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Peripheral Auditory Function in Young HIV-Positive Adults With Clinically Normal Hearing

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Abstract

Objective. Little is known about peripheral auditory function in young adults with HIV, who might be expected to show early evidence of hearing loss if HIV infection or treatment does affect peripheral function. The goal of this study was to compare peripheral auditory function in 2 age- and gender-matched groups of young adults with clinically normal hearing with and without HIV.

Study Design. Matched cohort study with repeated measures.

Setting. Infectious disease center in Dar es Salaam, Tanzania.

Methods. Participants included HIV-positive (n = 38) and HIV-negative (n = 38) adults aged 20 to 30 years who had clinically normal hearing, defined as type A tympanograms, air conduction thresholds $\leq$25 dB HL bilaterally from 0.5 to 8 kHz, and distortion product otoacoustic emissions (DPOAEs) >6 dB above the noise floor bilaterally from 1.5 to 8 kHz. Participants were tested multiple times over 6-month intervals (average, 2.7 sessions/participant) for a total of 208 observations. Primary outcome measures included tympanograms, air conduction audiograms, DPOAEs, and click-evoked auditory brainstem responses.

Results. HIV groups did not significantly differ in age, static immittance, or air conduction thresholds. HIV-positive status was independently associated with approximately 3.7-dB lower DPOAE amplitudes from 2 to 8 kHz (95% CI, 1.01-6.82) in both ears and 0.04-μV lower (95% CI, 0.003-0.076) auditory brainstem response wave I amplitudes in the right ear.

Conclusion. Young adults living with HIV have slightly but reliably smaller DPOAEs and auditory brainstem response wave I amplitudes than matched HIV-negative controls. The magnitude of these differences is small, but these results support measuring peripheral auditory function in HIV-positive individuals as they age.

Keywords

peripheral auditory function, HIV, distortion product otoacoustic emissions (DPOAEs), auditory brainstem response (ABR), tympanometry

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Human immunodeficiency virus (HIV) affects auditory pathways with evidence of abnormal peripheral and central involvement.[1-8] Our recent work has focused on central auditory dysfunction because of HIV's effects on the central nervous system.[2,8,9] Some clinical studies have shown peripheral auditory system abnormalities, based on tympanograms, threshold audiograms, distortion product otoacoustic emissions (DPOAEs), and auditory brainstem response (ABR) tests,[2,4,5,10,11] but no consistent pattern has emerged of peripheral hearing dysfunction in people living with HIV (PLWH). Yet, PLWH report higher levels of hearing complaints than controls.[4] Furthermore, results from previous studies have been complicated by imperfectly controlled confounds of presbycusis or age-related hearing loss, noise exposure, socioeconomic status, history of ear infections, and other factors.

The goal of the current study was to compare peripheral auditory function, as measured by DPOAEs and ABR parameters, between young PLWH and HIV-negative controls with clinically normal hearing. The data come from a longitudinal cohort of individuals in Tanzania who were studied over

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Table 1. Characteristics and Statistical Comparisons Between PLWH and HIV-Negative Groups.[a]

	HIV–	PLWH	*P* value
No. of subjects	38	38	—
Observations per subject	2.49 (1.40)	3.07 (1.78)	.076
Female, %	37	34	.271
Age, y	23.7 (2.94)	23.2 (2.81)	.118
Years of education	11.2 (2.94)	11.3 (2.04)	.866
Pure tone average, 0.5-4 kHz			
Right ear	3.82 (5.92)	5.02 (5.53)	.122
Left ear	3.44 (6.11)	3.83 (5.91)	.671
Duration of HIV infection, y		12.90 (5.08)	
Currently on antiretroviral therapy, %		100	

Abbreviations: HIV, human immunodeficiency virus; PLWH, people living with HIV.
[a]Values are presented as mean (SD) unless noted otherwise. A chi-square test was conducted on gender data, and *t* tests comparing independent means were conducted on all continuous data. No significant differences existed between groups.

the course of 4 years with a comprehensive test battery of peripheral and central auditory tests. With this large cohort of PLWH and HIV-negative individuals, we could provide objective evidence of subclinical deficits. If HIV infection or treatment does affect peripheral hearing, we might expect a relatively young cohort of PLWH to exhibit subtle decreases on tests of peripheral function. We therefore hypothesized that young, normal-hearing PLWH show subclinical decreases in DPOAE response amplitude as compared with age- and gender-matched HIV-negative controls. Through the examination of subclinical auditory changes and potential early identification of hearing deficits in young PLWH, we may aid in determining prognosis, changing therapies, reducing sensory deprivation, and improving quality of life for those with HIV.

