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Original Article

Association between frequency of adding salt to foods and risk of hearing loss: A population-based cohort study using UK Biobank data

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ABSTRACT

Objectives: To evaluate the association between the frequency of adding salt to food and the risk of incident hearing loss.
Design: A prospective cohort study.
Setting and participants: 492,168 UK Biobank participants aged 40–69 years who were free of hearing loss at baseline (2006–2010) and followed through 2023.
Measurements: Frequency of salt addition to foods was self-reported and categorized as "Never/Rarely," "Sometimes," "Usually," or "Always." Incident hearing loss was identified using ICD-10 codes H90 and H91. Cox proportional hazards models were applied to estimate hazard ratios (HRs) and 95% confidence intervals (CIs), adjusting for demographic, lifestyle, and medical confounders. Causal mediation analyses were conducted to investigate potential intermediating roles of systemic inflammation and vascular dysfunction.
Results: Over a mean follow-up of 11 years, 19,188 participants developed hearing loss. The incidence rate increased from 3.37 to 4.33 per 1,000 person-years across ascending salt use categories. Compared with the "Never/Rarely" group, the adjusted HR for the "Always" group was 1.23 (95% CI, 1.16–1.32), with a significant dose-response relationship (p for trend <0.001). Subgroup analyses showed stronger associations among younger participants, men, and individuals without diabetes or hypertension. Mediation analysis indicated that systemic inflammatory markers, particularly glycoprotein acetyls and CRP, significantly mediated part of the association, while blood pressure and arterial stiffness did not demonstrate a significant mediating effect.
Conclusions: Frequent addition of salt to food was associated with an increased risk of incident hearing loss in a dose-dependent manner. These findings suggest that salt intake may be a modifiable risk factor for hearing loss and implicate systemic inflammation as a potential biological pathway.

1. Introduction

Hearing loss is a prevalent condition that frequently accompanies aging [1,2], and while preventive options have historically been limited [3,4]. However, emerging evidence suggests that certain forms of hearing impairment may be delayed or avoided through targeted interventions [5,6]. This evolving understanding has spurred interest in identifying modifiable risk factors related to the onset and progression of hearing loss [7,8]. Hearing loss is a multifactorial sensory disorder influenced by genetic, environmental, and behavioral factors [6,9–11]. Although age-related degeneration and noise exposure remain prominent contributors, modifiable factors such as ototoxic drug use, chronic noise exposure, and systemic conditions including diabetes and cardiovascular disease (CVD) have gained attention for their roles in auditory decline [12–14]. Public health measures including newborn hearing screening, vaccinations, noise regulations, and safer medication practices have helped reduce the prevalence of hearing loss [15].

Although progress has been made in hearing loss prevention, the role of specific dietary behaviors remains poorly understood. Salt intake, a well-recognized determinant of cardiovascular and metabolic health [16,17], has been linked to hypertension, endothelial dysfunction, and vascular impairment [18,19], all of which may contribute to the

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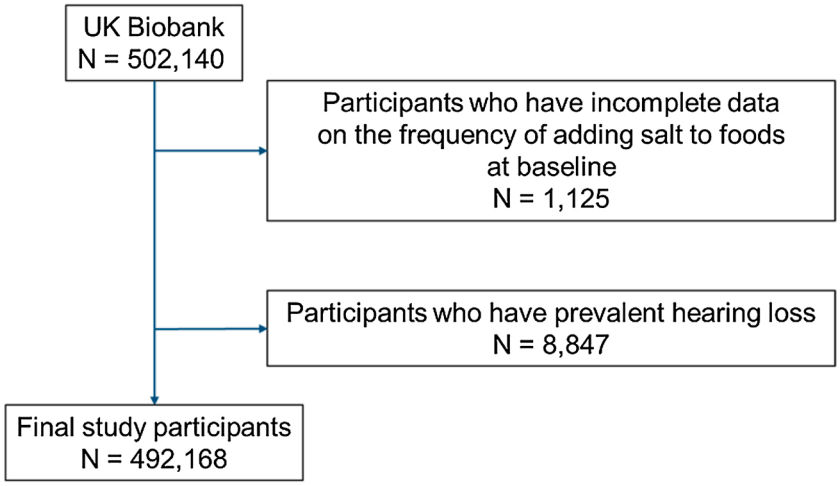


Fig. 1. Flowchart of participant selection.

pathophysiology of hearing loss. While previous research has examined the effects of blood pressure and cardiovascular health on auditory function [20,21], the existing evidence is constrained by several limitations. Many studies are cross-sectional in design [22], rely on relatively small or selective samples [23], where reverse causation is a particular concern. Furthermore, most investigations depend on self-reported hearing difficulties, which may underestimate or misclassify the true burden of disease [24]. Only a few studies have addressed the specific impact of dietary salt habits, such as the frequency of adding salt to foods, on auditory outcomes. To address these gaps, the present study leverages the UK Biobank, a large-scale prospective cohort with standardized follow-up, to examine incident hearing loss objectively defined by ICD-10 codes (H90/H91) from linked health records. By excluding prevalent cases at baseline and following participants longitudinally, we aimed to minimize the potential for reverse-causation bias.

Furthermore, focusing on a simple behavioral marker of sodium intake, the frequency of adding salt to foods, provides a pragmatic and potentially modifiable dietary target that has not been systematically evaluated in relation to hearing outcomes. The frequency of adding salt to foods is a simple yet meaningful behavioral marker of long-term sodium consumption [25–27], and may offer insights into preventable dietary contributors to hearing loss. In this study, we investigated whether a higher frequency of salt addition to foods is associated with an increased risk of incident hearing loss in a large prospective cohort. By examining this relationship, we aimed to evaluate whether this dietary habit represents a reasonable and modifiable target for hearing loss prevention. In addition, we conducted mediation analyses to explore potential biological pathways, demonstrating that systemic inflammatory markers, particularly glycoprotein acetyls and CRP, partially explained the observed association, whereas blood pressure and arterial stiffness did not. This mechanistic evidence further supports the plausibility of a causal link between excessive salt intake and auditory decline.

2. Method

2.1. Study population

Fig. 1 shows the flowchart. This is a prospective cohort study utilizing data from the UK Biobank, a large-scale cohort of UK adults aged 40–69 years at baseline (2006–2010). The UK Biobank dataset has been extensively documented [28]. Initially, 502,140 participants were considered. Participants with preexisting hearing loss at baseline were excluded (N = 1,125). Additionally, individuals who did not report their frequency of adding salt to foods or selected "Prefer not to answer" were excluded (N = 8,847). The final analytical sample comprised 492,168 participants, categorized into four groups based on self-reported salt intake frequency: "Never/Rarely," "Sometimes," "Usually," or "Always."

2.2. Exposure assessment

The primary exposure variable was the self-reported frequency of adding salt to foods (Data-Field 1478), assessed via a touchscreen questionnaire at baseline. Participants chose from the four response options: "Never/Rarely," "Sometimes," "Usually," or "Always."

