Association Between Occupational or Environmental Noise Exposure and Renal Function Among Middle-aged and Older Korean Population: A Cross-sectional Study

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Research Article

Keywords: Renal function, glomerular filtration rate, chronic kidney disease, occupational noise, environmental noise

Posted Date: August 7th, 2021

DOI: <https://doi.org/10.21203/rs.3.rs-778767/v1>

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Abstract

Exposure to occupational and environmental noise is closely linked to various diseases of auditory system. Few studies have focused on noise exposure effect on extra auditory system especially, urinary system. This study analyzed 17,202 participants aged 40–79 years from the Korea National Health and Nutrition Examination Survey between 2013 and 2018. A self-reported questionnaire was used to assess occupational or environmental noise exposure. Logistic regression was used to determine the differences in chronic kidney disease (CKD) prevalence considering noise exposure characteristics. For participants with noise exposure, linear regression was performed to determine the noise exposure period and estimated glomerular filtration rate (eGFR). In the noise exposure group, a higher CKD prevalence was associated with females who experienced long-term occupational noise (≥ 240 months), compared with the other groups (Adjusted OR = 2.86, 95% CI: 1.18–6.94). An increase of one month of occupational noise exposure was associated with a 0.0096 mL/min/1.73 m², 0.0080 mL/min/1.73 m², and 0.0159 mL/min/1.73 m² decrease of eGFR in total, in males, and in females, respectively. Our results suggest that noise exposure can be a risk factor for reduced renal function, especially long-term exposure to occupational noise. More precise studies are necessary to determine the relationship between noise and renal function and the underlying mechanisms.

Introduction

Chronic kidney disease (CKD) is a generic term for any condition leading to kidney damage or decreased renal function. Its severity is assessed by the glomerular filtration rate, albuminuria, and clinical diagnosis ¹. CKD is known to be a risk factor for premature death, cardiovascular disease, stroke, and poor quality of life ²⁻⁴. Globally, the prevalence of CKD is reported to be more than 10% and the prevalence in Korea has been estimated to be 7.9% ⁵. Because of population aging and an increase in diabetes and hypertension, two of the main causes of CKD, the burden of CKD has intensified ⁶. To reduce this burden, determining and avoiding risk factors would be beneficial.

Noise is considered to be an important risk factor for several diseases ⁷. Exposure to noise not only affects auditory acuity, it also leads to non-auditory adverse health outcomes, such as cardiovascular disease, metabolic outcomes, and cognitive impairment ⁸⁻¹⁰, especially when such exposure is long term ⁷. The biological mechanism of the non-auditory effects of noise can be explained by the induction of the sympathetic-adrenal-medullary (SAM) axis and the hypothalamic-pituitary-adrenal (HPA) axis. Activation of these axes releases epinephrine, norepinephrine, and cortisol, which alter blood flow and metabolism ^{11,12}. Environmental noise is defined as the noise created from all sources, except workplaces. Noise exposure in the workplace is called occupational noise ⁷.

Despite its known effect, there is a lack of studies on the impact of noise on renal function. Because renal hemodynamics are closely related to blood pressure, vascular reactivity, and endothelial function, renal function might be influenced by noise-induced stress ¹³. One study showed that residential proximity to

major roadways is related to the reduction of renal function in hospitalized patients with acute ischemic stroke¹⁴. In addition, a reduction in renal function has been associated with community noise exposure in male patients with cardiovascular disease¹⁵. However, few epidemiological studies have considered the types of exposure to noise related to renal diseases.

We therefore conducted a cross-sectional study to determine whether noise exposure is associated with renal function. We examined the association between the two types of noise exposure status and renal function, which were CKD and estimated glomerular filtration rate (eGFR). The hypothesis of this study is that noise exposure negatively affects renal function.