Methods

Recruitment

We recruited from a unique cohort of approximately 724 PLWH and HIV-negative individuals in Dar es Salaam, Tanzania, who have been performing central auditory, peripheral auditory, and cognitive testing at approximate 6-month intervals for the last 4 years. The research protocol was approved by the Committee for the Protection of Human Subjects of Dartmouth College and the Research Ethics Committee of Muhimbili University of Health and Allied Sciences. All participants gave written informed consent.

Study Procedures

Subjects completed a series of questionnaires and performed auditory tests at the Infectious Disease Center in Dar es Salaam. The questionnaires gathered data on self-reported hearing ability (hearing status questionnaire) and general health (health history questionnaire). The questions covered noise exposure, ear pathology, and ototoxic chemical exposure. The questionnaire also asked about education, HIV treatment, and gentamicin exposure, as well as the use of antimalarials, aspirin, and diuretics. All participants completed testing at approximately 6-month intervals.

We were specifically interested in variation in peripheral auditory function among young adults with clinically normal hearing; thus, we age- and gender-matched 2 groups of PLWH and HIV-negative individuals between the ages of 20 and 30 years and employed several data selection criteria. Individuals were excluded if they had any of the following: hearing sensitivity >25 dB HL from 0.5 to 8 kHz or abnormal middle ear function as indicated by type B or C tympanograms; DPOAEs <6 dB above the noise floor bilaterally from 1.5 to 8 kHz; a positive history of ear drainage, concussion, significant noise or chemical exposure, neurologic disease, mental illness, ototoxic antibiotics (eg, gentamycin), or chemotherapy; and age <20 or >30 years. This selection technique resulted in 78 individuals, with approximately 2.7 observations per subject. The demographics of this sample population are provided in **Table 1**.

Tympanometry

Tympanometry was conducted after otoscopy with cerumen removal as needed to ensure a clear ear canal. A Madsen Otoflex 100 (GN Otometrics) was used to perform tympanometry at 226 Hz. Measurements were collected for ear canal volume, static admittance, tympanometric peak pressure, tympanometric width, and tympanogram type (A, A_s, A_d, B, C). Type A tympanograms (including A_s and A_d) were required for inclusion in this study (pressure limits from −100 to +50 daPa and static admittance limits from 0.3 to 2.2 mmho).

Hearing Thresholds

Pure tone audiometry was completed with Creare LLC's wireless automated hearing test system as controlled through a laptop. The system allowed for testing in rooms with suboptimal noise levels, as the device speakers are mounted in the highly noise-attenuating ear cups. The attenuation provided

by this headset is on par with a portable single-walled sound booth, as measured by an independent laboratory according to the relevant ANSI standards (American National Standards Institute).[12] Pure tone air conduction thresholds were measured in octaves from 0.5 to 8 kHz plus the 6-kHz interoctave with a Békésy-like tracking procedure.[13] When the button was pressed, the tone decreased in 4-dB steps until the first reversal, when 2-dB steps were used. Upon releasing the button, the tones increased in 2-dB steps. Six reversals were counted to identify threshold. Normal peripheral hearing sensitivity (<25 dB HL at each frequency bilaterally) was required for all subjects. A pure tone average was calculated from 0.5 to 4.0 kHz.

