Original research

Hearing impairment, psychological distress, and incident heart failure: a prospective cohort study

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ABSTRACT

Background The relationship between objectively measured hearing ability and the risk of incident heart failure (HF) remains unclear. This study aimed to assess this association, explore potential modifying factors, and examine whether psychological factors mediate this relationship.

Methods We included 164 431 participants from the UK Biobank without HF at baseline. Speech-in-noise hearing ability was measured using the Digit Triplets Test and quantified by the speech-reception-threshold (SRT). Incident HF was identified through hospital admission and death records. Mediation analyses assessed the role of social isolation, psychological distress, and neuroticism.

Results Over a median follow-up of 11.7 years, 4449 (2.7%) participants developed incident HF. Higher SRT levels were associated with an increased risk of HF (adjusted HR per SD increment 1.05, 95% CI 1.02 to 1.08). Compared with those with normal hearing, participants with insufficient hearing, poor hearing, or hearing aid use had higher HF risks (adjusted HRs 1.15, 1.28, and 1.26, respectively). Psychological distress mediated 16.9% of the association between SRT levels and HF, while social isolation and neuroticism mediated 3.0% and 3.1%, respectively. The association was stronger in participants without coronary heart disease or stroke at baseline.

Conclusions Poor hearing ability is associated with an increased risk of incident HF, with psychological distress playing a notable mediating role. These findings suggest that hearing health and psychological well-being should be considered in cardiovascular risk assessment and prevention strategies.

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ Hearing impairment is associated with an increased risk of cardiovascular disease and mortality.
⇒ Hearing problems can lead to social detachment and impaired psychosocial well-being, which may contribute to cardiovascular risk.

WHAT THIS STUDY ADDS

⇒ Poor hearing ability, measured objectively, is associated with a higher risk of incident heart failure (HF) in the general population.
⇒ Psychological factors, particularly psychological distress, mediate a substantial proportion (16.9%) of the association between hearing impairment and HF risk.
⇒ The association between hearing impairment and HF is more pronounced in individuals without pre-existing coronary heart disease or stroke.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

⇒ Hearing impairment may serve as a potential marker for HF risk, highlighting the importance of integrating hearing health assessments into cardiovascular risk evaluation frameworks.

INTRODUCTION

Heart failure (HF) is a multi-faceted, life-threatening syndrome characterised by high morbidity and mortality, poor quality of life and high costs, affecting more than 64 million people worldwide and showing an alarming growth trend.[1] Therefore, early identification of modifiable risk factors to enhance primary prevention is an important public health issue to reduce the disease and economic burden of HF.

Hearing impairment is an increasingly common health problem that increases in prevalence with age,[2] leading to communication difficulties. Several studies have suggested that hearing problems may predict the development of cardiovascular disease and cardiovascular mortality.[3 4] Moreover, a recent study found that hearing loss was associated with a higher risk of HF.[5] However, the definition of hearing loss in this study was based on an algorithm that has not been validated by independent criteria such as audiology tests. To date, no study has comprehensively examined the association between hearing ability qualified by validated hearing tests and the risk of incident HF. In addition, prior studies have shown that the adverse impact of hearing problems on communication may lead to social detachment and thereby impair psychosocial well-being,[6 7] which is also an important risk factor or driver of HF risk.[8 9] Therefore, we hypothesise that psychosocial factors may partly mediate the relationship between hearing levels and the risk of incident HF. However, no studies have examined this hypothesis.

To address these knowledge gaps, using data from the UK Biobank, we aimed to assess the association between objectively measured hearing ability and incident HF, explore potential modifying factors,

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and examine whether social isolation, psychological distress and neuroticism mediate this association.

METHODS

Study population

UK Biobank is a large-scale prospective cohort study consisting of more than half one million adults recruited from 22 assessment centres across England, Wales and Scotland between 2006 and 2010. Participants completed touch screen questionnaires and verbal interviews, underwent a series of anthropometric measurements and provided biological samples for biomarker analysis. Details of the study's design and data collection procedures have been described elsewhere.[10]

Our current study included 165 500 participants who had completed Digit Triplet Test (DTT) data in the UK Biobank. After further excluding those who had a history of HF (self-reported HF or medical records for HF) at baseline (n=983) or lacked hearing aid usage data (n=86), 160 062 participants without hearing aid usage and 4369 with hearing aid usage were included in the final analyses (online supplemental figure 1).

