

RESEARCH ARTICLE

Sensory Processing

Hypoactivation of the central auditory system in listeners who are hypertolerant of background noise

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Abstract

Listeners exhibit varying levels of tolerance for background noise during speech communication. It has been proposed that low tolerance of background noise may be the consequence of abnormally amplified gain in the central auditory system (CAS). Here, using a dataset of young adults with normal hearing thresholds, we asked whether central gain mechanisms might also explain cases of hypertolerance of background noise, as well as cases of reduced, but not abnormal, tolerance. We used the auditory brainstem response to derive a measure of CAS gain (wave V/wave I ratio) to compare listeners' background noise tolerance while listening to speech, grouping them into three categories: hyper, high, and medium tolerance. We found that hypertolerant listeners had reduced CAS gain compared to those with high tolerance. This effect was driven by wave V not wave I. In addition, the medium tolerant listeners trended toward having reduced wave I and reduced wave V amplitudes and generally higher levels of exposure to loud sound, suggestive of the early stages of noise-compromised peripheral function without an apparent compensatory increase in central gain. Our results provide physiological evidence that 1) reduced CAS gain may account for hypertolerance of background noise but that 2) increased CAS gain is not a prerequisite for medium tolerance of background noise.

NEW & NOTEWORTHY Our findings strengthen the proposed mechanistic connection between background noise tolerance and auditory physiology by suggesting a link between hypertolerance and reduced central auditory gain, measured by the auditory brainstem response.

auditory brainstem response; background noise tolerance; central gain

INTRODUCTION

Listening problems in real-world settings include difficulty understanding speech in background noise and negative psychological and physiological responses to background noise, including stress, exhaustion, and annoyance in noisy communication environments $(1-3)$ $(1-3)$ $(1-3)$. These factors can lower an individual's tolerance for communicating in noisy environments, constraining social opportunities. However, listeners differ in their response to background noise, with a spectrum emerging from extreme tolerance (hypertolerance) to extreme intolerance (hypotolerance). Tolerance for background noise can be measured in a clinical or research setting using the

acceptable noise level (ANL) test [\(4\)](#page-8-2), which involves playing a speech passage at a comfortable sound level and then presenting increasing levels of background noise until the listener reports feeling tension or fatigue [see also Nabelek et al. [\(4\)](#page-8-2)]. Although tolerance for background noise is easily measured, its neural mechanisms and influencing factors are poorly understood. One mechanistic hypothesis is that background noise tolerance is driven by central auditory system (CAS) gain, with hypotolerance (i.e., intolerance) arising from excessive CAS gain [\(5](#page-8-3)–[7](#page-9-0)). CAS gain refers to the process by which auditory nuclei modify the strength of their response to increase or decrease their output relative to peripheral input. Abnormal increases in CAS gain have also been proposed to underlie hyperacusis, the extreme reaction to everyday

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sounds being too loud that sometimes co-occurs with tinnitus and hearing loss (reviewed in Refs. [8](#page-9-1), [9](#page-9-2)). Changes in CAS gain often follow cochlear damage from noise exposure. In such cases, decreased peripheral output from the cochlea may increase activity within the central auditory system to counteract cochlear damage [\(8](#page-9-1), [10](#page-9-3), [11](#page-9-4)), leading to sound intolerance.

CAS gain can be measured noninvasively using auditory brainstem responses (ABRs), scalp recordings of the electrical activity produced by the cochlear nerve and brainstem ([5](#page-8-4), [6,](#page-8-5) [8,](#page-9-1) [12](#page-9-5), [13](#page-9-6)). The human ABR has a stereotyped morphology composed of five primary waves, the first of which arises from peripheral receptors (wave I, cochlear nerve) and the fifth from more central receptors (wave V, rostral brainstem and midbrain) [\(14](#page-9-7)). Wave I amplitude has received recent attention as a noninvasive measure of the cochlear nerve synapse integrity, and it has been the target of multiple studies investigating the physiological consequences of aging and noise exposure, with reduced wave I amplitude being interpreted as a cochlear neuropathy biomarker [\(15](#page-9-8)–[17\)](#page-9-9). A comparison of wave I to wave V (expressed as the V/I or I/V ratio) has been used in the literature to measure CAS gain ([14,](#page-9-7) [18](#page-9-10)–[20\)](#page-9-11). Other studies of central gain have included ABR wave III—a wave intermediate to I and V that arises from the cochlear nucleus—as a means to delineate where along the central pathway neuroplastic changes originate [\(6,](#page-8-5) [21\)](#page-9-12). In two small studies using the ANL test and only female participants presumably at low risk of noise-induced hearing loss (NIHL) [\(5](#page-8-4), [6](#page-8-5)), Tampas and Harkrider found elevated wave V amplitudes in listeners with low tolerance for background noise compared with those with higher tolerance. Yet, no group differences for wave I, wave III, or suprathreshold otoacoustic emissions (OAEs) were observed. They concluded that low tolerance to background noise could arise from central auditory system hyperactivity (i.e., increased CAS gain) in listeners with normal cochlear nerve function, with this hyperactivity first emerging in the midbrain (i.e., wave V).

