuous variables with non-normal distribution, among the three groups. We also applied the Chi-square test and Wilcoxon rank sum test to recognize the differences in dichotomous and continuous variables between the three groups, respectively. In addition, non-parametric Spearman correlation coefficients were estimated to investigate the correlation between individual noise exposure, environmental noise levels, and octave-band frequencies of environmental noise in the workplace.

Because this retrospective cohort study only obtained one measurement of FBG in December 2012, each participant's baseline FBG was established at that measured at the time of employment. The average length of time between baseline non-hyperglycemia FBG and follow-up FBG measurements was 7.5 years (median: 5.3 years; IQR: 7.9 years). We summed the cases of hyperglycemia identified by the questionnaire ($n = 1$) or by FBG measurements ($n = 118$) as the health outcome to conduct the Cox proportional hazard regression analyses. The relative risks (RRs) with 95% confidence intervals (CIs) were

calculated to compare the differences in incident hyperglycemia with different groups. A basic model (Model 1) was first established to include age and sex for biological plausibility, and two dummy variables of exposure groups. Later, we extended this model to cover 2 variables (triglyceride level and hypertension) that were significantly associated with the incident hyperglycemia in simple Cox regressions (Model 2). The final model (Model 3) was set-up to include all variables in Model 2 and one confounder (the use of hearing-protection devices). We excluded the employment duration in the final model due to its high correlation with age in the present study (Spearman's correlation coefficient was 0.648, $p < 0.001$). The educational level and working activity were not included because both variables were not significant in simple Cox regressions. The stratified analyses were used to determine the effect modification of selected demographic characteristics and to test the interaction between high-exposure and low-exposure groups. We applied the SAS standard package for Windows version 9.4 (SAS Institute Incorporation, Cary, North Carolina, USA) to analyze data and set the significance level at 0.050 for all tests. The Bonferroni correction (i.e., the statistical significance level is decreased in proportion to the number of comparisons made (i.e. $n = 2$; $p < 0.025$)) was also used to avoid significant results in multiple comparisons purely by chance rather than real differences existing between groups.

Results

The demographic characteristics of the three study groups are presented in Table 1. Significant group-differences in mean age, employment duration, and SBP, and in the proportions of male sex, high educational levels, current smokers, regular exercisers, high working activity, and workers using hearing-protection devices were observed (all $p < 0.05$). The high- and medium-exposure groups had significantly higher means in SBP and higher proportions of male sex, current smokers, and high working activity, but lower means in employment duration and the proportions of high educational levels and regular exercise than those in the low-exposure group. In addition, workers in the high-exposure group were more likely to use hearing-protection devices than did the medium- and low-exposure groups (both $p < 0.05$).

Supplemental Table S1 shows the correlations between personal exposure, environmental levels, and octave-band frequencies of workplace noise. Personal noise levels correlated significantly with environmental levels and all octave-band frequencies (all $p < 0.050$), and higher correlations (correlation coefficients > 0.810) were observed at frequencies of 250, 500, and 1000 Hz.

Personal noise levels and octave-band analyses of environmental noise pertaining to different groups are presented in Table 2. Significantly higher mean levels of environmental and personal noise exposure were observed in the high- and medium-exposure groups compared with those measured in the low-exposure group (both $p < 0.05$). In addition, the high- and medium-exposure groups had significantly higher averages of noise levels at all octave-band frequencies than those measured in the low-exposure group (all $p < 0.05$).

The mean FBG and the RR of hyperglycemia for the three groups were shown in Table 3. A significant difference in FBG between groups was observed ($p < 0.05$). Both high-exposure and low-exposure groups had significantly higher mean FBG values than did the medium-exposure group (both p values < 0.05).

Table 4 presents the association between occupational noise exposure and risk of incident hyperglycemia. Workers exposed to 80 dBA had an increased RR for hyperglycemia of 1.78 (95% CI: 1.11, 2.84) compared with those exposed to < 70 dBA, before and after the Bonferroni correction. A direct exposure-response association was found between noise exposure and risk of hyperglycemia for all the three groups (Adjusted RR [ARR] = 1.28; 95% CI: 1.04, 1.59; $p = 0.023$). Sex was the only parameter found as the effect modification variable for comparisons between the high-exposure and low-exposure groups, as shown in Supplemental Figure S2 ($p = 0.016$). Women were more susceptible to an increased risk of incident hyperglycemia than were men (ARR = 4.77; 95% CI: 1.87, 12.17; $p = 0.001$).

Figure 1 shows the risk of incident hyperglycemia according to octave-band frequencies of workplace noise exposure by group. High-exposure groups at frequencies of 31.5, 63, 125, 250, 500, 1000, and 2000 Hz had significantly increased risks of incident hyperglycemia compared with low-exposure groups, before the Bonferroni correction (all $p < 0.050$), but only those at

frequencies of 31.5, 1000, and 2000 Hz had significant results after the Bonferroni correction (p values < 0.025). The strongest association was observed at 31.5 Hz. Participants exposed to 36.7 ± 3.1 dB at 31.5 Hz had an increased RR for hyperglycemia of 1.95 (95% CI: 1.26, 3.00) compared with those exposed to 25.4 ± 4.6 dB at the same frequency, after the Bonferroni correction ($p = 0.003$).