2.3. Outcome assessment

Hearing loss was identified using ICD-10 diagnosis codes H90 (Conductive and sensorineural hearing loss) and H91 (Other and unspecified hearing loss). The primary outcome was incident hearing loss, defined as the first occurrence of either H90 or H91 during follow-up. To operationalize this outcome, we used UK Biobank Data-Fields 131258 and 131260, which record the date of the first occurrence of each ICD-10 diagnosis. A binary variable was created, coded as 1 if any diagnosis of H90 or H91 occurred during follow-up and 0 otherwise. Prevalent cases (those with H90/H91 prior to baseline) were excluded, and person-years were calculated from baseline to the date of first diagnosis, death, loss to follow-up, or end of data availability, whichever came first.

2.4. Covariates

Potential confounders and covariates were identified based on prior literature and included demographic factors (age, sex, and race/ethnicity), Townsend deprivation index, lifestyle factors (smoking status, alcohol consumption, physical activity measured in metabolic equivalent of task [MET] hours/week), and body mass index (BMI). Health conditions, including dyslipidemia, diabetes mellitus, CVD, hypertension, tinnitus, and depression, were included only if they were formally diagnosed and documented in the medical records of the participants to ensure diagnostic accuracy. Self-reported conditions without a formal diagnosis were excluded from the analysis. Noise exposure was assessed based on self-reported exposure to loud music and occupational noise. A diet score was constructed based on adherence to five key dietary components, reflecting overall diet quality in line with public health recommendations. Dietary intake information was obtained from baseline touchscreen questionnaires in the UK Biobank. Each component was assigned a score of 1 (healthy) or 0 (unhealthy), resulting in a total score ranging from 0 to 5, with higher scores indicating better overall diet quality. The specific scoring criteria are detailed in

Supplementary Table S2. Missing values were imputed utilizing mode imputation.

2.5. Urinary sodium measurement

Urinary sodium was measured at baseline using random spot urine samples collected during the initial assessment visit (2006–2010). The analysis was conducted using the Ion Selective Electrode (ISE) method with the Beckman Coulter AU5400 clinical chemistry analyzer (Beckman Coulter UK Ltd), as part of the UK Biobank Biomarker Enhancement Project. All measurements were performed under ISO 17025-accredited quality control protocols, and only values within the validated analytical range (10–400 mmol/L) were included. Urinary sodium levels were used to validate the self-reported salt intake frequency categories and were analyzed for dose-response trends.

2.6. Measurement of vascular and inflammatory biomarkers used in mediation analysis

All mediators were measured at baseline during the initial assessment visit (2006–2010). Blood-based biomarkers were quantified from serum or plasma samples using standardized protocols under the UK Biobank Biomarker Enhancement Project. Glycoprotein acetyls (GlycA) were quantified via high-throughput nuclear magnetic resonance (NMR) spectroscopy using the Nightingale Health platform. This inflammatory marker reflects concentrations of multiple acute-phase glycoproteins and was measured from EDTA plasma. C-reactive protein (CRP) was measured from serum using a high-sensitivity immunoturbidimetric assay (Beckman Coulter AU5800, UK), with an analytical range of 0.08–80 mg/L. Pulse wave arterial stiffness index was derived from digital volume pulse measurements using finger photoplethysmography (PulseTrace PCA2, CareFusion) while the participant was seated. Systolic blood pressure (SBP), diastolic blood pressure (DBP), pulse pressure, and pulse rate were measured twice in the seated position using an Omron HEM-7015IT digital blood pressure monitor after at least 5 min of rest. The mean of the two readings was used in the analysis.

2.7. Statistical analysis

Differences in baseline characteristics across salt intake groups were examined using linear regression for continuous variables and logistic regression for categorical variables, with the ordinal salt intake variable modeled as a continuous term to test for linear trend [29].

Cox proportional hazard regression models were used to estimate hazard ratios (HRs) and 95% confidence intervals (CIs) for the relationship between salt intake frequency and hearing loss risk, with adjustments for potential confounders in the multivariable models. A trend test was conducted by treating salt intake as a continuous variable in the Cox regression models to evaluate the linear association with hearing loss risk. Additionally, a pairwise Jonckheere–Terpstra test was performed to compare differences between specific salt intake groups. Interaction analysis was conducted by adjusting for self-reported speech recognition threshold in noise (SRTn) values and testing for interaction terms to examine potential effect modification. Each ear was tested individually, and the SRTn of the better-performing ear was used for analysis. Based on this performance, participants were classified into two categories: normal hearing (SRTn < -5.5 dB) and insufficient hearing (-5.5 dB $\leq$ SRTn ≤ -3.5 dB) [30]. Stratified analyses were performed to examine whether the associations varied based on age, sex, race and ethnicity, BMI, Townsend deprivation index, smoking status, alcohol consumption, regular physical activity, hypertension, CVD, diabetes, tinnitus, loud music exposure frequency, and noisy workplace at baseline. Sensitivity analyses were performed to test the robustness of the findings by excluding participants with hypertension, dyslipidemia, diabetes, CVD, tinnitus, a history of loud music exposure, or noisy workplaces, alongside those who reported dietary changes within 5

Table 1
Baseline characteristics of participants by frequency of adding salt to foods.