Methods

Study participants

The Korea National Health and Nutrition Examination Survey (KNHANES) data from 2013 to 2018 were used in this study. KNHANES is a nationwide cross-sectional survey conducted in the Republic of Korea to assess the health and nutritional status of Koreans. Representatives are selected using multistage cluster sampling. Annually, 20 households throughout 192 regions are included as a new sample, and about 10,000 individuals aged one year old and older are targeted. This survey provides participants' information based on a health examination, a health interview, and a nutrition survey, conducted by trained staff members¹⁶.

The total number of KNHANES participants from 2013 to 2018 was 47,217. We only included participants aged 40 to 79 years old, as the noise exposure survey was performed at 40 years of age or older and the survey recorded all patients of 80 years or older as 80 years old. To minimize the effect of kidney disease intervention, participants with a previous diagnosis of renal failure were excluded. Missing or "participant refusal" values of the variables used in this study—such as hearing discomfort status, occupational noise exposure, environmental noise exposure, serum creatinine, educational level, HTN, DM, dyslipidemia, BMI, smoking status, high-risk alcohol consumption, and aerobic physical activity—were also excluded. The final sample size for analysis was 17,202 (Figure 2).

Renal function

Renal function was evaluated using the serum creatinine level. This was measured with a Hitachi Automatic Analyzer 7600-210 (Hitachi/JAPAN) and CREA reagent (Roche/Germany) using the Jaffe rate-blanked and compensated method. eGFR was calculated using the Chronic Kidney Disease Epidemiology Collaboration equation¹⁷. An eGFR lower than 60 mL/min/1.73 m² was classified as CKD.

Noise exposure

The KNHANES survey used self-reported questionnaires to assess noise exposure. For occupational noise, the question was "Have you ever worked in place with loud noise such as machines or generators

for more than three months? A loud noise means that you have to raise your voices for a conversation.” Those who answered yes were asked about their total working period, in months. The question addressing environmental noise was “Have you ever been exposed to loud noise for more than five hours a week, except for occupational noise? A loud noise means that you have to raise your voices for a conversation, such as cars, trucks, motorcycles, machines, or loud music (ex. singing room, concert hall).” If answered “yes,” the average minutes of exposure per day were asked. Long-term exposure to noise was defined as the third quartile of noise exposure period, ≥ 240 months for occupational noise, and ≥ 300 minutes for environmental noise. As noise exposure is closely linked to auditory problems, we conducted an additional analysis of the association between hearing discomfort and CKD or eGFR. For hearing discomfort, this question was asked: “Among the following, choose the most appropriate sentence to describe your hearing ability (without wearing a hearing aid).” The options were “comfortable,” “a little uncomfortable,” “very uncomfortable,” and “cannot hear at all.” We excluded participants who responded “cannot hear at all” and combined “a little uncomfortable” and “very uncomfortable” as just “uncomfortable.”

Other covariates

Our socioeconomic variables were age, sex, and educational status. Education level was classified as elementary school, middle school, high school, college, or higher as the highest level of completed education.

For chronic disease variables, we included HTN, DM, dyslipidemia, and BMI. HTN was defined as participants who satisfied at least one of the following criteria: (1) Systolic blood pressure ≥ 140 mmHg, (2) diastolic blood pressure ≥ 90 mmHg, or (3) diagnosed with hypertension before or with drugs for blood pressure control. Participants with any of the following conditions were considered to have DM: (1) fasting glucose level ≥ 126 mg/dL, (2) diagnosed with DM before, or (3) use oral hypoglycemic medications or insulin injections. Pregnant women were excluded because gestational diabetes was a transient state. Dyslipidemia was defined according to the 2018 Korean Dyslipidemia Management Guidelines¹⁸ and participants who were diagnosed before or used oral drugs were included. The guidelines describe dyslipidemia as (1) total cholesterol ≥ 240 mg/dL, (2) triglyceride ≥ 200 mg/dL, (3) LDL-C ≥ 160 mg/dL, or (4) HDL-C < 40 mg/dL. As LDL cholesterol levels were not measured, they were calculated using the Friedewald equation¹⁹. BMI was grouped into two categories: (1) < 25 kg/m² and (2) ≥ 25 kg/m².

