Research Square

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Acute traffic noise induces sympathetic overactivation and metabolic dysfunction in healthy adults while mask sound offers protection: A randomized crossover trial

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Abstract

Background: With urbanization accelerating, traffic noise has become a significant environmental factor in daily urban life.

Objective: This study investigates the effects of short-term traffic-noise exposure on energy metabolism and physiological status in healthy individuals, systematically characterizing the metabolic pathways induced by noise and exploring the protective role of mask sound (natural mixed sound) intervention.

Methods: A randomized crossover design was employed, with 26 healthy adults exposed to three types of sound environments: quiet (46.52 dBA (± 0.31), 8:00 AM - 7:00 AM the next day), traffic noise (76.23 dBA (± 1.21), 8:00 AM - 10:00 PM), and mask sound (76.15 dBA (± 1.32), 8:00 AM - 10:00 PM). Continuous monitoring tracked energy metabolism, physiological indicators, hormone levels, and subjective feelings, with comparisons across environments.

Results: Short-term traffic noise amplified sympathetic, HPA, and HPT axis activity, raising heart rate and blood pressure (HR +2.95%, diastolic BP +3.09%). Glycemic regulation worsened (glucose up 7.72%), metabolic flexibility declined (ΔNPRQ postprandial − fasting −42.11%), and rhythms/mood were disrupted. Women exhibited more pronounced autonomic and metabolic disturbances. Mask sound modestly mitigated adverse effects, improving cardiovascular measures (HR +2.55% decline, diastolic BP +0.3%), lowering insulin resistance (glucose down 4.39%), and enhancing metabolic flexibility (ΔNPRQ postprandial–fasting +41.67%), though protection was weaker in women. **Conclusions**: We concluded that short-term traffic noise triggers broad neuroendocrine activation and may elevate the risk of cardiovascular and metabolic diseases, particularly in women. Mask sound intervention effectively attenuates acute metabolic and physiological damage, suggesting potential nonpharmacological benefits for noise protection.

Trial registration: ChiCTR2500098645

Introduction

The rapid urbanization and motorization worldwide have made traffic noise an undeniable public health burden, directly challenging the goal of "inclusive, safe, and sustainable cities and communities" outlined in Sustainable Development Goal (SDG 11) [1]. The World Health Organization (WHO) 2018 guidelines on environmental noise recommend threshold levels that are frequently exceeded in many rapidly urbanizing large cities [2]. Epidemiological evidence suggests that with the acceleration of urbanization, traffic noise has emerged as a significant environmental risk factor affecting the public health of urban residents [3,4]. The WHO assessment of populations in Europe indicates that with each increase of 10 A-weighted decibels (dBA) in road traffic noise, the risk of ischemic heart disease rises by 8%, and the incidence of diabetes increases by 8% [2,5]. Beyond hearing impairment and emotional damage, traffic noise is closely associated with psychological disorders, sleep disturbances, cardiovascular diseases, and metabolic abnormalities [6-11].

Acute noise exposure triggers complex physiological responses beyond auditory processing. The central nervous system initiates neural and endocrine cascades, with the autonomic nervous system regulating cardiovascular control and activating the HPA axis, releasing stress hormones [4,12]. Noise also indirectly activates hypothalamic pathways by affecting sleep and releasing stress signals [13]. Mouse studies show noise disrupts sleep architecture and circadian rhythms [14]. Noise causes inflammation, oxidative stress, eNOS/nNOS uncoupling, and mitochondrial ROS production, contributing to vascular and neural dysfunction [14], and increasing the risk of aging and metabolic diseases [15]. Noise perturbs cardiovascular stability and insulin homeostasis in animals. Mice exposed to noise develop insulin resistance, linked to reduced Akt signaling and GLUT4 translocation [16]. Simulated aircraft noise elevates systolic blood pressure, norepinephrine, and angiotensin II in mice [13]. These effects form a multi-layered network driving injury progression [14]. While animal and human physiological thresholds are similar [17], human evidence on noise-induced stress and neuroendocrine responses is limited, often relying on long-term epidemiological studies linking noise to cardiovascular damage and insulin resistance [18,19]. Noise increases sympathetic nervous activity [20]. An RCT showed noise amplifies air pollution's effects on heart rate variability at high noise levels (>65.6 dBA) [21]. Overactive sympathetic nerves precede insulin resistance, promoting obesity and metabolic syndrome [22,23], but an RCT linking this to noise is lacking. Only one RCT reported noise's effect on glycemic and insulin homeostasis, showing traffic noise exposure reduced glycemic tolerance and insulin sensitivity in healthy subjects after four nights, but mechanisms weren't explored [11]. Human studies indicate noise causes sleep and circadian rhythm disturbances, but underlying reasons remain unclear [24,25]. The effects of traffic noise on human autonomic nervous system and metabolic health require further clinical investigation.

Traffic noise causes broad and far-reaching harm. Mitigation strategies typically involve blocking, eliminating, or mask sound, with mask sounds (e.g., flowing water) being the simplest and most cost-effective option [26,27]. Mask sounds may exert neuromodulatory effects, elevating positive affect, improving functional connectivity of the brain, and reducing irritability and stress responses. Experimental and observational studies show that mask sounds significantly enhance activation in the left medial prefrontal cortex and the right medial orbitofrontal cortex, with the associated emotional effects linked to pleasant experiences [28]. Meanwhile, mask sounds are thought to enhance mood and psychological comfort, and to reduce negative responses associated with noise [29]. The protective effect is more evident under higher noise levels; when natural soundscapes are overlaid with traffic noise, the positive emotional effects of natural sounds may decline, suggesting an intensity-dependent efficacy of mask sounds [30]. Additionally, some RCT studies report that mask sound can improve sleep and anxiety, and reduce blood pressure and heart rate during surgical stress. [31-34]. However, existing studies lack integrated physiological and metabolic monitoring in human evidence, and the protective mechanisms of mask sound on cardiovascular and metabolic health remain unclear. Therefore, exploring the potential of mask sound as a protective intervention may provide new insights into understanding the mechanisms of noise-induced health impacts.

To fill this gap, the current study has designed a prospective randomized crossover trial aimed at systematically assessing the effects of traffic noise on human neuroendocrine function, physiological metabolism, and subjective feelings for the first time in a population. Additionally, it will evaluate the potential protective effects of mask sound, in order to uncover the key links in the chain of noise stimulation-neuroendocrine response-metabolic physiological

changes. This study will provide new perspectives on understanding the relationship between noise and metabolic health and will offer an evidence base for the formulation of effective public health policies and personalized intervention strategies.

Methods

Study Participants

This study was approved by the ethics committee of the hospital where the researchers are based (ChiCTR2500098645). The study protocol, screening questionnaire, and informed consent document were approved by the local ethics committee and strictly adhered to the Declaration of Helsinki and international ethical standards. The participants for this study were recruited from March 2025 to October 2025 through questionnaire advertisements posted on social media and within the community. The research team conducted rigorous medical screening of potential participants to ensure they met the study criteria. All enrolled participants underwent blood tests to confirm normal levels of hematological parameters (including thyroid-related indicators, adrenal-related indicators, and glycemic and lipid metabolism-related indicators) and were assessed through hearing tests to ensure their hearing thresholds were within the normal range based on age and sex. Individuals with recent prolonged exposure to high-decibel environments (such as construction sites, factories, loud music, noisy conditions, or heavy traffic) were excluded from participation, as were those with long-term exposure to high-noise environments as part of their occupations, such as traffic police or airport workers. To ensure the accuracy of the study results, participants were also required to have good subjective sleep quality ((Pittsburgh Sleep Quality Index (PSQI) ≤ 5)) and no symptoms of daytime sleepiness ((Epworth Sleepiness Scale (ESS) ≤ 10)). Individuals who had taken long-haul flights across time zones within the month prior to recruitment were also excluded. This study only included non-smokers and individuals who had not taken any medications (including hormonal contraceptives). Female participants underwent progesterone testing before enrollment, and all female participants were scheduled to participate during the period from day 0 to day 11 of their menstrual cycle (the follicular phase) to control for potential hormonal cycle effects on the outcomes. Sensitivity to noise was assessed using the Noise Sensitivity Questionnaire (NoiSeQ). After the stringent screening process, a total of 174 potential participants (84 female) were evaluated, and ultimately 26 participants (13 female) were included in the analysis and completed all experimental phases (**Supplementary Fig. 1**). All participants signed a written informed consent form before officially beginning the experiment.

Pre-Experimental Conditions

To maintain a regular sleep-wake rhythm, participants were instructed to keep their habitual bedtime within ±30 minutes for one week prior to the study. They were required to spend 8 hours in bed and avoid napping. Participants were not allowed to be exposed to high noise environments (>70 dBA according to WHO guidelines). Additionally, they were asked to avoid the intake of stimulating foods (such as coffee, tea, and chocolate) and alcohol, to maintain a normal diet while avoiding greasy meals, and to refrain from engaging in vigorous physical activity to match laboratory conditions as closely as possible.

Laboratory Study Conditions

The experimental design is illustrated in **Fig. 1**, which details the design and implementation of the randomized crossover study. A total of 26 healthy participants (13 females) were randomly allocated (**Fig. 1A**), with the allocation sequence generated using random number tables. The study employed a crossover design where each participant underwent two exposure conditions in succession: traffic noise (Noise) and mask sound (Mask). The daily activities and measurement schedule are presented in **Fig. 1B**, which details the daily 24-hour routine and data collection time points during the experimental period.

Characteristics of traffic noise and mask sound

Sound stimuli were created using recorded segments of real-world sounds, with traffic noise data collected on July 26, 2023, aimed at capturing the noise characteristics during typical peak hours on a workday. The noise collection was primarily focused on the morning peak (from 8:51 to 8:58), and all data were gathered at 769 Zhaojiabang Road, located in the central area of Shanghai, which is a busy main road exhibiting typical urban traffic noise characteristics. The mask sound data utilized a mixture of natural sounds designed by Cai et al., including birdsong, water sounds, and rain sounds, which were processed to match the sound level of the traffic noise [35]. The background noise level during the noise day was recorded at 76.23 dBA (± 1.21), while the background noise level on the mask day was 76.15 dBA (± 1.32). There was no significant difference in sound levels between the noise day and the mask day (See supplementary materials for more acoustic information).