Characteristic	Never or rarely adds salt to food (N = 273,014)	Sometimes adds salt to food (N = 137,974)	Usually adds salt to food (N = 57,242)	Always adds salt to food (N = 23,938)	P for trend
Demographics and lifestyle characteristics					
Age, mean (SD)	56.49 (8.08)	56.39 (8.11)	56.96 (8.04)	55.90 (8.26)	0.7261
Sex, No. (%)					
Female	153,315 (56.16)	74,632 (54.09)	28,050 (49.00)	12,352 (51.60)	< 0.001
Male	119,699 (43.84)	63,342 (45.91)	29,192 (51.00)	11,586 (48.40)	< 0.001
Race and ethnicity (%)					
Asian	4,492 (1.65)	3,739 (2.71)	1,717 (3.00)	1,311 (5.48)	< 0.001
Black	3,671 (1.34)	2629 (1.91)	869 (1.52)	825 (3.45)	< 0.001
White	260,621 (95.46)	128,739 (93.31)	53,439 (93.36)	20,953 (87.53)	< 0.001
Multiracial[a]	1,389 (0.51)	903 (0.65)	393 (0.69)	242 (1.01)	< 0.001
Others[b]	1,882 (0.69)	1,512 (1.10)	615 (1.07)	495 (2.07)	< 0.001
Unknown[c]	959 (0.35)	452 (0.33)	209 (0.37)	112 (0.47)	< 0.001
BMI, mean (SD)	27.18 (4.73)	27.64 (4.81)	27.83 (4.81)	28.03 (5.10)	< 0.001
Smoking status, No. (%)	21,920 (8.03)	15,688 (11.37)	8,760 (15.30)	5,669 (23.68)	< 0.001
Alcohol status, No. (%)	250,875 (91.89)	127,882 (92.69)	52,900 (92.41)	20,937 (87.46)	< 0.001
Townsend deprivation index, mean (SD)	−1.49 (2.99)	−1.22(3.12)	−1.08 (3.18)	−0.18 (3.51)	< 0.001
MET, mean (SD)	43.99 (38.35)	43.99 (38.61)	43.89 (39.29)	46.06 (42.52)	< 0.001
Diet score, mean (SD)[d]	2.64 (1.24)	2.43 (1.22)	2.24 (1.23)	2.06 (1.21)	< 0.001
Health Conditions					
Depression status, No. (%)	17,807 (6.52)	9,097 (6.59)	3,990 (6.97)	1,830 (7.64)	< 0.001
Dyslipidemia, No. (%)	68,349 (25.03)	35,083 (25.43)	15,267 (26.67)	6,556 (27.39)	< 0.001
Diabetes, No. (%)	26,222 (9.60)	14,698 (10.65)	6,427 (11.23)	3,094 (12.93)	< 0.001
CVD, No. (%)	21,442 (7.85)	11,093 (8.04)	5,073 (8.86)	2,439 (10.19)	< 0.001
Hypertension, No. (%)	111,035 (40.67)	54,411 (39.44)	23,003 (40.19)	10,076 (42.09)	< 0.001
Hearing and Noise Exposure					
Tinnitus, No. (%)	9,665 (3.54)	4,887 (3.54)	2,012 (3.51)	832 (3.48)	0.9493
Loud music exposure, No. (%)	10,584 (3.88)	5,962 (4.32)	2,650 (4.63)	1,391 (5.81)	< 0.001
Noisy workplace, No. (%)	19,656 (7.20)	11,144 (8.08)	5,034 (8.79)	2,517 (10.51)	< 0.001
Hearing difficulty/ problems No. (%)	62,098 (22.75)	32,132 (23.29)	14,432 (25.21)	6,223 (26.00)	< 0.001
Hearing difficulty/ problems with background noise No. (%)	95,055 (34.82)	50,196 (36.38)	22,237 (38.85)	9,704 (40.54)	< 0.001
Hearing aid user No. (%)	6,600 (2.42)	3,461 (2.51)	1,622 (2.83)	724 (3.02)	< 0.001
Cochlear implant No. (%)	60 (0.02)	57 (0.04)	25 (0.04)	18 (0.08)	< 0.001

SD, standard deviation; BMI, body mass index; MET, metabolic equivalent of task; CVD, cardiovascular disease.

[a] Multiracial category combined the responses of the UK Biobank race and ethnicity questions of "mixed," "White and Black Caribbean," "White and Black African," "White and Asian," and "any other mixed backgrounds.".

[b] Other was defined as any race or ethnicity not otherwise specified in the provided categories.

[c] Unknown included participants with missing or unreported race/ethnicity data.

[d] Diet score included intake of fruits, vegetables, fish, unprocessed meat, and processed meat.

Table 2
Incident rate of hearing loss by frequency of adding salt to food.

Participants (N = 492,168)	Never or rarely add salt to food (N = 273,014)	Sometimes adds salt to food (N = 137,974)	Usually adds salt to food (N = 57,242)	Always adds salt to food (N = 23,938)
Hearing loss events (%)	10,334 (3.79)	5,355 (3.88)	2,425 (4.24)	1,074 (4.49)
Person-years	3.06×10^6	1.52×10^6	6.22×10^5	2.48×10^5
Incident rate (per 1,000 PY)	3.37	3.53	3.89	4.33

PY, person-years.

years prior to baseline. Also, a multivariable Cox regression model additionally adjusted for diet score was conducted to examine the robustness of the association between salt intake frequency and hearing loss. The diet score, was constructed according to the method proposed by Tang et al., based on UK Biobank food frequency questionnaire data and reflects overall dietary quality [25].

To evaluate potential mediating pathways, causal mediation analysis was conducted using a counterfactual framework. Salt intake was modeled as the exposure, hearing loss as the outcome, and inflammation and vascular biomarkers, including glycoprotein Acetyls (GlcA), C-reactive protein (CRP), pulse wave arterial stiffness index, systolic blood pressure, diastolic blood pressure, pulse pressure, and pulse rate, as potential mediators. The indirect effect (average causal mediation effect, ACME) and direct effect (average direct effect, ADE) were estimated using a combination of linear regression and Cox proportional hazards models. The Sobel test was used to assess the significance of the mediation effect, and the proportion mediated was calculated as the ratio of the indirect effect to the total effect. Nonparametric bootstrapping with 1,000 resamples was applied to generate 95% confidence intervals for ACME and the proportion mediated. All mediation analyses were stratified by salt intake frequency categories.

All statistics analyses were performed using Python (version 3.9.5, Python Software Foundation, Wilmington, Delaware).

3. Results

3.1. Baseline characteristics by salt intake frequency

Among the 492,168 participants, those who always added salt to food had distinct demographic and health profiles compared to individuals who never or rarely added salt (Table 1). Participants in the "Always" group were more likely to be male, less likely to be White, and had higher BMI and Townsend Deprivation Index scores, indicating greater socioeconomic deprivation. They also exhibited higher smoking prevalence and poorer diet quality, whereas their alcohol consumption was lower than those who never or rarely added salt. Higher salt intake was also linked to a greater prevalence of cardiometabolic conditions, including hypertension, diabetes, dyslipidemia, and CVD at baseline. Regarding hearing-related factors, frequent salt users were more likely to experience hearing difficulties, particularly in background noise, and had a slightly higher prevalence of hearing aid and cochlear implant use.

3.2. Frequency of salt added to food and risk of hearing loss

We observed a dose-dependent relationship between salt intake frequency and hearing loss incidence over time. Hearing loss incidence increased with greater salt intake (Table 2, Fig. 2).

This trend was consistent with the increasing hazard ratios observed across three models: (1) an unadjusted model; (2) a model adjusted for age and sex; (3) a fully adjusted model that included demographic variables. Table 3 presents the HRs for hearing loss risk based on salt intake frequency. Although the magnitude of the association was