Building on this literature, we investigate the physiological signatures of background noise tolerance in a dataset of young adults with normal hearing thresholds. The dataset is taken from our larger investigation on the early biological indicators of noise-induced hearing loss in young adults with clinically normal hearing thresholds [\(22](#page-9-13)–[25](#page-9-14)). In this line of work, we use college musicians as our primary noise exposure model, given that college musicians are routinely exposed to high noise levels [\(26](#page-9-15), [27\)](#page-9-16). In addition to treating music activities as a source of potential noise trauma for the auditory system, this line of work also examines the potential auditory benefits that arise from playing a musical instrument (quantified as the total years of playing an instrument). A complication of this approach is that lifetime noise exposure will scale as a function of the total years of musical activity, and participants who have played a musical instrument for a longer time are also more likely to be involved in more music activities than those with less ([25\)](#page-9-14). A previous analysis of the ANL data in this dataset focused on the association between noise exposure (measured from one week of personal noise dosimetry) and background noise tolerance (measured from the ANL test) with college music activities (e.g., marching band) identified as a primary contributor to

risk of NIHL ([24](#page-9-17)). We found that students whose daily average noise exposure exceeded recommended exposure limits had decreased tolerance to background noise tolerance compared with those below the limit who were, accordingly, at lower risk of NIHL. This previous report ([24](#page-9-17)) did not examine the physiological basis of decreased tolerance, leading us to undertake the current analysis. To our knowledge, the impact of decreased cochlear nerve function on background noise tolerance has not previously been investigated by others. However, some evidence suggests that noise-induced cochlear neuropathy (manifesting as decreased wave I amplitudes) leads to an exaggerated behavioral response to sound at moderate sound levels without changing the later, more central ABR waves [\(28\)](#page-9-18). Drawing from this hyperacusis literature [\(28](#page-9-18)), we hypothesize that decreased background noise tolerance in populations at risk for NIHL is linked to noise-induced cochlear nerve degeneration, leading to reduced ABR wave I in the participants least tolerant of background noise in our dataset.

In our dataset, the tolerated signal-to-noise ratios (SNRs) range from -3.33 to 12.0 dB SNR (N.B., -3.33 dB SNR means that the signal is 3.33 dB lower than the noise and 12 dB SNR means that the signal is 12 dB greater than the noise). Following standard criteria for the ANL test [\(5,](#page-8-4) [29\)](#page-9-19), tolerated $SNRs \leq 6$ dB are considered "high tolerance" for background noise, tolerated SNRs between 7 and 15 are considered "medium tolerance," and tolerated $SNRs > 16$ dB are considered "low tolerance." Based on these criteria, 25% of our cohort fell in the "medium tolerance range" and 75% fell into the "high tolerance." None fell into the "low tolerance range." This contrasts with the studies by Harkrider and Tampas reviewed earlier, which focused on the "low" range in comparison to the "high" range and only involved female listeners. To our knowledge, there have not been any investigations into the physiological signatures of "medium" background noise intolerance (a range intermediate to low and high), nor has there been an analysis of the signatures of those who are most tolerant ("hyper-tolerant"). The current investigation addresses both these gaps.

In addition to studying the link between noise injury and medium tolerance for background noise, we build from the Tampas and Harkrider [\(5,](#page-8-4) [6\)](#page-8-5) studies to ask whether CAS gain could serve as a potential mechanism underlying hypertolerance for background noise in healthy adults with normal audiometric thresholds at low risk of NIHL. To test the predicted relationship between CAS gain and hypertolerance, the current investigation cleaves the high tolerance range into two groups to create a subcategory of high tolerance called "hyper tolerance". We compare ABR wave amplitudes for the three groups (hyper, high, and medium background noise tolerance).

MATERIALS AND METHODS

Experimental procedures were approved by the Institutional Research Board (IRB) at the University of Connecticut. Participants provided their written informed consent before study enrollment. The study occurred during the academic semester (Spring 2017), when academic, enrichment, and employment activities were ongoing.

Following a mandatory 14-h quiet period, participants came to the laboratory for audiological threshold testing and electrophysiological assessments (ABRs). For an objective measurement of noise exposure, participants also completed 1 wk of personal noise dosimetry. Noise dosimetry measurements began immediately after in-laboratory testing and lasted for 168 continuous hours, spanning eight calendar days. The battery-powered noise dosimeters have a memory load that caps at 168 h, motivating our decision to adopt a weeklong protocol. Our previous work has also shown that 1 wk provides a representative sample of current noise exposure [\(30\)](#page-9-20).

Participants

Participants were 56 college students aged 18–24 yr. All participants were native speakers of American English with clinically normal hearing bilaterally (i.e., air conduction audiometric thresholds \leq 25 dB HL for octave frequencies from 0.25–8 kHz) and speech perception in noise within the clinically normal range as measured by the Quick Speech in Noise (QuickSIN, Etymotic, Inc.) test $(\leq 3$ dB SNR Loss). Of the 56, 21 were participating in collegiate music ensembles at the time of testing. All but two college musicians were active in the University of Connecticut Marching Band, which has over 300 members. The other 35 participants were not active in music activities at the time of testing but had a range of experience playing a musical instrument (0–18 yr).

Hearing Thresholds

Audiological threshold testing was performed in a singlewalled sound booth. Air conduction thresholds were obtained for the right and left ears at 0.125, 0.25, 0.50, 1, 1.5, 2, 3, 4, 6, and 8 kHz using insert earphones with a clinical audiometer (GSI 61 Audiometer, Grason-Stadler Inc.). If thresholds at 0.25, 0.50, 1, 2, or 4 kHz were >5 dB HL, bone conduction thresholds were obtained at those frequencies, and tympanometry was used to assess possible conductive pathology. Air-bone gaps >15 dB at two or more adjacent frequencies or abnormal tympanograms would have resulted in exclusion from the study due to possible middle-ear pathology; however, it was not necessary to exclude any of the participants based on these criteria.