The risk of incident hyperglycemia according to a 5-dBA increase in personal noise levels and 5-dB increase at octave-band frequencies is shown in Figure 2. Five-dB increases in environmental noise levels at frequencies of 31.5, 63, 125, 250, 500, and 1000 Hz were associated with the incidence of hyperglycemia (all $p < 0.05$), with the highest risk of 1.27 (95% CI: 1.10, 1.47) at 31.5 Hz ($p = 0.001$). Only the sex variable was observed to modify the association between exposure to occupational noise and the incident hyperglycemia, as shown in Supplemental Table S2 ($p = 0.025$). A 5-dBA increase in occupational noise was associated with a 1.48-fold increase in risk of incident hyperglycemia among women (95% CI: 1.12, 1.96; $p = 0.005$).

Discussion

Subjects exposed to occupational noise levels 80 dBA had a significantly higher risk of hyperglycemia compared with those exposed to <70 dBA before and after the Bonferroni correction. We also observed a significant exposure-response relationship among the high-, medium-, and low-exposure groups (ARR = 1.28; 95% CI: 1.04, 1.59; $p = 0.023$). These results are consistent with findings in a population-based cohort study of 57,053 residents, which found a significant and increased risk of incident diabetes (ARR = 1.11; 95% CI: 1.03, 1.19) per 10-dB increase in road traffic noise [19], and with findings in a case-crossover study from Madrid (Spain) (2001–2009), which reported a strong association between a rise of 0.5 dBA in night-time traffic noise at a 1-day lag and a 4.6% risk (95% CI: 1.5, 7.8) of diabetic mortality [20]. The present and previous two studies all support the possibility that noise-induced cardiovascular disease may result from the activation of impaired metabolism which lead to increased blood glucose levels [11, 12]. In contrast, a cross-sectional survey did not observe the significant association between self-reported occupational noise exposure and diabetes among 23 486 European participants [27]. The inconsistent finding may be due to the more accurate exposure assessment in measured and modeled noise levels [19, 20] compared with the subjective reported ones [27]. In addition to noise-induced hearing loss [28, 29] and cardiovascular disease [1–10], the possibility of developing hyperglycemia by occupational noise exposure should be considered in future studies.

Exposure to noise levels at 31.5, 63, 125, 250, 500, 1000, and 2000 Hz was associated with the incidence of hyperglycemia, and the highest risk was found at 31.5 Hz. Significant exposure-response relationships were also identified at 31.5 Hz (ARR = 1.39; 95% CI: 1.11, 1.74; $p = 0.004$), 1000 Hz (ARR = 1.33; 95% CI: 1.07, 1.66; $p = 0.011$) and 2000 Hz (ARR = 1.35, 95% CI: 1.08, 1.68; $p = 0.007$). These findings indicated that machinery and equipment manufacturing workers may be more sensitive to low and medium frequencies to elevate blood glucose. The real reason for such observations is unknown. A cross-sectional study also reported a strong association between diabetes and hearing loss at low- and medium-frequencies [30]. Annoyance and stress caused by the low-frequency noise at work may be the other reason to increase the risk of diabetes [22, 31]. Therefore, we recommend conducting additional studies in the future to investigate the association between the frequency spectrum of noise exposure and specific physiological functions.

We observed 5-dB increases at frequencies of 31.5, 63, 125, 250, 500, and 1000 Hz associated with an increased risk of hyperglycemia that may indicate the noise-induced-hyperglycemia involving multiple pathways in the biological mechanism. Low-frequency occupational noise exposure (i.e., 31.5, 63, 125, and 250 Hz) may pose hyperglycemia indirectly through responses such as annoyance and the disturbance experienced during activities requiring selective attention or while dealing with high-load information [11, 12, 32–34]. In contrast, middle-frequency occupational noise exposure (i.e., 500 and 1000 Hz) may cause hyperglycemia directly by repeated and prolonged stimulation of the autonomic nervous and endocrine systems [11, 12, 35, 36]. However, more evidence is required to elucidate the reasons for the association between frequency components of noise exposure and the incidence of hypertension.

The association between occupational noise exposure and incident hyperglycemia was significantly influenced by sex in the present study. Women had a higher risk of hyperglycemia compared with men. One previous study reported no significant effect modification by sex, but it also found a stronger relationship of hyperglycemia with road traffic noise among women (ARR = 1.11; 95% CI: 1.03, 1.20) compared to that among men (ARR = 1.05; 95% CI: 0.98, 1.13) [19]. Future studies are suggested to consider this modifier for investigating the association between noise exposure and hyperglycemia.

