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Jackson Heart Study: Aggregate cardiovascular disease risk and auditory profiles

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Abstract

Objectives: Evaluate the relationship between cardiovascular disease (CVD) risk factors and cochlear function in African Americans.

Methods: Relationships between hearing loss, cochlear function, and CVD risk factors were assessed in a cross-sectional analysis of 1106 Jackson Heart Study participants. Hearing loss was defined as puretone average ($PTA_{0.5,1,2,4}$) > 15 dB HL. Distortion product otoacoustic emissions (DPOAEs) were collected for $f_2 = 1.0$–8.0 kHz. Two amplitude averages were computed: $DPOAE_{low}$ ($f_2 \leq 4$ kHz) and $DPOAE_{high}$ ($f_2 \geq 6$ kHz). Based on major CVD risk factors (diabetes, current smoking, total cholesterol ≥240 mg/dL or treatment, and systolic blood pressure [BP]/diastolic BP ≥ 140/≥90 mmHg or treatment), four risk groups were created: 0, 1, 2, and ≥3 risk factors. Logistic regression estimated the odds of hearing loss and absent/reduced $DPOAE_{low}$ and $DPOAE_{high}$ by CVD risk status adjusting for age, sex, education, BMI, vertigo, and noise exposure.

Results: With multivariable adjustment, diabetes was associated with hearing loss (OR = 1.48 [95% CI: 1.04–2.10]). However, there was not a statistically significant relationship between CVD risk factors (individually or for overall risk) and DPOAEs.

Conclusion: Diabetes was associated with hearing loss. Neither individual CVD risk factors nor overall risk showed a relationship to cochlear dysfunction.

Level of Evidence: 2b.

KEYWORDS
audiology, cardiovascular disease risk factors, distortion product otoacoustic emissions, hearing loss, Jackson Heart Study, otology, sensorineural hearing loss

1 | INTRODUCTION

Hearing loss is a prevalent chronic condition that poses a major public health concern. Among persons in the United States aged ≥12 years, ~38 million are estimated to have hearing loss[1] and by 2040, ~63 million adults (≥20 years) are projected to have hearing loss.[2] Depression,[3] social isolation,[4] accelerated cognitive decline,[5,6] and increased fall risk[7] have been independently associated with hearing

loss hence understanding its risk factors is of public health and clinical significance.

Medical comorbidities including hypertension[8,9] and diabetes[10–13] have significant relationships to hearing loss, as have modifiable behaviors such as tobacco smoking.[14–18] Epidemiological studies have explored associations with various cardiovascular disease (CVD) risk metrics, which are used to determine overall risk factor load based on the status of multiple CVD risk factors. These studies have yielded mixed results. For example, metabolic syndrome was associated with hearing loss in the National Health and Nutrition Examination Survey (NHANES)[19] but not in the Korean NHANES.[20] Tan et al. reported a graded association between Framingham Risk Score and hearing loss in the Busselton Healthy Ageing Study.[21] The Epidemiology of Hearing Loss Study reported that a history of myocardial infarction was associated with cochlear (inner ear) impairment in older women.[22] That study assessed cochlear integrity with otoacoustic emissions (OAEs), low-level sounds produced by healthy cochleae that provide a barometer of auditory health independent of the behavioral audiogram. However, research on the association between CVD risk status and cochlear function remains limited, especially at the population level.

Current understanding of the relationship between CVD risk and auditory function is derived primarily from epidemiological studies which, historically, have predominantly enrolled non-Hispanic white participants. Results of these studies might not be generalizable to other races/ethnicities, particularly those with disproportionately high CVD risk factor burden. The African American population, compared with whites of European ancestry, have higher prevalence of CVD risk factors including obesity, diabetes, and hypertension.[23,24] Comorbidity of these risk factors is also more common in African Americans,[25] which increases overall CVD risk.

Because studies show that the African American population has a high CVD risk factor load, exploring these risk factors in the context of auditory function is of importance to clinicians and epidemiologists interested in supporting healthy hearing. The Jackson Heart Study (JHS) is a prospective, population-based, longitudinal study designed to explore factors related to the progression of CVD exclusively in African Americans.[26] In contrast to most population-based studies, audiological assessment in the JHS includes OAEs. To date, three JHS studies have assessed the relation between auditory status and CVD risk. First, in 2018, Sorrel et al.[27] identified a positive association between hearing loss (PTA >25 dB HL) and 10-year risk of atherosclerotic CVD in 1107 JHS participants. A subsequent study[28] used the same 10-year CVD risk metric and examined the relationship to cochlear health using distortion product (DP) OAEs. When restricted to a subgroup of participants with normal hearing (threshold ≤ 25 dB HL), those with the highest atherosclerotic CVD risk had poorer cochlear function than participants with the lowest risk. The third JHS study assessed the correlation between hearing loss and Life's Simple 7, a metric of overall cardiovascular health based on seven risk factors, finding that better hearing was associated with higher (healthier) Life's Simple 7 scores.[29]