Energy Expenditure and Substrate Oxidation

To accurately measure the energy expenditure levels and substrate oxidation (such as carbohydrates and fats) of the participants, the entire experiment was conducted within a metabolic chamber. Each chamber was a closed space of 30,000 liters, equipped with amenities including an adjustable bed, a desk, chairs, a cycle ergometer, a washbasin, and a toilet. The temperature within the chamber was tightly controlled at 25.0 ± 0.1°C, and the air was continuously extracted at a constant rate of 80 liters per minute. The concentrations of oxygen (O_2) and carbon dioxide (CO_2) in the sampled air were monitored in real-time using the Promethion integrated system (model GA-06/FG-01) manufactured by Sable Systems International, based in Las Vegas, USA. To ensure optimal performance, the gas analyzers were calibrated weekly with standard gases. The calculations for oxygen uptake (VO_2) and carbon dioxide production (VCO_2) utilized the Henning method. To minimize potential errors associated with the metabolic chamber, the accuracy of the measurements was verified using the propane combustion methodology. The results demonstrated that the accuracy of oxygen consumption measurements reached 99.3 ± 0.8%, while carbon dioxide production measurements achieved an accuracy of 100.0 ± 0.6%. The VO_2 and VCO_2 data obtained via the Henning method were initially recorded per minute and subsequently aggregated into hourly data for later statistical comparisons. The oxidation rates of macronutrients and total energy expenditure calculations were performed using the Weir formula, in conjunction with nitrogen excretion data from urine samples [36]. The measurement of

urinary nitrogen was conducted on 24-hour urine samples collected in segments. In order to correct the bias of body fat composition on substrate oxidation, the energy consumption results are calibrated with fat-free mass.

Physiological Measures

Non-invasive blood pressure, three-lead electrocardiogram (ECG), and peripheral pulse oxygen saturation (SpO_2) were continuously monitored using a cardiac telemetry system (WEP-5204C, Omegawave, Tokyo, Japan). During the study, blood pressure (BP) measurements were taken every 30 minutes for systolic and diastolic values. Heart rate, respiratory rate, and SpO_2 were recorded once per second throughout the study period.

Laboratory test

Laboratory test results for participants were recorded during each visit of the screening and intervention periods. The tested items and metrics included: Glycemic metabolism indicators and insulin: Venous blood glucose (VBG), insulin, and C-peptide (CP). Endocrine Hormones: Total triiodothyronine (T3), total free thyroxine (T4), thyroxine-binding globulin (TBG), reverse triiodothyronine (rT3), free triiodothyronine (FT3), free thyroxine (FT4), thyroid-stimulating hormone (TSH), metanephrine (MN), and normetanephrine (NMN). Urea Nitrogen: Blood urea nitrogen (BUN) and urine volume. Salivary Melatonin. During the screening period, participants underwent fasting venous blood collection for glycemic, insulin, CP, and all aforementioned endocrine hormones at 8:00 AM. Throughout the intervention period (Days 1-4), blood samples, saliva, and urine were collected at various time points each intervention day after the placement of a catheter in the antecubital vein or dorsal hand vein.

Statistical Analysis

This study collected continuous variable data, with the experimental group exposed to continuous traffic noise and the control group exposed to mask sound throughout the day. The significance level was set at $\alpha = 0.05$ and $\beta = 0.1$. The sample size was determined based on similar transportation noise intervention studies by L. Thiesse et al., which observed significant changes in the glycemic area under the curve (AUC) (mmol/L·min·10) for participants on the last morning of the experimental day, with a comparison of high-intensity noise versus low-intensity noise groups ($p < 0.0001$) [11]. Considering a dropout rate of 10%, we planned to recruit 26 participants to complete the experiment, which is expected to achieve a statistical power of 95%. Participant characteristics were summarized using descriptive statistics, with continuous variables expressed as means ± standard deviation and categorical variables as percentages. Differences between groups were estimated using a linear mixed-effects model, with the observed parameters as dependent variables, experimental day groupings (quiet, noise, and mask) as independent variables, and sequence and time as covariates, with participants treated as random effects. When significant differences were detected, post-hoc tests were conducted using Bonferroni correction. Correlation coefficients were calculated to analyze relationships between heart rate variability and indicators of HPA and HPT axis, glycemic, insulin sensitivity and energy metabolism parameters [37]. The statistical method employs Pearson correlation analysis and controls for the risk of false positives due to multiple comparisons through Bonferroni adjustment. The threshold for statistical significance was established at $p < 0.05$. Unless otherwise specified, data results are presented as means ± standard error (SE), and all statistical analyses were conducted using R version 5.0.

Results

The study design is illustrated in **Supplementary Fig. 1**. A total of 26 potential participants were screened for the study. Ultimately, 26 healthy participants (13 males and 13 females) successfully completed all experimental procedures and provided complete data. There were no significant differences in demographic and primary metabolic parameters among all participants at baseline (Day 0) ($p > 0.05$), specifically including age, height, weight, body mass index (BMI), and body composition parameters (such as body fat percentage, lean body mass, muscle mass, etc.), as detailed in **Table 1**. Moreover, baseline metabolic and endocrine-related indicators, including fasting glycemic, fasting insulin, and Homeostasis Model Assessment of Insulin Resistance (HOMA-IR), showed no statistically significant differences across groups. This further confirms the effectiveness of randomization, ensuring good comparability between groups.

Changes in HRV and catecholamines

Interpret autonomic nervous system homeostasis changes through HRV and catecholamine secretion. Regarding HRV (**Fig. 2**), the noise day exhibited significantly lower levels of SDNN (Standard Deviation of Normal-to-Normal intervals, **Fig. 2A**), TP (Total Power, **Fig. 2B**), and HF (High Frequency, **Fig. 2E**) compared to the quiet day (all $p < 0.001$). In contrast, LF (Low Frequency, **Fig. 2D**) and the LF/HF ratio (**Fig. 2C**) were substantially higher on the noise day than on the quiet day (both $p < 0.001$). In terms of catecholamines, noise exposure significantly elevated the average daily levels of MN (**Fig. 3A**) and NMN (**Fig. 3B**) during the noise day, with MN and NMN levels significantly higher than those on the quiet day (MN: noise, 36.71 ± 6.42 pg/ml vs. quiet, 31.74 ± 5.73 pg/ml, $p < 0.01$. NMN: noise, 52.7 ± 11.24 pg/ml vs. 44.91 ± 10.56 pg/ml, $p < 0.001$; mean ± SD).

Changes in HPA axis and HPT axis hormone secretion

In the HPA axis (**Supplementary Fig. 2A, 3B**), noise exposure significantly increases the secretion of ACTH and cortisol (ACTH: noise, 24.94 ± 7.42 pg/ml vs. quiet, 22.31 ± 8.32 pg/ml, $p < 0.05$. Cortisol: noise, 15.07 ± 3.89 pg/ml vs. quiet, 13.79 ± 3.78 pg/ml, $p < 0.05$. mean ± SD). Regarding the hypothalamic-pituitary-thyroid (HPT) axis (**Supplementary Fig. 2C - I**), short-term exposure to noise significantly increased thyroid hormone secretion. On the noise day, the average daily levels of T4 (**Supplementary Fig. 2D**), FT3 (**Supplementary Figure 2E**), FT4 (**Supplementary Fig. 2F**), TSH (**Supplementary Fig. 2G**), and TBG (**Supplementary Fig. 2I**) were significantly higher compared to the quiet day, with T4, FT4, and TBG showing the most pronounced increases (T4: noise, 101.52 ± 9.23 mmol/L vs. quiet, 95.32 ± 8.75 mmol/L, $p < 0.001$. FT4, noise, 13.25 ± 1.34 pmol/L vs. quiet, 12.67 ± 1.57 pmol/L, $p < 0.001$. TBG: noise, 19.45 ± 3.78 ng/mL vs. quiet, 17.23 ± 4.12 ng/mL, $p < 0.001$. TSH: noise, 1.55 ± 0.72 μIU/mL vs. quiet, 1.36 ± 0.63 μIU/mL, $p < 0.05$. FT3: noise, 4.52 ± 0.64 pmol/L vs. quiet, 4.35 ± 0.74 pmol/L, $p < 0.01$. mean ± SD).

Changes in cardiovascular parameters, glycemic-insulin homeostasis, energy metabolism, rhythm, and subjective perceptions