The Acceptable Noise Level Test

Tolerated signal-to-noise ratios (SNRs) were measured in a single-walled sound booth using the acceptable noise level (ANL) test [\(31\)](#page-9-21). The ANL test uses the Arizona Travelogue Davy Crockett Passage (Cosmos, Inc.) as the target speech signal and eight-talker speech babble as background noise. In our implementation, the test stimuli were delivered from a CD (Frye Electronics, Inc., Beaverton, Oregon) via a GSI 61 audiometer to a single speaker located in a sound booth. Participants were seated 1 m from the speaker at 0° azimuth and were verbally instructed on the test procedure using instructions adapted from Nabelek et al. and the test distributor (Frye Electronics). Consistent with administration in other ANL studies [\(32,](#page-9-22) [33](#page-9-23)), SNR adjustments were requested by the participant using a thumbs-up signal to increase the level, thumbs-down to decrease the level, and flat palm signals to stop adjustments.

Verbal instructions were given in steps:

- 1) You will listen to a story through the speaker. After a few moments, select the loudness of the story that is most comfortable for you, as if listening to the radio. The thumbs-up and thumbs-down gestures will allow you to make adjustments. First, turn the loudness up until it is too loud and then down until it is too soft. Finally, select the loudness level that is most comfortable for you.
- 2) You will listen to the same story with background noise of several people talking at the same time. After you have listened to this for a few moments, select the level of background noise that is the MOST you would be willing to accept or "put up with" without becoming tense and tired while following the story. First, turn the noise up until it is too loud and then down until the story becomes very clear.
- 3) Finally, adjust the noise (up and down) to the MAXIMUM noise level you would be willing to "put up with" for a long time while following the story."

In the standard implementation of this test, the target speech signal is played at the listener's most comfortable level (MCL). We followed this standard here. MCL is found by increasing the level of the speech passage from 30 dB HL in 5-dB steps until the participant signals to the experimenter that the MCL has been reached. As the speech passage continues to be played at MCL, the background noise level (BNL) is found by increasing the level of the background babble from 30 dB HL in 5-dB steps and bracketing in 2-dB steps until the participant indicates they have reached the maximum level of background noise they are willing to tolerate while still following the speech passage. The "tolerated SNR" is calculated as the difference between the MCL and BNL (tolerated $SNR = MCL - BNL$). In our dataset, this procedure was completed three times, and the trials were averaged to calculate the final tolerated SNR value.

"Tolerated SNR" is defined as a measure of how much background noise the listener "would be willing to accept or 'put up with' [i.e., tolerate] without becoming tense or tired while following the story" [\(34\)](#page-9-24). The test developers acknowledge that the term "tolerance" is often associated with loudness discomfort levels measured for a single stimulus, such as a pure tone, and that it is also used in describing hyperacusis. Therefore, in the interest of clarity, we emphasize that the term "tolerated SNR," when used in the current study, refers to the specific aspect of tolerance being measured by the ANL test, as described in the instructions above. For greater transparency, we have also chosen to refer to the final score as "tolerated SNR" instead of "ANL score" [\(5](#page-8-4), [6](#page-8-5), [32\)](#page-9-22).

In our dataset here, tolerated SNRs ranged from –3.33 to 12 dB SNR. Using common criteria in the literature, "high tolerance" was defined as tolerated SNRs \leq 6 (i.e., tolerating background noise up to 6 dB lower than the story) and "low tolerance" was defined as tolerated SNRs \geq 16 (i.e., tolerating background noise when it at least 16 dB lower than the story) [\(5,](#page-8-4) [29\)](#page-9-19). Within our full dataset described previously [\(24\)](#page-9-17), 41 participants (31 females) met the criteria for having high tolerance for background noise, and 15 participants (12 female) had "medium" tolerance, falling between the low and high categories. We further subdivided the high tolerance category

into two, using a tolerated SNR of 2 as the cutoff, with tolerated SNRs < 2 being classified as "hypertolerant," a subcategory of the high tolerance range. The hypertolerance group had tolerated SNRs from -3.33 to 1.33 ($n = 18$, 4 males, 4 college musicians). In this analysis, we reserve "high tolerance" to label listeners with tolerated SNR from 2 to 6 ($n = 23, 5$) males, 7 college musicians). The $<$ 2 dB SNR cutoff was selected as the cutoff for the hypertolerance group because 2 dB SNR is the level at which a person with normal hearing is expected to correctly recognize 50% of the keywords in the QuickSIN test (i.e., the expected threshold for speech understanding). The medium tolerance group had tolerated SNR ranges from 6.33 to 14 ($n = 15$, 2 males, 10 college musicians).

Auditory Brainstem Responses

ABRs were recorded using a clinical ABR system (the Biologic AEP, Natus, Inc.) using standard protocols and Ag-AgCl electrodes arranged in a vertical, ipsilateral montage. Recordings were made with the participant sitting reclined in a double-walled, electromagnetically shielded sound booth. After cleaning and scrubbing the skin, the noninverting electrode was placed on the central vertex of the head (Cz), the inverting electrode was placed on the right earlobe (A2), and the ground electrode was placed on the forehead. Contact impedance was \leq 5 k Ω for all electrodes during the recording.

ABRs were recorded to 100-ms rarefaction clicks presented at 75 dB nHL at eight presentation rates (3.4, 6.9, 10.9, 15.4, 31.25, 46.5, 61.5, and 91.25 Hz). This intensity was measured as 106.7 peSPL from the output of the ear insert using a 2-mL coupler attached to a 2250 Light-G4 B&K sound level meter. Because all participants demonstrated symmetric audiometric thresholds, only the right ear was stimulated to reduce testing time. The first presentation rate recorded was 31.25 Hz; normative data exist for this rate for the Bio-logic AEP system [\(35](#page-9-25)) allowing the experimenter to confirm the quality of the recordings and make any necessary adjustments before beginning the full ABR protocol. After the 31.25-Hz condition, the stimulus rates were presented from slowest to fastest. The current analysis focuses on the slowest five rates for which the waveform morphology is most stable across rates and therefore most directly comparable, and where wave I is most reliably detected ([22,](#page-9-13) [36](#page-9-26)).