Despite these studies, our understanding of cochlear health in the context of CVD risk factors remains limited in the African American population. There is a dearth of research on auditory health in African Americans, especially in relation to CVD condition. To date, there is only one published report of cochlear dysfunction (as measured by DPOAEs) and CVD risk in the JHS.[28] That study evaluated the relationship between DPOAEs and stroke risk but did not examine relationships to individual CVD risk factors (e.g., diabetes). Herein, we report on the relationship between cochlear integrity and CVD risk in the JHS cohort. Our study builds upon past work by examining independent associations with four prevalent CVD risk factors and uses a CVD risk metric distinctive from earlier JHS reports. The specific goals of this study were to assess relationships between cochlear function and (1) four individual CVD risk factors (diabetes, smoking, hypertension, and high cholesterol) and (2) overall CVD risk factor burden. We hypothesized that persons with the highest CVD risk burden (i.e., ≥3 risk factors) would have poorer cochlear health than those who were not high risk.

2 | MATERIALS AND METHODS

2.1 | Study sample

We report associations between CVD risk factors and auditory status (hearing sensitivity and cochlear function) among JHS participants. Between 2000 and 2004, the JHS enrolled 5306 individuals aged 21–94 years from Jackson, Mississippi.[30] Health and demographic information were collected during home interviews. Clinical data were during examinations in (1) 2000–2004; (2) 2005–2008; and (3) 2009–2013. Data analyzed for this study were obtained from participants who were evaluated at Exam 2 and underwent audiological evaluation in an ancillary study coinciding with Exam 2. The study protocol was approved by the Institutional Review Boards of Jackson State University, Tougaloo College, and the University of Mississippi Medical Center. Participants provided written informed consent.

2.2 | Audiological assessment

Participants underwent otoscopy, tympanometry, air and bone conduction puretone audiometry, and DPOAE testing. Details regarding these assessments can be found in earlier reports.[27,28,31] Briefly, tympanometry was performed using a Madsen Capella tympanometer (GN Otometrics).[32] Audiometry was performed from 0.25 to 8 kHz (air) and 0.5 to 4 kHz (bone) using a Madsen Conera audiometer (GN Otometrics) equipped with insert earphones (ER-3A, GN Otometrics, Denmark). Ear-specific puretone averages were computed based on thresholds at 0.5, 1.0, 2.0, and 4.0 kHz (hereafter $PTA_{0.5,1,2,4}$). Dependent variable hearing loss was defined as $PTA_{0.5,1,2,4}$ > 15 dB HL in the worse ear. This cutoff allowed us to capture cases of slight hearing loss.

Bilateral DPOAE evaluation was performed with a Madsen Capella instrument (Natus Medical, Tasstrup, Denmark) using stimulus parameters of $L_1/L_2 = 60/50$ dB SPL, $f_2/f_1 = 1.22$, f_2 frequencies of 1.0, 1.5, 2.0, 3.0, 4.0, 6.0, and 8.0 kHz, and a 32 kHz sampling rate. Frames were rejected if single-frame noise level exceeded 30 dB SPL. For additional detail, see Sorrel et al.[28] For this analysis, data were excluded on a frequency-specific basis if the noise floor exceeded 0 dB SPL. Two DPOAE amplitude averages were computed: low-frequency ($f_2 \leq 4$ kHz; $DPOAE_{low}$) and high-frequency ($f_2 \geq 6$ kHz; $DPOAE_{high}$). Participants were included in analysis if they had ≥2 data points per frequency bin. DPOAE amplitude status was defined as follows: (1) present and normal (hereafter, "normal;" amplitude >0 dB SPL and signal-to-noise ratio [SNR] ≥ 6 dB); (2) reduced (amplitude ≤0 but >−20 dB SPL and SNR ≥ 6 dB); (3) absent (amplitude <−20 dB SPL or SNR < 6 dB). For statistical analysis, the reduced and absent categories were collapsed so that comparisons were made between normal versus absent/reduced DPOAEs. Analysis was conducted separately for better and worse ears (defined by $PTA_{0.5,1,2,4}$).

2.3 | Determination of covariate and CVD risk factor status

Independent variables included age, sex, blood pressure (BP), diabetes, smoking status (obtained at Exam 1), and medication use (antihypertensives and statins). These data were obtained via home interviews and during clinical examinations. There were three education levels (<high school, high school/GED, and >high school). Body mass index (BMI) was calculated in kilograms per meters squared (kg/m^2). Fasting blood samples were collected and analyzed as per Carpenter et al.[33]