The health behavioral variables were smoking status, high-risk alcohol consumption, and aerobic physical activity. Smoking status was classified according to their current smoking status: never smoked, past smoker, or current smoker. High-risk alcohol consumption is described as averaging ≥ 7 drinks at a time and drinking at least twice a week for males, and an average of ≥ 5 drinks at a time and drinking at least twice a week for females. Aerobic physical activity refers to doing moderate-intensity physical activity (≥ 2.5 h) or high-intensity physical activity (≥ 1.25 h) or mixing moderate- and high-intensity

physical activity (1 min of high-intensity physical activity is equivalent to 2 minutes of moderate-intensity) per week.

Statistical analysis

We conducted a χ^2 test to examine the differences in CKD prevalence. A student's *t*-test and one-way ANOVA were used to show the variances in eGFR by sociodemographic characteristics. We assessed the association between CKD and noise exposure using a logistic regression adjusted for age, sex, educational status, HTN, DM, dyslipidemia, BMI, smoking status, high-risk alcohol consumption, and aerobic physical activity. A linear regression analysis was performed to examine the association between eGFR and noise exposure time. Participants who were not exposed to noise were excluded from the analysis. The adjusted covariates were the same as described above; however, age was not included to avoid overadjustment. Statistical analyses were performed using SAS version 9.4 (SAS Institute, Cary, NC, USA) and *R* software (version 4.1.0) was used to draw the plots in Figure 1. Two-tailed *p*-values less than 0.05 were considered to be statistically significant.

Ethics approval and consent to participate

The study was performed in accordance with the ethical standards of the Declaration of Helsinki (1964) and its subsequent amendment. KNHANES data were anonymized prior to its release to the authors. All participants in the survey provided written informed consent. The Institutional Review Board of the Gil Medical Center, Gachon University, approved the current study (IRB number: GCIRB2020-147).

Results

Participant characteristics

Participants' characteristics according to their eGFR are summarized in Table 1. There were 636 (3.7%) participants with CKD (eGFR < 60 mL/min/1.73 m²) and the mean eGFR value was 89.8 (±14.5) mL/min/1.73 m². The older group, aged from 60 to 79 years, had a higher prevalence of CKD (7.5%) than the younger group, aged 40 to 59 years (0.7%). The CKD prevalence in males was 4.7%, which was higher than that of the females (3.0%). Concerning educational status, participants who went to college or higher had the lowest CKD prevalence (1.5%), while those with an elementary school education had the highest (7.1%). The following health status showed a higher prevalence of CKD than the other groups: hypertension (HTN) (5.1%), diabetes mellitus (DM) (9.7%), dyslipidemia (5.1%), and high body mass index (BMI) (4.9%). CKD prevalence in smoking status, from high to low, was as follows: past smokers (5.1%), never smokers (3.3%), and current smokers (3.2%). The prevalence of CKD was lower in the high-risk alcohol consumption group (1.1%) than in the non-high-risk group (4.0%), and more participants who reported no aerobic physical activity had CKD (4.5%) than those who got no exercise (2.6%). Groups classified according to occupational noise and environmental noise did not show a statistical difference in CKD prevalence and mean eGFR (*p* > 0.05). Participants who reported discomfort in hearing conditions had higher CKD prevalence (7.3%) than those who did not (3.0%).

Noise exposure and CKD

To determine the association between noise exposure and CKD, a logistic regression analysis was performed (Table 2). Occupational noise and environmental noise did not show statistically significant results in either the crude or adjusted models. However, we analyzed long-term noise exposure, which was set at the upper 25% among participants with noise exposure. There was no association between long-term environmental noise exposure and CKD; however, for occupational noise, there were statistical associations in the crude model for the total group (95% CI: 1.54–3.52), the male group (95% CI: 1.08–2.88), and for the female group (95% CI: 1.53–7.97). The adjusted model showed statistically significant results only in the female group (95% CI: 1.18–6.94). As noise exposure results in a decrease in hearing ability, we regarded hearing discomfort as an index of crude noise exposure. In the unadjusted model, CKD was significantly associated with hearing discomfort (95% CI: 2.18–3.06); this tendency was maintained during gender stratification. Females showed a stronger association (95% CI: 2.42–3.96) than males (95% CI: 1.70–2.72). After adjustment, the total group (95% CI: 1.04–1.49) and female group (95% CI: 1.08–1.81) still showed an association, with a 95% confidence interval. However, there was no significant difference in the male group ($p = 0.4117$).

