

A longitudinal study investigating the effects of noise exposure on behavioural, electrophysiological and self-report measures of hearing in musicians with normal audiometric thresholds

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Highlights

- First longitudinal study of proxy measures of cochlear synaptopathy in musicians.
- Cumulative levels of noise exposure were similar for musicians and non-musicians.
- Measures of hearing remained largely consistent over a 2-year period.
- Noise exposure may impact outer hair cell function in those with normal audiograms.
- Tinnitus symptoms may improve with hearing conservation behaviours.

Abstract

Musicians are at risk of hearing loss and tinnitus due to regular exposure to high levels of noise. This level of risk may have been underestimated previously since damage to the auditory system, such as cochlear synaptopathy, may not be easily detectable using standard clinical measures. Most previous research investigating hearing loss in musicians has involved cross-sectional study designs that may capture only a snapshot of hearing health in relation to noise exposure. The aim of this study was to investigate the effects of cumulative noise exposure on behavioural, electrophysiological, and self-report indices of hearing damage in early-career musicians and non-musicians with normal hearing over a 2-year period. Participants completed an annual test battery consisting of pure tone audiometry, extended high-frequency hearing thresholds, distortion product otoacoustic emissions (DPOAE), speech-perception-in-noise, auditory brainstem responses, and self-report measures of tinnitus, hyperacusis, and hearing in background noise. Participants also completed the Noise Exposure Structured Interview to estimate cumulative noise exposure across the study period. Linear mixed models assessed changes over time. The longitudinal analysis comprised 64 early-career musicians (female n = 34; age range at T0 = 18-26 years) and 30 non-musicians (female n = 20; age range at T0 = 18-27 years). There were few longitudinal changes as a result of musicianship. Small improvements over time in some measures may be attributable to a practice/test-retest effect. Some measures (e.g., DPAOE indices of outer hair cell function) were associated with noise exposure at each time point, but did not show a significant change over time. A small proportion of participants reported a worsening of their tinnitus symptoms, which participants attributed to noise exposure, or not using hearing protection. Future longitudinal studies should attempt to capture the effects of noise exposure over a longer period, taken at several time points, for a precise measure of how hearing changes over time. Hearing conservation programmes for “at risk” individuals should closely monitor distortion product otoacoustic emissions to detect early signs of noise-induced hearing loss when audiometric thresholds are clinically normal.

Keywords

Longitudinal study, musicians, noise exposure, cochlear synaptopathy, tinnitus, otoacoustic emissions

Abbreviations

ABR – Auditory brainstem response

CRM – Coordinate response measure

DPOAE – Distortion product otoacoustic emissions

EHF – Extended high-frequency

FDR – False discovery rate

HQ – Hyperacusis Questionnaire

ICC – Intraclass correlation coefficient

LMM – Linear mixed model

NESI – Noise Exposure Structured Interview

OHC – Outer hair cell

PTA – Pure tone audiometry

SESMQ – Situational Communication Management Questionnaire

SNR – Signal-to-noise ratio

SPiN – Speech perception in noise

SRT – Speech reception threshold

THI – Tinnitus Handicap Inventory

TTS – Temporary threshold shift

1. Introduction

Musicians are at risk of noise-induced hearing problems due to regular exposure to high levels of noise (Couth et al., 2019; Di Stadio et al., 2018; Greasley et al., 2018; Jansen et al., 2009; Phillips et al., 2010). Hearing loss and tinnitus has serious implications for musicians, affecting their ability to perform (Vaisberg et al., 2019), and impacting on their careers and quality of life (Greasley et al., 2018; Vogel et al., 2014). The prevalence of noise-induced hearing damage in musicians may have been underestimated previously since signs of hearing damage may not be easily detectable using conventional clinical measures (i.e., pure tone audiometry). More specifically, high levels of noise exposure could lead to cochlear synaptopathy – a loss of synapses between inner hair cells and the auditory nerve – which could lead to tinnitus and difficulties with speech perception in noise (SPiN), while hearing thresholds remain intact (for reviews see Bramhall et al., 2019; Le Prell, 2019a; Shehabi et al., 2022).

Previous studies investigating noise-induced cochlear synaptopathy in musicians have produced mixed findings. For example, Kikidis et al. (2020) reported that musicians have a significantly smaller wave I amplitude of the auditory brainstem response (ABR) compared to non-musicians, consistent with cochlear synaptopathy. Similarly, Grose et al. (2017) showed that individuals with high levels of noise exposure, most of whom were musicians, showed a reduction in the wave I/V ratio of the ABR compared to those with low noise exposure. Liberman et al. (2016) demonstrated that the pre-synaptic summing potential of the ABR and its ratio to the action potential (i.e., wave I) were increased in high-risk individuals (mostly musicians), compared to low-risk, which the authors considered to be consistent with cochlear synaptopathy. In addition, the high-risk individuals in this study showed poorer word recognition in noise performance compared to low-risk. Conversely, Yeend et al. (2017) showed that noise exposure was not related to SPiN for high noise-exposed musicians. A follow-up electrophysiology study showed a moderate negative association between noise exposure and wave I amplitude (Valderrama et al., 2018). However, there was no relation between noise exposure and SPiN, and audiometric thresholds were not controlled for, so these findings could be due to poorer outer hair cell (OHC) function rather than cochlear synaptopathy. Similarly, Washnik et al. (2020) showed a weak negative association between noise exposure and wave I amplitude, although this was only found for low-intensity click stimuli (75 dB nHL) and not a high-intensity click (90 dB nHL), which is not consistent with the view that effects of cochlear synaptopathy should be maintained or enhanced at higher stimulus levels (Furman et al., 2013). Additionally, OHC damage was not controlled for in this study either, so these findings cannot be solely attributed to noise-induced cochlear synaptopathy. Patro and Srinivasan (2023, 2022) have also demonstrated greater stimulus

presentation rate dependent ABR wave I amplitude reductions and wave V latency shifts in musicians compared to non-musicians, but the authors argue that the effects are too subtle or too diffuse to cause perceptual deficits, such as to SPiN. Overall, some studies have shown a relationship between noise exposure and ABR measures, but many more do not, and any perceptual deficits because of noise damage remain unclear. These studies vary in terms of their sample sizes and demographics, measures of interest, and measurement and analysis techniques, and effect sizes are not always reported or cannot be calculated from the information provided. Therefore, it is difficult to directly compare these studies. Note, however, that studies which claim to demonstrate a link between noise exposure and proxy measures of cochlear synaptopathy had relatively small sample sizes (i.e., N range = 34-75), whereas the study which did not show a significant association between noise exposure and cochlear synaptopathy had the largest sample size of the studies reported ($N = 122$; Yeend et al., 2017).

In a recent cross-sectional study with a large participant sample ($N = 123$), we investigated the effects of lifetime noise exposure and musicianship on behavioural (i.e., SPiN), electrophysiological (i.e., ABR) and self-report (e.g., tinnitus, hyperacusis, and hearing in noise) indices of hearing damage (Couth et al., 2020). We found that SPiN and ABR measures were similar between early-career musicians ($n = 76$) and age-matched non-musicians ($n = 47$). Musicians were more likely to report experience of – and/or more severe – tinnitus, hyperacusis and hearing in noise difficulties (consistent with cochlear synaptopathy), but this was not related to noise exposure. One of the possible reasons for these mostly null differences between musicians and non-musicians could be due to the finding that lifetime noise exposure (as measured via self-report; Guest et al., 2018a) was not significantly different between musicians and non-musicians. Although musicians reported higher levels of occupational noise exposure, both musicians and non-musicians reported similar levels of recreational noise exposure, which accounted for most of the total noise exposure for most participants. We suggested that both groups were at an age (18-27 years) and period in life (university undergraduates) where they were experiencing relatively high levels of recreational noise exposure (e.g., concerts and nightclubs). This study emphasised that a cross-sectional design can only capture a snapshot of hearing status in relation to noise exposure, and that a longitudinal study design would provide a better insight into the effects of noise exposure on hearing.