The strength of this study lies in the retrospective cohort design, which was formulated to calculate the observed person-years and distinguish the noise-induced effect after the longitudinal follow up. Therefore, the temporal association between exposure to occupational noise and the incident hyperglycemia limited in cross-sectional studies could be assessed in this study. In addition, personal exposure assessment, environmental noise measurements, and octave-band analyses of workplace noise were conducted to provide a precise and accurate evaluation of noise exposure in a real-world workplace setting.

This study has some limitations that must be mentioned. The major restriction is the retrospective design to generate a healthy-worker effect that may produce the lower proportion of hyperglycemia cases in the high-noise-exposure group or the higher incidence of hyperglycemia in the lower-noise-exposure group. The second is to underestimate the observed person-years because missing information on the noise exposure history before participants began employment for the current company or the diagnosis date of diabetes by a physician. The third is no collection of data on noise exposure out of the workplace during the employment period. Exposure to aircraft and road traffic noise have been reported with the increased risk of diabetes in a meta-analysis study [22]. The fourth is the potential recall bias in lifestyle habits that affect diabetes are only measured in 2012. Finally, participants' information about sleep disturbance and noise annoyance at home is not obtained. Noise-induced sleep disturbance and annoyance are both possible mechanisms to generate the cardio-metabolic effects [22]. This restriction may overestimate the effect of occupational noise exposure on the incident hyperglycemia.

Conclusions

Regardless of these limitations, this study observed the association between occupational noise exposure and an increased risk of incident hyperglycemia. Positive and linear exposure-response relationships have been demonstrated at noise frequency components of 31.5, 63, 125, 250, 500, 1000, and 2000 Hz. The machinery and equipment manufacturing workers exposed to noise levels at 31.5 Hz may have the strongest risk of hyperglycemia. These findings provide a possible link between noise exposure and cardio-metabolic disease. We recommend future studies to determine the associations between hyperglycemia and octave-band frequencies of occupational noise exposure in the different industries.

List Of Abbreviations

ARR, adjusted relative risk; CI, confidence interval; dB, decibel; dBA, A-weighted decibel; DBP, diastolic blood pressure; FBG, fasting blood glucose; Leq, equivalent sound levels; RR, relative risk; SD, standard deviation; SBP, systolic blood pressure; SEG, similar exposure group.

Declarations

Ethical Approval and Consent to participate:

All participants provided informed consent and the protocol was reviewed and approved by the Institutional Review Board of the School of Public Health, China Medical University (No. 100-03-10-4).

Consent for publication

No applicable.

Availability of data and materials

The datasets generated and analyzed during the current study are not publicly available due the confidentiality agreement with participating companies but are available from the corresponding author on reasonable request.

Competing interests

The authors declare that they have no competing interests.

Funding

The National Science Council, Taiwan (NSC 100–2221-E–039–004) provided the financial support for this study.

Authors' contributions

TYC, CSL and LHY designed the study and directed its implementation, including quality assurance and control. LHY and TYC supervised the field activities and designed the study's analytic strategy. CSL and TYY conducted the literature review and prepare the Materials and Methods and the Discussion sections of the text. BYB and TYC interpreted the data and prepared the Results and the Discussion sections of this manuscript. All of authors have read and approved the final manuscript.

Acknowledgements

We thank the National Science Council, Taiwan (NSC 100–2221-E–039–004) to provide the financial support. We also thank all volunteers willing to join in this study.

Authors' information

Department of Occupational Safety and Health, College of Public Health, China Medical University, No. 91, Hsueh-Shih Road, Taichung 40402, Taiwan, Republic of China (Li-Hao Young, Tzu-Yi Yu, Ta-Yuan Chang); Department of Family Medicine, China Medical University Hospital, No. 2, Yuh-Der Road, Taichung 40447, Taiwan, Republic of China (Chiu-Shong Liu); School of Medicine, College of Medicine, China Medical University, Taichung, Taiwan, Republic of China (Chiu-Shong Liu); Department of Pharmacy, College of Pharmacy, China Medical University, Taichung, Taiwan, Republic of China (Bo-Ying Bao); Department of Nursing, Asia University, No. 500, Lioufeng Road, Wufeng, Taichung 41354, Taiwan, Republic of China (Bo-Ying Bao).

References

1. McNamee R, Burgess G, Dipnall WM, Cherry N: *Occupational noise exposure and ischaemic heart disease mortality. Occupational and environmental medicine* 2006, *63*(12):813–819.
2. Virkkunen H, Kauppinen T, Tenkanen L: *Long-term effect of occupational noise on the risk of coronary heart disease. Scandinavian journal of work, environment & health* 2005, *31*(4):291–299.
3. Virkkunen H, Harma M, Kauppinen T, Tenkanen L: *The triad of shift work, occupational noise, and physical workload and risk of coronary heart disease. Occupational and environmental medicine* 2006, *63*(6):378–386.
4. Gan WQ, Davies HW, Demers PA: *Exposure to occupational noise and cardiovascular disease in the United States: the National Health and Nutrition Examination Survey 1999–2004. Occupational and environmental medicine* 2011, *68*(3):183–190.