Noise exposure and eGFR

The linear analysis results for the noise exposure time and eGFR are presented in Table 3. We analyzed only the noise exposure group, so there were 2,921 participants:—1,725 males and 1,196 females—in the occupational noise group, and 303 participants, 131 males and 172 females, in the environmental noise group. Both crude and adjusted models showed a negative association between noise exposure time and eGFR. A one month increase in occupational noise exposure was associated with $-0.0198 (\pm 0.0019)$ mL/min/1.73 m² in the crude model and $-0.0096 (\pm 0.0019)$ mL/min/1.73 m² in the adjusted model. After stratifying according to sex, the association remained in both classes. The absolute value of regression coefficients of the female group (crude: $-0.0275 [\pm 0.0040]$ mL/min/1.73 m², adjusted: $-0.0159 [\pm 0.0037]$ mL/min/1.73 m²) was larger than that of the male group (crude: $-0.0134 [\pm 0.0023]$ mL/min/1.73 m², adjusted: $-0.0080 [\pm 0.0022]$ mL/min/1.73 m²). Figure 1 depicts the trend lines of the adjusted model on a scatterplot. In the environmental noise group, there were no statistically significant results for any model.

Discussion

This study aimed to determine the association between noise exposure and renal function. Regarding occupational noise, long-term exposure was associated with CKD prevalence in the unadjusted model for all classes; however, in the adjusted model, the association was only observed among females (95% CI: 1.18–6.94; Table 2). We used linear regression to examine the dose-response relationship between noise exposure and eGFR. Negative relationships between occupational noise exposure time and eGFR were found in all classes (Table 3). No relationships were found in the logistic and linear analyses for environmental noise. We also found that hearing discomfort was related to CKD prevalence (95% CI:

1.04–1.49). That is, participants who complained of hearing discomfort had a higher CKD rate. When stratified by gender, this tendency persisted among female participants (95% CI: 1.08–1.81; Table 2).

There have been few studies on the relationship between noise and renal function. Epidemiological research in 1,103 patients—from the Boston area, United States—with ischemic stroke showed lower eGFR for those living closer to a major roadway ¹⁴. In 217 patients with cardiovascular heart disease, increased exposure to day–evening–night noise levels (L_{den}) resulted in a decrease in eGFR among males who experienced ischemic heart disease or stroke and who were exposed to lower air pollution ¹⁵. Our findings are similar to these studies in terms of noise exposure. However, our study does not correspond with these studies from the perspective of environmental noise. There was no association between environmental noise exposure and renal function in any of the groups we analyzed; however, the focus of the previous studies was environmental noise. This inconsistency might be caused by the low number of participants who reported being affected by environmental noise in our data.

Some other studies produced results opposite to those in our research. A randomized single blinded control study compared renal hemodynamics after aircraft noise and sham procedures; they found no significant change in renal circulation, including GFR ²⁰. In addition, a cross-sectional single-center study found an association between noise annoyance, renal perfusion, and renal vascular resistance; however, there was no difference in measured GFR ²¹. Because the study populations and noise characteristics differ from those in our study, the results also differ. For both these studies, the study participants were all males, aged around their 30s and 40s; our data, however, included participants from 40 to 79 years old and of both sexes.