There have only been a small number of studies to measure hearing health longitudinally in musicians. Axelsson et al. (1995) conducted a 16-year follow-up study of hearing thresholds in pop/rock musicians, revealing only very modest increases in hearing thresholds, despite an estimated average noise exposure level of 90-105 dB A for 20-25 hours per week. Kähäri et al. (2001) also conducted a

16-year follow-up study of hearing thresholds in classical musicians, demonstrating that there was no substantial increase in hearing thresholds compared to normative values. Similarly, Behar et al. (2018) measured changes in hearing thresholds over a 5-year period in orchestral musicians, indicating no changes to thresholds in this period. Muller and Schneider (2018) measured audiometric thresholds in military musicians over a 13.3-year interval, revealing an improvement in thresholds <3 kHz, and small increases in 4-8 kHz thresholds that were less than what would be predicted by age-related hearing loss (3 to 8 dB lower than predicted by ISO standards). Collectively, these studies suggest no long-term changes to hearing thresholds in relation to noise exposure in musicians. However, these studies were limited to measuring audiometric thresholds which may not be sensitive to more insidious changes to hearing (e.g., cochlear synaptopathy or subclinical hair-cell damage); are restricted to a single follow-up measure rather than assessing patterns of change over time; and have estimated the effects of prolonged noise exposure on hearing using dosimetry measurements from a small number of rehearsals/performances only (if accounted for at all), rather than accounting for cumulative effects of noise exposure for a range of noisy activities.

To our knowledge, only two studies have explored noise-induced cochlear synaptopathy in humans longitudinally. In both studies, audiometric, electrophysiological, and SPiN measures were conducted pre- and post- exposure to loud recreational events (e.g., a music festival), with both studies reporting that very few participants experienced a temporary threshold shift one day after the event, and there were no permanent changes to hearing function up to one-week post exposure (Grinn et al., 2017; Maele et al., 2021). The authors of these studies suggest that there is little risk of cochlear synaptopathy in young normal-hearing individuals, or that the proxy measures of cochlear synaptopathy are not sensitive enough to detect noise-induced changes. Nevertheless, both studies investigated the effects of noise exposure on hearing for a single noisy recreational activity, and only followed up with participants for a short period (up to one week) after the exposure event.

The aim of the current experiment was to investigate the effects of cumulative noise exposure on behavioural, electrophysiological, and self-reported indices of hearing damage in early-career musicians and age-matched non-musicians over a 2-year period. This included proxy measures of cochlear synaptopathy (e.g., SPiN and ABR wave amplitudes and latencies; wave latencies may provide an alternative measure of synaptopathy to wave I amplitude which can be difficult to capture, and which has been measured and interpreted in different ways in previous research; Mehraei et al., 2016; Skoe and Tufts, 2018), whilst also accounting for changes to audiometric thresholds, extended high-frequency (EHF) thresholds, and OHC function (otoacoustic emissions). We focussed on early-career musicians to determine whether these sub-clinical noise-related effects are detectable even at a

relatively young age when interventions to protect hearing longevity may be vital, such as during this period of intensive musical training at the start of their careers.

2. Methods

2.1. Participants

At the baseline assessment (T0; conducted between Feb 2017 – Sept 2018), 76 early-career musicians and 47 non-musicians were included in the data analysis (for details see Couth et al., 2020). All participants were aged 18 years or older and were native English speakers or were highly fluent in English as second language speakers ($n = 9$; seven musicians). Participants reported no history of ear operations and no neurological conditions or severe head injuries. Musicians were recruited from the Royal Northern College of Music or the University of Manchester and were undertaking – or within one year of completing – a degree (bachelors or masters) in performance-based musical studies, hence all were deemed to be “early-career.” Non-musicians were recruited via the University of Manchester Research Volunteering website and mostly consisted of students and staff members. Participants were invited back to repeat the test battery on two further occasions; +1 year (± 2 weeks) following the baseline assessment (T1; conducted between Feb 2018 – Sept 2019), and +2 years (± 2 weeks) following the baseline assessment (T2; conducted between Feb 2019 – Mar 2020). At T1, 67 musicians and 32 non-musicians returned to complete the test battery, with four participants excluded due to failing the otoscopic examination (three musicians) and four participants excluded due to failing the tympanometric screen (one musician). At T2, 47 musicians and 17 non-musicians returned to complete the test battery¹, with one participant excluded due to failing the otoscopic examination (one musician) and two participants excluded due to failing the tympanometric screen (one musician).

For the longitudinal analysis, only participants who completed at least one follow-up assessment (T1 and/or T2) were included (see 2.4. Data Analysis). Consequently, 94 participants in total were included in the longitudinal analysis; 64 musicians (female $n = 34$; age range at T0 = 18-26 years) and 30 non-musicians (female $n = 20$; age range at T0 = 18-27 years). Musicians had an average of 13.4 years of musical experience at T0 (range = 8-20 years) and started playing music at an average age of 6.9 years (range = 2-13.5 years). Musicians reported an average of 14.0 hours of personal practice per week at T0 (range = 1-35 hours), 13.6 hours per week at T1 (range = 0-36 hours), and 13.4 hours per week at T2 (range = 0-35 hours). Two musicians reported having stopped personal practice at T1, and one reported having stopped personal practice at T2, but all musicians reported engaging in a range of

¹ Note that data collection for this last phase of testing was cut short due to the coronavirus pandemic, which partially explains the higher rate of attrition for T2.

other musical activities across the study period, such as group rehearsals and performances. Sixteen non-musicians reported having at least some musical experience, with these participants reporting an average of 9.0 years of musical experience at T0 (range = 1-22 years) and started playing music at an average age of 12.3 years (range = 4-23 years). Non-musicians with some musical experience reported practicing their instruments on average 0.9 hours per week at T0 (range = 0-7 hours), 0.3 hours per week at T1 (range = 0-4 hours), and 0.1 hours per week at T2 (range = 0-1 hours), and none of these were actively studying music or pursuing a career in music, and so were deemed to be hobbyists. Across all non-musicians, the average years of musical experience was 4.8 years at T0 (range = 0-22 years).

2.2. Procedure

Participants completed all testing sessions at the University of Manchester. Each testing session (T0, T1 and T2) took approximately three hours to complete the full test battery, and each session was completed in a single visit or split over two closely spaced visits. The study was approved by the University of Manchester Research Ethics Committee in accordance with the Declaration of Helsinki 2013. All participants provided informed consent at each visit.

2.2.1. Otoscopy

The ear canal and tympanic membrane were inspected visually to ensure normal anatomy, with no wax occlusion, foreign bodies, active infection, or other contraindications to the subsequent test battery.

2.2.2. Tympanometry

Tympanometry was performed using a GSI Tymptstar diagnostic middle-ear analyser. Tympanic membrane compliances between 0.3 and 1.6 ml and peak pressures in the range -150 to +50 daPa were considered normal (Clark et al., 2007). Five participants (three musicians) had slightly elevated (up to 1.8ml) or low (down to 0.2 ml) compliance which was consistent across all testing sessions that they completed. All five participants had present otoacoustic emissions and met the study level criteria, so were included in the analysis.

2.2.3. Noise exposure

A comprehensive estimate of lifetime noise exposure was obtained at T0 using the Noise Exposure Structured Interview (NESI; Guest et al., 2018a). In brief, the NESI prompts participants to identify noisy occupational and recreational activities (above ~80 dB A) that they have regularly engaged in throughout their lifetime. Noise exposure level is estimated based on vocal effort required to hold a

conversation or typical volume control settings for personal listening devices. For music performance-based activities where it would be difficult or impossible for the participant to talk (e.g., woodwind/brass instruments, singers), participants were instructed to estimate hypothetical vocal effort from the viewpoint of the listener rather than the speaker in those situations (i.e., how loud the communication partner would need to speak for the participant to hear them over their musical instrument). For each noisy activity, frequency, duration, level, and attenuation from hearing protection (if any), are combined to generate units of noise exposure linearly related to total energy of exposure. One noise exposure unit is equivalent to one working year (2080 hours) of exposure at a daily level of 90 dB A (for further details on the NESI see Guest et al., 2018a). Units for occupational, recreational and total noise exposure were log transformed to produce a normal distribution, and participants with at least one log unit of total noise exposure (i.e., equivalent to 10 working years of exposure at 90 dB A) at T0 were classified as having high noise exposure ($n = 34$; 22 musicians), and those with less than one log unit classified as having low noise exposure ($n = 60$; 42 musicians).

For subsequent testing sessions (T1 and T2), the NESI was completed based on the participants' noisy occupational and recreational activities in the period since the previous testing session (i.e., one year), which were added to the units from the previous session to provide cumulative measures of occupational, recreational and total noise exposure. Absolute NESI units were used for the longitudinal analysis rather than log transformed to capture rate of change over time.