Speech-in-noise hearing ability in the UK Biobank was measured by DTT and quantified using the speech-reception-threshold (SRT).[11] The English speech materials for the UK Biobank DTT were developed at the University of Southampton and have been described elsewhere.[12] Fifteen sets of three monosyllabic digits (eg, 2-3-7) were presented against background noise via circumaural headphones (Sennheiser HD-25, Wedemark, Germany). The background noise matched the spectrum of the speech stimuli, and the noise level varied adaptively after each triplet. SRT is defined as the signal-to-noise ratio (SNR) at which half of the presented speech can be correctly understood, and a higher score corresponds to a worse performance. In analyses, we used the SRT of the better hearing ear for each participant, and if the SRT was only available for one ear, we assumed that it was the better one. Participants without hearing aid usage were categorised into three groups according to their performance on the DTT: (1) normal (SRT < -5.5 dB); (2) insufficient (SRT ≥ -5.5 and ≤ -3.5 dB); and (3) poor (SRT > -3.5 dB) hearing status.[13]

Study outcome

The study outcome was incident HF, identified using linkage to hospital admission and death register data, according to the International Classification of Diseases, ninth revision codes (ICD-9) of 4280, 4281, and 4289, and 10th revision codes (ICD-10) of I11.0, I13.0, I13.2, I50.0, I50.1, and I50.9 (online supplemental table 1).[14] The follow-up time was calculated from the recruitment date to the date of the first diagnosis of HF, death, loss to follow-up, or end of follow-up, whichever occurred first.

Assessment of covariates

Information for each participant about sociodemographic characteristics, lifestyles, dietary intakes and medical history was collected by a touchscreen questionnaire at baseline, including age, sex, Townsend Deprivation Index (TDI), race, education levels, smoking status, alcohol consumption status, physical activity status, sedentary time, diabetes, hypertension, coronary heart disease, stroke, cholesterol-lowering drugs, and hearing aid usage.

Body mass index (BMI) was calculated as weight divided by height squared (kg/m^2). Optimal physical activity was defined as at least 150 min/week moderate intensity or 75 min/week vigorous intensity activity, or a combination of both, according to global recommendations on physical activity for cardiovascular health.[15] Prevalent diabetes was identified as a history of diabetes, medication for diabetes, or glycated haemoglobin (HbA1c) ≥6.5%.[16] Hypertension status at baseline was defined as a systolic blood pressure ≥140 mm Hg, a diastolic blood pressure ≥90 mm Hg, self-reported hypertension history, medication for hypertension, and/or medical records for hypertension. Coronary heart disease and stroke were defined as self-reported history and medical records. Low-density lipoprotein cholesterol (LDL-C; mmol/L) and C-reactive protein (CRP; mg/L) were measured by enzymatic protective selection analysis and immunoturbidimetric-high sensitivity analysis, respectively, on a Beckman Coulter AU5800.

Information on psychosocial factors assessment (social isolation, psychological distress and neuroticism) was collected via touchscreen questionnaires. Social isolation was assessed using a composite definition in the UK Biobank derived from three questions: number of people living in household (1 point for living alone), frequency of friend or family visits (1 point for friends and family visit less than once a month), and attendance of leisure or social activities (1 point for no participation in social activities at least weekly). Thus, participants could score a total of 0–3, with a score of 2 or 3 being classified as having social isolation, and 0 or 1 being classified as having no social isolation.[17] Psychological distress was assessed using a four-item version of the Patient Health Questionnaire (PHQ-4), which has been validated for the measure of depression and anxiety elsewhere.[18] Each item is rated on a 4-point Likert scale, with a score of 0 (not at all), 1 (several days), 2 (more than half the days), or 3 (nearly every day). Therefore, the total score of psychological distress ranges from 0 to 12. Neuroticism, a depression-related personality trait, was assessed using 12 questions from the Eysenck Personality Questionnaire-Revised Short Form, which has been highly validated.[19] Participants who answered 'yes' scored 1 point, which is summed to produce a neuroticism total score ranging from 0 to 12. Detailed questions for social isolation, psychological distress and neuroticism are listed in online supplemental table 2.