Responses were digitally sampled at 24 kHz and filtered online from 100–1,500 Hz. Artifact rejection using a $|23.8|$ mV criteria was used, and responses were averaged online over a 10 ms window that included 0.8 ms before stimulus onset. Two subaverages of 1,000 artifact-free trials per rate were averaged as a calculated waveform. During recordings, participants watched a self-selected, muted video with English captions as they sat in a reclined position. The video was projected onto the wall of the dimmed sound booth, using an LCD projector placed outside the booth window.

The broadband click stimulus was chosen for this study because it elicits a highly stereotyped response characterized by a series of waves. Wave I of the ABR originates from activity within the cochlear nerve and occurs at a latency of 1–2 ms for suprathreshold stimulation. Wave V is the most prominent and reliable wave, and its generators are within the lateral lemniscus and inferior colliculus, with an approximate

latency of 5–6 ms [\(37\)](#page-9-27). Though waves I and V were our primary focus in designing the study, the other intervening waves were also labeled to ensure accuracy in identifying waves I and V. We include wave III in the analysis to better isolate the neurobiological locus of any potential differences noted for wave V. For each rate, the ABR waves were identified on the calculated waveform by the experimenter at the time of testing, and their locations were later confirmed by two additional raters, including the first author, who were blind to the participants' tolerated SNRs and noise exposure data while visually inspecting the ABR waves. Custom MATLAB programs extracted wave amplitudes (peak-tobaseline amplitudes) from the hand-marked latencies. Waves I, III, and wave V were present in all participants at the five slowest rates. To overcome the inherent variability of ABR amplitude [\(38\)](#page-9-28), we averaged the amplitudes across these five slowest rates, and the analysis was performed on the average wave I amplitude, average wave III amplitude, average wave V amplitude, and the average CAS gain (V-avg/ $I-avg$).

Noise Dosimetry Protocol

After completing the auditory test battery, participants were trained to use a noise dosimeter (ER-200DW8 personal noise dosimeter; Etymotic, Inc.). They were instructed to wear it attached to their clothing near the ear, with the microphone inlet uncovered. They were told that they could remove the dosimeter when sleeping or showering, or during activities when the devices might be damaged (e.g., sports), but that they should keep it nearby, if possible. The power button was disabled by the experimenter so that participants could not accidentally shut off the dosimeter. Participants were asked to contact the research team if any issues relating to the dosimeter arose during the week. At the end of training, the experimenter turned on the dosimeter and immediately recorded the start time. Participants were scheduled to return in no less than 1 wk (\sim 168 h) to hand in the dosimeter and activity log and to receive monetary compensation for their participation in the study. In the activity logs, participants reported only minimal use of hearing protection devices such as earmuffs or earplugs.

The dosimeters were set to an 85-dBA criterion level, 3-dB exchange rate, and 75-dBA threshold, in conformance with the National Institute for Occupational Safety and Health criteria (NIOSH, 1998). They logged dose data in 3.75-min increments throughout the entire measurement period. The calibration of all dosimeters was checked periodically to ensure that the devices were operating properly. Calibration was done by generating a continuous 1,000-Hz narrowband signal at a nominal level of 90 dB SPL in an Audioscan Verifit test box and measuring its level with a calibrated type 1 sound level meter (Larson-Davis 824). The level was then measured using each dosimeter in "QuickCheck" mode. The microphones of the devices were positioned at the same location in the test box. Measured dosimeter levels fell within 2.5 dB of the average of three type 1 sound level meter measurements.

At the end of the recording period, each participant's dosimetry data were downloaded to a .txt file using the ER200D Utility Suite software (v. 4.04) and then processed individually

	Pure Tone Hearing Thresholds, dB HL $F(2,53) = 1.10 P = 0.34$, $n^2 = 0.04$			Daily Average Noise Dose, % $F(2,53) = 1.31, P = 0.28,$ $\eta^2 = 0.05$			QuickSIN score, dB SNR Loss $F(2,53) = 3.91, P = 0.03,$ $n^2 = 0.13$			Most Comfortable Listening Level, dB HL $F(2,53) = 1.95, P = 0.15,$ $\eta^2 = 0.07$			Years of Playing a Musical Instrument $F(2,53) = 3.85$, $P = 0.03$, $n^2 = 0.18$		
	Hyper	High	Medium	Hyper	High	Medium	Hyper	High	Medium	Hyper	High	Medium	Hyper	High	Medium
Mean	2.22	2.30	2.54	178.94	147.63	293.71	0.38	0.88	1.05	41.37	44.67	50.18	7.86	5.89	10.60
Std. Deviation	0.62	0.65	0.61	300.13	210.65	336.69	0.85	0.64	0.75	12.50	13.30	12.42	5.91	4.75	4.60
Minimum	1.32	1.22	0.94	5.62	0.66	10.70	-1.25	-0.25	0.50	22.67	22.67	18.00	0.00	0.00	3.00
Maximum	3.29	3.70	3.45	902.44	779.41	884.63	.75	2.25	3.00	63.33	72.67	68.67	17.00	15.00	20.00

Table 1. Descriptive statistics for daily average noise dose, pure tone hearing thresholds, QuickSIN, most comfortable listening level, and years of playing a musical instrument

Values are mean, standard deviation, minimum, and maximum for the hyper $(n = 18$ participants), high $(n = 23$ participants), and medium ($n = 15$ participants) tolerance groups. Group means were compared via ANOVA (\hat{F} , P, and effect sizes) and are reported below the header line.

using an in-house MATLAB routine that separated the data by date, using the dosimeter start time recorded by the investigator. The noise dose for each date was calculated using National Institute for Occupational Safety and Health (NIOSH) criteria. Finally, doses were averaged across days to derive the average daily noise exposure dose over the course of the measurement week. Individuals routinely exposed to noise over 100% of the recommended exposure limit (REL) are considered at risk for NIHL, but risk cannot be ruled out in cases of routine exposure to lower-level sound or even single exposures to high-level sound.