Distortion Product Otoacoustic Emissions

DPOAEs were collected with Creare LLC's system at f2 values of 1.5, 2, 3, 4, 6, and 8 kHz with an f2/f1 ratio of 1.2 and L1/L2 values of 65/55 dB SPL, similar to our previous work.[2] The f2-f1 frequency pair was delivered for a minimum of 4 seconds. After 4 seconds, if the difference between the DPOAE level and the averaged noise floor level was <10 dB SNR (signal to noise ratio), the frequency pair continued to be presented until 10 dB SNR was reached or 10 seconds had elapsed. The operators instructed the participants not to swallow during DPOAE testing, and an adaptive noise-rejection algorithm was used to remove extraneous noise. In-ear calibration was not used (ie, the speaker output was not adjusted in the ear canal). The level of harmonic distortion for each system was determined with a Brüel and Kjær Type 4157 Ear Simulator/Artificial Ear. Because consistent DPOAE probe placement is important for achieving consistent results over time, a position check (frequency sweep) was presented in the ear canal prior to DPOAE testing. Three position checks (0.5-5 kHz) at 65 dB SPL were averaged, smoothed, and displayed to the operator. A measured level below 20 dB SPL at 0.5 kHz was used to indicate a bad probe seal. In this case, the probe was reseated, and the chirps were repeated. If the probe was placed securely in the ear canal and the seal check passed, results from the position check were saved as a baseline for that participant. On subsequent visits, the baseline position check was displayed so that the operator could position the probe to match the frequency sweep within ±5 dB at each frequency if possible.

Auditory Brainstem Responses

A SmartEP system (Intelligent Hearing Systems) was used to record and analyze ABR measurements in the right ear. The ABR was collected with an electrode attached to the right earlobe as reference, a ground electrode at F_{pz}, and an electrode at the high forehead (F_z) as the noninverting electrode. The stimuli were 100-μs rarefaction clicks presented at a rate of 21.1/s (slow) or 61.1/s (fast) at 80 dB SPL to the right ear. Two repetitions of each click were recorded and averaged (total, 2000 sweeps). Responses were filtered from 0.1 to 1.5 kHz (second-order Butterworth). The absolute latencies and amplitudes of waves I, III, and V were measured from the zero line.

Statistical Analysis

Demographic characteristics of the groups were compared with a *t* test for independent groups and a chi-square distribution test for gender. Data were analyzed with a linear mixed effects model and Wilcoxon rank sum test for independent groups in MATLAB 2020a (MathWorks). The response variables were the audiologic measures (audiometric thresholds, DPOAE amplitudes, ABR parameters). The model fixed effect was *HIV status*, and the random effect was *subject* variability of repeated measures. Including *subject* as a random factor over repeated observations allowed us to estimate fixed effects that replicated over time. The primary hypothesis testing focused on the difference between HIV groups.

Results

Demographics

After screening, there were 38 adult PLWH aged 20 to 30 years with normal hearing, tested an average of 3.07 times each, and 38 HIV-negative adults, tested 2.49 times each. PLWH were similar in age, education, and number of observations to HIV-negative controls. The distribution of males and females was similar between groups ($\chi^2 = 0.97$, $P = .27$; **Table 1**).

Comparison of Auditory Measures Between Groups

We evaluated the difference between HIV groups using linear mixed effects models and Wilcoxon rank sum tests on tympanometric measures, air conduction thresholds, DPOAEs, and click-evoked ABR latencies and amplitudes. Linear mixed effects models and Wilcoxon rank sum tests showed no difference between groups on static immittance or air conduction thresholds. Overall, PLWH had significantly decreased DPOAE amplitudes from 3.0 to 8.0 kHz in the left ear (all $P \leq .017$) and at 2.0, 4.0, and 6.0 kHz in the right ear (all $P \leq .037$) as compared with HIV-negative controls. PLWH also had significantly reduced ABR wave I amplitude ($P = .041$) as compared with the HIV-negative group. **Tables 2** to **4** present results for linear mixed effects models and Wilcoxon analysis for tympanometry and auditory thresholds, DPOAEs, and ABR measures, respectively.

Figure 1 to **3** illustrate differences between groups on all auditory measures. **Figure 1** shows pure tone auditory thresholds with inlayed static admittance measures for both groups. Observed pure tone audiometry results are consistent with normal peripheral hearing ability, with all mean thresholds values less than 10 dB HL. According to **Figure 2**, DPOAE results are consistent with differences between groups at 3 to 8 kHz in the left ear and at 2, 4, and 6 kHz in the right ear. The large signal-to-noise ratio for both groups in both ears indicate high-quality DPOAE recordings. **Figure 3** presents ABR grand means for both groups in the right ear (ABRs were not conducted in the left ear). These panels indicate typical morphology of ABR component peaks with a smaller wave I amplitude in PLWH than in the HIV-negative group.