Definition of CVD risk status has been previously published.[34,35] Briefly, CVD risk strata were defined based on optimal, elevated/moderate, and high-risk levels of four major CVD risk factors. BP level was categorized as optimal (untreated systolic [SBP] < 120 and diastolic [DBP] < 80 mmHg), elevated/moderate (untreated SBP: 120–139 or DBP: 80–89 mmHg), and high risk (SBP ≥ 140 or DBP ≥ 90 mmHg or medication use). For statistical analysis, the optimal and elevated/moderate categories were collapsed so that BP was categorized as high risk versus not high risk. Total cholesterol was categorized as optimal (untreated serum total cholesterol <200 mg/dL), elevated (200–239 mg/dL, untreated), and high risk (≥240 mg/dL or on medication). Tobacco smoking status was classified as current (high risk), former (moderate), or never (optimal). Diabetes was defined as fasting glucose ≥ 126 mg/dL, or HbA1c ≥ 6.5%, or use of diabetic medication (actual or self-reported) within 2 weeks prior to the clinic visit, with having diabetes as high risk, and no diabetes as optimal. Because a small proportion of participants had all optimal factors (6.1%) and nearly half of the participants had ≥2 high-risk factors, our four CVD risk strata were categorized as: (A) not high-risk (0 high-risk levels of all 4 major CVD risk factors); (B) having any 1 high-risk factor; (C) having any 2 high-risk factors; and (D) having ≥3 high-risk factors.

2.4 | Exclusions

Based on audiological and CVD risk assessment, 1314 participants were eligible for study inclusion. From this group, participants were excluded if they met any of the following criteria: missing audiometric thresholds at 0.5, 1.0, 2.0, and/or 4.0 kHz ($n = 17$), non-Type A tympanogram ($n = 64$), air-bone gap at 0.5, 1.0, 2.0, and/or 4.0 kHz ≥15 dB ($n = 36$), missing CVD risk factor data ($n = 44$), and missing covariate data (e.g., age, sex, education status; $n = 47$). After exclusions, 1106 participants remained in the study sample (Figure 1). A second analytic sample was used for DPOAE analysis. Participants were excluded from DPOAE analysis if they were missing DPOAE data ($n = 37$), resulting in a sample of 1069 participants.

2.5 | Statistical analysis

Descriptive analyses were used to examine demographic, CVD risk, and audiological data. Continuous measures are reported as mean (SD) and categorical measures as number (percent). Continuous variables were compared between groups using variance tests. Categorical variables were compared using Chi-square tests. Logistic regression was used to determine associations between auditory outcomes (hearing loss [$PTA_{0.5,1,2,4}$ > 15 dB HL] and cochlear dysfunction [absent/reduced $DPOAE_{low}$, $DPOAE_{high}$]) and four discrete CVD risk factors as well as the association with overall CVD risk status. Three models were constructed: unadjusted, age-sex adjusted, and a fully adjusted multivariable which included age, sex, education, BMI, vertigo, and noise exposure (Yes/No) as covariates. Models for individual risk factors were adjusted for the other CVD risk factors. Values of $p < .05$ were considered statistically significant. Analysis was performed using SAS version 9.4 (SAS Institute Inc., Cary, North Carolina).

3 | RESULTS

Table 1 shows demographic and cardiometabolic risk characteristics of the 1106 JHS participants. The majority (772 [69.8%]) of participants were female. Compared to the highest CVD risk group (≥3 high-risk factors), participants in the lowest risk group were younger, less likely to be obese, and better educated. Among persons with ≥1 CVD risk factors, the use of antihypertensives and lipid-lowering medication were common (79.0% and 36.2%, respectively). Regarding audiological profiles, persons in the highest risk group had higher $PTA_{0.5,1,2,4}$, greater prevalence of hearing loss, and were more likely to report vertigo (Table 2). Overall, the prevalence of hearing loss was 64.7% (data not shown). Average DPOAE levels across all CVD risk groups ranged from −1.33 to −10.50 dB SPL. As CVD risk increased, DPOAE amplitudes tended to decrease (i.e., worsen; all $p < .0001$). Relatedly, the prevalence of normal DPOAEs (i.e., average emission amplitude >0 dB SPL and SNR ≥ 6 dB) significantly decreased as CVD risk increased (all $p < .05$). However, even in the lowest risk group, <50% of participants had normal DPOAEs. In the highest CVD risk stratum, fewer than 30% of participants had normal DPOAEs.

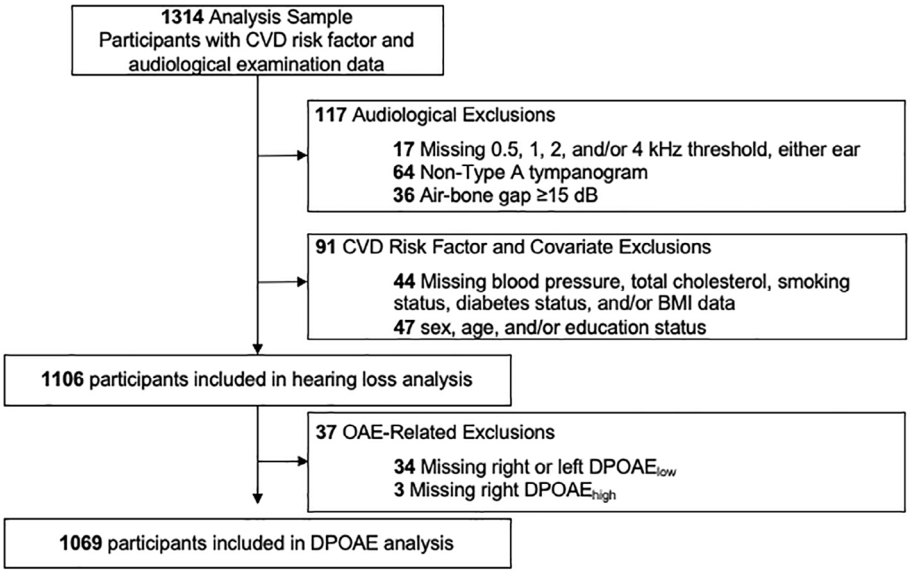

FIGURE 1 Flowchart of participant exclusions. DPOAE data missing if <2 data points contribute to the average. BMI, body mass index; CVD, cardiovascular disease; DPOAE, distortion product otoacoustic emission.