2.2.4. Hearing thresholds

Pure-tone air-conduction thresholds were measured at 0.25-8 kHz according to British Society of Audiology recommended procedures (British Society of Audiology, 2018), using a GSI Arrow audiometer coupled to TDH-39 supra-aural headphones with MX41 cushions. Normal hearing thresholds were defined as ≤ 20 dB HL for all frequencies.

At T0, three participants (two musicians) were deemed to have a mild unilateral hearing loss (25-40 dB HL) which was restricted to 8 kHz for all participants. At T1, five participants (four musicians) were deemed as having a mild unilateral loss which was restricted to 8 kHz for three participants, 2 kHz for one participant, and 4 kHz for one participant. At T2, five participants (four musicians) were deemed as having a mild unilateral loss which was restricted to 8 kHz for four participants, and .25 kHz for one participant. For three participants (two musicians), mild unilateral losses were consistent across testing sessions. For other participants, mild unilateral losses may reflect a small amount of variability between sessions (i.e., pure tone audiometry (PTA) test-retest results can vary by approximately 5-10 dB; Schmuziger et al., 2004), where their hearing thresholds may be at or near to normal limits (20 dB

HL). All of these participants were included in the data analysis since i) hearing thresholds were not found to be related to proxy measures of cochlear synaptopathy at T0 (Couth et al., 2020), and ii) the aim of this study was to explore changes to hearing over time, including both clinical and subclinical measures.

EHF thresholds were obtained using a three-alternative forced-choice paradigm via a MATLAB (Mathworks, Inc.) programme. Stimuli were delivered via Sennheiser HDA 200 circum-aural headphones driven by a Creative E-MU 0202 USB external soundcard at a sample rate of 48 kHz. Thresholds were measured at 12 and 16 kHz using 1/3-octave bands of noise centred at each of these frequencies. Steady-state duration was 180 ms, with the addition of 10-ms raised-cosine onset and offset ramps. Stimulus levels (dB SPL) were varied adaptively using a two-down, one-up rule, with four initial turnpoints (6 dB step size) and 10 subsequent turnpoints (2 dB step size). Thresholds were calculated as the average stimulus level of the final 10 turnpoints. Thresholds were obtained for each ear separately, with the order of frequencies (12 and 16 kHz) and test ear (left and right) selected at random.

Participants were seated in a double-walled sound-attenuating booth for both PTA and EHF procedures.

2.2.5. Otoacoustic emissions

OHC function was assessed by distortion product otoacoustic emissions (DPOAEs) using an Otodynamics ILO v6 measurement system. The ILO probe microphone was calibrated with a 2-cc cavity before each use. The frequency ratio of the two primary tones, f_2/f_1 , was 1.22. Responses were recorded for f_2 frequencies between 1 and 6 kHz, with two points per octave. The level of f_2 and f_1 tones was 65 dB SPL and 55 dB SPL, respectively. Ninety-six sweeps were measured for each frequency. DPOAEs were classified as present if the signal-to-noise ratio (SNR) was ≥ 3 dB. On occasions where the DPOAEs were classified as absent, the DPOAE level was set to the estimated system distortion level.

2.2.6. Speech perception in noise

SPiN abilities were assessed using the coordinate response measure (CRM; Bolia et al., 2000). Participants were presented with three concurrent speech utterances of the structure “Ready <call sign> go to <color><number> now”, in which there were eight different call signs, four different colors (Red, White, Blue and Green), and numbers ranging from 1 to 4. Participants were instructed to listen for the speech utterance that contained the call sign “Baron”, and to identify the color and number spoken by that talker using a computer interface.

Masker stimuli were presented at a combined level of 80 dB SPL, and in two spatial configurations; one where the maskers were presented centrally and one where they were spatially offset by -60° and $+60^\circ$ azimuth on either side of centre, as simulated by head-related transfer functions. The target was always presented centrally at a sound level which varied adaptively from trial-to-trial using a one-down, one-up rule, with four initial turnpoints (4 dB step size) and 10 subsequent turnpoints (2 dB step size). Speech reception thresholds (SRTs) were calculated as the average SNR of the final 10 turnpoints. Central and offset masker conditions were presented in separate blocks of trials and presented twice following an ABBA structure, with the offset condition always presented first and with an enforced break halfway through. For each separate testing session (T0-T2), participants were given a short practice consisting of separate blocks of central and offset conditions to familiarise themselves with the procedure.

Stimuli were delivered via Sennheiser HD 650 circum-aural headphones driven by a Creative E-MU 0202 USB external soundcard at a sample rate of 44.1 kHz. The test was conducted in a double-walled sound-attenuating booth.

2.2.7. Electrophysiology

At T0, ABRs were recorded using an ICS Chartr EP 200 (Optometrics-Natus) clinical system using ER3 insert earphones. Due to a product recall of the ICS Chartr EP 200 system in June 2019, recordings switched to an Interacoustics Eclipse clinical system. This meant that at T1, two participants (both non-musicians) had ABRs recorded using the Eclipse system, and at T2, 26 participants (20 musicians) had ABRs recorded using the Eclipse system. Identical montage configurations and recording parameters were used for both systems. A single-channel vertical montage configuration was used with the active electrode placed at Fz (high forehead), the reference electrode on the ipsilateral mastoid, and the ground electrode on the contralateral mastoid. Click stimuli were 100 μ s in duration and presented in alternating polarity at a rate of 11.1/s. Stimuli were presented at 60 and 80 dB nHL, to both left and right ears separately, in four separate blocks of trials. Responses were amplified with a gain of 50k and band-pass filtered between 100-1500 Hz. Data were collected over a 20 ms epoch, with a minimum of 6000 sweeps, with additional repetitions added for each rejection. An average of the 6000 sweeps was taken to form an average waveform for each ear and stimulus level. Participants lay in a comfortable supine position, were asked to remain still throughout the recordings, and were encouraged to fall asleep. The measurement took place in a double-walled sound-attenuating booth.

To further ensure consistency between the data collected from the two different systems, we applied a correction to each participants' wave amplitudes and latencies collected with the Eclipse, for each stimulus intensity. This procedure is explained and verified in Supplementary Materials 1.

Wave I and V from each participants' average waveform, for each ear and click intensity, were identified using an automated peak- and trough-picking procedure in MATLAB (Mathworks, Inc.) and visually inspected to verify the accuracy of the automated procedure (for further details see Couth et al., 2020). Wave I and V amplitudes were calculated peak-to-trough and were used to determine wave I/V amplitude ratio for each participant. Wave I and V latencies were calculated from stimulus onset to the peak of each wave.

2.2.8. Tinnitus

At T0, participants were asked whether they had ever experienced tinnitus, defined as *"an occasional sensation of a ringing, roaring, or buzzing sound in the ears or head even though no such sound is present, for a minimum duration of 5 min."* Participants who responded "Yes" were asked to complete the Tinnitus Handicap Inventory (THI; Newman et al., 1996). In subsequent testing sessions (T1 and T2), participants were asked whether there had been any change to their experience of tinnitus. If participants reported a change, they were asked to describe how it had changed (i.e., started, stopped, got worse, etc.), what they thought had caused the change, and to complete the THI.

2.2.9. Hyperacusis

At T0, participants were asked whether they had ever experienced hyperacusis, defined as *"an abnormal sensitivity to everyday sound levels or noises. Often there is also sensitivity to high pitched sounds. In some circumstances, certain sounds may become painfully loud."* Due to hyperacusis being a less familiar term for most individuals, all participants were asked to complete the Modified Khalifa Hyperacusis Questionnaire (HQ; Khalifa et al., 2002), which was also used to quantify hyperacusis severity. In subsequent testing sessions (T1 and T2), participants were asked whether there had been any change to their experience of hyperacusis. If participants reported a change, they were asked to describe how it had changed (i.e., started, stopped, got worse, etc.), what they thought had caused the change, and to complete the HQ.

2.2.10. Hearing in noise difficulties

At T0, participants were asked whether they *"find it difficult to follow a conversation if there is background noise, such as TV, radio, children playing"* and were asked to complete a subset of questions from the Self-efficacy for Situational Communication Management Questionnaire (SESMQ;

Jennings et al., 2014) relating to six challenging noisy situations (see Couth et al., 2020). In subsequent testing sessions (T1 and T2), participants were asked whether there had been any change to their experience of listening in noisy situations (i.e., started, stopped, got worse, etc.). If participants reported a change, they were asked to complete the subset of SESMQ questions.