Statistical Analysis

In our previously published analysis of this dataset [\(24](#page-9-29)), we grouped participants based on their risk level for noiseinduced hearing loss using 1 wk of personal noise dosimetry data as a representative measure of their routine noise exposure. We found that participants above the NIOSH daily recommended exposure limit (REL) were less tolerant of background noise than those below the REL. Participation in collegiate music activities was the primary source of noise exposure. From these initial findings, we hypothesized that high noise exposure levels may lead to incipient noiseinduced cochlear damage that manifests as lower tolerance for background noise, even before hearing thresholds are affected. The current analysis takes this same dataset from the perspective of the tolerated SNRs, grouping participants into three categories of background noise tolerance: hypertolerance, high tolerance, and medium tolerance.

Statistical analysis was carried out in MATLAB and/or JASP. The level of statistical significance was $P = 0.05$. The primary dependent variables were ABR wave I amplitude, wave III amplitude, wave V amplitude, V/I ratio (CAS gain). A repeated-measures ANOVA was conducted to examine

whether the three groups (dependent variable) showed different patterns for waves I, III, and V amplitudes (withinsubjects factor), with Greenhouse Geiser correction and two covariates. Given that background noise tolerance is impacted by the presentation level of the target stimulus ([6](#page-8-6), [7](#page-9-0), [39,](#page-9-30) [40](#page-9-31)), and musical training may positively influence speech perception in noise ([25,](#page-9-32) [41](#page-9-33), [42\)](#page-9-34), MCL and total years of playing a musical instrument (including voice) were added as covariates. Groups were also compared with respect to daily average noise dose (log-transformed to achieve a normal distribution).

Previous studies of the physiological signatures of tolerated SNR have included listeners with high tolerance for background noise but have not focused on listeners who have medium or hypertolerance. This motivated a post hoc exploration that compared, in a pair-wise fashion, the high tolerance group to each of the other two.

RESULTS

See [Tables 1](#page-4-0) and [2](#page-4-1) for mean, standard deviations, and statistical values. A comparison of the three groups shows that the three noise tolerance groups did not differ with respect to hearing thresholds, i.e., there was no main effect of group with all participants falling in the clinically "normal" hearing range [\(Table 1\)](#page-4-0). A visual inspection of the data shows that the medium group had higher daily average noise doses on average [\(Fig. 1](#page-5-0)A), with the other two groups being similar. However, there was not a significant group difference at the $P < 0.05$ level for the three groups ([Table 1](#page-4-0)), likely because of one participant in the hyper group with very high noise doses (above 900% of the REL). Yet, there was a statistically significant group difference for the QuickSIN test and years of playing a musical instrument. Consistent with the medium tolerance group being at higher risk for NIHL than the other

Table 2. Descriptive statistics for auditory brain stem response wave amplitudes and central gain (V/I ratio)

		Wave I amplitude, µV		Wave III amplitude, µV				Wave V amplitude, µV		V/I Ratio		
	Hyper	High	Medium	Hyper	High	Medium	Hyper	Hiah	Medium	Hyper	High	Medium
Mean	0.20	0.19	0.14	0.28	0.24	0.27	0.10	0.17	0.12	0.49	0.98	0.54
Std. Deviation	0.07	0.08	0.10	0.10	0.11	0.08	0.11	0.11	0.11	0.52	0.67	1.44
Minimum	0.08	0.07	-0.07	0.13	0.07	0.11	-0.12	0.02	-0.05	-0.64	0.14	-2.82
Maximum	0.32	0.36	0.25	0.44	0.55	0.42	0.30	0.38	0.32	1.47	3.00	3.50

Values are mean, standard deviation, minimum, and maximum for the hyper $(n = 18$ participants), high $(n = 23$ participants), and medium ($n = 15$ participants) tolerance groups. V/I ratio, comparison of wave I to wave V.

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Figure 1. Group comparisons for noise exposure (average daily noise dose %, log scale; A), QuickSIN [signal-to-noise ratio (SNR) loss; B], most comfortable listening (MCL) level (dB HL) (C), and years of playing a musical instrument (D). Hypertolerant listeners performed best on the QuickSIN and trended toward a lower MCL. Medium tolerance listeners trended toward higher noise exposure doses, poorer QuickSIN performance (albeit within the normal range), and slightly higher MCLs. Bars represent group means for the hyper (red, $n = 18$ participants), high (black, $n = 23$ participants), and medium (blue, $n = 15$ participants) tolerance groups, with error bars representing one standard error of the mean.

groups, they also had worse speech perception in noise on average on the QuickSIN test ([Fig. 1](#page-5-0)B), matching the predicted patterns of peripheral noise injury observed by others [\(16](#page-9-35)). Recall, however, that all participants had QuickSIN scores and audiometric thresholds within the clinically normal range, suggesting that this injury is "hidden" when using common clinical cutoffs. For the medium group, this injury could result from greater noise exposure across their lifetime due to having been a musician for a longer period. In addition to the main effect of group for QuickSIN and years of playing a musical instrument, the MCL trended up across the three groups, with the medium tolerance group having the highest MCLs [\(Fig. 1](#page-5-0)C).