5. Davies HW, Teschke K, Kennedy SM, Hodgson MR, Hertzman C, Demers PA: *Occupational exposure to noise and mortality from acute myocardial infarction. Epidemiology* 2005, *16*(1):25–32.
6. Willich SN, Wegscheider K, Stallmann M, Keil T: *Noise burden and the risk of myocardial infarction. European heart journal* 2006, *27*(3):276–282.
7. Bortkiewicz A, Gadzicka E, Siedlecka J, Szyjkowska A, Viebig P, Wrancich JK, Kurpesa M, Dziuba M, Trzos E, Makowiec-Dabrowska T: *Work-related risk factors of myocardial infarction. International journal of occupational medicine and environmental health* 2010, *23*(3):255–265.
8. Sbihi H, Davies HW, Demers PA: *Hypertension in noise-exposed sawmill workers: a cohort study. Occupational and environmental medicine* 2008, *65*(9):643–646.
9. Chang TY, Hwang BF, Liu CS, Chen RY, Wang VS, Bao BY, Lai JS: *Occupational noise exposure and incident hypertension in men: a prospective cohort study. American journal of epidemiology* 2013, *177*(8):818–825.
10. Liu CS, Young LH, Yu TY, Bao BY, Chang TY: *Occupational Noise Frequencies and the Incidence of Hypertension in a Retrospective Cohort Study. American journal of epidemiology* 2016, *184*(2):120–128.
11. Babisch W: *The Noise/Stress Concept, Risk Assessment and Research Needs. Noise & health* 2002, *4*(16):1–11.
12. Munzel T, Gori T, Babisch W, Basner M: *Cardiovascular effects of environmental noise exposure. European heart journal* 2014, *35*(13):829–836.
13. Recio A, Linares C, Banegas JR, Diaz J: *Road traffic noise effects on cardiovascular, respiratory, and metabolic health: An integrative model of biological mechanisms. Environmental research* 2016, *146*:359–370.
14. Wu HP, Cheng TJ, Tan CT, Guo YL, Hsu CJ: *Diabetes impairs recovery from noise-induced temporary hearing loss. The Laryngoscope* 2009, *119*(6):1190–1194.
15. Wu HP, Hsu CJ, Cheng TJ, Guo YL: *N-acetylcysteine attenuates noise-induced permanent hearing loss in diabetic rats. Hearing research* 2010, *267*(1–2):71–77.
16. Cui B, Gai Z, She X, Wang R, Xi Z: *Effects of chronic noise on glucose metabolism and gut microbiota-host inflammatory homeostasis in rats. Sci Rep* 2016, *6*:36693.
17. Liu L, Huang Y, Fang C, Zhang H, Yang J, Xuan C, Wang F, Lu H, Cao S, Wang Y *et al*: *Chronic noise-exposure exacerbates insulin resistance and promotes the manifestations of the type 2 diabetes in a high-fat diet mouse model. PloS one* 2018, *13*(3):e0195411.
18. Jang TW, Kim BG, Kwon YJ, Im HJ: *The association between impaired fasting glucose and noise-induced hearing loss. Journal of occupational health* 2011, *53*(4):274–279.
19. Sorensen M, Andersen ZJ, Nordsborg RB, Becker T, Tjonneland A, Overvad K, Raaschou-Nielsen O: *Long-term exposure to road traffic noise and incident diabetes: a cohort study. Environmental health perspectives* 2013, *121*(2):217–222.
20. Tobias A, Diaz J, Recio A, Linares C: *Traffic noise and risk of mortality from diabetes. Acta diabetologica* 2015, *52*(1):187–188.
21. Cai Y, Hansell AL, Blangiardo M, Burton PR, BioShaRe, de Hoogh K, Doiron D, Fortier I, Gulliver J, Hveem K *et al*: *Long-term exposure to road traffic noise, ambient air pollution, and cardiovascular risk factors in the HUNT and lifelines cohorts. European heart journal* 2017, *38*(29):2290–2296.