Short-term exposure to traffic noise exerted a significant impact on the cardiovascular system, with the average daily heart rate (HR, **Fig. 3C**) on the noise day significantly higher than that on the quiet day (HR: noise, 81.25 ± 13.44 bpm vs. quiet, 78.92 ± 11.23 bpm, $p < 0.001$. mean ± SD). Regarding blood pressure, systolic blood pressure (SYS, **Fig. 3D**) showed no significant change on the noise day, while diastolic blood pressure (DIA, **Fig. 3E**) was significantly higher than that on the quiet day (DIA: noise, 71.35 ± 8.46 mmHg vs. quiet, 69.21 ± 9.52 mmHg, $p < 0.01$. mean ± SD). Short-term exposure to traffic noise exerts multifaceted and significant effects on glycemic-insulin homeostasis and substrate utilization in healthy participants (**Fig. 4, Supplementary Fig. 3, Table 2**). Firstly, regarding the dynamic changes in glycemic and insulin, noise exposure significantly disrupted daytime glycemic homeostasis. As shown in **Fig. 4A** and **Supplementary Fig. 3A**, VBG levels peaked significantly higher on the noise day at both 2 hours and 12 hours post-exposure compared to the quiet day (2h VBG: noise, 5.89 ± 1.52 mmol/L vs. quiet, 4.97 ± 1.21 mmol/L, $p < 0.001$. 12h VBG: noise, 6.05 ± 1.42 mmol/L vs. quiet, 5.45 ± 1.51 mmol/L, $p < 0.001$. mean ± SD). The average daily glycemic levels during the exposure were also significantly higher on the noise day than on the quiet day (VBG: noise, 5.72 ± 0.87 mmol/L vs. quiet, 5.31 ± 0.46 mmol/L, $p < 0.001$. mean ± SD). For insulin levels, as illustrated in **Fig. 4B** and **Supplementary Fig. 3B**, insulin rapidly increased at 2 hours post-exposure on the noise day (2h Insulin: noise, 26.76 ± 8.73 pmol/L vs. quiet, 12.52 ± 4.32 pmol/L, $p < 0.001$. mean ± SD), and the daily average was significantly higher than the levels on quiet day (Insulin: noise, 28.74 ± 11.23 pmol/L vs. quiet, 23.42 ± 12.52 pmol/L, $p < 0.05$. mean ± SD). The changes in CP mirrored these results (**Fig. 4C** and **Supplementary Fig. 3C**): significant peaks in CP levels at 2 hours and 14 hours post-exposure in the noise day were observed, exceeding those of both the quiet and mask groups (2h vs quiet, $p < 0.001$). Additionally, the average CP levels during the first 2 hours of noise exposure were significantly the highest. According to **Table 2**, fasting glycemic after noise exposure was 4.98 mmol/L, and fasting insulin was 8.65 pmol/L, both significantly higher than those on the quiet day (4.7 mmol/L and 6.97 pmol/L, respectively), with the difference in fasting glycemic reaching statistical significance ($p = 0.024$). Notably, the HOMA-IR increased sharply from 1.46 on the quiet day to 1.95 after noise exposure ($p = 0.047$). In terms of glycemic tolerance, the postprandial glycemic level at 2 hours (G_{120}) also significantly elevated (5.9 mmol/L vs. 4.95 mmol/L, $p < 0.001$). The Matsuda index significantly decreased to 6.56 on the noise day ($p = 0.0015$). In terms of substrate utilization for energy metabolism, there were no significant differences in energy expenditure (EE, **Supplementary Fig. 3E**) among the groups. However, significant changes in substrate utilization were observed. The non-protein respiratory quotient (NPRQ, **Fig. 4D** and **Supplementary Fig. 3D**) was significantly lower in the noise day (NPRQ: noise, 0.75 ± 0.33 vs. quiet, 0.78 ± 0.29, $p < 0.05$. mean ± SD). This trend was also reflected in the fat oxidation rate (FOX, **Fig. 4F**), which showed a significantly higher average level throughout the exposure period ($p < 0.01$). Notably, protein oxidation rate (PROX, **Supplementary Fig. 3F**) significantly decreased on the noise day ($p < 0.01$), being markedly lower than on the quiet day, both overall and across time segments. The ΔNPRQ (**Fig. 4E**), an indicator of metabolic flexibility, also showed a significant decrease on noise days, whether during the daytime ($\Delta NPRQ_{Day\text{-}Night}$: noise, 0.051 ± 0.016 vs. quiet, 0.064 ± 0.021, $p < 0.05$. mean ± SD), post-meal ($\Delta NPRQ_{Postprandial\text{-}Fasting}$: noise, 0.011 ± 0.006 vs. quiet, 0.019 ± 0.008, $p < 0.001$. mean ± SD), or post-exercise ($\Delta NPRQ_{Post\ exercise\text{-}Pre\ exercise}$: noise, -0.041 ± 0.018 vs. quiet, -0.027 ± 0.011, $p < 0.001$. mean ± SD). Salivary melatonin levels, which reflect rhythmic disruption (**Supplementary Fig. 4**), indicated significant differences at the 13-hour mark after exposure started (21:00). The noise group exhibited significantly lower melatonin levels compared to the quiet day ($p < 0.001$). Notably, the noise group showed significantly higher melatonin levels the following morning compared to both the quiet and mask groups ($p < 0.001$, $p < 0.01$). As shown in **Fig. 5 and Supplementary Fig. 5**, exposure to traffic noise significantly impacted participants' subjective feelings and emotional states. In terms of sleep-related scores, the daytime sleepiness score (**Fig. 5A**) was significantly lower on both noise and mask days compared to the quiet day ($p < 0.01$), indicating that both types of sound exposure enhanced individuals' tendency to feel awake during the day. The Stanford Sleepiness Scale (SSSS, **Supplementary Fig. 5A**) showed no significant differences among the three groups, suggesting that noise exposure did not markedly alter participants' overall evaluation of sleep quality. Regarding emotional states, noise exposure triggered negative emotional responses. Anxiety scores (**Fig. 5B**) sharply increased on the noise day, significantly higher than those on the quiet day ($p < 0.001$). This indicates that noise exacerbated participants' feelings of anxiety. Simultaneously, happiness scores (**Fig. 5C**) were significantly lower on both the noise and mask days compared to the quiet day ($p < 0.001$), suggesting that both noise and mask sound reduced the experience of positive emotions. In terms of appetite and satiety, the hunger score (**Supplementary Fig. 5C**) was significantly higher on the noise day compared to the quiet day ($p < 0.05$), implying that noise could enhance feelings of hunger. The fullness level (**Supplementary Fig. 5D**) was significantly lower on the noise day than on the quiet day ($p < 0.05$), indicating that noise exposure may diminish individuals' subjective feelings of fullness. Participants' temperature perception (**Supplementary Fig. 5B**), stomach capacity (**Supplementary Fig. 5E**), willingness to eat (**Supplementary Fig. 5F**), and maximum food intake scores (**Supplementary Fig. 5G**) exhibited no significant changes among the three groups. In summary, short-term exposure to traffic noise leads to increased subjective wakefulness and anxiety, decreased well-being, and abnormal appetite regulation among participants.

The protective effect of masking sound

The protective effects of mask sound involve multiple facets. During the mask sound intervention, SDNN, TP, and HF levels were significantly higher than those on the noise day ($p < 0.001$), while LF and LF/HF were significantly lower than on the noise day ($p < 0.001$), returning close to quiet day levels (**Fig. 2**). The levels of MN and NMN in the mask sound group fell between those of the noise group and the quiet group, showing no statistical difference between the quiet and noise groups (**Fig. 3A, Fig. 3B**). In HPA axis, mask sound can alleviate the secretion of ACTH and cortisol, although not significantly (**Supplementary Fig. 2A, Supplementary Fig. 2B**). Regarding the HPT axis, the mask sound group exhibited some protective effects concerning these thyroid hormone indicators, as their daily average levels were lower than those on the noise day, with FT4 notably lower than on the noise day (FT4, mask, 12.85 ± 1.17 pmol/L vs. noise, 13.25 ± 1.34 pmol/L, $p < 0.05$. mean ± SD) (**Supplementary Fig. 2F**). Regarding cardiovascular responses, the mask sound alleviated the increase in heart rate (reduced by 2.55%), bringing it closer to levels observed on the quiet day. Although not statistically significant, the mask sound group showed a 0.3% reduction in DIA (**Fig. 3E**). In terms of glycemic and insulin homeostasis, the protective effects of mask sound were evident in the partial restoration of several metabolic indicators: although fasting glycemic and fasting insulin on the mask day were slightly higher than those on the quiet day, the differences were not statistically significant when compared to the noise day (**Fig. 4A, Fig. 4B, Table 2**). Additionally, G_{120} was significantly lower on the mask day than on the noise day ($p = 0.0032$). Importantly, the Matsuda index recovered to 8.31 on the mask day, with no significant differences from the quiet day (**Table2**). In terms of energy metabolism, the performance of the mask sound group in terms of respiratory quotient and protein oxidation rate was

generally intermediate between the quiet group and the noise group. However, ΔNPRQ $_{Day\text{-}Night}$ (**Fig. 4E**) also significantly decreased on mask day (ΔNPRQ $_{Day\text{-}Night}$: mask, 0.052 ± 0.019 vs. quiet, 0.064 ± 0.021, $p < 0.05$. mean ± SD). It is noteworthy that ΔNPRQ on mask day shows significant improvement before and after meals and before and after exercise compared with the noise days (ΔNPRQ $_{Postprandial\text{-}Fasting}$: mask, 0.016 ± 0.008 vs. noise, 0.011 ± 0.006, $p < 0.05$. ΔNPRQ $_{Post\ exercise\text{-}Pre\ exercise}$: mask, -0.032 ± 0.013 vs. noise, -0.041 ± 0.018, $p < 0.05$. mean ± SD). Regarding salivary melatonin, in contrast, the mask sound intervention appeared to alleviate this impact, leading to a secretion pattern that was closer to what was observed on quiet days (**Supplementary Fig. 4**). Mask sound can moderately improve subjective well-being. While mask sound significantly alleviated anxiety scores, its effects on appetite and overall well-being appear to be limited (**Fig. 5, Supplementary Fig. 5**).

Correlations and sex-stratified analysis

Fig. 6A and **Fig. 6B** show a significant correlation between sympathetic nervous activity (LF/HF ratio) and changes in catecholamines (MN, R = 0.45, p = 0.023; NMN, R = 0.72, $p < 0.001$). Female participants exhibit higher sensitivity to traffic noise (MN, R = 0.57, p = 0.043; NMN, R = 0.75, p = 0.003). On noise days, sympathetic activity is positively correlated with HPA axis secretion (**Supplementary Fig. 6C**,cortisol, R = 0.44, p = 0.025); moreover, in women, cortisol secretion on noise days shows a tighter association with sympathetic activity (R = 0.57, p = 0.043). In **Supplementary Fig. 6D - F and Supplementary Fig. 7A - D**, changes in T4, FT3, FT4, and TBG are positively correlated with sympathetic nervous activity (T4, R = 0.49, p = 0.011; FT3, R = 0.42, p = 0.034; FT4, R = 0.46, p = 0.019; TBG, R = 0.49, p = 0.011). Women also demonstrate higher sensitivity in their responses to these hormones (T4, R = 0.57, p = 0.041; FT3, R = 0.6, p = 0.03; FT4, R = 0.57, p = 0.041; TBG, R = 0.66, p = 0.013). Data in **Supplementary Fig. 6C - E** and **Supplementary Fig. 7B - D** show that mask sound also helps maintain hormonal secretion homeostasis in the HPA and HPT axes, especially in women. Changes in glycemic levels (VBG during exposure, **Figure 6C**, G_{120}, **Fig. 6D**), insulin (**Fig. 6E**), and glycemic-insulin homeostasis (HOMA-IR, **Fig. 6F**, Mastuda Index, **Supplementary Fig. 6A**) are also affected. The correlations between noise and VBG (R = 0.62, $p < 0.001$), G_{120} (R = 0.74, $p < 0.001$), insulin (R = 0.41, p = 0.037), HOMA-IR (R = 0.6, $p < 0.001$), and Mastuda Index (R = -0.41, p = 0.0035) show that noise significantly disrupts metabolic homeostasis. Women show higher sensitivity in VBG (R = 0.67, p = 0.012), G_{120} (R = 0.79, p = 0.001), insulin (R = 0.57, p = 0.042), and insulin resistance (HOMA-IR, R = 0.65, p = 0.017; Mastuda Index, R = -0.6, p = 0.031). The use of mask sound is associated with improvements in glycemic and insulin levels, with data in **Figure 6C - 6F** and **Supplementary Fig. 6A** demonstrate that the changes in glycemic and insulin in the mask sound group are smaller, with less disruption to glycemic-insulin homeostasis, particularly in women.