2.4. *Data analysis*

Linear mixed models (LMM) were conducted using SPSS 25 (IBM Corp.) in line with the procedure proposed by Peugh and Enders (2005) and Shek and Ma (2011), and were visualised according to Howard (2021). LMMs study the rate of change using individual growth curves which model within-subject and between-subject developments across time (e.g., T0, T1 and T2). This analysis method is preferable to generalised linear models as it does not require balanced data (e.g., unequal group sizes and missing data), allows the study of intra- and inter-individual differences in growth parameters (e.g., slope and intercepts), and other predictors of individual growth (e.g., noise exposure) can be flexibly added.

First, an unconditional means model was used to assess whether there was sufficient variability in individuals' scores averaged over time. If the within-subject variance is statistically significant, there is substantial variation of individuals' averaged scores, meaning that time-varying predictors (e.g., total cumulative noise exposure) can be added to the model. If the between-subject variance is statistically significant, there is substantial variation of initial status (i.e., intercept) due to individual differences, that may be predicted by time-invariant predictors (e.g., musical experience at T0). Second, an unconditional linear growth curve model was used to examine any significant variations in individual trajectory changes over time. If there was no significant inter-individual difference in trajectory change over time, no further model testing was conducted. On occasions where the unconditional growth model indicated significant differences in individual growth trajectories over time, additional predictors were added to the model to explore their effect on individual growth trajectories (i.e., the interaction with time). For cumulative noise exposure as a dependent variable, predictors included two dichotomous independent variables; musician status (musician vs. non-musician; coded as -1 and 1, respectively) and noise exposure group at T0 (low vs. high; coded as -1 and 1, respectively). This enabled us to model group differences in cumulative noise exposure. For all other dependent variables, predictors included two continuous independent variables; years of musical training and total noise exposure (absolute NESI units). Dependent and predictor variables for each LMM can be seen in Supplementary Materials 2.

The data collection for this study commenced prior to Manchester Centre for Audiology and Deafness' commitment to pre-registering all research studies (Munro and Prendergast, 2019; Prendergast et al., 2023). Although the analysis investigating the relationships between noise exposure, musicianship and hearing outcome measures were pre-planned based on our previous cross-sectional study (Couth et al., 2020), there was no pre-registration of the data analysis plan for the present study, and some aspects of the analysis approach were decided upon post hoc. We did not make any specific hypotheses about the results prior to data collection, and so the interpretation of the results should generally be regarded as hypothesis-generating rather than hypothesis-confirming. Given the exploratory approach to the data analysis, there was no control of the familywise error rate when conducting multiple statistical tests (e.g., Bonferroni correction) to allow for trends in results to be observed. Nevertheless, all results which are statistically significant ($p < .05$) should be interpreted with caution.

3. Results

The LMM for each measure is presented in table format in Supplementary Materials 2.

3.1. Noise exposure

For occupational, recreational, and total noise exposure (Tables S1, S2 and S3, respectively), the unconditional linear growth models all indicated significant effects of time (all $p < .05$), demonstrating predictable cumulative increases in noise exposure over the study period. Musician group and noise exposure group, plus their interactions with time, were added as fixed effects to each of the conditional linear growth models. For occupational noise exposure, both musician group and noise exposure group were significant (both $p < .05$) indicating a higher level of occupational noise exposure overall for musicians compared to non-musicians, and for participants deemed as having high levels of noise exposure at T0 compared to those deemed as having low noise exposure. A significant interaction between time and musician group ($\beta = -.739, p = .037$) indicated that musicians reported higher rates of growth of occupational noise exposure (1.62 NESI units per year) compared to non-musicians (0.14 NESI units per year). For recreational noise exposure, the conditional linear growth model indicated no significant interactions (all $p > .05$), suggesting that the rate of growth of recreational noise exposure did not significantly differ between musicians (1.48 NESI units per year) and non-musicians (2.61 NESI units per year), nor between low (1.41 NESI units per year) and high noise exposure groups (2.68 NESI units per year). However, the high noise exposure group reported significantly higher levels of recreational noise exposure overall compared to the low noise exposure group ($\beta = 19.529, p < .001$). For total noise exposure, the conditional linear growth model indicated

that the high noise exposure group reported significantly higher levels of total noise exposure overall compared to the low noise exposure group ($\beta = 21.246$, $p < .001$). There was also a significant interaction between time and noise exposure group ($\beta = 1.121$, $p = .042$), demonstrating that participants identified as having high levels of noise exposure at T0 reported higher rates of growth of total noise exposure (4.02 NESI units per year) compared to participants with low levels of noise exposure (1.78 NESI units per year) (Fig. 1). No further significant interactions were found (all $p > .05$).

At all time points, the majority of total noise exposure was due to recreational activities, for both musicians and non-musicians. Recreational noise exposure was primarily from music related activities, such as attending concerts and nightclubs, and listening to music at high levels via earphones/headphones and hi-fi systems.

[Figure 1 here]

3.2. *Hearing thresholds*

Average hearing thresholds were calculated across low frequencies (PTA low; .25-1 kHz), high frequencies (PTA high; 2-8 kHz), and EHF (12 and 16 kHz), across both ears, for each participant at each time point.

For low- and high- frequency PTA thresholds (Tables S4 and S5, respectively), the unconditional linear growth models indicated significant effects of time ($\beta = -1.546$, $p < .001$ and $\beta = -.521$, $p = .001$, respectively), suggesting a small decrease (i.e., improvement) in thresholds each year. For completeness, we added musical experience and total noise exposure as predictor variables into each model to see whether these factors could account for the improvement in thresholds. For low-frequency PTA thresholds, there were no significant interactions with time (all $p > .05$; Fig. 2a), but there was a significant interaction between total noise exposure and time for high-frequency PTA thresholds ($\beta = 0.014$, $p = .002$). This suggests that higher than average levels of cumulative noise exposure offset the rate of improvement in high-frequency PTA thresholds over time, although the effect is very small (low noise exposure = .60 dB improvement per year; high noise exposure = .38 dB improvement per year; Fig. 2b). There were no other significant interactions (all $p > .05$).

For EHF thresholds (Table S6), there was a small but significant increase (i.e., worsening) in thresholds over time ($\beta = .661$, $p < .001$). However, the conditional linear growth model did not show any significant fixed effects of total noise exposure or musical experience, and no interactions (all $p > .05$; Fig. 2c). To explore this effect further, we included average low and high PTA thresholds and participants' age at T0 into the model, which were all found to be non-significant predictors of the change in EHF thresholds (all $p > .05$).

[Figure 2 here]

3.3. *Otoacoustic emissions*

Average DPOAEs were calculated across all measured frequencies (1-6 kHz), across both ears, for each participant at each time point. The unconditional linear growth model indicated that there was no significant change in average DPOAEs over the course of the study ($\beta = .006$, $p = .956$). Since participants with the highest levels of noise exposure had significantly lower DPOAEs at T0 compared to those with the lowest levels of noise exposure (top and bottom 20%, respectively; Couth et al., 2020), we performed an exploratory conditional linear growth model with the effects of noise exposure group and musicianship group (categorical variables) added as fixed effects. This analysis revealed a significant main effect of noise exposure group with overall significantly lower average DPOAEs for the high noise exposure group compared to low ($\beta = -.915$, $p = .034$; Table S7; Fig. 3).

[Figure 3 here]

3.4. *Speech perception in noise*

The unconditional growth model for the CRM task with offset maskers indicated a significant decrease (i.e., improvement) in SRTs each year ($\beta = -1.435$, $p < .001$). For completeness, we added musical experience and total noise exposure as predictor variables into the conditional linear growth model (Table S8). There were no significant interactions between musical experience and time, nor between noise exposure and time (both $p > .05$), but there was a significant three-way interaction between musical experience, noise exposure and time ($\beta = -.001$, $p = .037$). This interaction suggests that, for those with low musical experience, the improvement in SRTs may be reduced slightly by increasing levels of noise exposure (.95 dB improvement per year; Fig. 4a), whereas no such effect is seen for those with higher levels of musical experience (1.85 dB improvement per year; Fig. 4b).