Genetic risk score of HF

Detailed information about genotyping, imputation, and quality control in the UK Biobank study has been described in previous studies.[20] A total of 12 single nucleotide polymorphisms (SNPs) (online supplemental table 3), which showed independently significant genome-wide association with HF based on recent studies,[14] were selected to calculate a genetic risk score (GRS) of HF for each individual using a weighted method. A higher score indicates a higher genetic predisposition to HF.

Statistical analyses

Baseline characteristics of participants were presented as mean (SD) for continuous variables and number (%) for categorical variables. χ^2 tests (for categorical variables) or analysis of variance (ANOVA) tests (for continuous variables) were used to compare differences in baseline characteristics according to hearing status (normal, insufficient, poor and hearing aid usage).

The cumulative event rates of incident HF according to hearing status were estimated using the Kaplan-Meier method. The crude and adjusted hazard ratios (HR) and their 95% confidence intervals (95% CI) were estimated by Cox proportional hazard regression models to investigate the association of baseline SRT levels (in participants without hearing aid usage) and hearing status with incident HF. Potential confounders that were

identified as traditional or suspected risk factors for incident HF were selected as the covariates in multivariate models. Model 1 was adjusted for age and sex. Model 2 was additionally adjusted for BMI, TDI, race, education levels, smoking status, alcohol consumption status, optimal physical activity, CRP, LDL-C, hypertension, diabetes, and use of cholesterol-lowering drugs. If the proportion of missing data of covariates was <1%, the missing data of categorical variables were encoded with the reference group, and the missing data of continuous variables were encoded with the mean value. If the proportion of missing data for the covariates was ≥1%, including CRP (6.9%), LDL-C (6.9%), and optimal physical activity (18.2%), a separate missing category was created. Potential mediators were not included in the adjusted models to avoid overadjustment. Schoenfeld residuals testing was used to assess the proportional hazards assumption. If any covariates violated the proportional hazards assumption, a stratified Cox model was constructed to determine whether these covariates significantly affected the observed findings.

Stratified analyses were performed to assess the possible effect modifiers on the association of SRT levels (per SD increment) and incident HF in participants without hearing aid usage, according to age, sex, BMI, CRP, LDL-C, diabetes, hypertension, coronary heart disease, stroke, and HF genetic risk score. Interactions between SRT levels and subgroups were evaluated by likelihood ratio testing.

Mediation analyses were performed to explore the potential mechanisms driven by psychosocial factors in the association between SRT levels and incident HF in participants without hearing aid usage. Three models were estimated with adjustments for the same covariates included in model 2. First, multivariate linear regression models were used to assess the association between SRT levels and the three mediators. Second, multivariate Weibull regression models were used for the association of SRT levels and the mediators with incident HF. Third, the mediation models were conducted to estimate the mediation effects of the three mediators by combining the linear regression model and the survival regression model using the R package *mediation*. To quantify the magnitude of mediation, the mediation proportions were estimated.

To test the robustness of the findings, several sensitivity analyses were performed. First, we repeated the analyses by excluding participants who developed incident HF within the first 2 years of follow-up. Second, the GRS of HF, psychosocial mediators and sedentary time were further adjusted, respectively.

All statistical analyses were performed using R 4.1.1 software, and the two-sided p value <0.05 was considered to be statistically significant.