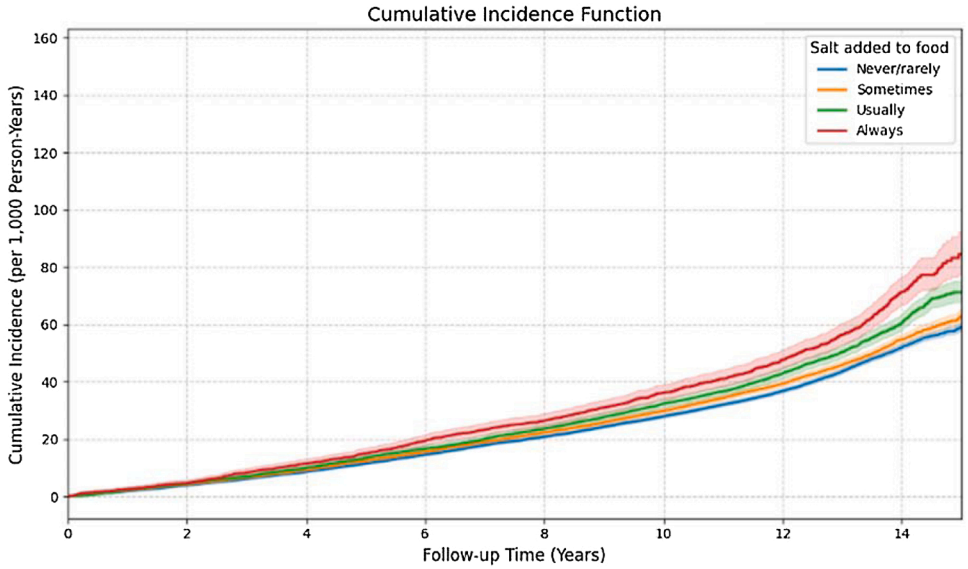


Fig. 2. Cumulative incidence function (CIF) of hearing loss by frequency of adding salt to food.
This figure illustrates the Nelson–Aalen estimated cumulative incidence of hearing loss per 1,000 person-years across four salt intake frequency groups: "Never/rarely," "Sometimes," "Usually," and "Always." The analysis is based on follow-up data up to 17.5 years. Solid lines represent cumulative hazard estimates, and shaded areas indicate 95% confidence intervals. A dose-response trend is observed, with higher cumulative incidence in groups reporting more frequent addition of salt to food, most notably in the "Always" group.

Table 3
Frequency of adding salt to foods and hearing loss risk.

Model	Frequency of adding salt to food, HR (95% CI)				P for trend
	Never or rarely adds salt to food	Sometimes adds salt to food	Usually adds salt to food	Always adds salt to food	
Univariable	1 [Reference]	1.06 (1.02–1.09)	1.17 (1.12–1.22)	1.33 (1.25–1.41)	< 0.001
Sex- and age-adjusted	1 [Reference]	1.05 (1.02–1.09)	1.10 (1.05–1.15)	1.33 (1.25–1.38)	< 0.001
Multivariable adjusted[a]	1 [Reference]	1.04 (1.01–1.08)	1.08 (1.03–1.13)	1.23 (1.16–1.32)	< 0.005
Multivariable adjusted[a] + Diet score	1 [Reference]	1.03 (1.00–1.07)	1.07 (1.02–1.12)	1.21 (1.14–1.29)	< 0.005

HR, hazard ratio; CI, confidence interval.
[a] Adjusted for sex, age, race and ethnicity, body mass index, Townsend deprivation index, smoking, drinking, regular physical activity, hypertension, cardiovascular diseases, diabetes, tinnitus, loud music exposure frequency, and noisy workplace at baseline.

Table 4
Stratified analysis of salt intake and hearing loss risk.

Subgroups		N	Frequency of adding salt to food, HR (95%CI)[a]			
			Never or rarely adds salt to food (N = 273,014)	Sometimes adds salt to food (N = 137,974)	Usually adds salt to food (N = 57,242)	Always adds salt to food (N = 23,938)
Age, y	≤ 60	304,138	1 [Reference]	1.08 (1.03–1.14)	1.16 (1.08–1.25)	1.53 (1.38–1.68)
	> 60	198,525	1 [Reference]	1.05 (1.00–1.09)	1.09 (1.03–1.15)	1.29 (1.19–1.40)
Sex	Female	268,349	1 [Reference]	1.02 (0.97–1.07)	1.05 (0.98–1.13)	1.44 (1.31–1.58)
	Male	223,819	1 [Reference]	1.10 (1.05–1.15)	1.16 (1.09–1.23)	1.34 (1.23–1.46)
Race and ethnicity	White	463,751	1 [Reference]	1.06 (1.02–1.09)	1.12 (1.07–1.17)	1.39 (1.31–1.49)
	Others[a]	28,417	1 [Reference]	1.15 (0.98–1.36)	1.08 (0.86–1.35)	1.19 (0.91–1.56)
Townsend Deprivation Index score, quintile	1 (Low)	98,317	1 [Reference]	1.05 (0.98–1.13)	1.12 (1.01–1.24)	1.26 (1.06–1.49)
	2–4 (Intermediate)	295,617	1 [Reference]	1.04 (1.00–1.09)	1.06 (1.00–1.13)	1.25 (1.15–1.36)
	5 (High)	98,234	1 [Reference]	1.03 (0.96–1.11)	1.09 (0.99–1.20)	1.19 (1.06–1.34)
Diet score	≥ Median	247,077	1 [Reference]	1.03 (0.99–1.08)	1.08 (1.00–1.15)	1.22 (1.10–1.36)
	< Median	245,091	1 [Reference]	1.04 (0.99–1.09)	1.07 (1.01–1.14)	1.21 (1.12–1.31)
Smoking status	Noncurrent	440,131	1 [Reference]	1.05 (1.01–1.09)	1.11 (1.06–1.17)	1.35 (1.26–1.45)
	Current	52,037	1 [Reference]	1.11 (1.00–1.24)	1.04 (0.91–1.18)	1.33 (1.15–1.54)
Alcohol status	Noncurrent	40,158	1 [Reference]	1.01 (0.91–1.13)	1.12 (0.96–1.30)	1.31 (1.10–1.57)
	Current	452,010	1 [Reference]	1.07 (1.04–1.11)	1.12 (1.07–1.18)	1.39 (1.30–1.48)
Baseline diabetes	No	441,727	1 [Reference]	1.11 (1.06–1.17)	1.06 (1.03–1.1)	1.35 (1.26–1.45)
	Yes	50,441	1 [Reference]	0.99 (0.85–1.15)	0.98 (0.90–1.06)	0.99 (0.89–1.10)
Baseline dyslipidemia	No	366,913	1 [Reference]	1.06 (1.02–1.11)	1.15 (1.08–1.21)	1.35 (1.24–1.46)
	Yes	125,255	1 [Reference]	1.03 (0.98–1.08)	1.16 (1.08–1.24)	1.22 (1.11–1.34)
Baseline hypertension	No	293,643	1 [Reference]	1.10 (1.04–1.15)	1.13 (1.06–1.21)	1.38 (1.25–1.52)
	Yes	198,525	1 [Reference]	1.02 (0.97–1.06)	1.07 (1.00–1.13)	1.21 (1.11–1.31)

[a] Adjusted for sex, age, race and ethnicity, body mass index, Townsend deprivation index, smoking, drinking, regular physical activity, hypertension, cardiovascular diseases, diabetes, tinnitus, loud music exposure frequency, and noisy workplace at baseline. HR, hazard ratio; CI, confidence interval.

slightly attenuated with progressive adjustment, the overall pattern of increased risk with higher salt intake persisted. After additional adjustment for overall diet quality using the diet score, the association remained robust, indicating that general dietary patterns did not fully explain the relationship. The test for trend showed a statistically significant increase, confirming a dose-dependent relationship between salt intake frequency and hearing loss risk.