Table 2. Linear Mixed Effects Model and Wilcoxon Rank Sum Test for Static Admittance and Pure Tone Audiometry From 0.5 to 8.0 kHz for the Right and Left Ears.[a]

	Mixed effects model			Wilcoxon rank sum	
Ear: measure	Estimate	*P* value	95% CI	*U* value	*P* value
Right					
Static admittance	–0.103	.485	–0.395 to 0.188	–0.593	.553
500 Hz	–1.038	.479	–3.923 to 1.846	–0.622	.531
1000 Hz	–0.387	.773	–3.027 to 2.252	–0.496	.619
2000 Hz	–1.598	.278	–4.495 to 1.299	–1.319	.187
4000 Hz	–1.694	.208	–4.339 to 0.951	–1.433	.088
6000 Hz	0.027	.933	–4.714 to 5.128	0.034	.973
8000 Hz	0.680	.840	–6.063 to 7.423	0.272	.789
Left					
Static admittance	0.024	.863	–0.249 to 0.297	0.805	.420
500 Hz	1.590	.260	–1.184 to 4.364	0.406	.684
1000 Hz	–0.225	.865	–2.818 to 2.369	–0.414	.678
2000 Hz	–1.164	.478	–4.394 to 2.067	–0.172	.362
4000 Hz	–1.235	.443	–4.407 to 1.936	–1.163	.244
6000 Hz	–2.784	.315	–8.303 to 2.735	–1.197	.203
8000 Hz	–0.878	.797	–7.732 to 5.976	–0.942	.634

[a]Model estimates reference the HIV-negative group. Model specification: measure ~ HIV status + 1|subject. No significant difference in static admittance or pure tone audiometry existed between groups.

Table 3. Linear Mixed Effects Model and Wilcoxon Rank Sum Test for DPOAEs for the Right and Left Ears.[a]

	Mixed effects model			Wilcoxon rank sum	
Ear: DPOAE signal frequency, Hz	Estimate	*P* value	95% CI	*U* value	*P* value
Right					
1500	2.050	.171	–0.896 to 4.997	1.810	.073
2000	2.324	.037	0.141 to 4.544	2.651	.008
3000	1.108	.303	–1.008 to 3.224	1.745	.081
4000	4.345	.002	1.678 to 7.011	3.704	<.001
6000	3.840	.026	–0.262 to 7.942	2.565	.010
8000	2.717	.169	–1.163 to 6.596	1.498	.134
Left					
1500	0.767	.576	–1.934 to 3.468	0.398	.690
2000	1.956	.163	–0.796 to 4.707	1.726	.084
3000	3.319	.005	1.002 to 5.635	4.015	<.001
4000	3.871	.006	1.127 to 6.615	3.103	.001
6000	3.788	.017	0.340 to 6.816	1.574	.007
8000	5.291	.007	1.435 to 9.148	2.762	.005

Abbreviations: DPOAE, distortion product otoacoustic emission; HIV, human immunodeficiency virus; PLWH, people living with HIV.
[a]The light gray highlight indicates that the DPOAE amplitude values were significantly different between HIV groups at the $P < .05$ level. Significant differences indicating lower DPOAE amplitudes in PLWH were found at 2, 4, and 6 kHz in the right ear and from 3 to 6 kHz in the left ear.