TABLE 1 Descriptive characteristics among participants by CVD risk status (Jackson Heart Study, Jackson, Mississippi, United States).

	CVD risk status[a]				
	Not high risk ($n = 210$)	1 high-risk factor ($n = 390$)	2 high-risk factors ($n = 325$)	≥3 high-risk factors ($n = 181$)	
	Mean (SD) or *n* (%)	Mean (SD) or *n* (%)	Mean (SD) or *n* (%)	Mean (SD) or *n* (%)	*p*-value
Age group					<.0001
25–<45 years	57 (27.14)	41 (10.51)	14 (4.31)	6 (3.31)	
<55 years	84 (40)	111 (28.46)	73 (22.46)	34 (18.78)	
<65 years	42 (20)	117 (30)	105 (32.31)	64 (35.36)	
<75 years	19 (9.05)	96 (24.62)	92 (28.31)	57 (31.49)	
≥75 –97 years	8 (3.81)	25 (6.41)	41 (12.62)	20 (11.05)	
Age (years)	51.3 (10.80)	58.35 (11.09)	61.95 (10.33)	62.46 (9.73)	<.0001
Female	143 (68.1)	262 (67.18)	233 (71.69)	134 (74.03)	0.3017
Educational level					<.0001
<High school	10 (4.76)	37 (9.49)	52 (16)	28 (15.47)	
High school/GED	24 (11.43)	61 (15.64)	56 (17.23)	38 (20.99)	
>High school	176 (83.81)	292 (74.87)	217 (66.77)	115 (63.54)	
Body Mass Index category					.0015
Normal weight; <25 kg/m^2	32 (15.24)	44 (11.28)	31 (9.54)	13 (7.18)	
Overweight; 25–<30 kg/m^2	80 (38.1)	128 (32.82)	106 (32.62)	43 (23.76)	
Obese; ≥30 kg/m^2	98 (46.67)	218 (55.9)	188 (57.85)	125 (69.06)	
BMI (kg/m^2)	30.98 (6.43)	32.35 (7.52)	32.15 (6.48)	33.68 (6.41)	0.0016
Smoking status					NA
Never	180 (85.71)	290 (74.36)	219 (67.38)	105 (58.01)	
Former	30 (14.29)	71 (18.21)	73 (22.46)	33 (18.23)	
Current	0 (0%)	29 (7.44)	33 (10.15)	43 (23.76)	
Blood pressure category					NA
Optimal[b]	117 (55.71)	54 (13.85)	12 (3.69)	1 (0.55)	
Elevated/moderate[c]	93 (44.29)	59 (15.13)	10 (3.08)	2 (1.1)	
High[d]	0 (0%)	277 (71.03)	303 (93.23)	178 (98.34)	

TABLE 1 (Continued)

	CVD risk status[a]				
	Not high risk ($n = 210$)	1 high-risk factor ($n = 390$)	2 high-risk factors ($n = 325$)	≥3 high-risk factors ($n = 181$)	
	Mean (SD) or *n* (%)	Mean (SD) or *n* (%)	Mean (SD) or *n* (%)	Mean (SD) or *n* (%)	*p*-value
Systolic blood pressure, mmHg	116.88 (12.03)	126.30 (17.59)	129.66 (18.18)	130.49 (20.26)	NA
Diastolic blood pressure, mmHg	72.77 (7.92)	75.37 (10.06)	74.63 (9.86)	73.08 (10.38)	NA
Antihypertensives, yes	0 (0%)	250 (64.10)	286 (88.0)	172 (95.03)	<.0001
Total cholesterol category					NA
Optimal[e]	129 (61.43)	207 (53.08)	87 (26.77)	11 (6.08)	
Elevated[f]	81 (38.57)	118 (30.26)	35 (10.77)	1 (0.55)	
High[g]	0 (0%)	65 (16.67)	203 (62.46)	169 (93.37)	
Total cholesterol, mg/dL	190.19 (27.23)	196.75 (35.47)	202.59 (44.32)	198.61 (55.31)	NA
Lipid-lowering medication, yes	0 (0%)	26 (6.67)	151 (46.46)	147 (81.22)	<.0001
Diabetes, yes	0 (0%)	19 (4.87)	111 (34.15)	160 (88.4)	NA
Stroke, yes	1 (0.48)	10 (2.56)	11 (3.38)	7 (3.87)	.131
History of coronary heart disease, yes	2 (0.95)	15 (3.85)	23 (7.08)	16 (8.84)	.0008
History of chemo/radiation, yes	7 (3.33)	23 (5.9)	20 (6.15)	10 (5.52)	.5124
Head injury, yes	12 (5.71)	43 (11.03)	34 (10.46)	17 (9.39)	.1823