Discussion

Traffic noise has broad adverse health effects, yet prospective human studies are scarce, and daytime physiological/metabolic impacts remain limited [4]. Mask sound is a potential nonpharmacological intervention; early studies suggest benefits, but robust evidence across physiological, metabolic, and psychological domains is still needed [38]. In this randomized cross-over study, we assessed short-term physiological and energy-metabolic responses to traffic noise and evaluated the protective role of mask sounds. For the first time in a healthy population, we show that brief traffic-noise exposure activates the sympathetic nervous system and the HPA/HPT axes, disrupts autonomic and endocrine balance, increases cardiovascular load, perturbs glycemic–insulin homeostasis, and impairs metabolic flexibility. Mask sound provided protective effects across multiple domains by maintaining autonomic balance and reducing excessive HPA/HPT activity, thereby mitigating noise-related cardiovascular, insulin resistance, and metabolic-flexibility impairments. Our findings suggest that optimizing the acoustic environment and timely use of mask sound offer a practical, nonpharmacological intervention for those exposed to noise pollution.

Neural and endocrine pathways linking traffic noise to autonomic dysregulation

Animal and human studies using fMRI and electrophysiology show noise activates the cochlea–auditory cortex–amygdala pathway, shaping downstream neural and endocrine responses [39,40]. HRV is a key mechanistic indicator of autonomic state, with LF reflecting sympathetic activity, HF reflecting vagal activity, and the LF/HF ratio serving as a sympathovagal balance index [41,42]. Kraus found that 6-hour daytime noise exposure alters HRV: L_{Aeq} <65 dBA, each 5 dBA increase raises LF/HF by 4.89%, while LF and HF decline (−3.77%, −8.56%), and SDNN rises with noise levels <65 dBA (5.74%). This aligns with our finding that traffic noise reduces HRV overall, lowering SDNN, TP, HF, raising LF, and increasing LF/HF, indicating sympathetic predominance. Walke et al. reported opposite autonomic results: 40 minutes of noise reduced HRV, with low-frequency noise decreasing HF, LF, and SDNN by 32%, 34%, and 16%, respectively, and high-frequency noise reducing LF by 21%; no clear sympathetic/parasympathetic shift emerged, possibly due to mild stress and coordinated rebalancing [45]. From electrophysiology, traffic noise appears to trigger sympathetic activation, with catecholamines (adrenaline, noradrenaline, MN/NMN) as indirect endocrine markers [46]. Some studies found no nighttime catecholamine changes with noisy nights, which may reflect differences in exposure or methods [47,48]. Our randomized exposure study with real-time blood analyses shows noise significantly affects catecholamine secretion. Animal data corroborate this: 29-day exposure to airport noise at 75–80 dBA raises plasma normetanephrine [49], supporting population-level links. In sum, electrophysiological and endocrine evidence converge on traffic noise inducing sympathetic activation and disrupted autonomic balance, reflected in HRV and catecholamine dynamics.

Traffic noise activates sympathetic, HPA, and HPT axes

Studies show that, beyond sympathetic activation, noise stimulates endocrine axes via the hypothalamus [4]. Animal and mechanistic reviews consistently report that environmental noises, including traffic-like continuous noise, activate the HPA axis and raise stress hormones. Reviews on environmental pollution indicate traffic-related pollution (particulates plus noise) can trigger the HPA axis and increase glucocorticoids, elevating allostatic load and potentially harming the CNS [50]. Mice exposed to 75 dBA long-term had elevated ACTH [51], aligning with our findings. In piglet transport trials, noise increased ACTH and cortisol after brief transport, supporting noise-induced HPA activation [52], concordant with our ACTH/cortisol results. Correlations show higher cortisol with greater sympathetic activity on days with noise, indicating cooperation between HPA and sympathetic pathways in response to

noise [53]. The HPT axis also participates in stress, though its response depends on energy status, stress type, and exposure duration; it activates under energy-demanding conditions [54,55]. Few studies treat noise as a direct stressor; our data show noise over-stimulates FT3, FT4, and TSH via the HPT axis. Analogies with other stressors suggest noise may affect the HPT axis through energy metabolism and mood–sleep pathways. Masoud et al. followed 297 male workers for four years and found that every 10 dBA noise increase raised TSH, especially at mid-frequencies, implying long-term noise exposure may impair HPT homeostasis, paralleling our short-term findings [56]. We also found positive correlations between HRV and FT3/FT4 during noise exposure, indicating HPT involvement in neuroendocrine regulation. Collectively, this study provides the first human corroboration that traffic noise affects downstream metabolism and homeostasis through coordinated regulation of the sympathetic system, HPA axis, and HPT axis.

Multisystem neuroendocrine mechanisms of traffic noise: cardiovascular, glycemic-insulin homeostasis and metabolic flexibility

From an exposure–response perspective, activating the sympathetic nervous system and the HPA axis triggers cardiovascular responses, altering heart rate and blood pressure [57]. In mice, simulated traffic/aircraft noise increases systolic blood pressure and heart rate, with vascular dysfunction and inflammation [58]. Münzel et al. exposed mice to aircraft noise peaks of 85 dBA (avg 72 dBA) for 4 days, raising systolic blood pressure, NMN, and angiotensin II [12]. These findings align with ours. Human evidence on noise and cardiovascular responses is mixed. Haralabidis et al. reported 15-minute aircraft events increasing systolic blood pressure by 6.2 mmHg and diastolic blood pressure by 7.4 mmHg [59], while we found traffic noise raises heart rate by ~2.95% and diastolic pressure by ~3.09%. Roberto et al. describe temporary blood pressure and HR increases during exposure and for 2–3 hours after, with higher blood pressure variability [60]. Walke et al. found no blood pressure changes after 40 minutes of low-frequency noise [44], likely due to differences in exposure duration, intensity, measurement timing, and population. Thyroid hormone elevation raises myocardial oxygen demand, heart rate, and contractility, and increases catecholamine sensitivity, mechanistically supporting higher HR and blood pressure [55]. Our results suggest short-term noise stimulates the HPT axis, contributing to greater cardiovascular load. Notably, noise increased diastolic blood pressure but not systolic blood pressure, likely due to sympathetic-driven peripheral vasoconstriction (elevated normetanephrine) with limited changes in cardiac output and baroreceptor adjustments [61]. Vascular-type hypertension features higher peripheral resistance and diastolic blood pressure with modest systolic blood pressure changes, typical of stress, while exercise-type stress elevates cardiac output and systolic blood pressure [62,63]. Thus, traffic noise–induced sympathetic and HPA/HPT activation may raise cardiovascular risk. In multivariable analyses, long-term LF/HF power was independently linked to insulin sensitivity, implying sympathetic imbalance may drive insulin resistance and type 2 diabetes risk [64]. Natural daylight rapidly reshapes glycemic metabolism via the SCN–hypothalamus–autonomic pathway [65]. Noise can perturb cardiovascular/metabolic homeostasis through central autonomic networks and the HPA/HPT axes, offering a plausible noise–metabolism link. No RCTs directly confirm neuroendocrine activation by traffic noise disrupting glycemic homeostasis, though animal studies link 95 dBA noise to insulin resistance in mice, with duration-related persistence [16]. Prolonged noise elevates glycemic and corticosterone levels, reduces hepatic insulin sensitivity, and sustains insulin signaling activation [66], consistent with our findings (glycemia up 7.72% during exposure; day after, HOMA-IR up 33.56%) and supports noise-induced insulin resistance via sympathetic and HPA activation [67–69]. Chronic stress and glucocorticoids augment gluconeogenesis and suppress insulin signaling, promoting insulin resistance and type 2 diabetes [70]. For metabolic flexibility, we first quantified ΔNPRQ under noise. Noise shifted substrate use toward more fat oxidation, reduced protein oxidation, and impaired metabolic flexibility, especially after meals and exercise ($\Delta NPRQ_{\text{Day-Night}}$ 20.31% lower; $\Delta NPRQ_{\text{postprandial-fasting}}$ 42.11% lower; $\Delta NPRQ_{\text{post-exercise}}$ 51.85% lower) — aligning with obesity, insulin resistance, and type 2 diabetes traits [71]. Metabolic flexibility, a measure of system resilience, decreases with metabolic ill-health and links to obesity, metabolic syndrome, fatty liver, CVD, and worse outcomes [72]. Mechanistically, sympathetic activity, HPA activation, and HPT activation from noise may impair metabolic flexibility. Chronic noise can sustain catecholamine elevations, promoting insulin resistance, visceral fat, and low-grade inflammation, placing the body in a state of high glycemic/high fatty acid availability but reduced insulin sensitivity, hindering carbohydrate–fat oxidation switching across states [73]. In mice with limited catecholamine release, elevated sympathetic activity drives insulin resistance and fatty liver; reducing sympathetic activity protects against these conditions [67]. HPA activation increases glucocorticoids, upregulating gluconeogenic genes and opposing insulin signaling, affecting lipid metabolism and fat redistribution [74]. Long-term noise exposure in animals elevates glycemic indices and lipids while suppressing thyroid axis activity, suggesting duration-dependent effects on the HPT axis; acute stress may transiently activate the HPT axis to meet energy demands, with FT3/FT4 promoting fatty acid oxidation alongside hepatic lipogenesis [55,76]. Elevated ACTH, cortisol, and thyroid hormones may also disrupt mood and circadian rhythms [77,78]. Consistent with prior work, traffic noise worsens subjective distress and disrupts rhythms, increasing appetite and reducing satiety. Prolonged daytime noise (65–100 dBA, 17 days) raises body weight and food intake in adult female rodents, with lower energy expenditure during exposure and recovery [79]. Five weeks of high-intensity noise (87.5 dBA) in juvenile males increases intake and weight, with CART up and leptin receptor down, indicating energy conservation with higher intake [80]. Engineered noise exposure also raises fat mass, adipocyte size, and lowers HDL [81], aligning with our findings of compensatory eating and fat accumulation. Circadian disruption includes reduced nighttime melatonin and morning melatonin shifts, with sleep disruption affecting leptin/ghrelin and energy intake [79]. Stress and circadian disturbances may worsen insulin resistance and metabolic flexibility. In summary, traffic noise activates the sympathetic, HPA, and HPT axes, elevating heart rate and blood pressure, promoting lipolysis and insulin resistance, and impairing metabolic flexibility, circadian rhythm, and mood, thereby increasing obesity and diabetes risk.