- 22.Zare Sakhvidi MJ, Zare Sakhvidi F, Mehrparvar AH, Foraster M, Dadvand P: *Association between noise exposure and diabetes: A systematic review and meta-analysis. Environmental research* 2018, 166:647–657.
- 23.American Diabetes A: *Standards of medical care in diabetes—2013. Diabetes care* 2013, 36 Suppl 1:S11–66.
- 24.Chang TY, Liu CS, Young LH, Wang VS, Jian SE, Bao BY: *Noise frequency components and the prevalence of hypertension in workers. The Science of the total environment* 2012, 416:89–96.
- 25.Hwang LC, Chen CJ, Tsieng WP: *A nested case control study on multiple risk factors for acute fatal cerebrovascular accident and coronary heart disease. Chin J Fam Med (Taiwan)* 1997, 7:121–130.
- 26.Nulhausen JR, Damiano J: *A strategy for assessing and managing occupational exposure*, 2nd edn. Fairfax, VA: American Industrial Hygiene Association 1998.
- 27.Dzhambov AM: *Exposure to self-reported occupational noise and diabetes - A cross-sectional relationship in 7th European Social Survey (ESS7, 2014). International journal of occupational medicine and environmental health* 2017, 30(4):537–551.
- 28.Rubak T, Kock SA, Koefoed-Nielsen B, Bonde JP, Kolstad HA: *The risk of noise-induced hearing loss in the Danish workforce. Noise & health* 2006, 8(31):80–87.
- 29.Seixas NS, Neitzel R, Stover B, Sheppard L, Feeney P, Mills D, Kujawa S: *10-Year prospective study of noise exposure and hearing damage among construction workers. Occupational and environmental medicine* 2012, 69(9):643–650.
- 30.Bamanie AH, Al-Noury KI: *Prevalence of hearing loss among Saudi type 2 diabetic patients. Saudi medical journal* 2011, 32(3):271–274.
- 31.Leventhall HG: *Low frequency noise and annoyance. Noise & health* 2004, 6(23):59–72.
- 32.Pawlaczyk-Luszczynska M, Dudarewicz A, Waszkowska M, Sliwinska-Kowalska M: *Assessment of annoyance from low frequency and broadband noises. International journal of occupational medicine and environmental health* 2003, 16(4):337–343.
- 33.Bengtsson J, Waye KP, Kjellberg A: *Evaluations of effects due to low-frequency noise in a low demanding work situation. J Sound Vib* 2004, 278(1–2):83–99.
- 34.Pawlaczyk-Luszczynska M, Dudarewicz A, Waszkowska M, Szymczak W, Sliwinska-Kowalska M: *The impact of low-frequency noise on human mental performance. International journal of occupational medicine and environmental health* 2005, 18(2):185–198.
- 35.Truswell WHt, Randolph KJ, Snyder GG, 3rd: *The effect of static tympanic pressure gradients on hearing sensitivity in normal subjects. The Laryngoscope* 1979, 89(2 Pt 1):306–310.
- 36.Maurizi M, Paludetti G, Ottaviani F, Rosignoli M: *Auditory brainstem responses to middle- and low-frequency tone pips. Audiology: official organ of the International Society of Audiology* 1984, 23(1):75–84.

Tables

Table 1. Demographic characteristics for subjects of the study conducted in 2012 in Taichung, Taiwan.

Variable	Noise exposure group						Total subjects (n = 905)			P value			
	High (n = 281)			Medium (n = 260)			Low (n = 364)						
	No.	%	Mean (SD)	No.	%	Mean (SD)	No.	%	Mean (SD)				
Age, years			38.1(9.3) ^c			33.9(6.7) ^d			39.0(7.8)			37.3(8.3)	<0.001 ^a
Employment duration, years			8.7(7.4) ^{c,d}			7.0(6.3) ^d			11.6(8.2)			9.4(7.7)	<0.001 ^a
Body mass index ^a			24.3(3.6)			24.3(3.8)			24.0(3.8)			24.2(3.7)	0.454 ^a
Systolic BP			126.7 (13.4) ^d			125.3 (11.6) ^d			123.3 (14.5)			124.9 (13.4)	<0.001 ^a
Diastolic BP			82.3 (10.2)			82.4 (8.8)			82.2 (10.8)			82.3 (10.1)	0.824 ^a
Triglyceride level, mg/dl			127.8(187.1)			117.8(67.4)			124.9(98.7)			123.8(126.8)	0.363 ^a
Total cholesterol level, mg/dl			189.0(37.8)			187.4(33.5)			189.8(34.1)			188.9(35.1)	0.514 ^a
Gender, male	241	85.8 ^e		226	86.9 ^e		259	71.2		726	80.2		<0.001 ^b
Educational level, >12 years	78	27.8 ^{e,f}		157	60.4 ^e		300	82.4		535	59.1		<0.001 ^b
Current smoker, yes	96	34.2 ^e		70	26.9 ^e		58	15.9		224	24.8		<0.001 ^b
Alcohol consumption, yes	36	12.8		37	14.2		34	9.3		107	11.8		0.145 ^b
Tea consumption, yes	143	50.9		135	51.9		196	53.9		474	52.4		0.746 ^b
Coffee consumption, yes	124	44.1		104	40.0		157	43.1		385	42.5		0.598 ^b
Regular exercise, yes	92	32.7 ^e		87	33.5 ^e		166	45.6		345	38.1		<0.001 ^b
Hypertension, yes	84	29.9		67	25.8		87	23.9		238	26.3		0.224 ^b
Family history of diabetes, yes	49	17.4		48	18.5		78	21.4		175	19.3		0.407 ^b
Working activity, high	116	41.3 ^e		114	43.9 ^e		35	9.6		265	29.3		<0.001 ^b
Use of hearing-protection devices at work, yes	33	11.2 ^{e,f}		8	3.1		3	0.8		44	4.9		<0.001 ^c

BP, blood pressure; SD, standard deviation. ^a Kruskal-Wallis test of difference between the three groups. ^b Chi-square test of difference between the three groups. ^c Wilcoxon rank sum test of the significant difference (p<0.05) compared with the medium-exposure group. ^d Wilcoxon rank sum test of the significant difference (p<0.05) compared with the low-exposure group. ^e Chi-square test of the significant difference (p<0.05) compared with the low-exposure group. ^f Chi-square test of the significant difference (p<0.05) compared with the medium-exposure group.