RESULTS

Baseline characteristics of the participants

Of the 164 431 participants included (online supplemental figure 1), the mean (SD) age was 56.7 (8.1) years and 89 818 (54.6%) were women. Of the 160 062 participants without hearing aid usage, the mean (SD) SRT was −7.4 (1.7) dB, and there were 140 839 people with normal hearing (88.0%), 16 759 people with insufficient hearing (10.5%), and 2464 people with

Table 1 Baseline characteristics of participants by hearing status (normal, insufficient, poor hearing and hearing aid usage)*

Characteristics	Hearing status				P value
	Normal (SRT < −5.5 dB)	Insufficient (SRT ≥ −5.5 and ≤ −3.5 dB)	Poor (SRT > −3.5 dB)	Hearing aid usage	
N	140 839	16 759	2464	4369	
Age, year	56.12 (8.13)	59.70 (7.51)	60.03 (7.62)	62.02 (6.33)	<0.001
Female, n (%)	77 365 (54.9)	9291 (55.4)	1210 (49.1)	1952 (44.7)	<0.001
BMI, kg/m^2	27.34 (4.80)	27.69 (4.91)	28.09 (4.98)	28.13 (4.86)	<0.001
TDI	−1.22 (2.89)	−0.66 (3.13)	0.14 (3.38)	−1.13 (2.95)	<0.001
White, n (%)	130 660 (93.1)	13 911 (83.5)	1775 (72.6)	4173 (96.0)	<0.001
College or university degree	49 579 (35.5)	4689 (28.5)	513 (21.5)	1121 (26.0)	<0.001
Smoking status, n (%)					
Never	77 998 (55.6)	9126 (54.8)	1368 (56.1)	2075 (47.7)	<0.001
Previous	48 407 (34.5)	5699 (34.2)	783 (32.1)	1910 (43.9)	
Current	14 001 (10.0)	1829 (11.0)	289 (11.8)	363 (8.3)	
Alcohol consumption, n (%)					
Never or special occasions only	26 926 (19.1)	4941 (29.5)	972 (39.5)	1019 (23.4)	<0.001
1 time/month – 4 times/week	84 504 (60.0)	8789 (52.5)	1139 (46.3)	2453 (56.2)	
Daily or almost daily	29 310 (20.8)	3006 (18.0)	347 (14.1)	892 (20.4)	
Optimal physical activity, n (%)	64 725 (55.8)	7407 (56.6)	1032 (56.6)	1920 (55.3)	0.215
LDL-C, mmol/L	3.56 (0.86)	3.50 (0.89)	3.44 (0.91)	3.47 (0.90)	<0.001
CRP, mg/L	2.48 (4.22)	2.83 (4.60)	3.12 (4.85)	2.91 (4.45)	<0.001
Hypertension, n (%)	75 511 (53.8)	10 642 (63.9)	1667 (68.5)	2922 (67.3)	<0.001
Diabetes, n (%)	8108 (5.8)	1619 (9.7)	341 (13.8)	447 (10.2)	<0.001
Coronary heart disease, n (%)	6465 (4.6)	1266 (7.6)	235 (9.6)	482 (11.1)	<0.001
Stroke, n (%)	2079 (1.5)	425 (2.5)	90 (3.7)	141 (3.2)	<0.001
Cholesterol-lowering drugs, n (%)	23 513 (16.8)	4131 (24.9)	715 (29.7)	1354 (31.3)	<0.001
Social isolation, n (%)	12 851 (9.2)	1853 (11.4)	330 (14.1)	401 (9.4)	<0.001
Psychological distress	1.58 (2.09)	1.77 (2.33)	2.25 (2.73)	1.74 (2.29)	<0.001
Neuroticism	4.09 (3.26)	4.13 (3.34)	4.43 (3.44)	4.24 (3.30)	<0.001

*Data are presented as mean (SD) for continuous variables or n (%) for categorical variables.
BMI, body mass index; CRP, C-reactive protein; LDL-C, low-density lipoprotein cholesterol; SRT, speech-reception-threshold; TDI, Townsend Deprivation Index.