Multisystem protective effects of mask sound against traffic noise: cardiovascular, endocrine and metabolic

Natural white noise is widely used in environmental interventions to improve sleep and reduce stress. A study by Marsman et al. found that white noise significantly enhanced HRV compared to various music genres [82]. Mask sound, defined as white noise added to ambient noise, similarly inhibits sympathetic nerve excitation, improving HRV. Unlike detrimental noise like traffic noise, natural white noise at equivalent levels has no adverse effects on the sympathetic nervous system or HPA axis, nor does it induce oxidative stress, inflammation, or endocrine disorders, as shown in animal studies [12]. Our research demonstrates that mask sound reduces excessive ACTH and cortisol secretion by the HPA axis, aligning with animal studies. We are the first to report mask sound's protective effect on the HPT axis in humans, mitigating noise-induced FT4 increases. This is significant as stress elevates FT4 in animal

models [55]. This suggests that thyroid activation and related metabolic axes are curtailed. We are the first to identify mask sound's protective effects on the sympathetic nervous system, HPA axis, and HPT axis in humans, benefiting cardiac autonomic regulation and metabolism. Elevated HRV is linked to reduced cardiovascular disease risk. Animal experiments show white noise, unlike aircraft noise, does not elevate blood pressure [42], a finding supported by our human study where mask sound significantly lowered heart rate and diastolic blood pressure during noise exposure, nearing quiet-state levels. This indicates improved cardiac rhythmicity and vascular tone, reducing cardiovascular stress. Mask sound also reduced sympathetic excitation, and by lowering FT4 secretion, it contributed to reducing cardiovascular burden, as elevated FT4 is linked to increased heart rate and blood pressure [83,84]. Our study also reveals mask sound's positive role in glycemic-insulin homeostasis. Core indicators like glycemic and HOMA-IR levels were lower in the mask group, closer to quiet levels. This suggests mask sound alleviates glycemic-insulin damage by reducing sympathetic excitation. Mask sound also partially reverses noise-induced metabolic imbalances, improving respiratory quotient and fat/protein oxidation, promoting balanced metabolism and preventing unhealthy energy mismatch. It demonstrates a significant protective effect on metabolic flexibility, mitigating impaired substrate-switching caused by traffic noise, thereby helping prevent obesity and diabetes. FT4 modulates peripheral metabolism and energy allocation during stress [85]. The protective mechanism involves the combined actions of the sympathetic nervous system and the HPT axis. Ebben et al. found white noise significantly reduced wakefulness and nocturnal awakenings in individuals with noise-induced sleep disturbances [86]. Systematic reviews show white noise improves multiple sleep parameters [87]. Our study found mask sound positively impacts subjective mood and physiological rhythms. Although melatonin levels initially decreased, they recovered by early morning. Participants reported reduced anxiety and improved psychological well-being. Rhythm and subjective perception abnormalities are linked to insulin resistance [88]. Our study is the first human trial to reveal mask sound's protective effect on subjective mood and physiological rhythms. Improved neural and endocrine activation states, specifically ameliorated sympathetic nerve activation and HPT axis secretion, are key to alleviating rhythms and mood, consistent with previous conclusions [89]. In summary, mask sound may alleviate cardiovascular burden by mitigating sympathetic nervous system and HPT axis activation, with additional HPA axis support. It improves glycemic-insulin homeostasis, metabolic flexibility, subjective mood, and rhythms by reducing stress on the sympathetic nervous system and HPT axis. Experimental studies suggest women may have higher baseline sympathetic nervous system activation under stress, making them more susceptible to noise's health impacts [90]. W. Babisch et al. found elevated catecholamines in women living near high-traffic streets [91], consistent with our findings that traffic noise strongly stimulates catecholamine secretion in women. Females may be more vulnerable to noise-induced HPA axis activation due to hormonal differences [90], aligning with our findings. Vella and colleagues noted that stress effects on the HPT axis are "biphasic by sex" and early-life stress impacts the female HPT axis more [92]. Our findings support this: women have a higher risk of excessive HPT axis secretion under noise-induced sympathetic activation. Noise stimulation in women leads to stronger activation of the sympathetic, HPA, and HPT axes, causing more severe physiological and metabolic damages. A large cohort study found women had a greater increased risk of diabetes from long-term traffic noise exposure than men [5], suggesting females are more susceptible to glycemic homeostasis disruption from noise-related sympathetic activation. Subgroup analysis showed that while mask sound didn't significantly activate the sympathetic pathway in females, its correlation with metabolic disorders was higher in females than males. Despite women's higher sensitivity to acoustic stimuli, mask sound still offers significant protection in females compared to noise exposure. These findings suggest women are increasingly primary victims of traffic noise, and mask sound intervention for susceptible populations may need to be combined with other approaches (e.g., earplugs) for optimal outcomes.

Strengths and limitations

Although this study has made preliminary findings regarding the mechanistic links and intervention effects, there are certain limitations. Firstly, the sample size is relatively small, with only 26 healthy participants, which limits the stability and generalizability of the statistical conclusions, making it difficult to represent the overall effects in a broader population. Secondly, this study involved a short-term intervention with subjects limited to healthy young individuals, and thus, it cannot assess the effects of long-term exposure or intervention on specific populations, such as the elderly or individuals with chronic diseases. Furthermore, future research should strengthen collaborations with basic research, integrating animal models and molecular mechanisms to provide more robust causal inferences and biological explanations for observational findings in human populations. Therefore, it is recommended that future studies involve larger sample sizes and more diverse populations, expand mechanism indicators, refine circadian dynamic monitoring, assess the feasibility of long-term interventions, and deepen cross-disciplinary studies with basic experiments. This is aimed at providing a more solid theoretical basis for noise-related chronic disease prevention and individual health management.

Conclusion

This study systematically reveals that short-term exposure to traffic noise can activate the sympathetic nervous system and neuroendocrine axes (the HPA and HPT axes), leading to increased cardiovascular load, impaired glycemic–insulin homeostasis and metabolic inflexibility, and disruptions in circadian rhythm and mood, thereby elevating the risk of metabolic diseases such as cardiovascular diseases as well as obesity and diabetes. Mask sound intervention can partly mitigate the adverse effects of noise, including improvements in sympathetic activation and excessive endocrine axes activation, thereby ameliorating downstream cardiovascular stress, insulin resistance and energy metabolism abnormalities. This highlights its potential as a simple, non-pharmaceutical protective measure. Due to their sensitivity to noise, the protective effect of mask sound is less effective in women compared to men, necessitating the use of multiple protective measures. Therefore, the rational application of mask sound for populations affected by noise pollution is expected to become an important supplementary intervention for chronic disease prevention and control.

List of abbreviations

SDG 11, Sustainable Development Goal; WHO, World Health Organization; dBA, A-weighted decibels; HPA, Hypothalamic Pituitary Adrenal; CVD, Cardiovascular Disease; eNOS, endothelial Nitric Oxide Synthase; nNOS,neuronal Nitric Oxide Synthase; ROS, reactive oxygen species; RCT, Randomized Controlled Trial; HRV, heart rate variability; PSQI, Pittsburgh Sleep Quality Index; ESS, Epworth Sleepiness Scale; NoiSeQ, Noise Sensitivity Questionnaire;

BMR, Basal Metabolic Rate; VAS, Visual Analog Scale; LAeq, A-weighted equivalent continuous sound level; S, Sharpness; FS, Fluctuation Strength; RMR, Resting Metabolic Rate; PAL, Physical Activity Level; O_2, Oxygen; CO_2, Carbon Dioxide; VO_2, Oxygen Uptake; VCO_2, Carbon Dioxide Production; ECG, Electrocardiogram; SPO_2, Oxygen Saturation; BP, Blood Pressure; VM, Vector Magnitude; DXA, Dual-Energy X-ray Absorptiometry; VBG, Venous Blood Glucose; CP, C-peptide; T3, Triiodothyronine; T4, Total Free Thyroxine; TBG, Thyroxine-Binding Globulin; rT3, reverse Triiodothyronine; FT3, Free Triiodothyronine; FT4, Free Thyroxine; TSH, Thyroid-Stimulating Hormone; MN, metanephrine; NMN, normetanephrine ;BUN, Blood Urea Nitrogen; AUC, Area Under the Curve; SE, Standard Error; BMI, Body Mass Index; HOMA-IR, Homeostasis Model Assessment of Insulin Resistance; SDNN, Standard Deviation of Normal-to-Normal Intervals; HPT, Hypothalamic-Pituitary-Thyroid; TP, Total Power; HF, High Frequency; LF, Low Frequency; EE, Energy Expenditure; NPRQ, Non-Protein Respiratory Quotient; FOX, Fat Oxidation; PROX, Protein Oxidation; HR, Heart Rate; SYS, Systolic Blood Pressure; DIA, Diastolic Blood Pressure; SSSS, Stanford Sleepiness Scale; GR, Glucocorticoid Receptor; HR_{IQR}, hazard ratio per interquartile range increase; SD, Standard Deviation; FFM, Fat-Free Mass; CHO, Carbohydrate oxidation rate; CI, Confidence Interval.

Declarations

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CRediT authorship contribution statement

Weiqing Wang, Shijia Pan, and Guang Ning were responsible for the trial's conception and design. Riqiang Bbao and Yixiang Hu made significant contributions to the protocol and study design. Riqiang Bao, and Rui Xu were instrumental in the study's successful implementation. Yuanyuan Hu, Zhihong Huang, Yixiang Hu, Yuhan Guo, Yixiang Hu and Yashu Zhu played key roles in data collection. Rui Xu took the lead in manuscript preparation.

The final version of the manuscript has been reviewed and approved by all authors, and the order of authorship has been mutually agreed upon.

Data and materials availability

The authors declare that all data supporting the findings of this study can be found in the article and/or its Supplementary Information file.

Conflict of interest

The authors declared no conflict of interest.

Ethics approval and consent to participate

This study was approved by the ethics committee of the Ruijin hospital, Shanghai Jiao Tong University School of Medicine. ClinicalTrials.gov Identifiers: ChiCTR2500098645.

https://trialsearch.who.int/Trial2.aspx?TrialID=ChiCTR2500098645

Consent for publication

Not applicable.

References

1. Russell C (2018) SDG 11 sustainable cities and communities from backyards to biolinks: royal botanic gardens victoria's role in urban greening. BGjournal 15(1):31-33.
2. Organization WH (2018). Environmental Noise Guidelines for the European Region.
3. Hahad O, et al. (2025) Noise and mental health: evidence, mechanisms, and consequences. J Expo Sci Env Epid 35(1):16-23.
4. Garg R. (2025) A narrative review of environmental noise and cardiovascular health: From molecular mechanisms to public health impact. Env Dis 10(1):6-14.
5. Shin S, et al. (2020) Association Between Road Traffic Noise and Incidence of Diabetes Mellitus and Hypertension in Toronto, Canada: A Population-Based Cohort Study. J Am Heart Assoc 9(6):e013021.
6. Vermeulen R, Schymanski EL, Barabási AL, Miller GW (2020) The exposome and health: Where chemistry meets biology. Science 367(6476):392-396.
7. Fredianelli L, Bolognese M, Fidecaro F, Licitra G (2021) Classification of Noise Sources for Port Area Noise Mapping. Environments 8(2):12.