We conducted stratified analyses to evaluate effect modifications by age, sex, race/ethnicity, Townsend Deprivation Index, smoking, drinking, and lifestyle-related diseases (Table 4, Fig. 3). The association between higher salt intake and hearing loss risk was stronger in younger participants (≤60 years) than in older adults (>60 years). In the sex-stratified analysis, women who reported "Sometimes" or "Usually" adding salt to food did not show a statistically significant increase in the risk of hearing loss compared to those who "Never or rarely" added salt. However, a significantly increased risk was observed in the "Always" group. In contrast, among men, the risk of hearing loss increased consistently across all higher frequency groups, indicating a dose–response relationship. The risk was also greater among White participants but weaker and non-significant among non-White individuals. Stratification by Townsend Deprivation Index showed stronger associations in individuals with lower deprivation scores. The association was more evident in non-smokers and current drinkers and remained significant only in participants without diabetes or hypertension. Consistent findings were observed in the stratified analysis by overall diet quality, as measured by the diet score. When participants were divided by the median diet score, the association between salt intake frequency and hearing loss remained significant in both strata, indicating comparable effect sizes. These results suggest that frequent discretionary salt use may increase the risk of hearing loss regardless of background diet quality. Sensitivity analyses excluding participants with preexisting conditions (hypertension, dyslipidemia, diabetes, and CVD), as well as those with hearing-related risk factors (tinnitus, loud music exposure, and noisy workplace), or recent dietary changes, confirmed the robustness of the association between higher salt intake and hearing loss risk (Supplementary Table S1). Although excluding participants with hearing-related risk factors or dietary changes slightly weakened the estimates, the overall trend was unchanged. Additionally, in the fully adjusted model incorporating diet score, the association persisted, suggesting that overall diet quality does not fully explain the observed relationship.

3.3. Urinary sodium and hearing loss risk

To determine whether baseline hearing status influenced the association between salt intake and hearing loss, we conducted stratified analyses by hearing ability (Supplementary Fig. S1). In participants with normal hearing (SRTn < -5.5 dB), higher salt intake was significantly associated with an increased risk of hearing loss, showing a clear dose–response relationship. In contrast, the associations were weaker and