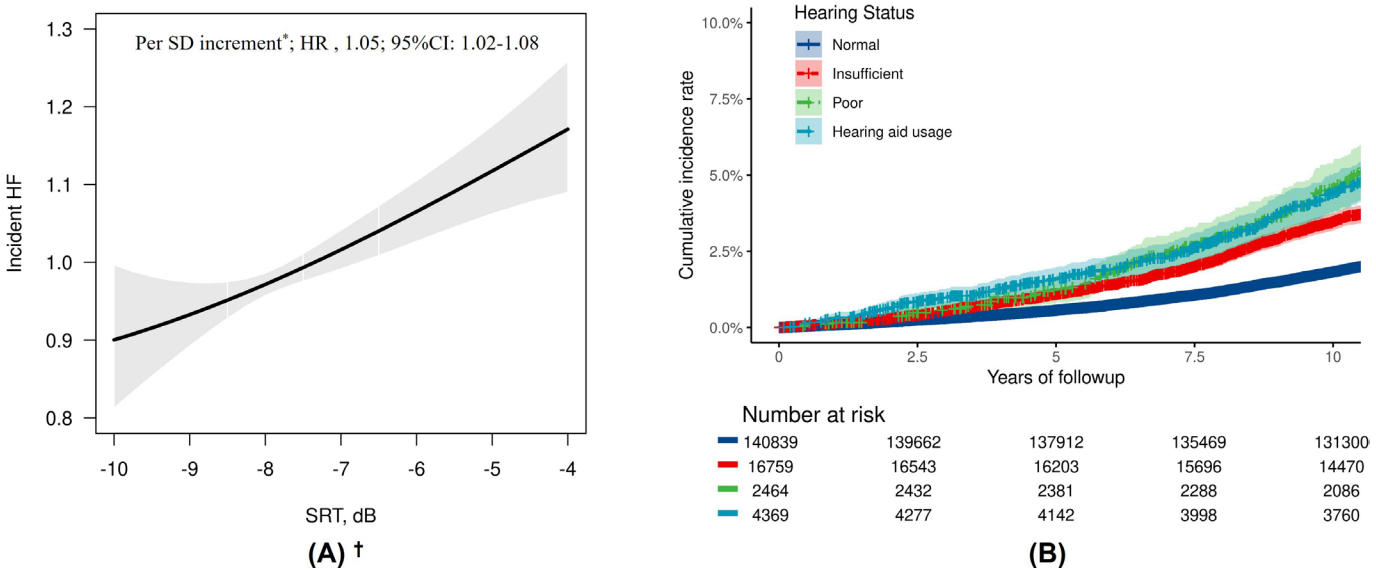

Figure 1 Association of speech-reception-threshold (SRT) levels (A) and hearing status (normal, insufficient, poor hearing and hearing aid usage) (B) with incident heart failure. *Adjusted for age, sex, body mass index, Townsend Deprivation Index, race, education, smoking status, alcohol drinking status, low-density lipoprotein cholesterol, C-reactive protein, optimal physical activity, hypertension, diabetes, and use of cholesterol-lowering drugs. †This analysis was limited to those without hearing aid usage.

poor hearing (1.5%). Participants with poor hearing status or using hearing aids were older, had higher levels of BMI and CRP, lower levels of education and LDL-C, higher prevalence of hypertension, diabetes, coronary heart disease, stroke and neuroticism, and higher frequency of using cholesterol-lowering drugs (table 1).

Association between hearing ability and incident HF

During a median follow-up of 11.7 years, 4449 (2.7%) participants developed incident HF. There was a significant positive association between SRT levels and the risk of incident HF (per SD increment; adjusted HR 1.05, 95% CI 1.02 to 1.08) in participants without hearing aid usage (figure 1A). Compared with those with normal hearing status, the adjusted HR (95% CI) of incident HF for insufficient hearing status, poor hearing status and using hearing aid were 1.15 (1.06 to 1.25), 1.28 (1.08 to 1.52) and 1.26 (1.11 to 1.43), respectively (table 2, figure 1B). Excluding participants who developed incident HF in the first 2 years of follow-up (online supplemental table 4), or further adjusting the HF GRS, psychosocial mediators, or sedentary time (online supplemental table 5), did not significantly change the results.

The Schoenfeld residuals testing revealed that certain covariates violated the proportional hazards assumption. Nevertheless, in stratified Cox models, the results were essentially the same as those observed from the non-stratified model (online supplemental table 6).