Until now, the non-auditory effects of noise have been investigated in terms of cardiovascular, metabolic, and cognitive outcomes ⁸⁻¹⁰. The probable mechanisms have been suggested from many perspectives. First, noise acts as a stressor that induces sympathetic nerve activity. Stress responses are mediated by the HPA axis and the SAM axis. The SAM axis secretes catecholamines as a reaction to acute stress, while the HPA secretes glucocorticoids, such as cortisol, which prolongs stress ²². These can alter glucose metabolism, increase blood pressure and free fatty acids, inhibit insulin, and adversely affect lymphocytes ^{23,24}. In addition, inflammation and oxidative stress can be activated by both axes, leading to endothelial dysfunction ²². As renal function is influenced by vascular function ²⁵, the non-auditory effects of noise exposure on renal function could share similar mechanisms. Recently, a possible explanation was provided from the perspective of epigenetic transformation. In epigenome-wide association studies using SAPALDIA data, some CpG sites related to C-reactive protein (CRP), BMI, and eGFR were methylated in the noise exposure group ²⁶. These explanations can help elucidate the relationship between renal function and noise exposure.

The female group in our study seemed to have a stronger association with noise exposure and renal function. The results of our logistic regression on hearing discomfort and long-term occupational noise exposure showed a positive relationship only in the female group (Table 2). In addition, the linear

regression coefficients were larger among females (Table 3). Therefore, in terms of biological mechanisms, we can assume that females are more vulnerable to noise-induced stress. These results are in line with the general concept that there are more stress-related psychiatric disorders among females^{27,28}. In addition, according to an analysis in European countries, there was a significant elevation of saliva cortisol levels among females in relation to aircraft noise, but not among males²⁹. However, there are conflicting results. Concerning cardiovascular disease, several studies found that noise exposure has a more severe impact on males^{30,31}. As the results are different in types of stress, population characteristics, biomarkers, and the like³²⁻³⁴, more research is needed to clarify the interpretation.

Despite its obvious contributions, this study has several limitations. First, there are limitations arising from the cross-sectional study. We were unable to confirm causal relationships, and the order of events cannot be considered; logically, it can be agreed that noise exposure influences the reduction of renal function, but not vice versa. Additional studies are needed to clarify this relationship. Second, although we used a national representative dataset from a survey conducted and managed by the Korea Disease Control and Prevention Agency, the results were not free from bias, such as recall and nonresponse bias. Third, there is the matter of definitions. For example, eGFR was calculated using a single measurement of serum creatinine. As the precise definition of CKD presents abnormalities of kidney structure or function for at least three months¹, misclassification can occur. However, this is the first method used in the clinical field. Finally, there are no direct measurements of noise exposure. Further studies using objective data are required.

Overall, noise exposure is associated with decreased renal function, based on our logistic and linear regressions of Korean middle-aged and older populations, especially in females. The long-term occupational noise exposure group was particularly significant in both males and females. The results of our study suggest that noise exposure might be a risk factor for reduced renal function. A more precise study to determine the relationship between noise and renal function and the underlying mechanisms should be implemented in future.

Abbreviations

CKD: Chronic kidney disease

KNHANES: Korea National Health and Nutrition Examination Survey

SAM: Sympathetic-adrenal-medullary (axis)

HPA: Hypothalamic-pituitary-adrenal (axis)

eGFR: Estimated glomerular filtration rate

HTN: Hypertension status

DM: Diabetes mellitus

BMI: Body mass index

Declarations

Consent for publication

Not applicable

Availability of data and materials

Data openly available in a public repository (Korea National Health & Nutrition Examination Survey, <https://knhanes.kdca.go.kr/knhanes/eng/index.do>).

Competing interests

The authors declare that we have no competing interests.

Funding

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Authors' contributions

W. Lee had full access to all of the data in the study and take responsibility for the integrity of the data and accuracy of the data analysis. Study design and concept: Y. Kim and W. Lee. Acquisition, analysis, or interpretation of data: Y. Kim, W.-J. Choi, and W. Lee. Drafting of the manuscript: Y. Kim, W.-J. Choi, S. Ham, S.-K. Kang, and W. Lee. Statistical analysis: Y. Kim and W. Lee. Administrative, technical, or material support: S.-K. Kang and W. Lee. Study supervision: W. Lee. The authors read and approved the final manuscript.