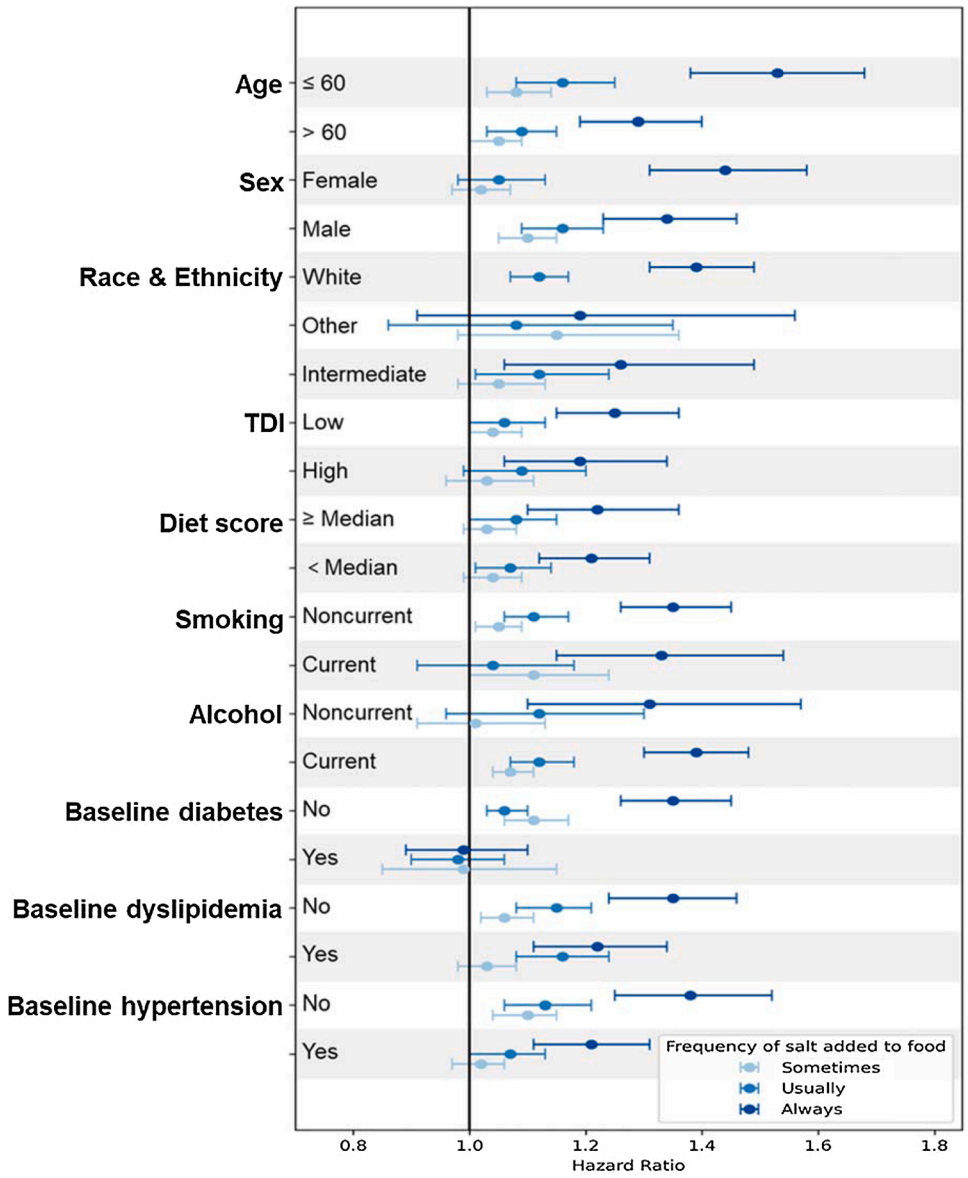


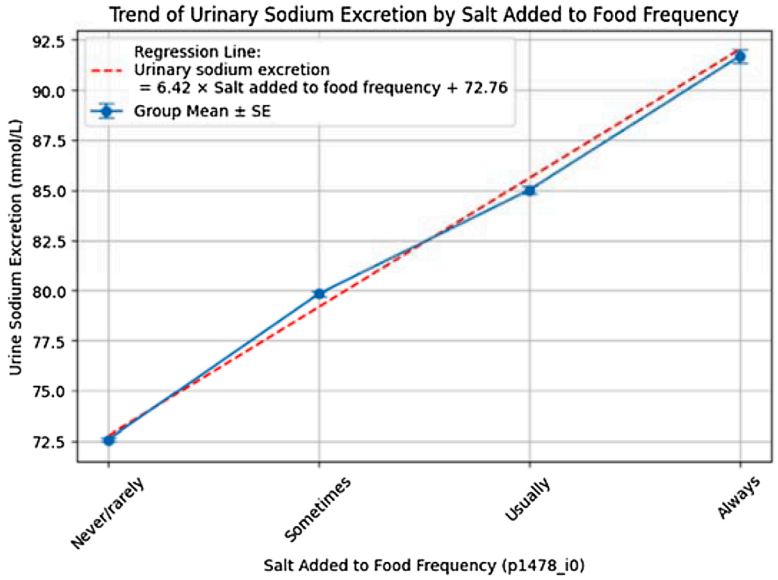
Fig. 3. Subgroup analyses of the association between frequency of adding salt to food and incident hearing loss.
Hazard ratios (HRs) and 95% confidence intervals (CIs) are shown for three groups according to the self-reported frequency of adding salt to foods: Sometimes, Usually, and Always, using Never/Rarely as the reference category (HR = 1.0).
Subgroup analyses were stratified by age (≤60 vs. >60 years), sex, race/ethnicity, Townsend Deprivation Index (TDI), smoking status, alcohol consumption, diabetes, dyslipidemia, and hypertension.
All models were adjusted for age, sex, race/ethnicity, body mass index, Townsend deprivation index, smoking status, alcohol intake, physical activity, hypertension, cardiovascular disease, diabetes mellitus, tinnitus, loud music exposure, and workplace noise exposure at baseline. The vertical line at HR = 1.0 represents no association.

not statistically significant in the insufficient hearing group (SRTn ≥ -5.5 dB to ≤ -3.5 dB). Although the interaction term between salt intake and baseline hearing ability was not significant, the stratified analysis suggests a stronger association between salt intake and hearing loss risk in individuals with normal hearing.

To validate self-reported salt intake and its association with hearing loss, we analyzed urinary sodium excretion (Fig. 4). Urinary sodium levels increased consistently with higher self-reported salt use, supporting the validity of self-reported dietary sodium intake assessments. When participants were stratified into quartiles based on urinary sodium excretion, the risk of hearing loss showed a modest but significant increase with higher sodium excretion relative to the lowest quartile. However, the risk plateaued in the highest quartile, suggesting a potential threshold beyond which additional sodium exposure does not further elevate hearing loss risk.

3.4. *Mediation effects of inflammatory and vascular biomarkers on the association between salt intake and hearing Loss*

We performed causal mediation analysis using a counterfactual framework to explore potential mediating mechanisms underlying the association between salt intake and hearing loss (Table 5). Glycoprotein Acetyls (GlycA), C-reactive protein (CRP), pulse wave arterial stiffness index, systolic blood pressure, diastolic blood pressure, pulse pressure, and pulse rate were evaluated as candidate mediators. Among the inflammatory biomarkers, GlycA and CRP exhibited statistically significant mediation effects across all salt intake groups. The effects were most pronounced in the "Always" group, suggesting that chronic low-



Urinary sodium excretion (mmol/L)			
	Mean	Std	SE
Never/rarely	72.58	42.34	0.08
Sometimes	79.84	44.56	0.13
Usually	85.01	46.38	0.20
Always	91.68	49.67	0.34

Urinary sodium excretion	Hearing loss HR (95% CI)
Q1 [10.00 – 42.60] (n=113,350)	1 [Reference]
Q2 [42.60 – 68.00] (n=113,096)	1.07 (1.03-1.12)
Q3 [68.00 – 103.10] (n=112,938)	1.08 (1.04-1.13)
Q4 [103.10 – 380.70] (n=112,889)	1.05 (1.01-1.10)

Fig. 4. Association of urinary sodium excretion with salt intake frequency and risk of hearing loss.
HR, hazard ratio; CI, confidence interval; Q1–Q4, quartiles of urine sodium concentration.

grade inflammation may partially explain the relationship between salt intake and increased risk of hearing loss. However, in the mediation analysis of the pulse wave arterial stiffness index, no significant direct effect was observed, which may be attributable to limited statistical power. The proportion of the total effect mediated by these inflammatory pathways remained modest overall.

In contrast, vascular biomarkers demonstrated limited mediation effects. Systolic blood pressure showed small but statistically significant mediation across all salt intake groups. Diastolic blood pressure, pulse pressure, and pulse wave arterial stiffness index did not exhibit significant mediation, except for a minor negative effect observed for diastolic blood pressure in one subgroup. Pulse rate showed borderline significance only in the "Always" group, with a low proportion of the total effect mediated.

4. Discussion

This large-scale prospective study provides novel and robust evidence that frequent addition of salt to food is associated with an increased risk of incident hearing loss in a dose-dependent manner. Using UK Biobank data, we identified progressively elevated risk across intake categories, and associations remained consistent after adjusting for multiple sociodemographic, behavioral, and medical variables. These findings suggest that discretionary salt use may represent a modifiable dietary risk factor for auditory dysfunction with broad implications for clinical nutrition and public health.

Causal mediation analysis indicated that systemic low-grade inflammation, particularly GlycA and CRP, partially explained the observed association. This supports the hypothesis that chronic inflammatory burden contributes to auditory decline, consistent with prior longitudinal findings linking elevated CRP to hearing impairment in middle-aged adults [31]. In contrast, some studies in older populations found null associations [32], highlighting the limitations of CRP as a single-point biomarker. GlycA, with its longer half-life and stability, may better reflect long-term inflammatory exposure [33]. Its significant mediation effect in our study strengthens the plausibility that chronic inflammation is a pathway through which sodium contributes to cochlear damage. By contrast, vascular markers such as blood pressure and arterial stiffness showed weaker or inconsistent effects, suggesting inflammation may play a more central role than hemodynamic stress [34,35].

Several plausible biological mechanisms may explain this relationship. The cochlea is an energy-intensive organ that depends on tightly regulated ion transport to sustain auditory signaling [36,37]. In particular, low sodium (Na^+) and high potassium (K^+) concentrations in the endolymph are essential for hair cell depolarization and repolarization, which convert mechanical sound waves into neural signals [38]. Excessive salt intake, the main dietary source of Na^+, can elevate plasma and interstitial sodium levels, thereby disrupting cochlear ion homeostasis. The positive correlation between self-reported salt use and urinary sodium excretion in our study supports this pathway, providing indirect evidence that higher sodium exposure increases hearing loss risk. Vascular integrity is also critical for maintaining ionic gradients. Previous studies also suggested that the cochlea relies on a delicate microvascular network, particularly the stria vascularis, to generate and sustain the endocochlear potential and produce endolymph [39,40]. Chronic hypertension and endothelial dysfunction, both established consequences of high sodium intake [41], may impair cochlear perfusion, promote microvascular remodeling, and damage the stria vascularis [42], may impair cochlear perfusion, promote microvascular remodeling, and damage the stria vascularis [40,43]. These vascular alterations reduce oxygen and nutrient delivery to the cochlea, accelerating degeneration of sensory hair cells. Because these cells lack regenerative capacity [44,45], sodium-related vascular and ionic stress can ultimately result in permanent sensorineural hearing loss [46].