8. Li J, Masullo M, Maffei L, Pascale A, Chau CK, Lin M (2024) Improving informational-attentional masking of water sound on traffic noise by spatial variation settings: An in situ study with brain activity measurements. Appl Acoust 218:109904.
9. Schmidt F, et al. (2015) Nighttime aircraft noise impairs endothelial function and increases blood pressure in patients with or at high risk for coronary artery disease. Clin Res Cardiol 104(1):23-30.
10. Herzog J, et al. (2019) Acute exposure to nocturnal train noise induces endothelial dysfunction and pro-thromboinflammatory changes of the plasma proteome in healthy subjects. Basic Res Cardiol 114(6):46.
11. Thiesse L, et al. (2018) Adverse impact of nocturnal transportation noise on glucose regulation in healthy young adults: Effect of different noise scenarios. Environ Int 121(Pt 1):1011-1023.
12. Münzel T, et al. (2017) Effects of noise on vascular function, oxidative stress, and inflammation: mechanistic insight from studies in mice. Eur Heart J 38:2838-2849.
13. Münzel T, et al. (2020) Adverse Cardiovascular Effects of Traffic Noise with a Focus on Nighttime Noise and the New WHO Noise Guidelines. Annu Rev Public Health 41:309-328.
14. Münzel T, Daiber A, Engelmann N, Röösli M, Kuntic M, Banks JL (2025) Noise causes cardiovascular disease: it's time to act. J Expo Sci Env Epid 35(1):24-33.
15. Arregi A, et al. (2024) Road traffic noise exposure and its impact on health: evidence from animal and human studies-chronic stress, inflammation, and oxidative stress as key components of the complex downstream pathway underlying noise-induced non-auditory health effects. Environ Sci Pollut Res Int 31(34):46820-46839.
16. Liu L, et al. (2016) Effects of Noise Exposure on Systemic and Tissue-Level Markers of Glucose Homeostasis and Insulin Resistance in Male Mice. Environ Health Perspect 124(9):1390-1398.
17. Campeau S, et al. (2010) Hypothalamic pituitary adrenal axis responses to low-intensity stressors are reduced after voluntary wheel running in rats. J Neuroendocrinol 22(8):872-88.
18. Munzel T, Schmidt FP, Steven S, Herzog J, Daiber A, Sorensen M (2018) Environmental Noise and the Cardiovascular System. J Am Coll Cardiol 71(6):688-697.
19. Clark C, Sbihi H, Tamburic L, Brauer M, Frank LD, Davies HW (2017) Association of Long-Term Exposure to Transportation Noise and Traffic-Related Air Pollution with the Incidence of Diabetes: A Prospective Cohort Study. Environ Health Perspect 125(8):087025.
20. Jacob Rodrigues MC, Postolache O, Cercas F (2023) The Influence of Stress Noise and Music Stimulation on the Autonomous Nervous System. IEEE Trans Instrum Meas 72:1-19.
21. Huang J, Deng F, Wu S, Lu H, Hao Y, Guo X (2013) The impacts of short-term exposure to noise and traffic-related air pollution on heart rate variability in young healthy adults. J Expo Sci Environ Epidemiol 23(5):559-64.
22. Moreira MCdS, et al. (2015) Does the sympathetic nervous system contribute to the pathophysiology of metabolic syndrome? Front Physiol 6:234.
23. Thorp AA, Schlaich MP (2015) Relevance of sympathetic nervous system activation in obesity and metabolic syndrome. J Diabetes Res 2015(1):341583.
24. Buxton OM, et al. (2012) Sleep disruption due to hospital noises: a prospective evaluation. Ann Intern Med 157(3):170-179.
25. Micic G, et al. (2023) Establishing the acute physiological and sleep disruption characteristics of wind farm versus road traffic noise disturbances in sleep: a randomized controlled trial protocol. Sleep Adv 4(1):zpad033.
26. Wang J, Qin M, Xu X, Cai J (2022) Effects of familiarity and likability on music masking of aircraft fly over noise for young people. Appl Acoust 199:109000.
27. Yu N, Cai J, Xu X, Sun J (2021) Improvement on perception of welding noise by fountain sound masking: Insights from fMRI. Appl Acoust 171:107563.
28. Yu N, Cai J, Xu X, Yang Y, Sun J (2020) Masking effects on subjective annoyance to aircraft flyover noise: An fMRI study. Hum Brain Mapp 41:3284-3294.
29. Zhang S, Chen L (2023) Acoustic information masking effects of natural sounds on traffic nois e based on psychological health in open urban spaces. Front Public Health 11.
30. Gilmour L, Bray I, Alford C, Lintott P (2024) Natural soundscapes enhance mood recovery amid anthropogenic noise pollution. PLoS ONE 19(11):e0311487.
31. Shaikh H, Chung P, Jubran A, Tobin M, Laghi F (2020) Does Noise Masking Improve Sleep Consolidation In Patients Weaning From Prolonged Mechanical Ventilation? Sleep 43(Supplement_1):A279-A279.
32. Shu H, Song Y, Zhou H (2018) Assessment of Music and Water Sounds for Urban Noise Masking. TENCON 2018 - 2018 IEEE Region 10 Conference 2018:1451-1455.
33. Xie H, Tian Y, Xiao K, Lv W, Liu C (2025) Environmental noise exposure and natural sound intervention in hospital interventional therapy rooms: Effects on patient perception, physiological and psychological outcomes. Build Environ 280:113149.
34. Saadatmand V, et al. (2013) Effect of nature-based sounds' intervention on agitation, anxiety, and stress in patients under mechanical ventilator support: A randomised controlled trial. Int J Nurs Stud 50(7):895-904.
35. Cai J, Liu J, Yu N, Liu B (2019) Effect of water sound masking on perception of the industrial noise. Appl Acoust 150:307-312.
36. Weir JB (1949) New methods for calculating metabolic rate with special reference to protein metabolism. J Physiol 109(1-2):1-9.
37. Mason IC, et al. (2022) Light exposure during sleep impairs cardiometabolic function. Proc Natl Acad Sci U S A 119(12):e2113290119.
38. Vincens N, et al. (2026) Pink noise reduces impact of traffic noise on sleep and the blood metabolome: a cross-over pilot study. Commun Med 6(1):114
39. Natarajan N, Batts S, Stankovic K (2023) Noise-Induced Hearing Loss. J Clin Med 12(6):2347.

40. Dewey R, et al. (2020) The association between subcortical and cortical fMRI and lifetime noise exposure in listeners with normal hearing thresholds. NeuroImage 204:116239.
41. Von Falkenhausen A, et al. (2025) Traditional and advanced electrocardiographic measures of autonomic function in the population-based KORA-F3 study. Eur J Epidemiol 40:815-832.
42. Münzel T, et al. (2017) Effects of noise on vascular function, oxidative stress, and inflammation: mechanistic insight from studies in mice. Eur Heart J 38(37):2838-2849.
43. Kraus U, et al. (2013) Individual Daytime Noise Exposure during Routine Activities and Heart Rate Variability in Adults: A Repeated Measures Study. Environ Health Perspect 121:607-612.
44. Walker ED, Brammer A, Cherniack MG, Laden F, Cavallari JM (2016) Cardiovascular and stress responses to short-term noise exposures-A panel study in healthy males. Environ Res 150:391-397.
45. Ulrich-Lai Y, Herman J (2009) Neural regulation of endocrine and autonomic stress responses. Nat Rev Neurosci 10:397-409.
46. Robertson D, Johnson G, Robertson RM, Nies A, Shand D, Oates J (1979) Comparative Assessment of Stimuli That Release Neuronal and Adrenomedullary Catecholamines in Man. Circulation 59:637.
47. Carter N, Hunyor S, Crawford G, Kelly D, Smith A (1994) Environmental noise and sleep--a study of arousals, cardiac arrhythmia and urinary catecholamines. Sleep 17(4):298-307.
48. Schmidt FP, et al. (2013) Effect of nighttime aircraft noise exposure on endothelial function an distress hormone release in healthy adults. Eur Heart J 34(45):3508-14a.
49. Di Gq, Zhou B, Li Zg, Lin Ql (2011) Aircraft noise exposure affects rat behavior, plasma norepinephrine levels, and cell morphology of the temporal lobe. J Zhejiang Univ Sci B 12(12):969-975.
50. Jafari Z, Kolb BE, Mohajerani MH (2020) Noise exposure accelerates the risk of cognitive impairment and Alzheimer's disease: Adulthood, gestational, and prenatal mechanistic evidence from animal studies. Neurosci Biobehav Rev 117:110-128.
51. Yan Z, et al. (2024) PPARα suppresses low-intensity-noise-induced body weight gain in mice: the activated HPA axis plays an critical role. Int J Obes 48:1274-1282.
52. Fu Q, et al. (2025) A new therapy to alleviate the inflammatory injury of piglet intestine caused by short-distance transportation——music. PLOS One 20(2):e0313354.
53. Lei AA, et al. (2025) Chronic Stress-Associated Depressive Disorders: The Impact of HPA Axis Dysregulation and Neuroinflammation on the Hippocampus—A Mini Review. Int J Mol Sci 26(7):2940.
54. Petrowski K, Kahaly G (2025) Stress & Thyroid Function - From Bench to Bedside. Endocr Rev 46(5):709-735.
55. De Oca MAP-M, Jaimes-Hoy L, Garduño K, García-Herrera R, Charli J, Joseph-Bravo P (2025) Long-Term Effects of Stress During Adolescence on the Sex-Dependent Responses of Thyroid Axis and Target Tissues to Exercise in Male and Female Wistar Rats. Int J Mol Sci 26(19):9425.
56. Khosravipour M, Ghanbari Kakavandi M, Nadri F, Gharagozlou F (2021) The long-term effects of exposure to noise on the levels of thyroid hormones: A four-year repeated measures study. Sci Total Environ 792:148315.
57. Burford N, Webster N, Cruz-Topete D (2017) Hypothalamic-Pituitary-Adrenal Axis Modulation of Glucocorticoids in the Cardiovascular System. Int J Mol Sci 18.
58. Molitor M, et al. (2023) Aircraft noise exposure induces pro-inflammatory vascular conditioning and amplifies vascular dysfunction and impairment of cardiac function after myocardial infarction. Cardiovasc Res 119(6):1416-1426.
59. Haralabidis A, et al. (2008) Acute effects of night-time noise exposure on blood pressure in populations living near airports. Eur Heart J 29(5):658-664.
60. Fogari R, Zoppi A, Corradi L, Marasi G, Vanasia A, Zanchetti A (2001) Transient but not sustained blood pressure increments by occupational noise. An ambulatory blood pressure measurement study. J Hypertens 19(6):1021-1027.
61. Regecová V, Kellerová E (1995) Effects of urban noise pollution on blood pressure and heart rate in preschool children. J Hypertens 13(4):405-412
62. Matthews KA, Cottington EM, Talbott E, Kuller LH, Siegel JM (1987) Stressful work conditions and diastolic blood pressure among blue collar factory workers. Am J Epidemiol 126(2):280-291.
63. Kurl S, Laukkanen JA, Rauramaa R, Lakka TA, Sivenius J, Salonen JT (2001) Systolic blood pressure response to exercise stress test and risk of stroke. Stroke 32(9):2036-41.
64. Svensson MK, et al. (2016) Alterations in heart rate variability during everyday life are linked to insulin resistance. A role of dominating sympathetic over parasympathetic nerve activity? Cardiovasc Diabetol 15(1):91.
65. Harmsen JF et al. (2026) Natural daylight during office hours improves glucose control and whole-body substrate metabolism. Cell Metab 38(1):65-81.
66. Cui B, Gai Z, She X, Wang R, Xi Z (2026) Effects of chronic noise on glucose metabolism and gut microbiota–host inflammatory homeostasis in rats. Sci Rep 6(1):36693.
67. Sakamoto K et al (2024) Overnutrition causes insulin resistance and metabolic disorder through increased sympathetic nervous system activity. Cell Metab 37(1):121-137.
68. Cipolletta E, et al (2009) The G protein coupled receptor kinase 2 plays an essential role in beta-adrenergic receptor-induced insulin resistance. Cardiovasc Res 84(3):407-415.
69. Montaner M, et al. (2023) A neuronal circuit driven by GLP-1 in the olfactory bulb regulates insulin secretion. Nat Commun 15(1):6941.