Acknowledgements

None.

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Tables

Table 1. Characteristics of study participants by kidney function status.

Characteristics	Total	CKD, <i>n</i> (%)		<i>p</i> - <i>value</i> [†]	eGFR	<i>p</i> - <i>value</i> [‡]
		No	Yes		Mean ±SD	
Total participants	17,202 (100)	16,566 (96.3)	636 (3.7)		89.8 ±14.5	
Socioeconomic status						
Age (years)				< 0.0001		
40–59	9,625 (56.0)	9,560 (99.3)	65 (0.7)		96.1 ±12.0	
60–79	7,577 (44.0)	7,006 (92.5)	571 (7.5)		81.9 ±13.6	
Sex				< 0.0001		< 0.0001
Male	7,438 (43.2)	7,092 (95.3)	346 (4.7)		86.9 ±14.4	
Female	9,764 (56.8)	9,474 (97.0)	290 (3.0)		92.0 ±14.2	
Educational status				< 0.0001		< 0.0001 [§]
Elementary school	4,495 (26.1)	4,176 (92.9)	319 (7.1)		83.6 ±14.4	
Middle school	2,409 (14.0)	2,320 (96.3)	89 (3.7)		88.6 ±13.7	
High School	5,475 (31.8)	5,320 (97.2)	155 (2.8)		92.5 ±14.3	
College or higher	4,823 (28.0)	4,750 (98.5)	73 (1.5)		93.2 ±13.3	
Chronic diseases						
Hypertension				< 0.0001		< 0.0001
No	6,135 (35.7)	6,066 (98.9)	69 (1.1)		94.3 ±12.8	
Yes	11,067 (64.3)	10,500 (94.9)	567 (5.1)		87.3 ±14.8	
Diabetes				<		< 0.0001

				0.0001	
No	14,507 (84.3)	14,132 (97.4)	375 (2.6)		90.8 ±13.8
Yes	2,695 (15.7)	2,434 (90.3)	261 (9.7)		84.5 ±17.1
Dyslipidemia				< 0.0001	< 0.0001
No	8,352 (48.6)	8,166 (97.8)	186 (2.2)		92.3 ±13.7
Yes	8,850 (51.4)	8,400 (94.9)	450 (5.1)		87.5±15.0
BMI				< 0.0001	< 0.0001
<25 kg/m ²	10,940 (63.6)	10,611 (97.0)	329 (3.0)		90.9 ±14.1
≥25 kg/m ²	6,262 (36.4)	5,955 (95.1)	307 (4.9)		88.0 ±15.2
Health behavioral					
Smoking				< 0.0001	< 0.0001 [§]
Never	10,317 (60.0)	9,977 (96.7)	340 (3.3)		91.1 ±14.3
Past	4,012 (23.3)	3,808 (94.9)	204 (5.1)		85.5 ±14.4
Current	2,873 (16.7)	2,781 (96.8)	92 (3.2)		91.3 ±14.5
Alcohol consumption				< 0.0001	< 0.0001
None or social	15,397 (89.5)	14,780 (96.0)	617 (4.0)		89.4 ±14.7
High-risk	1,805 (10.5)	1,786 (98.9)	19 (1.1)		93.5 ±12.6
Physical activity				< 0.0001	< 0.0001
Regular	7,371 (42.8)	7,177 (97.4)	194 (2.6)		90.6 ±13.9
None	9831 (57.2)	9,389 (95.5)	442 (4.5)		89.3 ±15.0

Noise exposure

Occupational noise				0.1065	0.2580
No	14,281 (83.0)	13738 (96.2)	543 (3.8)	89.8 ±14.6	
Yes	2,921 (17.0)	2828 (96.8)	93 (3.2)	90.1 ±14.2	
Environmental noise				0.7118	0.4583
No	16,899 (98.2)	16273 (96.3)	626 (3.7)	89.8 ±14.6	
Yes	303 (1.8)	293 (96.7)	10 (3.3)	90.4 ±13.6	
Hearing discomfort				< 0.0001	< 0.0001
Comfort	14,250 (82.8)	13,829 (97.0)	421 (3.0)	91.0 ±14.2	
Discomfort	2,952 (17.2)	2,737 (92.7)	215 (7.3)	84.4 ±15.1	