Discussion

The goal of the current study was to examine peripheral auditory function in young, normal-hearing individuals with and without HIV. To determine the variability of peripheral auditory function, we performed an analysis of age- and gender-matched PLWH and HIV-negative individuals in Tanzania using a comprehensive audiologic test battery. We hypothesized that PLWH would demonstrate subclinical decreases in peripheral auditory function as compared with HIV-negative controls. While tympanometry and pure tone thresholds were not significantly different, we found

Table 4. Linear Mixed Effects Model and Wilcoxon Rank Sum Test for ABR Amplitude and Latency Component Measures in the Right Ear.[a]

	Mixed effects model			Wilcoxon rank sum	
Click stimulus speed: measure (component)	Estimate	*P* value	95% CI	*U* value	*P* value
Slow					
Amplitude					
I	0.041	.033	0.003 to 0.076	2.004	.023
III	–0.007	.622	–0.037 to 0.022	–0.525	.599
V	0.006	.777	–0.036 to 0.048	–0.913	.361
Latency					
I	–0.154	.069	–0.320 to 0.012	–2.286	.053
III	–0.073	.341	–0.226 to 0.079	–0.334	.738
V	–0.107	.293	–0.308 to 0.094	–1.302	.192
Fast					
Amplitude					
I	0.023	.137	–0.007 to 0.053	1.484	.137
III	0.009	.492	–0.017 to 0.034	0.491	.623
V	–0.013	.440	–0.046 to 0.020	–1.530	.122
Latency					
I	–0.051	.604	–0.244 to 0.143	–0.457	.647
III	–0.086	.236	–0.230 to 0.057	–0.734	.462
V	–0.069	.505	–0.275 to 0.136	–0.317	.751

Abbreviations: ABR, auditory brainstem response; HIV, human immunodeficiency virus; PLWH, people living with HIV.
[a]ABR wave I amplitude with a slow click stimulus (21.1/s) was significantly reduced in PLWH vs HIV-negative controls. No other component measure yielded a significant difference between groups.

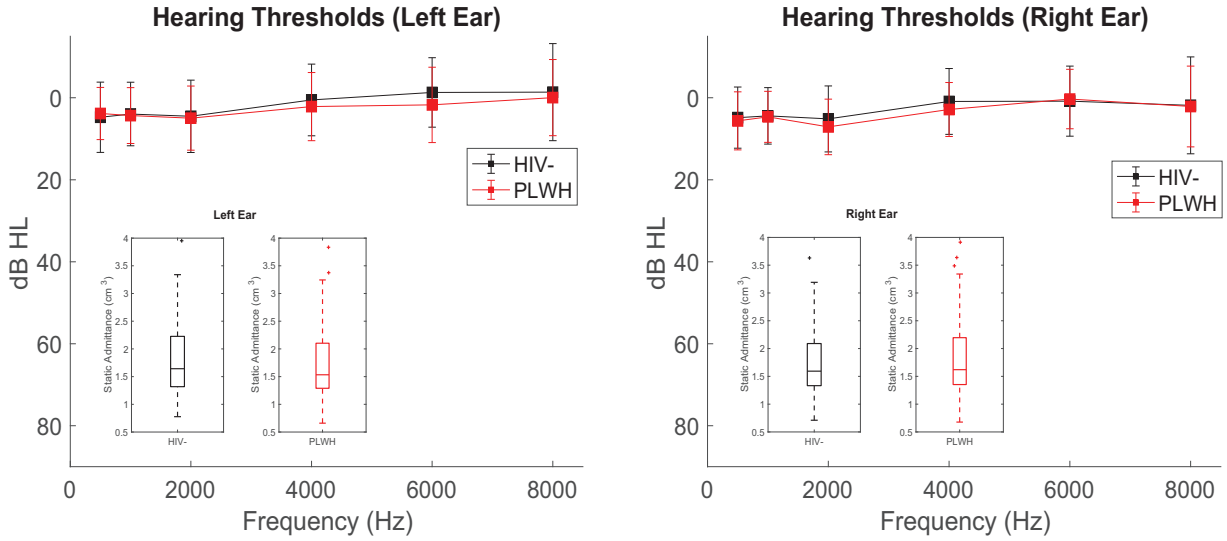

Figure 1. Pure tone thresholds (audiogram format) are displayed from 0.5 to 8 kHz with inlayed static admittance measures. Pure tone thresholds: values are presented as mean; error bars indicate ±1 SD. Static admittance: values are presented as median (line), interquartile range (box), 95% CI (error bars), and outlier (plus sign). HIV negative, black; PLWH, red. HIV, human immunodeficiency virus; PLWH, people living with HIV.