70. Beaupere C, Liboz A, Fève B, Blondeau B, Guillemain G (2021) Molecular Mechanisms of Glucocorticoid-Induced Insulin Resistance. Int J Mol Sci 22(2):623.
71. Galgani J, Fernández-Verdejo R (2020) Pathophysiological role of metabolic flexibility on metabolic health. Obes Rev 22(2):e13131.
72. Smith R, Soeters M, Wüst R, Houtkooper R (2018) Metabolic Flexibility as an Adaptation to Energy Resources and Requirements in Health and Disease. Endocr Rev 39:489-517.
73. Reilly S, Saltiel A (2017) Adapting to obesity with adipose tissue inflammation. Nat Rev Endocrinol 13:633-643.
74. Menke A (2023) The HPA Axis as Target for Depression. Curr Neuropharmacol 22:904-915.
75. Amjad R, et al. (2024) Exploring the effects of noise pollution on physiology and ptilochronology of birds. PLOS ONE 19(6):e0305091.
76. Martínez-Sánchez N, et al. (2017) Hypothalamic AMPK-ER Stress-JNK1 Axis Mediates the Central Actions of Thyroid Hormones on Energy Balance. Cell Metab 26:212-229.
77. Lightman S, Birnie M, Conway-Campbell B (2020) Dynamics of ACTH and Cortisol Secretion and Implications for Disease. Endocr Rev 41(3):bnaa002.
78. Ikegami K, Refetoff S, Van Cauter E, Yoshimura T (2019) Interconnection between circadian clocks and thyroid function. Nat Rev Endocrinol 15(10):590-600.
79. Coborn JE, Lessie RE, Sinton CM, Rance NE, Perez-Leighton CE, Teske JA (2019) Noise-induced sleep disruption increases weight gain and decreases energy metabolism in female rats. Int J Obes 43(9):1759-1768.
80. De Jenlis AB, Del Vecchio F, Delanaud S, Gay-Quéheillard J, Bach V, Pelletier A (2019) Impacts of Subchronic, High-Level Noise Exposure on Sleep and Metabolic Parameters: A Juvenile Rodent Model. Environ Health Perspect 127(5):57004.
81. Lee J, et al. (2024) Effects of Environmental Noise Stress on Mouse Metabolism. Int J Mol Sci 25(20).
82. Marsman E, et al. (2024) Listen to your heart: The effect of music on heart rate variability. Eur Heart J 45(Supplement_1).
83. Go MT, George A, Tahsin B, Fogelfeld L (2022) Tachycardia in hyperthyroidism: Not so common. PLoS ONE 17(9):e0273724.
84. Delitala A, Scuteri A, Fiorillo E, Lakatta E, Schlessinger D, Cucca F (2019) Role of Adipokines in the Association between Thyroid Hormone and Components of the Metabolic Syndrome. J Clin Med 8(6):764.
85. Sinha R, Yen P (2024) Metabolic Messengers: Thyroid Hormones. Nat Metab 6:639-650.
86. Ebben MR, Degrazia MQ, Krieger AC (2018) The Effect of White Noise on Individuals Complaining of Poor Quality Sleep in a High Noise Environment in New York City. Sleep 41(suppl_1):A145-A145.
87. Ding Y, et al. (2025) Impact of white noise on sleep quality across age groups and in critically ill/non-critically ill patients: A systematic review and meta-analysis of randomized controlled trials. Sleep Med 136:106869.
88. Stenvers DJ, Scheer FAJL, Schrauwen P, la Fleur SE, Kalsbeek A (2019) Circadian clocks and insulin resistance. Nat Rev Endocrinol 15(2):75-89.
89. Sato S, et al. (2021) Rapid-acting antidepressants and the circadian clock. Neuropsychopharmacology 47:805-816.
90. Ali N, Nitschke J, Cooperman C, Baldwin M, Pruessner J (2020) Systematic manipulations of the biological stress systems result in sex-specific compensatory stress responses and negative mood outcomes. Neuropsychopharmacology 45(10):1672-1680.
91. Babisch W, Fromme H, Beyer A, Ising H (2001) Increased catecholamine levels in urine in subjects exposed to road traffic noise: The role of stress hormones in noise research. Environ Int 26(7):475-481.
92. Vella K, Hollenberg A (2021) Early life stress affects the HPT axis response in a sexually dimorphic manner. Endocrinology 162(9):bqab137.

Tables

Table 1 Basal characteristics of participant.

Characteristics	Female	Male
No. of participants (n = 26)	13	13
Age (years)	25.54(4.24)	25.76(3.72)
Height (cm)	163.46(5.93)	177.03(4.77)
Weight (kg)	59.39(8.86)	72.49(7.85)
BMI (kg/m^2)	22.17(2.46)	23.12(2.28)
Fat (%)	33.83 (4.94)	23.87 (4.7)
FFM (kg)	38.97 (4.0)	55.04 (5.69)
Metabolic Rate and Physiology		
RMR (kJ/min)	4.75 (1.08)	4.50 (1.18)
Heart rate (beats/min)	61.62(5.05)	64.47(9.37)
Diastolic blood pressure (mmHg)	60.71(3.75)	61.18(5.42)
Systolic blood pressure (mmHg)	100.47(5.32)	100.27(4.18)
Baseline metabolic variables		
Fasting Glycemic (mmol/L)	4.78(0.34)	4.58(0.44)
Fasting CP (μg/L)	1.63(0.45)	1.8(0.33)
Fasting Insulin (pmol/L)	6.95(1.49)	7.4(1.91)
HOMA-IR	1.49(0.3)	1.51(0.43)
Matsuda index	16.57(4.98)	14.54(4.16)

Participant characteristics were summarized using descriptive statistics, with continuous variables expressed as means ± standard deviation (SD) and categorical variables as percentages. BMI, Body Mass Index; FFM, Fat Free Mass, RMR, Resting Metabolic Rate; HOMA-IR, Homeostasis Model Assessment of Insulin Resistance.

Table 2 Comparison of metabolic status and insulin sensitivity under different sound conditions.

	Quiet (mean ± SE)	Noise (mean ± SE)		Mask (mean ± SE)		vs. Quiet (p-value)				vs. Post-Intervention (p-value)	
		Pre-Noise	Post-Noise	Pre-Mask	Post-Mask	Pre-Noise	Post-Noise	Pre-Mask	Post-Mask	Pre-Noise	Pre-Mask
Fasting state											
Fasting glycemic (mmol/L)	4.7(0.44)	4.72(0.46)	4.98(0.34)	4.74(0.43)	4.87(0.32)	0.65	0.024*	0.97	0.12	0.03*	0.16
Fasting insulin (pmol/L)	6.97(4.02)	8.07(3.2)	8.65(4.45)	7.75(3.22)	8.35(5.21)	0.11	0.15	0.31	0.34	0.92	0.83
HOMA-IR	1.46(0.88)	1.52(0.68)	1.95(0.58)	1.57(0.6)	1.74(0.65)	0.5	0.047**	0.33	0.13	0.033*	0.46
Post load indexes											
Matsuda index	11.58(1.73)	6.56(0.71)		8.31(0.72)		0.0015**		0.74			

The fasting status indicators for the intervention day are collected before the onset of sound (8:00 am). The post-intervention day indicators were collected at 8:00 am on the morning of the second day after the intervention (Fig 1). The glycemic data for the fasting phase and the two-hour postprandial phase were obtained from venous blood draw results. Differences between groups were estimated using a linear mixed-effects model, with the observed parameters as dependent variables, experimental day groupings (Quiet, Noise, and Mask) as independent variables, and sequence and time as covariates, with participants treated as random effects. When significant differences were detected, post-hoc tests were conducted using Bonferroni correction. Data were presented as raw mean ± SE. *, p<0.05, **, p<0.01, ***p<0.001.