Table 2. Means and standard deviations of noise exposure and frequency components for participants measured in 2012 in Taichung, Taiwan.

Variable	Noise exposure group						Total	p-value	
	High Mean (SD)	Median (IQR)	Medium Mean (SD)	Median (IQR)	Low Mean (SD)	Median (IQR)			Mean (SD)
Personal level (dBA)	84.3(3.6)	83.3 (4.7) ^{b,c}	73.6(6.4)	75.2 (10.4) ^c	67.7(3.8)	66.4 (6.6)	74.6 (8.4)	75.2 (15.1)	<0.001 ^a
Environmental level (full frequency, dBA)	79.3(4.2)	79.2 (5.0) ^{b,c}	69.0(8.9)	71.0 (15.5) ^c	57.1(3.1)	55.0 (3.4)	67.4 (10.9)	68.5 (21.6)	<0.001 ^a
31.5 Hz (dB)	35.3(4.0)	34.9 (4.3) ^{b,c}	28.7(4.1)	28.8 (4.8) ^c	25.4(4.6)	21.7 (7.8)	29.4 (6.0)	29.3 (10.1)	<0.001 ^a
63 Hz (dB)	46.9(4.1)	46.3 (6.1) ^{b,c}	39.5(6.4)	41.9 (10.9) ^c	33.1(4.8)	30.1 (4.7)	39.2 (7.7)	41.5 (14.7)	<0.001 ^a
125 Hz (dB)	55.5(4.3)	55.4 (6.2) ^{b,c}	47.9(6.5)	50.4 (10.4) ^c	38.6(5.3)	34.9 (5.7)	46.5 (9.0)	48.2 (15.3)	<0.001 ^a
250 Hz (dB)	62.7(4.2)	62.9 (4.2) ^{b,c}	54.7(6.9)	58.1 (9.8) ^c	45.2(3.8)	42.5 (4.0)	53.4 (8.9)	53.9 (15.9)	<0.001 ^a
500 Hz (dB)	68.4(4.4)	67.2 (5.7) ^{b,c}	60.7(6.8)	63.2 (12.3) ^c	51.8(4.1)	48.5 (5.9)	59.5 (8.6)	60.0 (14.9)	<0.001 ^a
1000 Hz (dB)	71.2(4.5)	71.3 (5.9) ^{b,c}	62.3(7.0)	64.4 (14.4) ^c	52.7(3.9)	49.8 (4.7)	61.2 (9.3)	61.2 (17.1)	<0.001 ^a
2000 Hz (dB)	72.1(4.5)	72.3 (4.1) ^{b,c}	61.9(6.5)	65.3 (12.5) ^c	55.5(1.6)	55.8 (2.1)	62.5 (8.2)	59.4 (14.8)	<0.001 ^a
4000 Hz (dB)	73.0(4.8)	73.7 (6.4) ^{b,c}	62.6(7.1)	65.4 (14.7) ^c	53.6(1.4)	54.7 (2.5)	62.2 (9.4)	59.9 (17.6)	<0.001 ^a
8000 Hz (dB)	70.2(4.9)	70.6 (6.7) ^{b,c}	60.1(6.5)	60.6 (10.8) ^c	51.4(0.8)	52.1 (1.6)	59.7 (9.1)	59.3 (16.7)	<0.001 ^a

dB, decibel; dBA, A-weight decibel; IQR, interquartile range; SD, standard deviation. ^a Kruskal-Wallis test for the difference ($p < 0.05$) between the three groups. ^b Wilcoxon rank sum test for a significant difference ($p < 0.05$) compared with the medium-exposure group. ^c Wilcoxon rank sum test for a significant difference ($p < 0.05$) compared with the low-exposure group.

Table 3. Fasting blood glucose and relative risk of hyperglycemia in the study group.

Noise group	exposure n	FBG, mg/dl, mean (SD)	HG cases, n	Person-years	Incident rate	Crude RR (95% CI)	p-value
Low	364	90.9±9.5	47	3149.7	1.49×10 ⁻²	1.00	--
Medium	260	89.1±15.7 ^c	26	1485.5	1.83×10 ⁻²	1.34(0.82-2.18)	0.237
High	281	91.4±12.6 ^b	46	2175.8	2.06×10 ⁻²	1.46(0.97-2.20)	0.070
P<0.001 ^a							

CI, confidence interval; FBG, fasting blood glucose; HG, hyperglycemia; RR, relative risk; SD, standard deviation. ^a Kruskal-Wallis test for the difference between the three groups. ^b Wilcoxon rank sum test for a significant difference ($p < 0.05$) compared with the medium-exposure group. ^c Wilcoxon rank sum test for a significant difference ($p < 0.05$) compared with the low-exposure group.