significant differences between PLWH and HIV-negative individuals on multiple DPOAE frequencies and ABR wave I amplitude. DPOAE results showed reduced amplitude for PLWH at mid- to high frequencies (2.0-8.0 kHz) in both ears. We also found reduced ABR wave I amplitude for PLWH versus HIV-negative controls. Results from this study provide evidence of reduced peripheral auditory function in young, normal-hearing PLWH. Some studies reported a significantly higher prevalence of hearing loss and otoacoustic emission abnormalities in PLWH when compared with controls matched by age, gender, race, and working environment.[14] The current findings, in combination with the literature, indicate that there are changes in the peripheral auditory function related to HIV infection or treatment.[14,15] Long-term studies on auditory

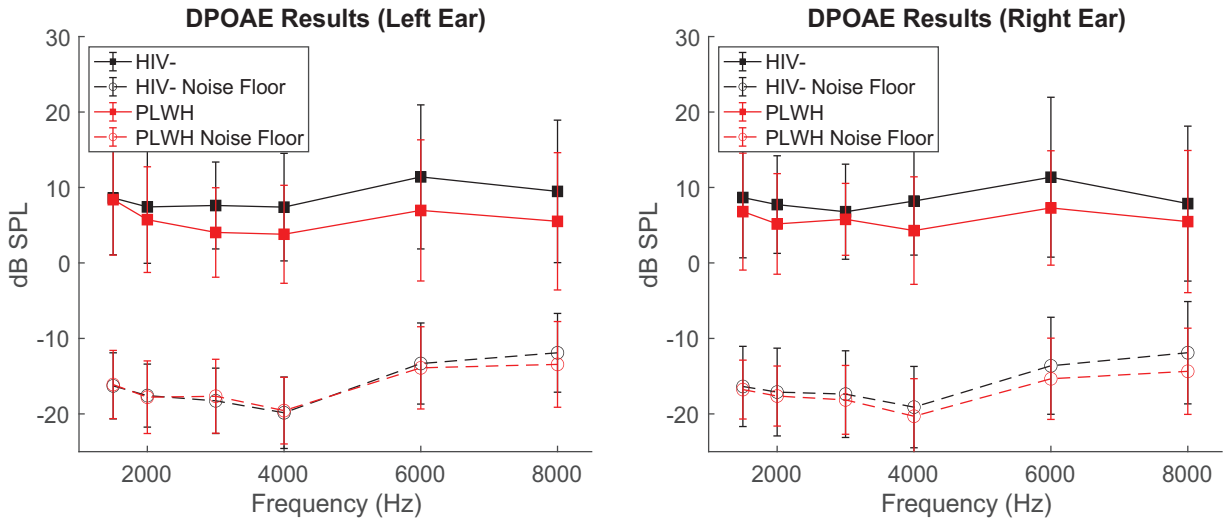

Figure 2. Distortion product otoacoustic emission (DPOAE) amplitudes (squares) and noise floors (circles) are plotted for each measured frequency. Values are presented as mean; error bars indicate ± 1 SD. HIV negative, black; PLWH, red. HIV, human immunodeficiency virus; PLWH, people living with HIV.

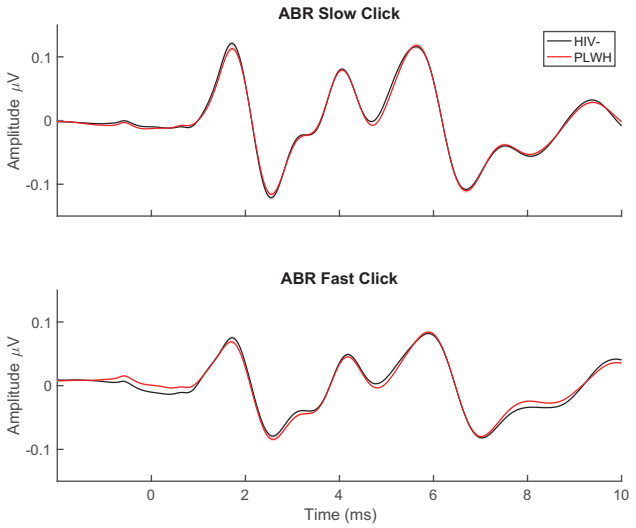

Figure 3. Auditory brainstem response (ABR) grand means for both groups. Top panel, slow click stimulus; bottom panel, fast click stimulus. HIV negative, black; PLWH, red. HIV, human immunodeficiency virus; PLWH, people living with HIV.

function in PLWH are needed to better understand the pathogenesis and to monitor and track disease progression.