Figures

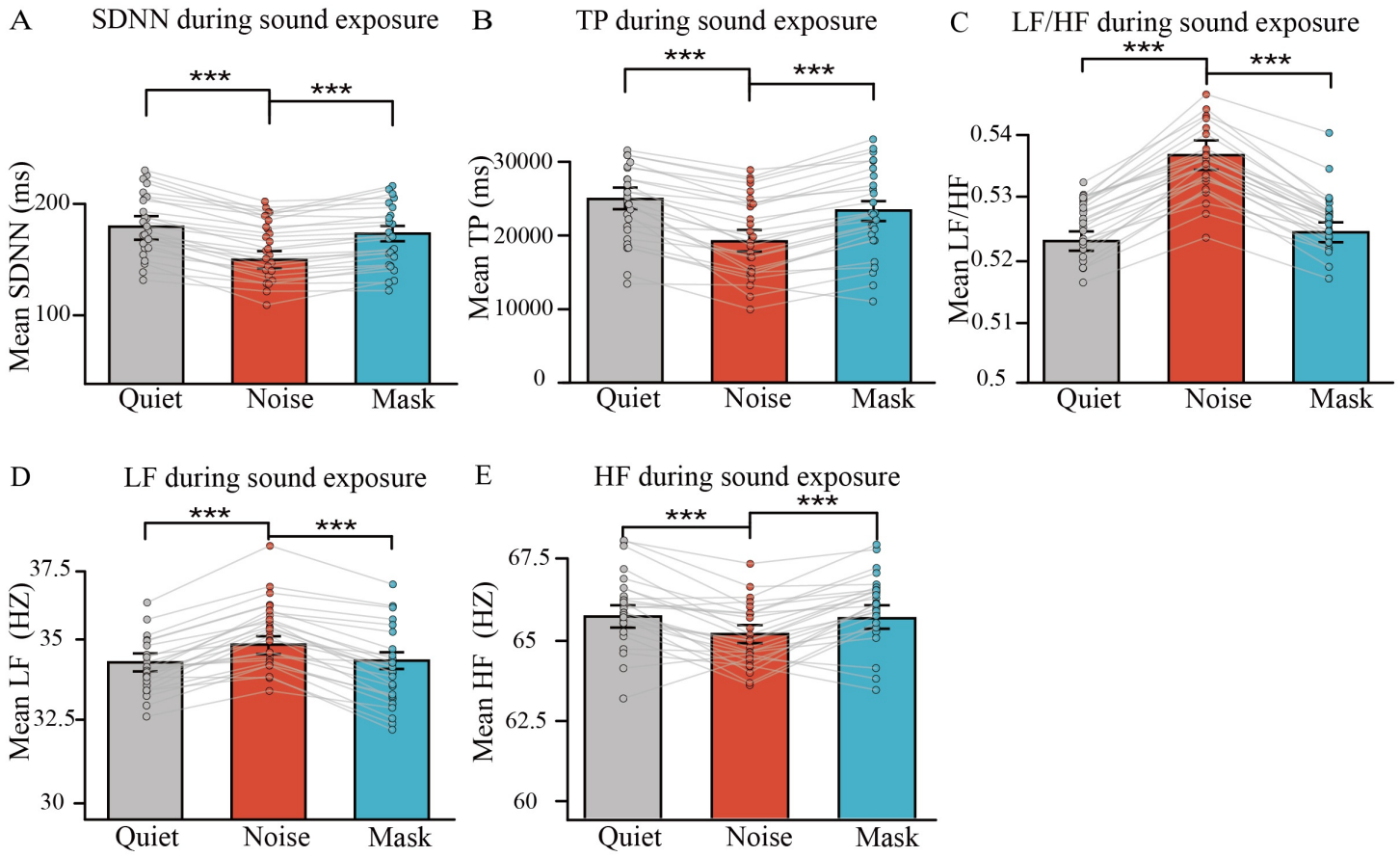


Figure 2

Changes in HRV during the experiment.

The following parameters were compared between groups during the exposure period based on overall daily averages: (A) Standard deviation of heart rate variability, (B) Total power, (C) Low-frequency/high-frequency ratio, (D) Low-frequency power, (E) High-frequency power. Differences between groups were estimated using a linear mixed-effects model, with the observed parameters as dependent variables, experimental day groupings (Quiet, Noise, and Mask) as independent variables, and sequence and time as covariates, with participants treated as random effects. When significant differences were detected, post-hoc tests were conducted using Bonferroni correction. All data are presented as Mean ± SE. Asterisks indicate significant differences when compared to the quiet day (Quiet). The significance levels are defined as follows: *, p<0.05, **, p<0.01, ***p<0.001. This format allows for a clear understanding of the differences in heart rate variability parameters between the noise exposure and mask sound exposure conditions, highlighting the physiological impact of these auditory environments.

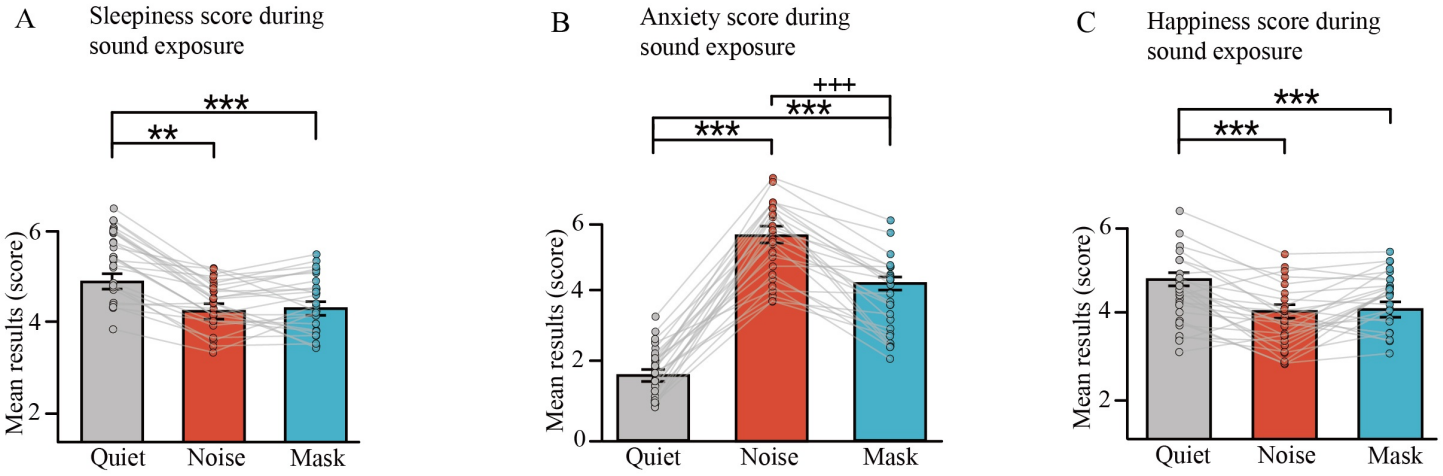


Figure 5

Changes in subjective rating scales questionnaires.

The following parameters were assessed for changes in daily averages: (A) Sleepiness Score, (B) Anxiety Score, (C) Happiness Score. All data are presented as Mean ± SE. Differences between groups were estimated using a linear mixed-effects model, with the observed parameters as dependent variables, experimental day groupings (Quiet, Noise, and Mask) as independent variables, and sequence and time as covariates, with participants treated as random effects. When significant differences were detected, post-hoc tests were conducted using Bonferroni correction. Asterisks indicate significant differences when compared to the quiet day (Quiet); Plus signs indicate significant differences between the noise day (Noise) and mask day (Mask). The significance levels are defined as follows: *, p<0.05, **, p<0.01, ***p<0.001; $^{+}$p < 0.05, $^{++}$p < 0.01, $^{+++}$p < 0.001.

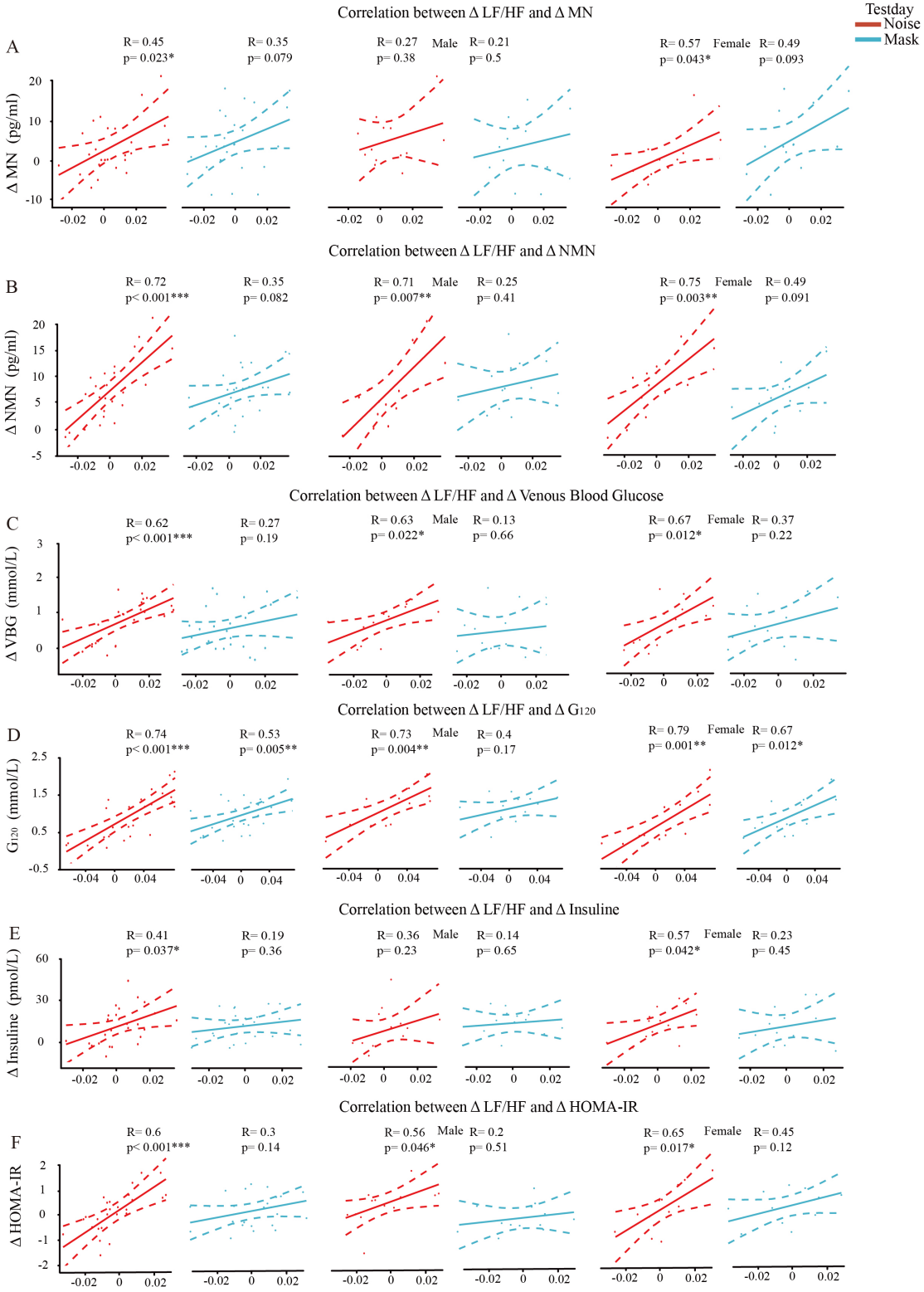


Figure 6

Heart rate variability and its relationship with glycemic, insulin sensitivity and catecholamines during sound stimulation.

Changes in low frequency/high frequency ratio measured by heart rate variability, with average values of various correlation analysis indicators taken during sound exposure. The statistical method employs Pearson correlation analysis and controls for the risk of false positives due to multiple comparisons through Bonferroni adjustment. The dashed lines represent the 95% confidence interval (CI).

Supplementary Files

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

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