Turning now to the ABR measures, a RMANOVA showed a significant wave-by-group interaction $[F(3.86, 98.523) = 3.11,$ $P = 0.02$, $\eta_{\text{p}}^2 = 0.109$, as well as a significant main effect of wave $[F(2, 102) - 3.63 P - 0.031 n^2 - 0.06]$ but no main wave $[F(2, 102) = 3.63 P = 0.031, \eta_{\text{p}}^2 = 0.06]$ but no main
effect of group $[F(2, 51) - 1.07 P = 0.35 \eta^2] = 0.04$ effect of group $[F(2, 51) = 1.07, P = 0.35, \eta_{p}^{2} = 0.04]$. There were also no main effects or interacting effects for either MCL $[F(1, 51) = 0.11, P = 0.74, \eta^2 p = 0.002]$ or total years of playing a musical instrument $[F(1, 51) - 2.105, P - 0.15, \eta^2]$ playing a musical instrument $[F(1, 51) = 2.105, P = 0.15, \eta^2]_p = 0.041$ so they were dropped from the post boc analyses 0.04], so they were dropped from the post hoc analyses.

The interaction between wave and group led us to conduct post hoc tests for main effects of group for each wave. The main effect of the group was trending for wave I amplitude $[F(2, 53) = 2.316, P = 0.11, \eta^2_p = 0.08]$, wave V
 $[F(2, 53) = 2.55, P = 0.09, \eta^2 = 0.09$ and central gain $[F(2, 53) = 2.55, P = 0.09, \eta_{p}^{2} = 0.09]$, and central gain
 $[F(2, 53) = 1.53, P = 0.16, \eta_{p}^{2} = 0.067]$. No main effect of $[F(2, 53) = 1.53, P = 0.16, \eta^{2}]_{P} = 0.067$. No main effect of group was found for wave III amplitude [$F(2, 53) = 0.63$] group was found for wave III amplitude $[F(2, 53) = 0.63,$ $\overline{P} = 0.64$, η^2 _p = 0.53].
A visual inspection

A visual inspection of the group means [\(Fig. 2](#page-6-0) and [Table 2\)](#page-4-1) indicates that for wave I, the medium tolerance group's peak amplitude is reduced on average compared with the high and hypertolerance groups, whose amplitudes are similar. At wave III, the three groups' amplitudes are similar on average. At wave V, the high tolerance group has a higher average amplitude than the medium and hypertolerance groups, whose amplitudes are like each other. As expected from the patterns for I and V, central gain (V/I) is higher on average for the high group but similar across the other two groups. Planned post hoc analyses compared the high tolerance group to each of the other two groups in a pair-wise fashion.

These post hoc analyses focused on wave I amplitude, wave V amplitude, the V/I ratio, and daily average noise dose; wave III amplitude was excluded from the analyses because the main effect of group was not significant or trending for wave III. Compared with the high tolerance group, the hyper tolerance group had reduced wave V amplitudes $[t(39) = -2.17, P = 0.04, Cohen's d = -0.68]$ and reduced central gain [V/I ratio: $t(39) = -2.59 = -2.49$, $P = 0.01$, Cohen's $d = -0.81$] but the groups were matched for wave I amplitude $[t(39) = -0.816, P = 0.77, \text{Cohen's d} = 0.09, \text{all}$ uncorrected P values]. These two groups were also matched for daily average noise dose $[t(39) = 0.03, P = 0.98, \text{ Cohen's}$ $d = 0.32$. When the high tolerance group was compared with the medium group, the medium group trended toward having daily noise doses and reduced having reduced ABR amplitudes [daily average noise dose: $t(36) = 1.72$, $P = 0.09$, Cohen's $d = 0.57$; wave I amplitude: $t(36) = 1.7$, $P = 0.1$, Cohen's $d =$ 0.56; wave V amplitude: $t(36) = 1.4$, $P = 0.14$, Cohen's d = 0.49; V/I central gain: $t(36) = 1.3$, $P = 0.2$, Cohen's d = 0.43]. In the case of V/I, this could be because of the large standard deviations of the medium group.

The results above suggest a complex, nonlinear relationship between tolerated SNRs and auditory function, as measured by ABR wave I and wave V. For the subset of participants tolerated SNRs < 6 (i.e., the hyper and high tolerant listeners), there is a linear relationship between central auditory activation and background noise tolerance, such that tolerance increases as wave V amplitude and the V/I ratio decreases (Pearsons' correlation wave V: $r = 0.40$, $P =$ 0.005; V/I ratio: $r = 0.38$, $P = 0.02$). There is no linear relationship between tolerated SNRs and wave I in this subset $(r = 0.14, P = 0.38)$ or when the full dataset is considered (wave V: $r = 0.14$, $P = 0.27$; wave I: $r = -010$, $P = 0.46$; V/I ratio: $r = 0.08$, $P = 0.58$].

DISCUSSION

Listening to speech in noisy backgrounds is part of everyday human communication; however, most studies focus on speech recognition in background noise rather than the listener's tolerance for background noise. Studies of background noise tolerance are important because they help

Figure 2. Top: group comparisons for ABR wave I amplitude (A), ABR wave III amplitude (B), ABR wave V amplitude (C), and central gain (D). Bars represent group means for the hyper (red, $n = 18$ participants)), high (black, $n = 23$ participants), and medium (blue, $n = 15$ participants) tolerance groups, with error bar representing one standard error of the mean. Bottom: group average ABR waveforms. To optimize the ability to compare the amplitude ABR waveforms for the three groups visually, waveforms were aligned with respect to wave V latency before generating the group average waveforms. ABR, auditory brainstem response.

yield insight into understanding human communicative behaviors and outcomes. For instance, they may help to explain why some people avoid certain auditory environments or have less success with assistive listening devices ([4\)](#page-8-2). It is also likely that if a person has reduced tolerance for background noise, they may not even bother to try to understand what someone is saying. In this way, background noise tolerance may be an antecedent to recognition.