Table 4. Association between occupational noise exposure and risk of incident hyperglycemia among participants.

Variable	Model 1 ^a			Model 2 ^b			Model 3 ^c		
	ARR	95% CI	p-value	ARR	95% CI	p-value	ARR	95% CI	p-value
Noise exposure group									
Low	1.00	Referent	–	1.00	Referent	–	1.00	Referent	–
Medium	1.23	0.74, 2.03	0.434	1.10	0.66, 1.83	0.716	1.09	0.66, 1.82	0.728
High	1.39	0.92, 2.11	0.118	1.49	0.98, 2.25	0.063	1.64	1.08, 2.50	0.021
Triglyceride level, mg/dL									
<99				1.00	Referent	–	1.00	Referent	–
≥99				1.66	1.10, 2.49	0.016	1.63	1.08, 2.45	0.020
Hypertension									
No				1.00	Referent	–	1.00	Referent	–
Yes				1.66	1.14, 2.42	0.008	1.65	1.13, 2.40	0.009
The use of hearing-protection devices									
No							1.00	Referent	–
Yes							0.36	0.13, 0.99	0.049

ARR, adjusted relative risk; CI, confidence interval; dBA, A-weighted decibel. ^a The Cox regression model adjusted for age and gender. ^b The Cox regression model adjusted for age, gender, triglyceride level, and hypertension (yes versus no). ^c The Cox regression model adjusted for age, gender, triglyceride level, hypertension (yes versus no) and the use of hearing-protection devices (yes versus no)