For the condition with centrally located maskers, the unconditional growth model also revealed a significant improvement in SRTs each year ($\beta = -1.262$, $p < .001$), but the conditional growth model did not reveal any effects of musical experience or noise exposure, and no interactions with time (all $p > .05$; Table S9; Fig. 4c and d).

[Figure 4 here]

As an additional analysis, we measured spatial release from masking by subtracting performance in the offset condition from performance in the central condition for each participant at each time point. The effect of time in the unconditional linear growth model was not significant ($\beta = -.176$, $p = .579$).

This suggests that the levels of improvements in central and offset conditions was approximately equal across the study period.

3.5. *Electrophysiology*

Given that ABRs were more reliably captured (i.e., less noisy recordings) for the high intensity stimuli compared to low, and that effects of synaptopathy should be at least as evident at high stimulus levels as at low stimulus levels (Furman et al., 2013), statistical analysis of the ABRs was completed for the 80 dB nHL click conditions only. Average wave amplitudes and latencies were taken over both ears, for each participant at each time point. Average ABR wave forms for each time point are shown in Supplementary Materials 3, along with explanations of wave amplitudes and latencies.

3.5.1. Wave amplitudes

The unconditional growth model indicated a small but significant increase in ABR wave I amplitudes each year ($\beta = .023$, $p = .001$; Fig. 5a). The conditional growth model did not reveal any effects of musical experience or noise exposure, and no interactions with time (all $p > .05$; Table S10). Similarly, the unconditional growth model indicated a small but significant increase in wave V amplitudes each year ($\beta = .018$, $p = .015$; Fig. 5b), but no significant effects of musical experience or noise exposure, and no interactions with time in the conditional growth model (all $p > .05$; Table S11). Given the significant improvement in hearing thresholds over time, we included average low and high PTA thresholds into the model, which were found to be non-significant predictors of the change in ABR wave amplitudes (all $p > .05$).

We also analysed wave I/V ratios, which account for individual differences that may affect wave I amplitudes (e.g., age, sex, anatomical differences) by normalising to wave V amplitudes, and has also been suggested to be an indicator of increased central gain (larger wave V) to compensate for reduced sensory input (smaller wave I) as a result of cochlear synaptopathy (Grose et al., 2017; Schaette and McAlpine, 2011). There was no significant change in wave I/V ratios across the study period ($\beta = .015$, $p = .348$). At T0, we found that musicians had significantly larger ABR wave I/V ratios compared to non-musicians (Couth et al., 2020). Accordingly, we ran the conditional growth model with musician group and noise exposure group (categorical variables), which revealed that wave I/V ratios were significantly larger for musicians than non-musicians across overall ($\beta = -.078$, $p = .009$; Table S12). This effect was driven by musicians having slightly larger wave I amplitudes and slightly smaller wave V amplitudes than non-musicians (both non-significant; $p > .05$). There were no other significant main effects or interactions (all $p > .05$).

3.5.2. Wave latencies

The unconditional growth model indicated a significant increase in ABR wave I latencies each year ($\beta = .016, p = .021$). The conditional growth model revealed a significant interaction between years of musical experience and time ($\beta = .003, p = .027$), suggesting a slight increase in wave I latencies each year for those with higher-than-average years of musical experience (.05 ms increase per year), or a slight decrease for those with lower-than-average years of musical experience (.02 decrease per year) (Fig. 5c). There were no other main effects or interactions (all $p > .05$, Table S13).

There were no significant changes in wave V latencies across the study period ($\beta = -.004, p = .705$; Fig. 5d) and no significant changes in wave I-V interpeak interval ($\beta = -.020, p = .180$) (Table S14 and 15). At T0, we found significantly increased wave V latencies and wave I-V interpeak intervals for the high noise exposure group compared to low (Couth et al., 2020). To explore the effects of noise exposure further, we ran conditional growth models with noise exposure group and musician group (categorical variables) as predictor variables for wave V latency and wave I-V interpeak interval. There were no significant main effects or interactions for wave V latency or wave I-V interpeak interval (all $p > .05$). We repeated these exploratory analyses by adding years of musical experience and total noise exposure (continuous variables) as predictors in the conditional growth models. There were no significant main effects or interactions for wave V latency (all $p > .05$), however there was a significant interaction between years of musical experience and time for wave I-V interpeak interval ($\beta = -.007, p < .016$), indicating a slight reduction in interpeak interval over time with increasing years of musical experience (low musical experience = .03 ms decrease per year; high musical experience = .01 ms decrease per year). This effect is driven by increasing wave I latency over time with increasing years of musical experience, as per the wave I latency analysis above.

[Figure 5 here]

3.6. Self-report measures

3.6.1. Tinnitus

Thirty-five participants (26 musicians) reported a change in their experience of tinnitus from T0-T1. Of these, only 14 (13 musicians) reported a worsening of their tinnitus, with the majority (21; 13 musicians) reporting an improvement. Nineteen participants (15 musicians) reported a change from T1-T2, with only eight of these participants (six musicians) reporting a worsening of their tinnitus, and the majority (11; nine musicians) reporting an improvement. Fourteen participants (12 musicians) reported changes from T0-T1 and T1-T2, with 10 of these (eight musicians) reporting a consistent improvement across the test sessions and two (both musicians) reporting a consistent worsening of

symptoms across the test sessions. Due to the low numbers reporting a consistent change, change in the THI was not modelled using LMM.

For those who noticed a worsening from T0-T1, 6/14 attributed this to more exposure to noise in the previous year, 1/14 suggested that they had become more attentive/aware of experiencing tinnitus since the previous testing session, 1/14 attributed it to stress, and 6/14 were unsure why it had worsened. For those who noticed an improvement from T0-T1, 11/21 attributed this to less exposure to noise in the previous year, 4/21 attributed this to using hearing protection devices more often, 1/21 attributed this to receiving tinnitus therapy, and 5/21 were unsure why it had improved. For those who noticed a worsening from T1-T2, 5/8 attributed this to more exposure to noise in the previous year, 2/8 suggested that they had become more attentive/aware of experiencing tinnitus since the previous testing session, and 1/8 suggested attributed this to using their hearing protection less often. For those who noticed an improvement from T1-T2, 7/11 attributed this to less exposure to noise in the previous year, 2/11 attributed this to using hearing protection devices more often, and 2/11 were unsure why it had improved (Fig. 6).

[Figure 6 here]

3.6.2. Hyperacusis

Fifteen participants (eight musicians) reported a change in hyperacusis severity from T0-T1, with 12 of these participants (seven musicians) indicating a worsening of symptoms, and three (one musician) reporting an improvement. Fourteen participants (10 musicians) reported a change in hyperacusis severity from T1-T2, with all of these indicating a worsening of symptoms. Only four participants (three musicians) consistently reported a change to hyperacusis severity across T0-T1 and T1-T2, with three of these participants (two musicians) reporting a worsening of symptoms on both occasions. Due to these low numbers reporting a consistent change over time, change to HQ scores was not modelled using LMM.

For those who noticed a worsening from T0-T1, 8/12 suggested that their symptoms were related to specific sound sources (e.g., traffic noise, café noise, lecture theatres, etc.) and 4/12 were unsure why it had worsened. For those who noticed an improvement from T0-T1, all (3/3) were unsure why it had improved. For T1-T2, 11/14 suggested that their symptoms were related to specific sound sources and 3/14 were unsure why it had worsened.

3.6.3. Hearing in noise difficulties

Only seven participants (five musicians) reported a change to their hearing in noise abilities (2 improved) from T0-T1 and only four participants (three musicians) reported a change (one improved) from T1-T2. Only two participants (one musician) reported a consistent change from T0-T1 and T1-T2 (both worsened). Due to these low numbers reporting a consistent change over time, change to SESMQ scores was not modelled using LMM. Reasons for the change in participants' experiences of hearing in noise difficulties were not collected.

3.7. Additional analyses

3.7.1. Repeatability of T0 findings

To assess the repeatability of the cross-sectional findings at T0, we conducted Spearman's rho correlation analyses between years of musical experience, total noise exposure, average low PTA, average high PTA, average EHF thresholds, average DPOAE levels, spatial release from masking from the CRM task, average wave I amplitudes at 80 dB nHL, average wave I/V ratio at 80 dB nHL, and average wave I and V latencies at 80 dB nHL, at each of the separate time points (see Supplementary Materials 4). To limit the proportion of false positives, we applied the false discovery rate (FDR) method with a p -value of .05 and a q -value of .10 (McDonald, 2014), at each time point.