Abbreviations: CVD, cardiovascular disease; DBP, diastolic blood pressure; PTA, pure-tone average; SBP, systolic blood pressure; SD, standard deviation.
[a]CVD risk status was defined based on four established CVD risk factors with four categories: (A) not high-risk (defined as not having any high-risk levels of all four major CVD risk factors including serum total cholesterol level ≥240 mg/dL or use of cholesterol-lowering medication; SBP/DBP ≥ 140/≥90 mmHg or use of antihypertensives; current smoking; and having diabetes); (B) having any 1 risk factor listed in (A); (C) having any two risk factors listed in (A); and (D) having ≥3 risk factors listed in (A).
[b]Defined as SBP < 120 and DBP <80 mmHg, untreated.
[c]Defined as SBP: 120–139 or DBP: 80–89 mmHg, untreated.
[d]Defined as SBP ≥ 140 or DBP ≥ 90 mmHg or on medication.
[e]Defined as total cholesterol <200 mg/dL, untreated.
[f]Defined as total cholesterol 200–239 mg/dL, untreated.
[g]Defined as total cholesterol ≥240 mg/dL or on medication.

Table 3 shows the odds of hearing loss by CVD risk status. In the unadjusted model, diabetes, high BP (SBP/DBP ≥ 140/≥90 or medication use), high cholesterol (≥240 mg/dL or medication), former smoking, and having 1, 2, or ≥3 CVD high-risk factors were associated with hearing loss. With age-sex adjustment, diabetes was associated with hearing loss (OR = 1.62 [95% CI: 1.15–2.29]). The fully multivariable model adjusted for age, sex, education, BMI, vertigo, and noise exposure revealed a significant relationship only with diabetes (multivariable adjusted OR [MVOR] = 1.48 [95% CI: 1.04–2.10]).

Table S1 shows unadjusted and age-sex adjusted odds of absent/reduced $DPOAE_{low}$ by CVD risk factor status. Although some associations were significant in the unadjusted model (e.g., former tobacco smoking and presence of 2 and≥3 high-risk factors), statistical significance was not maintained in the age-sex adjusted model. Likewise, Table S1 reports unadjusted and age-sex adjusted associations for absent/reduced $DPOAE_{high}$ by CVD risk factor status. Associations that were significant in the unadjusted model (e.g., high-risk BP level, high cholesterol, former smoking, and high CVD risk status) were not significant in the age-sex adjusted model.

Last, Table 4 shows ear-specific (better/worse) MVORs of absent/reduced $DPOAE_{low}$ and $DPOAE_{high}$ by CVD risk factor status. In this fully adjusted multivariable model, none of the associations between absent/reduced $DPOAE_{low}$ and individual CVD risk factors reached statistical significance. In terms of overall CVD risk, the fully adjusted model did not reveal any significant associations between absent/reduced $DPOAE_{low}$ and the presence of 1, 2, or ≥3 CVD risk factors. Results for $DPOAE_{high}$ were similar: neither the relationship to individual CVD risk factors nor overall CVD risk was significant.

4 | DISCUSSION

We explored the relationship between CVD risk factors (individually and in terms of overall risk) and cochlear function in a community-based population of African Americans. We hypothesized that increasing risk factor load would be associated with greater odds of hearing loss and cochlear dysfunction as assayed by DPOAEs. This study identified a significant relationship between diabetes and

TABLE 2 Audiological outcomes by CVD risk status (Jackson Heart Study, United States).

	CVD risk status[a]				
	Not high risk ($n = 210$)	1 high-risk factor ($n = 390$)	2 high-risk factors ($n = 325$)	≥3 high-risk factors ($n = 181$)	
	Mean (SD) or *n* (%)	Mean (SD) or *n* (%)	Mean (SD) or *n* (%)	Mean (SD) or *n* (%)	*p*-value
Exposure to loud noise, yes	50 (23.81)	125 (32.05)	103 (31.69)	45 (24.86)	.0685
Vertigo, yes	34 (16.19)	82 (21.03)	88 (27.08)	54 (29.83)	.0029
Hearing loss[b], yes	97 (46.19)	250 (64.1)	231 (71.08)	137 (75.69)	<.0001
Better ear $PTA_{0.5,1,2,4}$, dB HL	14.15 (7.49)	18.07 (10.30)	19.29 (9.88)	19.99 (9.57)	<.0001
Worse ear $PTA_{0.5,1,2,4}$, dB HL	16.79 (7.80)	21.89 (11.71)	23.19 (12.05)	23.57 (11.10)	<.0001
	Not high risk ($n = 204$)	**1 high-risk factor ($n = 376$)**	**2 high-risk factors ($n = 311$)**	**≥3 high-risk factors ($n = 178$)**	***p* value**
Mean (SD) DPOAE amplitude, dB SPL					
Better ear $DPOAE_{low}$[c]	−1.33 (8.17)	−3.81 (9.81)	−4.36 (9.08)	−5.90 (9.17)	<.0001
Worse ear $DPOAE_{low}$[c]	−3.13 (9.04)	−5.89 (10.18)	−6.58 (9.86)	−7.75 (10.05)	<.0001
Better ear $DPOAE_{high}$[d]	−3.34 (11.08)	−6.27 (11.16)	−8.55 (10.63)	−9.67 (11.19)	<.0001
Worse ear $DPOAE_{high}$[d]	−5.03 (11.24)	−8.20 (11.71)	−9.92 (11.06)	−10.50 (11.18)	<.0001
Present and normal DPOAE[e], *n* (%)					
Better ear $DPOAE_{low}$[c]	98 (48.04)	151 (40.16)	113 (36.33)	52 (29.21)	.0016
Worse ear $DPOAE_{low}$[c]	77 (37.75)	118 (31.38)	86 (27.65)	44 (24.72)	.0268
Better ear $DPOAE_{high}$[d]	88 (43.14)	104 (27.66)	70 (22.51)	39 (21.91)	<.0001
Worse ear $DPOAE_{high}$[d]	68 (33.33)	90 (23.94)	64 (20.58)	30 (16.85)	.0008