Our findings align with these mechanistic pathways and also provide important clinical insights. In stratified analyses, the association between salt intake and hearing loss was more pronounced among individuals with preserved auditory function at baseline, defined by speech reception thresholds in noise (SRTn) of less than –5.5 dB. This suggests that sodium-related cochlear injury may begin before the onset of clinically detectable impairment. In addition, stronger associations were observed in younger individuals aged 60 years or younger, indicating that dietary behaviors in early or midlife may have lasting implications for auditory health. These results point to a potential window of opportunity for preventive interventions. Alternatively, this pattern may also reflect a statistical effect [47]. Participants with better baseline auditory function may have had greater measurable capacity for deterioration, making subsequent decline more easily detectable. In contrast, those with already impaired hearing may have experienced a ceiling effect, limiting the ability to observe further decline. This possibility should be considered when interpreting stratified results.

Table 5
Mediation analysis of the association between salt intake and hearing loss through inflammatory and hypertension biomarkers.

Outcome	Group	Effect	Estimate	95% CIL	95% CIH	p-value
Glycoprotein Acetyls (n = 268,995)	Sometimes (n = 75,686)	Average Causal Mediation Effect	0.0019	0.0009	0.0030	<0.0001
		Average Direct Effect	0.0348	−0.0095	0.0790	0.1233
		Total Effect	0.0368	−0.0074	0.0810	0.1028
		Proportion Mediated	0.0519	−0.2622	0.5189	0.5060
	Usually (n = 31,229)	Average Causal Mediation Effect	0.0026	0.0012	0.0042	<0.0001
		Average Direct Effect	0.0476	−0.0123	0.1076	0.1192
		Total Effect	0.0508	−0.0091	0.1107	0.0966
		Proportion Mediated	0.0517	−0.3331	0.3189	0.4960
	Always (n = 13,031)	Average Causal Mediation Effect	0.0050	0.0022	0.0075	<0.0001
		Average Direct Effect	0.2250	0.1407	0.3093	<0.0001
		Total Effect	0.2308	0.1466	0.3151	<0.0001
		Proportion Mediated	0.0215	0.0094	0.0397	0.4770
C-reactive protein (n = 459,228)	Sometimes (n = 128,767)	Average Causal Mediation Effect	0.0016	0.0011	0.0022	<0.0001
		Average Direct Effect	0.0411	0.0068	0.0755	0.0188
		Total Effect	0.0432	0.0089	0.0775	0.0135
		Proportion Mediated	0.0370	0.0170	0.1573	0.5080
	Usually (n = 53,437)	Average Causal Mediation Effect	0.0026	0.0018	0.0035	<0.0001
		Average Direct Effect	0.0820	0.0359	0.1280	0.0005
		Total Effect	0.0852	0.0392	0.1312	<0.0001
		Proportion Mediated	0.0307	0.0172	0.0728	0.4910
	Always (n = 22,252)	Average Causal Mediation Effect	0.0050	0.0033	0.0066	<0.0001
		Average Direct Effect	0.2132	0.1476	0.2788	<0.0001
		Total Effect	0.2192	0.1536	0.2847	<0.0001
		Proportion Mediated	0.0228	0.0141	0.0359	0.4590
Pulse wave Arterial Stiffness index (n = 166,309)	Sometimes (n = 46,584)	Average Causal Mediation Effect	0.0003	−0.0005	0.0011	0.2226
		Average Direct Effect	0.0534	−0.0072	0.1141	0.0844
		Total Effect	0.0536	−0.0071	0.1143	0.0835
		Proportion Mediated	0.0059	−0.0320	0.0684	0.5330
	Usually (n = 19,063)	Average Causal Mediation Effect	0.0004	−0.0002	0.0013	0.2318
		Average Direct Effect	0.0832	0.0008	0.1655	0.0478
		Total Effect	0.0832	0.0009	0.1656	0.0477
		Proportion Mediated	0.0043	−0.0063	0.0383	0.5350
	Always (n = 8,120)	Average Causal Mediation Effect	0.0008	−0.0005	0.0024	0.2163
		Average Direct Effect	0.0734	−0.0507	0.1976	0.2464
		Total Effect	0.0746	−0.0496	0.1987	0.2391
		Proportion Mediated	0.0109	−0.1270	0.1234	0.5360
Systolic blood pressure (n = 463,223)	Sometimes (n = 129,388)	Average Causal Mediation Effect	0.0013	0.0008	0.0018	<0.0001
		Average Direct Effect	0.0407	0.0066	0.0747	0.0192
		Total Effect	0.0425	0.0085	0.0765	0.0144
		Proportion Mediated	0.0297	0.0126	0.1826	0.4830
	Usually (n = 53,512)	Average Causal Mediation Effect	0.0024	0.0016	0.0032	<0.0001
		Average Direct Effect	0.0882	0.0426	0.1338	<0.0001
		Total Effect	0.0902	0.0445	0.1358	<0.0001
		Proportion Mediated	0.0267	0.0158	0.0607	0.5400
	Always (n = 22,375)	Average Causal Mediation Effect	0.0026	0.0016	0.0037	<0.0001
		Average Direct Effect	0.1898	0.1239	0.2558	<0.0001
		Total Effect	0.1922	0.1263	0.2582	<0.0001
		Proportion Mediated	0.0135	0.0073	0.0235	0.4950
Diastolic blood pressure (n = 463,228)	Sometimes (n = 129,390)	Average Causal Mediation Effect	−0.0004	−0.0008	−0.0001	0.0366
		Average Direct Effect	0.0426	0.0085	0.0766	0.0142
		Total Effect	0.0425	0.0084	0.0765	0.0145
		Proportion Mediated	−0.0088	−0.0466	0.0001	0.5050
	Usually (n = 53,512)	Average Causal Mediation Effect	−0.0001	−0.0006	<0.001	0.6774
		Average Direct Effect	0.0903	0.0447	0.1359	<0.0001
		Total Effect	0.0902	0.0446	0.1358	<0.0001
		Proportion Mediated	−0.0011	−0.0079	0.0042	0.6840
	Always (n = 22,375)	Average Causal Mediation Effect	−0.0004	−0.0010	<0.001	0.3079
		Average Direct Effect	0.1925	0.1266	0.2585	<0.0001
		Total Effect	0.1922	0.1263	0.2582	<0.0001
		Proportion Mediated	−0.0019	−0.0062	0.0020	0.5160
Pulse pressure (n = 463,223)	Sometimes (n = 129,388)	Average Causal Mediation Effect	0.0014	0.0008	0.0020	<0.0001
		Average Direct Effect	0.0408	0.0067	0.0748	0.0190
		Total Effect	0.0425	0.0085	0.0765	0.0144
		Proportion Mediated	0.0324	0.0134	0.1593	0.5020
	Usually (n = 53,512)	Average Causal Mediation Effect	0.0022	0.0014	0.0031	<0.0001
		Average Direct Effect	0.0883	0.0427	0.1339	<0.0001
		Total Effect	0.0902	0.0445	0.1358	<0.0001
		Proportion Mediated	0.0249	0.0133	0.0559	0.5230
	Always (n = 22,375)	Average Causal Mediation Effect	0.0026	0.0015	0.0037	<0.0001
		Average Direct Effect	0.1899	0.1239	0.2558	<0.0001
		Total Effect	0.1922	0.1263	0.2582	<0.0001
		Proportion Mediated	0.0134	0.0076	0.0218	0.4970
Pulse rate (n = 463,223)	Sometimes (n = 129,390)	Average Causal Mediation Effect	<0.0001	<0.0001	<0.0001	0.0611
		Average Direct Effect	0.0421	0.0080	0.0761	0.0154