Mediation analyses

As shown in online supplemental figure 2, SRT levels were significantly positively associated with social isolation (β=0.007, p<0.001), psychological distress (β=0.053, p<0.001), and neuroticism (β=0.045, p<0.001) in participants without hearing aid usage. Social isolation, psychological distress and neuroticism significantly mediated the association between SRT levels and incident HF in participants without hearing aid usage, with mediating proportions of 3.0% (95% CI 1.5% to 7.0%), 16.9% (95% CI 10.0% to 52.0%) and 3.1% (95% CI 1.4% to 8.0%), respectively (table 3).

Table 2 Association of speech-reception-threshold levels, hearing status (normal, insufficient, poor hearing and hearing aid usage) with incident heart failure

			Model 1*		Model 2†	
	N	Cases (%)	HR (95% CI)	P value	HR (95% CI)	P value
SRT, continuous, per SD increment‡	160 062	4200 (2.6)	1.12 (1.09 to 1.15)	<0.001	1.05 (1.02 to 1.08)	0.001
Category						
Normal (SRT < −5.5 dB)	140 839	3361 (2.4)	Ref		Ref	
Insufficient (SRT ≥ −5.5 and ≤ −3.5 dB)	16 759	695 (4.1)	1.32 (1.22 to 1.44)	<0.001	1.15 (1.06 to 1.25)	0.001
Poor (SRT > −3.5 dB)	2464	144 (5.8)	1.75 (1.48 to 2.07)	<0.001	1.28 (1.08 to 1.52)	0.004
Hearing aid usage	4369	249 (5.7)	1.43 (1.26 to 1.63)	<0.001	1.26 (1.11 to 1.43)	0.001

*Model 1 was adjusted for age and sex.
†Model 2 was adjusted for age, sex, body mass index, Townsend Deprivation Index, race, education, smoking status, alcohol drinking status, low-density lipoprotein cholesterol, C-reactive protein, optimal physical activity, hypertension, diabetes, and use of cholesterol-lowering drugs.
‡This analysis was limited to those without hearing aid usage.
SRT, speech-reception-threshold .

Table 3 Mediation analysis of social isolation, psychological distress and neuroticism depressed mood in the association of speech-reception-threshold* levels and incident heart failure in participants without hearing aid usage†

Mediators	Proportions, % (95% CI)	P value
Social isolation	3.0 (1.5 to 7.0)	<0.001
Psychological distress	16.9 (10.0 to 52.0)	0.004
Neuroticism	3.1 (1.4 to 8.0)	<0.001

*This analysis was limited to those without hearing aid usage.
†Adjusted for age, sex, body mass index, Townsend Deprivation Index, race, education, smoking status, alcohol drinking status, low-density lipoprotein cholesterol, C-reactive protein, optimal moderate/vigorous physical activity, hypertension, diabetes, and use of cholesterol-lowering drugs.

Among participants without any missing data for social isolation, psychological distress, and neuroticism, when the three factors were combined, with a total score ranging from 0 to 27 (including 0–3 scores of social isolation, 0–12 scores of psychological distress, and 0–12 scores of neuroticism), the combined mediating effect was 9.3%, which was less than the sum of the individual mediating effects (19.4%) (online supplemental table 7); this suggests that there is overlap and interaction among the three mediators—social isolation, psychological distress, and neuroticism. As expected, there were significant correlations between any two of the three mediators. Among them, the Spearman correlation coefficient between psychological distress and neuroticism reached 0.554 (online supplemental table 8).

Stratified analyses

The association of SRT levels with incident HF was more pronounced in those without coronary heart disease (adjusted HR 1.07, 95% CI 1.04 to 1.10 vs participants with coronary heart disease, adjusted HR 0.99, 95% CI 0.94 to 1.04; p for interaction=0.006) or stroke (adjusted HR 1.06, 95% CI 1.03 to 1.08 vs participants with stroke, adjusted HR 0.93, 95% CI 0.84 to 1.03; p for interaction=0.009) at baseline in participants without hearing aid usage. Other variables, including age, sex, BMI, CRP, LDL-C, diabetes, hypertension, and GRS of HF did not significantly modify the association between SRT levels and incident HF (all p for interaction >0.05 (figure 2).