As per T0 ($r_s = -.259$, $n = 123$, $p = .004$; Couth et al., 2020), total noise exposure was significantly correlated with average DPOAE levels at T1 ($r_s = -.340$, $n = 91$, $p = .001$) and T2 ($r_s = -.303$, $n = 61$, $p = .018$) (Fig. 7a and b). Average wave V latency was also found to be significantly correlated with total noise exposure at T0 ($r_s = .293$, $n = 120$, $p = .001$; Couth et al., 2020) and T1 ($r_s = .271$, $n = 91$, $p = .009$), and a similar non-significant pattern was observed at T2 ($r_s = .229$, $n = 58$, $p = .083$) (Fig. 7c and d).

[Figure 7 here]

Years of musical experience was not found to be significantly correlated with any measures after applying FDR corrections at T0. This was consistent at T1, but there was a significant correlation between average DPOAE levels and years of musical experience at T2 ($r_s = -.361$, $n = 61$, $p = .004$). Given that there was a correlation between years of musical experience and noise exposure at T2 (albeit not deemed to be a significant finding after applying FDR; $r_s = .276$, $n = 61$, $p = .031$), and the majority of participants at T2 were musicians with high levels of musical experience, we conducted a partial correlation between years of musical experience and average DPOAE levels while controlling for total noise exposure. A correlation was still present after controlling for noise exposure ($r(58) = -.285$, $n = 61$, $p = .027$), however this is no longer deemed significant after applying FDR.

3.7.2. Reliability of measures

To assess the reliability of the measures between time points, we conducted two-way mixed-effects intraclass correlation coefficients (ICC; Shrout and Fleiss, 1979) with an absolute agreement definition (Koo and Li, 2016), between hearing measures at T0-T1 and T1-T2. ICC values are shown in Table 1. Most of the ICC values would be generally described as reflecting fair to excellent reliability (Cicchetti, 1994). Reliability was greater for T1-T2 compared with T0-T1 for the CRM measures, especially for the central condition which had poor reliability from T0-T1. Reliability was also much greater for T1-T2 compared with T0-T1 for the ABR measures.

[Table 1 here]

4. Discussion

In our previous cross-sectional study, we found very few effects of noise exposure on hearing, including proxy measures of cochlear synaptopathy (Couth et al., 2020). In the current study, we aimed to explore the cumulative effects of noise exposure on behavioural, self-report and electrophysiological measures of hearing over a 2-year period in early-career musicians and non-musicians. Our results have shown very few longitudinal changes to hearing due to noise exposure, with findings remaining consistent at each of the three measurement time points. For the first time (to our knowledge), these findings suggest that there is no evidence of cochlear synaptopathy with cumulative noise exposure in young normal hearing adults. In fact, some of the crucial proxy indices of cochlear synaptopathy, such as ABR wave amplitudes and SPiN thresholds, showed a significant improvement over time in the current study. Nevertheless, there were some detrimental effects of noise exposure, such as on outer hair cell function as measured by DPOAE level, which was consistently related to total noise exposure at each time point, but did not show a significant change over time. This raises some interesting questions regarding longitudinal changes to hearing function and its relation to noise exposure.

4.1. What are the detrimental effects of noise exposure on hearing function?

Consistent with the cross-sectional findings at T0 (Couth et al., 2020), we found a significant negative association between lifetime noise exposure and DPOAE level at both T1 and T2. This could be indicative of poorer OHC functioning due to noise exposure, even though hearing thresholds were classified as normal. The effects of noise exposure on OHC function are well documented (Hamdan et al., 2008; Job et al., 2009; Lapsley Miller et al., 2006; Lucertini et al., 2002; Mansfield et al., 1999). Nevertheless, we did not observe a significant change to OHC function across the study period due to cumulative noise exposure, and so it is not possible to assume a causal relationship based on these three separate cross-sectional findings.

There have been several studies that have monitored longitudinal changes to OAEs in relation to noise exposure (for a systematic review see Helleman et al., 2018). For example, Helleman et al (2010) showed a ~2-4 dB deterioration in DPOAE levels in the 4-8 kHz range for noise-exposed workers over a 17-month period. Similarly, Moukos et al. (2014) conducted a 9-year follow-up of noise-exposed industrial workers, demonstrating a significant reduction in DPOAE levels compared to baseline levels by ~5-10 dB in 3-5 kHz range. However, there was no direct comparison to a low noise exposed group in either of these studies, and the cumulative effects of noise exposure over the study period were not analysed. Seixas et al. (2012, 2005) conducted a 10-year prospective study of cumulative noise exposure on DPOAEs in young construction workers and non-noise exposed controls (mean age at baseline = 27.6 years), demonstrating a 1-2 dB decrease in DPOAE amplitude at 4 kHz for a 10 dB increase in noise exposure over 10 years. This relatively small noise-related change to OAEs, restricted to a single frequency, and in a slightly older participant group, could explain why we did not observe a significant change in average DPOAE levels over a 2-year period in the current study.

These previous longitudinal findings raise some important considerations. First, it is crucial to determine when these detrimental effects of noise exposure on OHC function become apparent (i.e., before 18 years of age), and second, to monitor OHC function over a longer period to determine the rate of change. In addition, several factors such as OAE stimulus and measurement parameters may affect the sensitivity of the recording, and thus the ability to detect change over time (Lapsley Miller and Marshall, 2001; Marshall et al., 2001). Therefore, it is crucial to determine clinical standards for OAE measurements that are sensitive to detecting change in “at risk” individuals.

Also consistent with the cross-sectional findings at T0 (Couth et al., 2020), we saw a positive association between noise exposure and ABR wave V latency at T1 and T2. However, there was no significant change over time. Moreover, it is not clear whether increases in ABR wave V latency are indicative of cochlear synaptopathy (Skoe and Tufts, 2018). As we have argued previously, delays to ABR wave V latency, but not to wave I, are consistent with a delayed propagation of signals in the auditory brainstem pathway more centrally, rather than in the auditory periphery (Couth et al., 2020). It may be possible to tease out the effects of noise exposure on the signal transduction pathway by measuring latency shifts in response to varying the stimulus presentation rate (Burkard et al., 1997; Burkard and Hecox, 1987; Lasky, 1997; Shi et al., 2013; Skoe and Tufts, 2018) or by presenting the stimulus in varying levels of background noise (Burkard et al., 1997; Burkard and Hecox, 1987; Guest et al., 2019b; Mehraei et al., 2016). For example, Burkard and Hecox (1987) showed that increasing background noise level and stimulus presentation rate produced an increase in wave I-V interpeak intervals in which wave III-V interpeak intervals were greater than wave I-III interpeak intervals,

suggesting that a central auditory mechanism produces wave V latency shifts. More recently, Skoe and Tufts (2018) demonstrated a correlation between noise exposure and rate-dependent changes to wave I-V interpeak interval, which the authors propose could be due to demyelination of the central auditory system, rather than cochlear synaptopathy (see S. E. Kim et al., 2013).

Cumulative noise exposure had little effect on proxy measures of cochlear synaptopathy in the current study. This finding builds on previous cross-sectional studies from our lab (Couth et al., 2020; Guest et al., 2019a, 2018b, 2017; Prendergast et al., 2019, 2018, 2017b, 2017a; Shehabi et al., 2023a) suggesting that normal hearing young adults are resilient to noise-induced cochlear synaptopathy. Alternatively, it is possible that the levels of noise exposure experienced prior to and during this study period were not sufficient to cause widespread hair cell and synaptic damage that would translate into detectable deterioration of hearing function. In support, a recent investigation from our lab has suggested that a minimum of 2 log NESI units of lifetime noise exposure (equivalent to working 13 years at 99 dB A for 40 hours per week) may be required before the effects of noise exposure on SPiN start to become apparent (Shehabi et al., 2023b). It is also possible that exposure to high peak-intensity noise, such as that from military noise and firearms, is required to induce cochlear synaptopathy (Bramhall et al., 2022, 2021, 2020, 2018, 2017; Kameron et al., 2022). An additional explanation for these null findings is that the proxy measures used in this study are not sensitive to detecting cochlear synaptopathy, or that the noise exposure measures are too imprecise to determine the effect of noise on hearing. We provided similar explanations for the null findings at T0, which were discussed at length (see Couth et al., 2020). For a comprehensive overview of and explanation for the mostly null findings in human cochlear synaptopathy research, we direct the reader to a recent narrative review by Shehabi et al. (2022).