To better understand the peripheral and central processes that may underlie individual differences in noise tolerance, we compared ABRs in listeners who were grouped based on their tolerated SNRs, building from smaller-scale studies that compared auditory evoked potentials in listeners with high versus low tolerance [\(5,](#page-8-3) [6\)](#page-8-6). Our findings suggest distinct physiological underpinnings of hypertolerance and medium tolerance: hypertolerance of background noise is associated with reduced levels of CAS gain (smaller V/I ratios) and medium tolerance is associated with higher risks of NIHL and trends toward reduced peripheral and central output (i.e., reduced ABR amplitudes).

In two small studies involving only female listeners, Tampas and Harkrider found elevated wave V amplitudes in listeners with low tolerance for background noise, compared with those with high tolerance. Yet, no group differences for wave I or suprathreshold OAEs were observed. The authors concluded that low tolerance to background noise could arise from central auditory system hyperactivity in female listeners with normal cochlear function. In other words, they concluded that more gain is associated with less tolerance.

Building on these preliminary findings, we applied a similar logic to investigate the possibility that hypertolerance to background noise is associated with reduced levels of central gain (i.e., CAS hypoactivity). We stratified young adult listeners within the previously defined high tolerance range to separate out those with the very highest tolerance, who we operationally defined as being hypertolerant of background noise. Wave I amplitude did not differ between the subgroups but wave V amplitude and central gain did (smaller V and V/I ratios in the hypertolerance group). These two variables (wave V and V/I ratio) also correlated significantly with tolerated SNR in this subset of participants. When melded with the work by Harkrider and Tampas, our findings suggest that CAS gain could serve as a potential mechanism underlying both hypertolerance and hypotolerance (i.e., reduced tolerance) for background noise in listeners at low risk for noise damage to peripheral function, with hypertolerance being the result of reduced levels of central gain (i.e., hypoactivation) and hypotolerance being the result of increased levels of central gain (i.e., hyperactivation). These differential levels of central activation appear to arise first in the midbrain (wave V), as differences were not noted for wave III here or in the Hakrider and Tampas work in groups matched with respect to wave I amplitude. In cases with minimal or no noise damage to cochlear function, differences in midbrain activation may be mediated by individual differences in the expression of GABA, an inhibitory neurotransmitter ([43\)](#page-9-36). However, we also acknowledge other possibilities: differences in central

activation could reflect differences in neural synchrony or anatomical variations that shape the signal detected at the scalp.

Important takeaways from our findings are that the V/I metric must be interpreted in the context of the amplitude of the individual waves, and the individual waves may show complex nonlinear relationships with respect to tolerated SNRs. To help illustrate the nature of this complexity, we transformed our data into a second-degree polynomial and overlaid the best-fit lines for waves I and V ([Fig. 3\)](#page-7-0). Although the medium and hypertolerance groups have similar ratios, the medium group has, on average, lower ABR wave I and V amplitudes. However, the group differences are not statistically significant and should be interpreted cautiously. Given that the medium group has higher noise exposure levels, as measured by personal noise dosimetry and more years of playing a musical instrument, these lower amplitudes could potentially evince an early stage of noise-induced damage to peripheral function (reduced wave I and decrease QuickSIN) that has not triggered a compensatory increase in midbrain function and/or is highly variable across individuals in the medium tolerance group. In the medium group, reduced tolerance for background could instead be due to compensatory changes in the auditory cortex ([10,](#page-9-37) [44\)](#page-9-38). Given that wave III is quite robust in this group [\(Fig. 1](#page-5-0)), it is also possible that reduced tolerance could also arise from a compensatory increase in gain at the cochlear nucleus that is then modulated down in the midbrain. Both interpretations are speculative at this point.

In cases where tolerated SNR cannot be explained by peripheral differences (i.e., wave I) or differences in noise exposure risk, what exogenous and endogenous factors might account for atypical levels of CAS gain? One school of thought is that tolerated SNR is an inherent psychological quality potentially tied to personality or self-control ([45](#page-9-39)). This hypothesis is supported by studies showing high test-retest reliability of noise-tolerance levels over durations as long as a year [\(46](#page-9-40)). Others propose that tolerated SNR is linked to CAS arousal, with evidence that

Figure 3. Hypothesized relationship between central auditory system (CAS) gain and background noise tolerance. SNR, signal-to-noise ratio.

tolerated SNRs can fluctuate with arousal state. For instance, studies using within-subject designs have shown individuals exhibit higher tolerance when under the influence of prescribed stimulants compared with when they are unmedicated [\(7](#page-9-0)). Similarly, short-term caffeine consumption has been associated with increased background noise tolerance ([47](#page-9-41)). However, ABR studies are at odds with this explanation; ABR data show increased wave V amplitudes, not decreased, after a similar time window following a comparable level of caffeine consumption [\(48](#page-9-42)) (wave I did not change).