DISCUSSION

Our study demonstrated a significant positive association between SRT levels and incident HF, suggesting that the speech-in-noise hearing ability was negatively associated with the risk of incident HF. Psychological factors, especially psychological distress, play a significant mediating role in the association between SRT levels

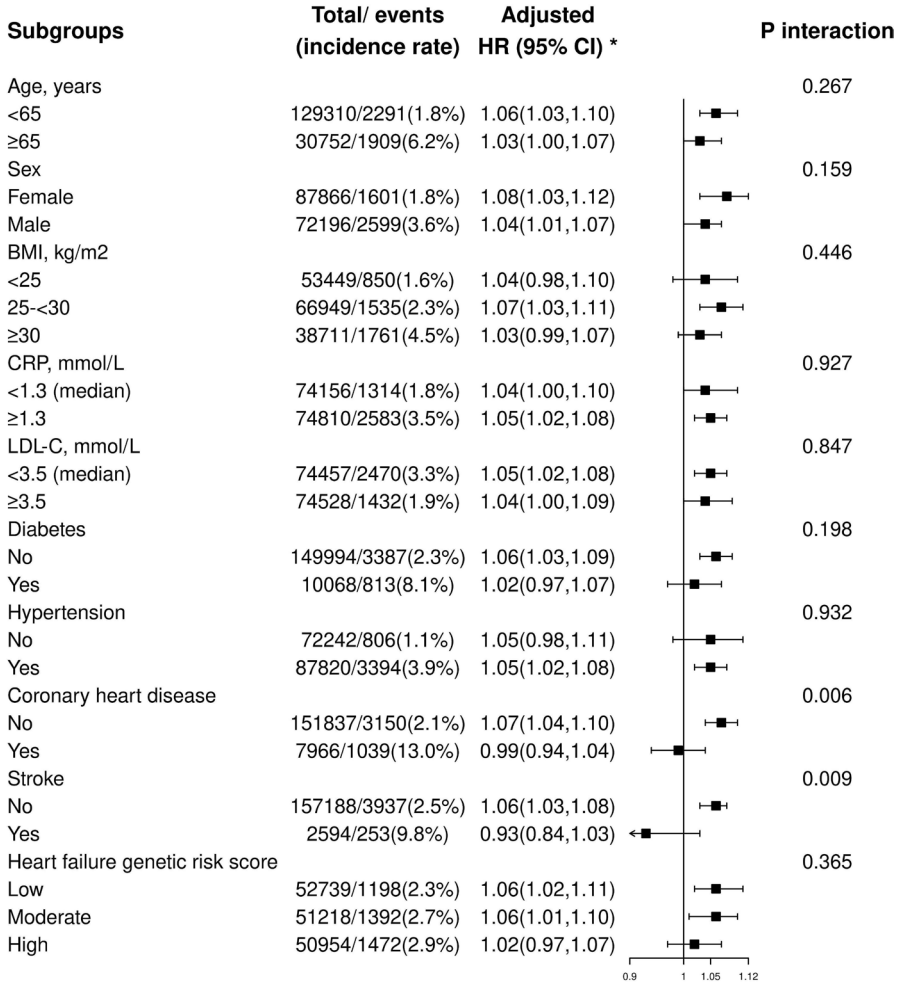

Figure 2 Subgroup analyses of speech-reception-threshold (SRT) levels (per SD increment†) and incident heart failure in participants without hearing aid usage. *Adjusted for age, sex, body mass index (BMI), Townsend Deprivation Index, race, education, smoking status, alcohol drinking status, low-density lipoprotein cholesterol (LDL-C), C-reactive protein (CRP), optimal physical activity, hypertension, diabetes, and use of cholesterol-lowering drugs. †This analysis was limited to those without hearing aid usage.

and incident HF. Moreover, the positive association of SRT levels with incident HF was more pronounced in those without coronary heart disease or stroke at baseline.