4.2. *Can hearing function improve over time?*

While OHC function did not change over the study period, there were some subtle longitudinal changes to hearing that were related to noise exposure in the current study. For average high-frequency (2-8 kHz) PTA thresholds there was a significant interaction between time and noise exposure, indicating that high levels of cumulative noise exposure were associated with accelerated growth (i.e., worsening) of thresholds. Yet, the overall trend showed an improvement in thresholds, suggesting that noise exposure could slightly offset this improvement over time. Improvements were also seen for average low-frequency (0.25-1 kHz) PTA thresholds, which were not affected by noise exposure.

It is unclear why average low- and high- frequency hearing thresholds improved in this study. In the Baltimore Longitudinal Study of Ageing, Pearson et al. (1995) also showed an improvement in thresholds ranging from 0.1 to 1.9 dB from the first to subsequent testing sessions. The authors attribute this to a learning effect which can be observed in subsequent testing sessions over a period of several years. The authors suggest that longitudinal studies with only one or two closely separated repeated measures may be biased by learning effects whereby improvements in hearing measures may mask some of the underlying changes to hearing thresholds. Note that we used a three-alternative forced-choice paradigm for measuring EHF (12-16 kHz) thresholds in the current study, which did show a significant increase (i.e., worsening) across the study period (discussed below). This could imply that using an automated forced-choice testing procedure may be less susceptible to learning/practice effects and/or experimenter variability, and thus may be better at detecting true changes to hearing. Alternatively, it may be beneficial to use objective hearing measures which are not susceptible to learning effects, such as DPOAEs, which showed the highest test-retest reliability in the current study (see Table 1).

Improvements in performance were also seen for the CRM task; one of the proxy measures of cochlear synaptopathy. The ICC analysis demonstrated that test-retest reliability was greater for T1-T2 compared with T0-T1 for the CRM task, especially for the central condition which had poor reliability from T0-T1 (Table 1). This likely reflects a learning effect from T0-T1 for both conditions of the CRM task, which was less pronounced from T1-T2. Learning effects for SPiN measures have been noted previously (Kollmeier et al., 2015; Maele et al., 2021), including the CRM task (Jakien et al., 2017). This is problematic since true decreases in SPiN performance due to noise damage could be masked by learning effects. We attempted to mitigate learning effects on the CRM task by providing participants with a short practice prior to conducting the test. For future longitudinal studies, it may be beneficial to use SPiN measures which are less susceptible to learning effects (e.g., Digits-in-Noise test; De Sousa et al., 2020), or to allow participants a longer practice session at each visit.

Significant increases in ABR wave amplitudes, another proxy measure of cochlear synaptopathy, were also found over the study period. Previous studies from our lab investigating test-retest reliability of the ABR also showed small (albeit non-significant) increases in ABR wave amplitudes from test to retest (Guest et al., 2019b; Prendergast et al., 2018). In these previous studies, the test-retest reliability of the ABR wave I amplitude yielded ICCs ~ 0.85 (Guest et al., 2019b; Prendergast et al., 2018), and wave V amplitude yielded an ICC of 0.80 (Prendergast et al., 2018). In the current study, the test-retest reliability from T1-T2 was comparable to these previous studies with ICCs of 0.92 and

0.81 for wave I and wave V amplitudes, respectively². However, the test-rest reliability from T0-T1 was much lower with ICCs of 0.50 and 0.65 for wave I and wave V amplitudes, respectively (Table 1). Guest et al. (2019b) suggested that ABR wave I can be a highly reliable measure as long as there is consistency in electrode placement, participant state, and other factors influenced by the investigator. Participants in the current study may have become more familiar with the ABR procedure and were more relaxed during subsequent testing sessions, especially from T0-T1, thus providing better quality and more consistent ABR recordings in follow-up sessions (i.e., a test-retest effect). These improvements in data capture could explain the small but significant increases in wave I amplitudes across the study period. Similarly, changes in test-retest reliability could be attributed to an experimenter-related effect (e.g., different electrode placements).

We also found that, for the participants who reported a change to their experience of tinnitus, the majority reported an improvement in their symptoms, such as experiencing tinnitus less often or a reduction in severity. Most of these participants attributed this improvement to a reduction in noise exposure or to increasing their use of hearing protection since the previous testing session. Conversely, for the small number of participants who noticed a worsening of tinnitus symptoms, most of these attributed this to an increase in noise exposure or non-use of hearing protection. This suggests that noise exposure can have a detrimental effect on tinnitus experience, and that hearing conservation measures had a positive effect. The potential long-term benefits of hearing conservation programs have been demonstrated previously in a 10-year longitudinal study of Danish workers, which showed no significant impact of noise exposure on hearing thresholds, which the authors attributed to declining industrial noise levels and increased use of hearing protection across the study period (Frederiksen et al., 2017). In support, a report from the Health and Safety Executive Workplace Health Expert Committee (Workplace Health Expert Committee, 2023) estimates that the prevalence of occupational noise-induced hearing loss has decreased over the past 40 years due to the introduction of noise control measures and greater use of hearing protection. However, it is not clear whether these findings also apply to the music industry.

As well as a reduction in tinnitus symptoms, it is plausible that an increased use of hearing protection and less regular participation in noisy events could also explain the small improvements in hearing

² Note that the ABR test-retest reliability shown by Prendergast et al. (2018) was measured using the same ICS Chartr EP 200 (Optometrics-Natus) clinical system used in the current study prior to its recall. The ICCs in this previous study were calculated from two closely separated time points (average 3.5 days apart, ranging from 1-12 days), demonstrating very similar test-retest reliability to what was observed between T1-T2 in the current study when forced to change to a different system (Interacoustics Eclipse). Accordingly, we are confident that the corrections applied to account for the change in systems are accurate (Supplementary Materials 1).

thresholds in the current study. Some participants may have been showing a small amount of temporary threshold shift (TTS) at T0 due to recent noise exposures without the use of hearing protection, but then subsequent uptake of hearing conservation measures may have reduced or prevented TTS, and thus given the impression that hearing thresholds were improving at later testing sessions. Additionally, with evidence from guinea pig studies showing that auditory nerve ribbon synapses in the cochlea are largely repairable (Liu et al., 2012; Puel et al., 1998, 1995; Pujol and Puel, 1999; Shi et al., 2013), and with the propensity for synaptic plasticity in humans (Bucillo et al., 2011; Kaas, 2001; Martino et al., 2011), it is possible that the adoption of hearing conservation practices could have provided an opportunity for damaged auditory nerve synapses to heal, which could also explain the small improvements observed in ABR amplitudes, and thus enhanced performance on the SPiN task (although note that there was no significant correlations between these measures). Future longitudinal studies could more closely monitor hearing function, including TTS and ABRs, immediately after participation in various noisy events over a prolonged period (cf. Grinn et al., 2017; Maele et al., 2021), and determine whether the use of hearing protection offsets acute and prolonged effects of noise exposure.

An alternative explanation for the improvements in hearing measures could be due to ongoing cortical maturation in our relatively young participant sample. Schneider et al. (2023) showed that white-to-gray matter ratios (i.e. the degree of myelination) in the Planum temporale continues to increase from young adulthood (19-29 years) into middle age (30-67 years). This could signify maturational plasticity with increasing age. The authors also showed that maturation of Heschl's gyrus begins to plateau at around 11-13 years. The authors suggest that Heschl's gyrus is specialised in the processing of lower-level sound features, whereas the Planum temporale is involved in more integrative, multimodal, higher complex functions. Therefore, it is unlikely that ongoing maturation to the Planum temporale in adulthood could explain the improvements in PTA and ABRs seen in the current study, since these are less related to higher-order processing, but it could potentially explain the improvements in SPiN performance.