Abbreviations: CVD, cardiovascular disease; dB HL, decibels hearing level; dB SPL, decibels sound pressure level; DPOAE, distortion product otoacoustic emission; PTA, pure-tone average.
[a]Defined as per Table 1.
[b]Defined as $PTA_{0.5,1,2,4} > 15$ dB HL in the worse ear.
[c]Defined as average DPOAE level at $f_2 \leq 4$ kHz.
[d]Defined as average DPOAE level at $f_2 \geq 6$ kHz.
[e]Present and normal defined as DPOAE amplitude >0 dB SPL and SNR ≥6.

hearing loss. Our unadjusted analysis revealed significant relationships between absent/reduced DPOAEs and individual risk factors (diabetes, high BP, and former smoking) and overall CVD risk. However, contrary to expectation, we did not find evidence of a harmful relationship between cochlear function and CVD risk factors (individually or upon consideration of overall CVD risk) in fully adjusted multivariable models.

Of the four CVD risk factors examined here, the relationship between hearing loss and diabetes is arguably the most established. In agreement with previous reports,[11–13] we observed a significant relationship of diabetes to hearing loss. Previously, Bishop et al.[31] did not find an association between diabetes and hearing loss in the JHS, but their cutoff for normal hearing was ≤25 dB HL, whereas we defined hearing loss as $PTA_{0.5,1,2,4} > 15$ dB HL. Our decision to use a more conservative definition of hearing loss was motivated in part by recent evidence linking subclinical hearing loss to decreased cognition and symptoms of depression.[36,37] Further, studies have suggested that the presence of normal hearing (defined using the standard clinical cutoff of 25 dB HL for frequencies ≤8 kHz) is not a sufficient indicator of cochlear health.[38,39] Because this study was concerned with outer hair cell health, our strict definition of normal hearing was fitting.

Histopathological analysis has revealed outer hair cell loss[40] and thickening of strial capillaries in diabetic rat cochleae.[41] Human temporal bone studies have also identified thickened strial vessels and outer hair cell loss in diabetics, most notably in the cochlear base.[42,43] Because DPOAEs assess outer hair cell function, they are ideal for noninvasively detecting cochlear pathology in individuals with diabetes or other cardiometabolic risk factors. In our study, although not statistically significant, the better-ear MVORs for absent/reduced $DPOAE_{low}$ and $DPOAE_{high}$ were 1.31 and 1.06, respectively. Longitudinal study of DPOAE changes in African Americans with diabetes would provide additional insight regarding the extent to which diabetes is related to cochlear dysfunction.

Unlike past reports,[15,44] current tobacco smoking was not associated with hearing loss in our study. The fully adjusted model did show higher odds of hearing loss in current smokers (MVOR, 1.05) but this finding was not statistically significant. Using a less conservative definition of hearing loss (PTA > 25 dB HL), an earlier JHS report also failed to find an association between smoking and hearing loss.[29] Though our unadjusted analysis showed increased odds of absent/reduced $DPOAE_{low}$ and $DPOAE_{high}$ in former smokers compared with nonsmokers, the relationship to smoking was not significant in

TABLE 3 Odds ratios (95% CI) for hearing loss (defined as $PTA_{0.5,1,2,4}$ > 15 dB HL in the worse ear) by individual CVD risk factors and overall risk status.