Only one previous population-based retrospective cohort study[5] found that hearing loss was associated with a higher risk for HF. However, hearing loss was only defined using an algorithm that has not been validated against an independent standard such as audiological testing in this study. Therefore, the association between objectively measured hearing ability and the risk of incident HF remains uncertain. Our study, with a large sample size and objective and validated audiological speech-in-noise tests, addresses the above knowledge gap in a timely manner.

Our study provides some new insights. First, there was a negative association between speech-in-noise hearing ability and the risk of incident HF. Our findings are biologically plausible. The rich distribution of capillaries in the stria vascularis of the cochlea and the high metabolic demand of the inner ear[21] may render these regions more sensitive to systemic vascular disorders rather than just local circulatory issues. Therefore, hearing impairment may reflect vascular health and serve as an early and sensitive predictor of cardiovascular disease, including HF.[3] Of note, both the participants who used hearing aids and those with poor hearing status had a similarly significant increase in the risk of incident HF, suggesting that while hearing aids can improve auditory function, they may not address the underlying vascular issues that contribute to the risk of HF. Second, we found that the positive association between poor hearing status and incident HF was significantly mediated by psychological factors. Because hearing problems can lead to difficulties in speech comprehension and poor engagement in social activities,[22 23] people with hearing impairment are more likely to experience social isolation, psychological distress, anxiety and depression[7 24 25] than people without hearing impairment. These psychological factors may increase the activity of the sympathetic nervous system and the hypothalamic-pituitary-adrenal axis, and enhance inflammation and oxidative stress, thereby accelerating atherosclerosis, increasing peripheral stress, and promoting the development of cardiac remodelling in the pre-HF period,[26 27] thus significantly increasing the risk of HF.[8 9] Our findings suggest that psychosocial factors may be an important link between hearing impairment and HF risk, as well as a possible intervention target for preventing HF risk in people with hearing loss.

The association of hearing impairment with incident HF was more pronounced in those without coronary heart disease or stroke at baseline. Consistent with previous studies,[28 29] our study also observed a significantly higher risk of HF in patients with coronary heart disease or stroke than those without, which may offset or mask the adverse effects of hearing impairment on the risk of incident HF. This finding highlights the importance of assessing hearing ability in the general population without coronary heart disease or stroke for early detection of those at high risk of cardiovascular diseases (especially HF) and strengthening primary prevention. Future studies are needed to confirm our findings and further explore the underlying mechanisms.

Our study has several limitations. First, due to the nature of observational studies, residual confounding bias cannot be completely ruled out even though many important covariates have been adjusted. Second, data on hearing status were collected only at baseline; more frequent measurements can provide more information. Finally, the participants in the current study were mainly of European descent and healthier than the UK general population,[30] limiting the generalizability of the results to other populations. Overall, it is necessary to confirm our findings further in more studies.

CONCLUSION

We have been the first to demonstrate that poor hearing ability is significantly associated with a higher risk of incident HF in the general population. Psychological factors, especially psychological distress, play a significant mediating role in this association. If further confirmed, hearing impairment may be a potential risk factor or marker for incident HF in the general population, highlighting the importance of integrating hearing health assessments into broader cardiovascular risk evaluation frameworks. Moreover, strengthening psychological intervention in people with hearing impairment may be an important path and strategy to reduce the risk of HF.

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Contributors YH and XQ designed and conducted the research; YH, YaZ and ZY performed the data management and statistical analyses; YH and XQ wrote the manuscript. All authors reviewed/edited the manuscript for important intellectual content. All authors read and approved the final manuscript. XQ is the guarantor.

Competing interests None declared.

Patient and public involvement Patients and/or the public were involved in the design, or conduct, or reporting, or dissemination plans of this research. Refer to the Methods section for further details.

Patient consent for publication Not applicable.

Ethics approval This study involves human participants. The UK Biobank was approved by the North West Research Ethics Committee (11/NW/0382). All participants gave written consent at enrolment to participate in the study before taking part.

Provenance and peer review Not commissioned; externally peer reviewed.

Data availability statement Data are available in a public, open access repository. The UK Biobank data are available on application to the UK Biobank.

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