Environmental sound conditioning is another proposed explanation for individual differences in central gain and behavioral noise tolerance. Short-term sound deprivation, resulting from using earplugs, can induce temporary changes in central gain as measured by acoustic reflexes and ABRs ([49](#page-9-43)–[51](#page-10-0)). Brotherton et al. [\(43](#page-9-36)) reported short-term changes to wave V amplitude but not wave I following 4 days of monaural auditory deprivation, suggesting that central gain is downregulated in loud or noisy environments, leading to greater sound tolerance. Sound desensitization training, in which the background noise intensity is slowly increased over a period of days, has also been reported to increase tolerance for background noise in listeners with low tolerance, with a mean change in tolerance of 9.95 dB SNR on the ANL test [\(52](#page-10-1)). Comparisons of listeners with high and low tolerance provide further evidence for the role of sound conditioning ([29](#page-9-44)). Franklin et al. found that listeners with high tolerance for background noise spent more time in environments with background noise than those with low tolerance of background noise (ANL scores between 15 and 20 dB SNR) ([29](#page-9-44)). This was gauged using the ANL test and a body-worn recording device called an Environmeter, which tracked how much time the wearer spent in different auditory environments (i.e., environments containing only noise, only speech, speech in noise, or quiet). In that study, environmental sound levels were not different between the two groups. However, a limitation of the Environmeter is that its upper level of sensitivity is 80 dB SPL, which means the level for a 100 dB SPL environment, for example, would be coded identically as an environment with an 85 dB sound level. This sensitivity contrasts with the personal noise dosimeters used in our study, which are sensitive up to 130 dB. In our study, the high and hyper groups did not differ in their daily environmental sound levels, suggesting that they might have similar short- and long-term sound exposures. The two groups also reported playing a musical instrument for a similar amount of time, suggesting potentially similar musicrelated benefits (Of course, two people with similar total years could have very different levels of weekly play, making years of practice an imperfect measure). Also, unlike the Franklin et al. study, we do not have data on how much time the listeners spent in different environmental signal-to-noise ratios—only the gross sound intensity of their environmental exposure. Another limitation of our dosimetry protocol is that it was limited to 1 wk and, therefore, is unlikely to represent lifetime noise exposure fully. Our recent work found close correspondence in noise exposure across 3 wk sampled over 6 mo, suggesting that 1 wk is a decent proxy of current exposure levels [\(30](#page-9-45)). One week also exceeds the measurement duration of other studies comparing sound exposure to ANL or ABR profiles $(18, 29)$ $(18, 29)$ $(18, 29)$ $(18, 29)$.

Factors other than daily environmental sound levels, playing a musical instrument, and peripheral and central auditory function that might have influenced participants' ANL test results can be speculated on but not tested, as information about variables such as personality traits and stimulant consumption were not collected. We also acknowledge the unknown impact that the pretest quiet period might have played in our measurement of tolerated SNR. All auditory testing, including ANL testing and ABRs, was completed following a mandatory 14-h quiet period, during which participants were asked to refrain from any loud activities. This quiet period was incorporated into the study design to minimize the influence of temporary threshold shifts, especially for roughly one-third of our sample participants who regularly engaged in loud activities such as collegiate music ensembles. Adherence to this quiet period was based on self-report. For listeners with the highest environmental sound levels, this quiet period may have deviated significantly from their typical sound exposures, and this short period of quiet could have influenced their tolerated SNR. Because the personal noise dosimetry was administered after all auditory testing (and not before), it is also possible that the two groups might have had different environmental sound levels during the week before the ANL test was administered. Ongoing investigations in the laboratory are addressing these open questions. The relationship between tolerated SNR and speech recognition is also not entirely clear for this dataset, as we did not measure speech recognition at the tolerated SNR. However, using the QuickSIN test, we did find that speech recognition in noise was not the same across the three groups, with the hypertolerance group having the best scores and the medium tolerance group having the lowest scores on average. That said, there are hints in the literature that speech intelligibility contributes more to background noise tolerance for listeners with lower compared with higher background noise tolerance (53) (53) (53) .

In our study, the group defined as "hypertolerant" was tolerant of background noise levels that either were close to the level of the target speech passage $(\sim\!\!1$ dB below the speech) or exceeded the speech level, with the most tolerant listeners exhibiting a tolerated SNR of –3.33 dB. There are previous reports of tolerated SNRs with a negative value [\(29,](#page-9-19) [54](#page-10-3)), although the prevalence is difficult to estimate from how group data are typically presented and the complications of comparing studies with different stimulus sets and instructions [\(55](#page-10-4), [56\)](#page-10-5). That said, it is premature to consider –3.33 dB to be the nadir for the ANL test, and we leave open the possibility that future research will reveal a rarer subcategory of listeners even more tolerant of background noise than we reported in our study. Indeed, in a large study of older adults $(n = 264)$ using Australian English materials, a small number of participants ($n = 7$) had tolerated SNRs below this, with the lowest being –11 dB SNR [\(57](#page-10-6)). Noise tolerance may also interact with factors such as talker effects, speaking style, test language/dialect, characteristics of the background noise, and interactions between factors. As such, we consider the label "hypertolerant" to be a relative and not absolute label.

Conclusions

To understand the physiological factors that underpin the spectrum of tolerated SNRs observed in listeners with clinically normal hearing thresholds, both peripheral and central activation must be considered. If only one of these dimensions is considered, it may give an incomplete picture. Clarifying the role of peripheral and central functions in background noise tolerance and other contributing exogenous and endogenous factors may reveal strategies for improving the experience of communicating in noise to allow listeners to achieve greater tolerance of background noise in social, educational, and occupational settings.

DATA AVAILABILITY

Data will be made available upon reasonable request.

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DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the authors.

AUTHOR CONTRIBUTIONS

E.S. conceived and designed research; S.P. performed experiments; E.S. analyzed data; E.S. and S.P. interpreted results of experiments; E.S. and S.P. prepared figures; E.S. drafted manuscript; E.S. and S.P. edited and revised manuscript; E.S. and S.P. approved final version of manuscript.

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