Figures

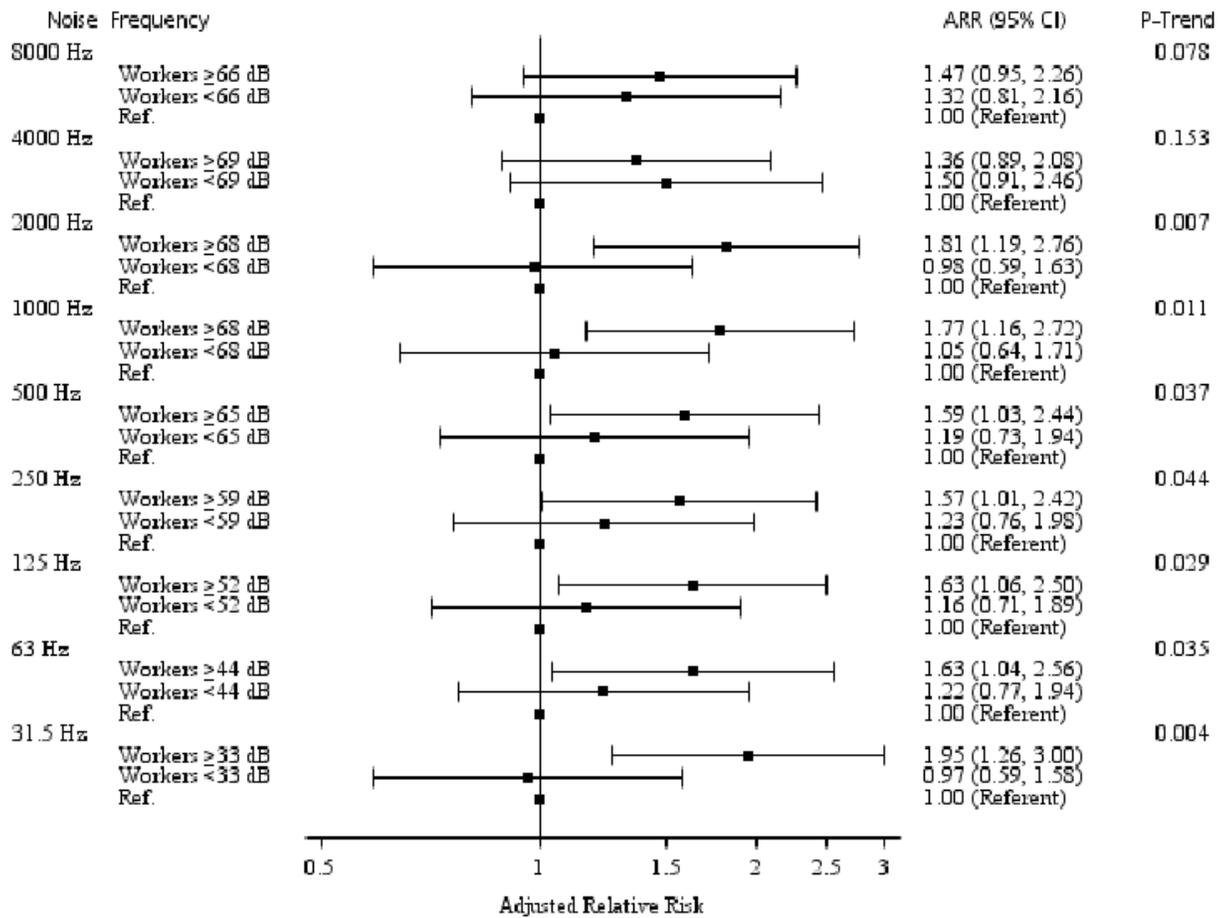


Figure 1

Adjusted relative risk (ARR) of incident hyperglycemia according to octave-band frequencies of occupational noise exposure for participants. ARR, adjusted relative risk; CI, confidence interval; dB, decibel; Ref, reference (i.e., officers). a Cox regression model adjusted for age, sex, triglyceride level, hypertension, and the use of hearing-protection devices.

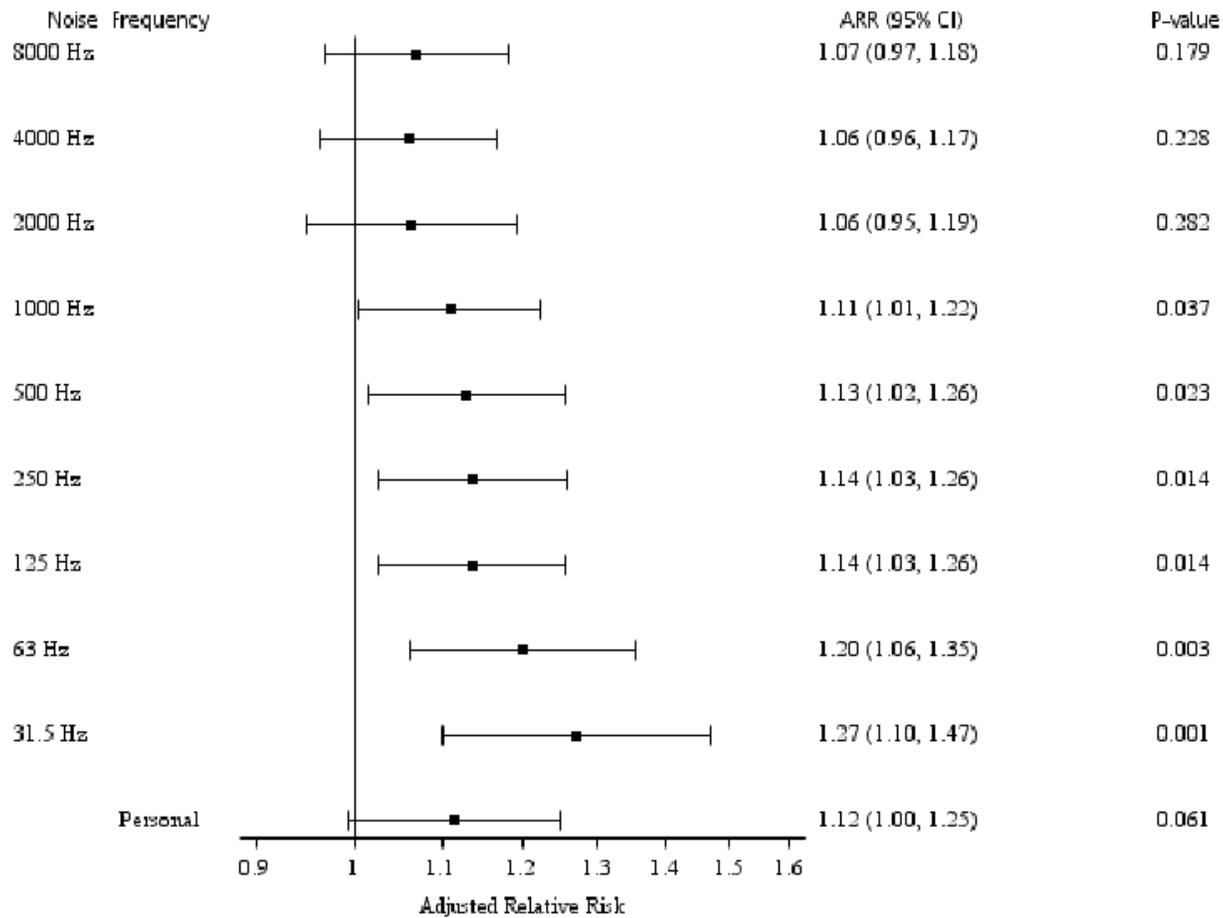


Figure 2

Adjusted relative risk (ARR)^a of incident hyperglycemia according to 5-dBA increase for personal noise levels and 5-dB increase for octave-band frequencies among participants. ARR, adjusted relative risk; CI, confidence interval; dB, decibel; dBA, A-weighted decibel. a Cox regression model adjusted for age, sex, triglyceride level, hypertension, and the use of hearing-protection devices.

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- [ONFandFBGSupplementalMaterial20191102finalEH.docx](#)