Our previous work revealed significant differences in DPOAE amplitudes between PLWH and HIV-negative controls.[4] Results from this cross-sectional study showed that PLWH had reduced DPOAE levels when compared with HIV-negative individuals, but their peripheral auditory thresholds and tympanometry results were similar. This study, however, analyzed individuals across a range of ages (18-62 years) and included subjects with hearing loss (defined as pure tone thresholds >25 dB HL from 0.5-4.0 kHz). Also, the PLWH and HIV– groups were not well matched on age, gender, noise exposure, and other factors. Although statistical procedures (propensity score matching) were implemented to control for age in our previous work, clinical differences in pure tone thresholds may have resulted in DPOAE differences between groups. In the present study, the groups are well matched for age, and individuals with a significant history of noise exposure or ototoxic drug exposure were excluded.

Nevertheless, we still found reduced DPOAE amplitudes in PLWH. To our knowledge, only one other study has examined DPOAE differences between normal-hearing HIV+ and HIV– groups.[16] Ranjan and Bhat[16] measured DPOAEs in 12 HIV+ and 15 HIV− individuals within an age range of 20 to 40 years. In PLWH, the SNR of DPOAEs was reduced from 1 to 4 kHz in 25% to 50% of individuals or completely absent (DPOAE response not sufficiently higher than the noise floor) in 50% of the individuals. The SNR needed to determine the presence of DPOAE response was not clearly stated, however, and results for HIV-negative individuals were not presented. In comparison, our results indicate high SNRs in both groups but decreased amplitude of DPOAE responses in PLWH versus the HIV-negative group.

An ABR wave I amplitude difference, while not as robust as DPOAE differences, was seen between the groups. Studies have found prolonged peak and interpeak latencies, but to our knowledge, no other study has reported a difference in ABR wave I amplitude between young PLWH and HIV-negative controls.[10,17-19] As ABR wave I represents the distal function of the auditory nerve at the spiral ganglion cells, we might interpret this as a slight reduction in neural strength from the hair cell to the cochlear nucleus in PLWH.[19,20] Potential cochlear synaptic dysfunction may not be apparent in peripheral auditory measures such as pure tone audiometry, perhaps explaining why audiometric thresholds were similar between groups.[21] Our previous data from Tanzania demonstrated that PLWH report hearing problems, particularly difficulty in understanding speech in noise, so it is possible that cochlear

synaptic dysfunction, revealed in a wave I abnormality, may be contributing to these complaints.

Several possible explanations exist for these findings. Pathologic studies have shown that HIV and infections related to HIV can produce problems at multiple sites within the auditory system, such as middle ear infections, hearing loss (conductive, mixed, and sensorineural), decreased DPOAE amplitude, vestibular symptoms, and subcortical and cortical pathologies.[1,2,4,5,9,19,22] Pappas et al found extracellular viral-like particles with morphologic characteristics of HIV on the cochlear tectorial membrane in 3 HIV+ cases postmortem (temporal bone dissection).[23] Just as the central nervous system can serve as a reservoir for HIV, this could occur in the cochlea as well, perhaps leading to reduced DPOAE and ABR amplitudes. Also, HIV infection is characterized by sustained immune activation and inflammation.[24] There may be low-grade inflammation in the cochlea in patients with HIV that could manifest as reductions in DPOAE and ABR amplitudes.