Obtained by: † Chi-squared test, ‡ Student's t-test, and § One-way ANOVA

Table 2. Results of the logistic regression analysis for noise exposure and CKD

	Crude			Adjusted		
	OR	95% CI	<i>p-value</i>	OR	95% CI	<i>p-value</i>
Occupational noise						
Total	0.83	0.67–1.04	0.1071	0.93	0.73–1.18	0.5289
Male	0.79	0.60–1.03	0.0849	1.02	0.76–1.37	0.8861
Female	0.70	0.47–1.05	0.0850	0.77	0.50–1.18	0.2259
Long-term occupational noise†						
Total	2.33	1.54–3.52	<0.0001	1.51	0.95–2.40	0.0842
Male	1.76	1.08–2.88	0.0240	1.24	0.73–2.10	0.4269
Female	3.49	1.53–7.97	0.0030	2.86	1.18–6.94	0.0205
Environmental noise						
Total	0.89	0.47–1.68	0.7120	0.87	0.45–1.71	0.6924
Male	0.98	0.43–2.25	0.9689	0.99	0.41–2.43	0.9869
Female	0.78	0.29–2.10	0.6168	0.74	0.26–2.07	0.5621
Long-term environmental noise‡						
Total	1.46	0.40–5.29	0.5675	1.01	0.20–5.21	0.9898
Male	1.34	0.23–7.64	0.7430	0.01	<0.001–4.39	0.1266
Female	1.90	0.26–13.81	0.5275	2.25	0.23–22.52	0.4888
Hearing discomfort						
Total	2.58	2.18–3.06	<0.0001	1.24	1.04–1.49	0.0194
Male	2.15	1.70–2.72	<0.0001	1.11	0.86–1.44	0.4117
Female	3.09	2.42–3.96	<0.0001	1.40	1.08–1.81	0.0121

Adjusted for age, sex, educational state, smoker, high-risk drink, aerobic physical activity, hypertension, diabetes, BMI, dyslipidemia.

† Only for participants with occupational noise ($n = 2,921$). Long-term means occupational noise time ≥ 240 months

‡ Only for participants with environmental noise ($n = 303$). Long-term means environmental noise time ≥ 300 minutes per day

Table 3. Results of linear regression analysis for noise exposure time and eGFR

	Crude			Adjusted		
	B	SE	<i>p-value</i>	B	SE	<i>p-value</i>
Occupational noise						
Total (<i>n</i> = 2,921)	-0.0198	0.0019	< 0.0001	-0.0096	0.0019	< 0.0001
Male (<i>n</i> = 1,725)	-0.0134	0.0023	< 0.0001	-0.0080	0.0022	0.0003
Female (<i>n</i> = 1,196)	-0.0275	0.0040	< 0.0001	-0.0159	0.0037	< 0.0001
Environmental noise						
Total (<i>n</i> = 303)	-0.0022	0.0041	0.5923	0.0001	0.0004	0.9751
Male (<i>n</i> = 131)	-0.0030	0.0063	0.6292	0.0024	0.0058	0.6788
Female (<i>n</i> = 172)	-0.0028	0.0054	0.6050	-0.0011	0.0052	0.8315

Adjusted for sex, educational state, smoker, high-risk drink, aerobic physical activity, hypertension, diabetes, BMI, dyslipidemia.

Figures

Figure 1

Association with noise exposure time and eGFR. Adjusted for sex, educational state, HTN, DM, dyslipidemia, BMI, smoking status, high-risk drink, and aerobic physical activity. HTN, hypertension. DM, diabetes mellitus. BMI, body mass index. eGFR, estimated glomerular filtration rate.

Figure 2

Flow chart for Study participants