(*continued on next page*)

Table 5 (*continued*)

Outcome	Group	Effect	Estimate	95% CIL	95% CIH	p-value
		Total Effect	0.0425	0.0084	0.0765	0.0145
		Proportion Mediated	0.0041	0.0002	0.0209	0.4780
		Average Causal Mediation Effect	0.0006	<0.0001	0.0011	0.0131
	Usually (n = 53,512)	Average Direct Effect	0.0895	0.0439	0.1351	<0.0001
		Total Effect	0.0902	0.0446	0.1358	<0.0001
		Proportion Mediated	0.0068	0.0019	0.0160	0.5180
		Average Causal Mediation Effect	0.0015	0.0005	0.0026	0.0095
	Always (n = 22,375)	Average Direct Effect	0.1908	0.1249	0.2568	<0.0001
		Total Effect	0.1922	0.1263	0.2582	<0.0001
		Proportion Mediated	0.0076	0.0022	0.0154	0.5080

Notably, the positive association between the frequency of adding salt to food and the risk of hearing loss was more pronounced among individuals without baseline diabetes or hypertension. These findings suggest that pharmacological therapies commonly prescribed for diabetes and hypertension, such as metformin, GLP-1 receptor agonists, ACE inhibitors, and calcium channel blockers, may confer vascular-protective effects that mitigate the detrimental impact of high sodium intake on cochlear integrity [48–50]. In diabetic individuals, preexisting microangiopathic changes may limit the marginal effect of additional sodium-related vascular stress [51]. Alternatively, these medications may improve endothelial function and reduce microvascular stress, thereby attenuating hearing loss risk [52,53]. Chronic hyperglycemia may also alter serum osmolality and electrolyte distribution [54], potentially obscuring the impact of dietary sodium. Further research stratified by treatment status, metabolic control, and inner ear-specific pathology is warranted.

Variation by sex, ethnicity, and socioeconomic status was also observed. The consistent increase in risk across all salt use categories in men may reflect greater susceptibility to sodium-related vascular or metabolic stress, while women showed significant risk only in the highest intake group, suggesting a possible threshold or protective modulation. These findings underscore the importance of considering sex as a biological variable in hearing loss research. Associations were stronger in White participants and in individuals with lower deprivation scores, whereas they were weaker and nonsignificant in non-White groups, possibly due to smaller sample sizes. Differences in diet quality, healthcare access, and social determinants of health [55,56] further highlight the need for culturally and contextually tailored dietary interventions. Participants who frequently added salt to food tended to have lower diet scores, reflecting less healthy dietary profiles. However, the association between salt use and hearing loss remained significant after adjustment for diet score and persisted across both low and high diet quality strata, supporting its independence from general dietary patterns.

From a clinical and public health perspective, these findings broaden the relevance of sodium reduction beyond cardiovascular health to sensory preservation. Hearing loss has often been considered an inevitable consequence of aging or noise exposure [57]. However, the identification of discretionary salt use as a modifiable factor highlights opportunities for prevention. At the individual level, clinicians in primary care, geriatrics, and audiology may incorporate sodium reduction counseling into routine care. At the population level, sodium reduction targets for food manufacturers, front-of-package labeling, and nationwide education campaigns may yield substantial benefits [58]. Incorporating auditory health into dietary and chronic disease surveillance systems may further enhance public health frameworks.

This study has several strengths, including its large sample size, prospective design, and use of both self-reported and biomarker-based sodium measures, which support internal validity and minimize recall bias. Nonetheless, some limitations remain. Hearing loss classification relied on ICD-10 codes, which may not fully capture subclinical or milder cases, although UK diagnoses typically involve audiometry [59]. While speech-in-noise tests provided additional standardized assessment [60], outcome misclassification is still possible. Urinary sodium was measured from spot rather than 24-h collections, potentially reducing precision. Finally, salt intake was assessed only at baseline, preventing evaluation of changes over time, and residual confounding cannot be excluded. Although salt intake frequency was measured at several time points, we used only baseline data, so changes over time were not captured and exposure misclassification may have attenuated associations.

Future studies should aim to replicate these findings in more diverse populations using longitudinal data that include repeated nutritional assessments and objective biomarkers. Studies incorporating markers such as serum osmolality, aldosterone, or vasopressin may clarify the physiological mechanisms that link sodium intake to auditory dysfunction. Interventional studies, ideally randomized controlled trials, are also needed to evaluate whether sodium reduction can prevent or slow the progression of hearing loss.

5. Conclusion

In conclusion, this study provides compelling evidence that frequent addition of salt to food is associated with an increased risk of incident hearing loss. These findings support the view that hearing loss, similar to hypertension and cardiovascular disease, may be influenced by modifiable dietary behaviors. Efforts to reduce sodium intake through individualized counseling and public health strategies may offer dual benefits by protecting both cardiovascular and auditory health. Integrating hearing preservation into clinical nutrition and chronic disease prevention frameworks represents an important and timely step toward addressing a growing and often overlooked public health concern.

This large-scale prospective study provides evidence that habitual addition of salt to food is associated with an increased risk of incident hearing loss in a dose-dependent manner. The findings highlight the potential role of salt intake as a modifiable lifestyle factor that may influence auditory function. Causal mediation analysis indicated that systemic low-grade inflammation, reflected by elevated GlycA and C-reactive protein, may partly explain the observed relationship. These results support the inclusion of auditory outcomes in dietary guidelines and public health strategies. Reducing discretionary salt use may offer benefits not only for cardiovascular and metabolic health but also for the preservation of hearing.

CRediT authorship contribution statement

YH conducted data analysis and contributed to manuscript drafting. KYL and IS contributed to study conceptualization and critical review of the manuscript. KYL also provided funding support. DJJ had primary responsibility for the final content. All authors read and approved the final manuscript.

Ethics approval and consent to participate

Ethical approval for the UK Biobank study is granted by the North West Multi-Centre Research Ethics Committee, with written informed

consent from all participants during the UK Biobank baseline recruitment. The current study received special approval from the UK Biobank, application number 146430.

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Availability of data materials

Access to the phenotypic and genotypic data of UK Biobank participants is governed by an application process via https://www.ukbiobank.ac.uk. These data are not publicly available due to data use restrictions and are accessible only under approved research applications. However, the data may be available from the authors upon reasonable request and with permission from the UK Biobank.

Declaration of competing interests

None declared

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Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at https://doi.org/10.1016/j.jnha.2025.100663.

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