HIV treatment, which typically involves multiple antiretroviral drugs, may affect peripheral auditory function.[14,25] Our previous study showed stable audiometric results in PLWH after starting antiretroviral treatment (ART), suggesting that there are no major ototoxic effects from ART.[4] Nevertheless, DPOAE amplitudes did decrease after ART was started, although the rate of decline did not differ from the HIV– group. In the current study, all individuals infected with HIV were receiving ART, but the specific drug regimens differed. Therefore, our results cannot distinguish between the effect of HIV and its treatment. Longitudinal effects of long-term ART (>5 years) may produce peripheral auditory dysfunction and contribute to early-onset presbycusis, suggesting that long-term studies are warranted to assess the effects of ART in this population.

While the DPOAE differences may reflect effects of HIV infection or treatment on the cochlea, they might also be due to differences in the efferent auditory system. As DPOAEs are primarily a function of descending projections from the brainstem (ie, superior olivary complex and inferior colliculus) to outer hair cells in the cochlea,[26] damage within the central nervous system due to HIV infection or treatment could allow for increased damage or degeneration of this pathway in PLWH as compared with uninfected controls. These small but significant differences could indicate disease progression or provide a marker for central nervous system dysfunction in HIV.

Generalizability of this study is limited. With a young, narrow age range and recruitment from one center in Dar es Salaam, additional analyses with a multisite recruitment procedure and broad age range are needed to confirm these findings. Additional audiometric measures, such as transient evoked and spontaneous otoacoustic emissions, are needed to extend the conclusions from this study. Nevertheless, this study provides evidence of subclinical deficits in DPOAE amplitude and ABR wave I amplitude in young, normal-hearing individuals living with HIV. We are currently conducting a longitudinal study in Dar es Salaam to better understand the trajectory of peripheral and central auditory function in those with HIV.

Future studies should examine how HIV may be affecting the peripheral auditory system, including DPOAEs, to better screen, track, and even predict auditory dysfunction in HIV. This supports tracking of auditory function by hearing health professionals (ie, audiologists) to provide serial auditory testing of peripheral auditory ability in this population. More comprehensive testing parameters, such as ABR thresholds or responses to more challenging stimuli (eg, speech), might reveal more differences associated with HIV status.

Conclusion

In summary, we found subtle but reliable differences in peripheral auditory function in young, normal-hearing individuals living with HIV as compared with age- and gender-matched HIV-negative controls. Specifically, we showed similar pure tone thresholds and tympanometry but reduced DPOAE amplitudes and a reduction ABR wave I in PLWH when compared with HIV-negative controls. While these effects were statistically reliable, their small magnitude means that they are unlikely to account for the higher incidence of hearing complaints among PLWH. Central auditory processing and aural cognition may contribute to these hearing difficulties, as shown in previous studies.[2,8,9] Still, this study indicates a subclinical difference in peripheral auditory function in young individuals living with HIV. Future work aims to continue following these individuals to determine if the early signs of dysfunction augur subsequent clinical hearing loss. If so, routine audiologic monitoring may be warranted for young adults living with HIV.

Author Contributions

Christopher E. Niemczak, completed final data and statistical analyses, created tables and figures, and completed the final draft of the manuscript as corresponding author; **Travis White-Schwoch**, participated in the analysis plan, statistical analyses, interpretation of findings, and helped complete the final draft of the manuscript; **Abigail Fellows**, verified data integrity, trained the assessment team, help to lead quality assurance for testing and data management, coordinated standard operating procedures, and supported institutional review board approvals; **Albert Magohe**, supervised the assessment team, coordinated participant scheduling and follow-up, provided quality assurance, reviewed data, and supported institutional review board approvals; **Jiang Gui**, assisted in statistical analysis and manuscript review; **Catherine Rieke**, verified data integrity and assisted with data interpretation; **Trent Nicol**, assisted with manuscript preparation and editing; **Enica R. Massawe**, assisted with study management, and data review; **Ndeserua Moshi**, assisted with study design and was primarily responsible for all aspects of study oversight in Tanzania; **Nina Kraus**, participated in all phases of study conceptualization, study design, proposal writing, analysis plan, study implementation of protocols, interpretation, and manuscript editing; **Jay C. Buckey**, was study primary investigator, and also participated in all phases of study conceptualization, study design, proposal writing, analysis plan, study implementation of protocols, interpretation, and manuscript editing.

Disclosures

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