	Unadjusted	Age-sex adjusted	Multivariable adjusted[a]
	Odds ratio (95% CI)	Odds ratio (95% CI)	MVOR (95% CI)
Diabetes			
No diabetes	Ref	Ref	Ref
Diabetes	**1.63 [1.18–2.23]**	**1.62 [1.15–2.29]**	**1.48 [1.04–2.10]**
Blood pressure			
Not high-risk	Ref	Ref	Ref
High risk[b]	**1.73 [1.32–2.28]**	0.97 [0.71–1.34]	0.86 [0.62–1.19]
Total cholesterol			
Optimal[c]	Ref	Ref	Ref
Elevated[d]	1.17 [0.84–1.63]	1.07 [0.74–1.54]	1.12 [0.77–1.61]
High[e]	**1.46 [1.09–1.96]**	1.06 [0.76–1.47]	1.08 [0.78–1.51]
Tobacco smoking			
Never	Ref	Ref	Ref
Former	**1.52 [1.08–2.16]**	0.90 [0.61–1.32]	0.87 [0.59–1.30]
Current	1.08 [0.70–1.66]	1.06 [0.67–1.68]	1.05 [0.65–1.68]
CVD risk status[f]			
Not high risk	Ref	Ref	Ref
1 high-risk factor	**2.08 [1.48–2.93]**	1.24 [0.84–1.81]	1.10 [0.75–1.63]
2 high-risk factors	**2.86 [1.99–4.11]**	1.29 [0.86–1.95]	1.11 [0.73–1.69]
≥3 high-risk factors	**3.63 [2.35–5.60]**	1.62 [0.99–2.63]	1.30 [0.79–2.15]

Note: Bolded values indicate statistically significant values ($p < .05$).
Abbreviations: BMI, body mass index; CI, confidence interval; CVD, cardiovascular disease; MVOR, multivariable adjusted odds ratio.
[a]Adjusted for age, sex, education, BMI, vertigo, noise exposure.
[b]Defined as SBP ≥ 140 or DBP ≥90 mmHg or on medication.
[c]Defined as total cholesterol <200 mg/dL, untreated.
[d]Defined as total cholesterol 200–239 mg/dL, untreated.
[e]Defined as total cholesterol ≥240 mg/dL or on medication.
[f]Defined as per Table 1.

adjusted models. The most likely reason for these findings is the low prevalence of current tobacco smoking, which was <10% in this study. Former smoking was more common but reported by only ~16% of participants. Our study also differs from earlier reports in that our participants were all African American. Previous studies supporting a relationship between smoking and hearing loss were primarily conducted in non-Hispanic white[18] or Asian[14,17] populations, although a study of black and white elders did not find evidence that smoking influenced hearing sensitivity.[45] Additional research is needed to better understand the relationship between cochlear dysfunction and smoking in persons of African American descent.

In unadjusted analyses, participants with high BP demonstrated increased odds of hearing loss and absent/reduced $DPOAE_{high}$. These findings are reasonable given past reports showing cochlear damage in aged hypertensive rats[46] and human clinical studies.[47,48] Once we accounted for all covariates, however, the statistical significance of these relationships was not retained. Three factors may contribute to this finding. First, ~83% of participants had elevated or high BP and ~64% of all participants ($n = 708$) were on antihypertensives. Second, it is well established that BP is correlated with age.[49] Third, even in the lowest CVD risk group, normal DPOAEs were observed in less than half of the participants (Table 2). These factors limited group sample sizes. It is also worth recognizing that normative DPOAE data are not race-specific. The African American population is not well represented in clinical guidelines, which may affect assessment and management of hearing loss. Additional study of cochlear function in this population, particularly as related to CVD risk, is therefore warranted.

Some studies have demonstrated associations between hearing loss and CVD risk metrics (e.g., Framingham Risk Score[21]) although these studies examined primarily Western Europeans. Here, we did not observe an association between overall CVD risk and hearing loss or cochlear dysfunction. Only one other study has examined DPOAEs in the context of CVD risk in African Americans. Sorrel et al.[28] found a significant relationship between DPOAE SNR and stroke risk when stratified by age, sex, and ear. However, the significant findings were scattered across age, sex, frequency, and ear categorization which limits clinical translation. Despite the high CVD risk burden experienced by African Americans,[24] studies have generally found this population to have a lower prevalence of hearing loss than non-Hispanic whites.[31,45] The reason for this discrepancy is unclear but cochlear melanin concentration may be a contributing factor. Strial

melanocytes produce melanin, which has been proposed to serve an otoprotective role.[50] It follows that black race may decrease risk of peripheral auditory damage even with higher overall CVD risk.

The Nord–Trøndelag Health study in Norway (an all-Caucasian sample) showed a relationship between CVD risk factors and hearing loss but CVD factors only explained 0.2%–0.4% of the variance.[51] Cochlear function as measured by DPOAEs, similar to studies of pure tone audiometry, are likely dominated by age, sex, and noise exposure, therefore significant relationships in cross-sectional analysis will be challenging to isolate. Longitudinal studies using audiometry and DPOAEs and monitoring changes with acquired CVD will likely be of greater power.

TABLE 4 Multivariable adjusted[a] odds ratios (95% CI) for reduced/absent (vs. normal) $DPOAE_{low}$ and $DPOAE_{high}$ by individual CVD risk factors and overall risk status.

	$DPOAE_{low}$	$DPOAE_{high}$
	MVOR (95% CI)	MVOR (95% CI)
Better ear		
Diabetes	1.31 [0.94–1.83]	1.06 [0.73–1.54]
Blood pressure		
Not high risk	Ref	Ref
High risk[b]	0.71 [0.52–0.98]	0.73 [0.52–1.04]
Total cholesterol		
Optimal[c]	Ref	Ref
Elevated[d]	0.83 [0.58–1.19]	0.71 [0.49–1.05
High[e]	0.98 [0.71–1.34]	1.00 [0.70–1.42]
Tobacco smoking		
Never	Ref	Ref
Former	0.90 [0.62–1.31]	1.16 [0.75–1.79]
Current	0.90 [0.58–1.41]	1.39 [0.84–2.32]
CVD risk status[f]		
Not high risk	Ref	Ref
1 risk factor	0.81 [0.55–1.19]	1.16 [0.77–1.74]
2 risk factors	0.77 [0.51–1.16]	1.10 [0.71–1.72]
≥3 risk factors	1.04 [0.65–1.69]	1.04 [0.62–1.76]
Worse ear		
Diabetes	1.15 [0.80–1.64]	0.96 [0.65–1.41]
Blood pressure		
Not high risk	Ref	Ref
High risk[b]	0.69 [0.49–0.96]	0.81 [0.57–1.17]
Total cholesterol		
Optimal[c]	Ref	Ref
Elevated[d]	0.83 [0.57–1.21]	0.86 [0.58–1.29]
High[e]	0.85 [0.60–1.19]	0.95 [0.65–1.38]
Smoking status		
Never	Ref	Ref
Former	0.92 [0.61–1.39]	0.94 [0.59–1.48]
Current	1.01 [0.63–1.63]	1.02 [0.61–1.72]

TABLE 4 (Continued)

	$DPOAE_{low}$	$DPOAE_{high}$
	MVOR (95% CI)	MVOR (95% CI)
CVD risk status[f]		
Not high risk	Ref	Ref
1 high-risk factor	0.71 [0.47–1.06]	0.83 [0.54–1.27]
2 high-risk factors	0.66 [0.43–1.03]	0.71 [0.45–1.13]
≥3 high-risk factors	0.73 [0.44–1.22]	0.85 [0.49–1.50]

Note: Normal defined as DPOAE amplitude >0 dB SPL and SNR ≥ 6 dB. Present but reduced defined as DPOAE amplitude ≤0 dB SPL but >−20 and SNR ≥ 6 dB. Absent defined as DPOAE amplitude <−20 dB SPL or SNR < 6 dB.

Abbreviations: BMI, body mass index; CI, confidence interval; CVD, cardiovascular disease; DPOAE, distortion product otoacoustic emission.

[a]Adjusted for age, sex, education, BMI, vertigo, noise exposure.

[b]Defined as SBP ≥140 or DBP ≥90 mmHg or on medication.

[c]Defined as total cholesterol <200 mg/dL, untreated.

[d]Defined as total cholesterol 200–239 mg/dL, untreated.

[e]Defined as total cholesterol ≥240 mg/dL or on medication.

[f]Defined as per Table 1.

Clinical translation of this research remains in an early stage. Some authors (e.g., Spankovich & Yerraguntla[52]) have suggested audiological monitoring of persons with diabetes, especially those with exposure to noise and/or ototoxic agents. Our analysis supports a relationship between hearing loss ($PTA_{0.5,1,2,4}$) and diabetes. These findings suggest that obtainment of baseline auditory status in persons of African American descent recently diagnosed with diabetes is prudent. Whether or not early evaluation of cochlear function in individuals with cardiometabolic risk factors would be clinically advantageous is inconclusive. A prospective study would lend itself to more actionable clinical recommendations.

An additional consideration is our definition of "not high risk." This group was not limited to persons with optimal BP and cholesterol levels and permitted inclusion of individuals with elevated levels that were not high enough to be considered major CVD risk factors. In this study, <20% of individuals were classified as not high risk. In this lowest risk group, 44.3% of participants had elevated BP and 38.6% had elevated cholesterol. Further, because obesity is so common in this cohort - even in the lowest risk group, 46.7% of participants were obese - we did not use BMI to classify a person's overall CVD risk although our fully adjusted multivariable models did include BMI as a covariate. Due to sample size limitations, we were unable to construct a low-risk reference group with optimal BP and cholesterol levels, thus potentially obscuring between-group differences in auditory outcomes.

This study has limitations. First, most participants were female and the JHS is exclusive to African Americans recruited from one geographic location, limiting generalizability to other races/ethnicities. Due to sample size limitations, we were unable to construct a low-risk group with optimal BP and cholesterol levels and our definition of CVD risk excluded BMI as a defining factor. Use of lipid- and BP-lowering medications were common, which may have limited our ability to assess associations with auditory function. Finally, this study

was cross-sectional and consequently, conclusions cannot be drawn regarding causality. A longitudinal design would be more informative to subclinical changes in cochlear function over time with development of CVD risk factors.

5 | CONCLUSION

We identified a relationship between diabetes and hearing loss. We did not find evidence to support an association between cochlear dysfunction and the four CVD risk factors discretely or in combination.

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CONFLICT OF INTEREST STATEMENT

The authors declare no conflicts of interest.

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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