4.3. *Are musicians at a greater risk of noise-induced hearing damage?*

Using the NESI to estimate year-on-year increases in noise exposure, we found that there was no significant difference in cumulative total lifetime noise exposure between early-career musicians and non-musicians. The reason for this finding may be due to the relatively high levels of recreational noise exposure for both participant groups, which accounted for most of the total noise exposure across the study period for the majority of participants. Accordingly, early-career musicians may be at no greater risk of noise-induced hearing damage than non-musicians (Axelsson et al., 1995; Kähäri et al., 2001;

Müller and Schneider, 2018). Nevertheless, there was a greater rate of growth of occupational noise exposure for musicians than non-musicians. It is plausible that, at a certain point in time, young adults begin to reduce the amount of recreational noise exposure that they expose themselves to (e.g., attending nightclubs and concerts), but for musicians, they may continue to be exposed to high levels of occupational noise over several decades. A longitudinal study of musicians' hearing health across the career span would enable a more precise evaluation of the prolonged effects of noise exposure.

Despite self-reported noise exposure being similar for both musicians and non-musicians, there were some subtle effects of musicianship on hearing. Although most participants reported no change in their experience of tinnitus, and most of those that did notice a change reported an improvement, nearly all of those that reported a worsening were musicians (T0-T1 = 13/14; T1-T2 = 6/8; T0-T2 = 2/2). Furthermore, most participants who noticed a worsening of their tinnitus attributed this to an increase in noise exposure or non-use of hearing protection. Given the small numbers of participants affected, we did not statistically model change in tinnitus experience or severity (i.e., THI score), and so we are unable to ascertain whether this was due to an increase in noise exposure as measured using the NESI. At T0, it was found that musicians reported more severe tinnitus symptoms than non-musicians, but this was not related to lifetime noise exposure measured using the NESI (Couth et al., 2020). Instead, it was proposed that musicians may be more aware or concerned about the effects of noise on hearing, and so may be more likely to report hearing problems such as tinnitus. This could also be apparent in the current study, whereby musicians who were more concerned about noise exposure and tinnitus were more likely to report a negative change and to attribute this to increased noise exposure. Caution should be taken in the interpretation of these findings given the small number of participants affected.

An additional longitudinal effect of musicianship was found for ABR wave I latency, demonstrating an increase in wave I latency for those with higher-than-average years of musical experience (i.e., >12 years) and a decrease in wave I latency for those with lower-than-average years of musical experience (i.e., < 12 years). There was no interaction with noise exposure and it is not clear what is causing this effect, and may simply be a chance finding due to random variation in the data (see 4.6. Limitations). Note that wave V latency was not influenced by years of musicianship, suggesting no further delay to signal propagation along the auditory brainstem after the initial delayed auditory nerve (wave 1) response, but rather a faster signal transmission with increasing years of musicianship (i.e., a reduction in wave I-V interpeak interval). The effect of musicianship on auditory brainstem evoked potentials has been investigated previously (for a review see Sanju and Kumar, 2016). Choi and Cho (2019) showed that college-aged musicians had significantly shorter wave I, wave V and wave I-V interpeak

intervals compared to age-matched non-musicians. Samelli et al. (2012) showed no significant differences in ABR wave latencies between musicians and non-musicians, although there was a slight trend for reduced latencies in musicians compared to non-musicians, even for musicians who were defined as having a hearing loss. These findings could imply enhanced neural plasticity in the brainstem due to musical training, which may be able to overcome hair cell damage. In the current study, individuals with more years of musical experience had faster wave I responses at T0, but this slowed down over time to the extent that wave I latency was greater (i.e., slower) than those with less musical experience at T2 (Fig. 5c). To our knowledge, no other studies have investigated longitudinal changes to the ABR in musicians previously, and additional studies may be able to verify or refute this finding, and to provide an explanation for this effect.

4.4. *Does musical training benefit hearing?*

Previous research has suggested that cognitive and auditory processing skills may be enhanced with musical training, which could improve SPiN performance (Coffey et al., 2017) and offset the negative effects of noise-induced cochlear synaptopathy on SPiN (Grose et al., 2017; Valderrama et al., 2018; Yeend et al., 2017). Our results from T0 showed no differences between musicians and non-musicians on the CRM task, and no interactions with noise exposure (Couth et al., 2020). Nevertheless, the current longitudinal study showed a three-way interaction between time, noise exposure and musicianship on the spatially offset condition of the CRM, which can be attributed to a reduced rate of improvement for those with less musical experience and higher levels of noise exposure (although see 4.6. Limitations). This pattern of results suggests that improvement in SPiN performance (i.e., the practice effect) could be negated by noise exposure, and that musical training may lessen this impact. More specifically, musicians may develop more robust strategies to aid and improve performance than non-musicians, such as increased reliance on spatially offset cues due to enhanced spatial processing abilities (Brochard et al., 2004; Glenn Schellenberg, 2001; Patston et al., 2007; Strong and Mast, 2019), which may be less susceptible to the effects of noise-induced hearing damage. In support, Swaminathan et al. (2015) demonstrated that musicians performed better than non-musicians in the spatially offset condition of their SPiN task (i.e., greater SRFM). Whilst this highlights the potential perceptual benefits of musical training, it also emphasises that SPiN can be influenced by factors other than noise exposure, and therefore might not be a particularly reliable measure of cochlear synaptopathy. Additional data on cognitive functioning (e.g., auditory working memory) and auditory processing abilities (e.g., spatial and temporal processing) should be collected as part of the test battery to control for these effects on SPiN performance (Beach, 2018; Gordon-Salant and Fitzgibbons, 1997; Heinrich, 2020; Heinrich et al., 2015; Kameron et al., 2019).

4.5. *Do EHF thresholds increase over time?*

EHF thresholds were also included in the current study since these may provide an early indication of hearing loss, even when audiometric thresholds in the standard frequency range (.25 – 8 kHz) are normal (for reviews see Hunter et al., 2020; Lough and Plack, 2022). We observed year-on-year increases (i.e., worsening) of average EHF thresholds (12 and 16 kHz) in the current study, but this was not related to noise exposure. Instead, it has been suggested that age is a primary factor in changes to EHF thresholds. For example, Groh et al. (2006) measured EHF thresholds in normal hearing children and adolescents aged 6 to 25 years, showing that hearing thresholds at 16 kHz start to increase at age 20 years. Stelmachowicz et al. (1989) also reported that thresholds above 14 kHz were 10-20 dB higher for listeners aged 20-29 years compared to listeners aged 10-19 years, despite there being no differences in thresholds at 8 kHz between these age groups. Given that the participants in our study were aged 18-27 years at T0, they may have been at an age where EHF hearing loss was beginning to show. However, it has also been suggested that decreased hearing sensitivity in the EHF region may be apparent at age 10 years or younger (Hemmingsen et al., 2021). Longitudinal assessments of EHF from an early age are required to determine the point at which EHF thresholds begin to increase and the rate of this increase across the lifespan. Lee et al. (2005) monitored EHF thresholds over a period of 3 to 11.5 years in adults aged 60-81 years old. The authors found that the average rate of change was approximately 1.23 dB per year at 12 kHz and the rate of change increased significantly with age. In comparison, the rate of change in average EHF thresholds in the current study was approximately .68 dB per year, suggesting that the rate of EHF loss across the lifespan might not be linear.

4.6. *Methodological considerations*

Although the strength and novelty of this study was its longitudinal design with a comprehensive test battery, one of the main limitations is that data was only captured over a 2-year period. Therefore, we might not have been able to detect any changes to hearing during this time frame, which may have been compounded by the difficulty in detecting the effects of noise exposure on sub-clinical hearing function in young normal hearing adults (Shehabi et al., 2022). In addition, hearing function was assessed on a small number of occasions for each participant (up to three). This meant that we were limited to using linear growth models, yet it is plausible that growth rates are not constant over time whereby changes to hearing may follow a non-linear trend. Indeed, it is unlikely that the improvements to hearing function captured in the present study would continue to increase in a linear fashion. Instead, it is expected that these would plateau and then would decrease over time with increasing age, irrespective of noise exposure (Lee et al., 2005). Note also that the cumulative gains in noise exposure across the study period were relatively modest compared to the levels of lifetime noise

exposure at T0, which could explain why relatively few changes in hearing function in relation to noise exposure were found, and could also mean that we missed the point at which noise exposure impacted on hearing (i.e., OHC function) earlier in life (i.e., pre- 18 years old). Future experiments should attempt to capture the effects of noise exposure on hearing over a longer time period, using a comprehensive test battery, and taken at several time points; it is recommended that a minimum of six time points are needed for a precise measurement of nonlinear change over time (Graves and Frohwerk, 2009). Additionally, future experiments should attempt to recruit adolescents prior to significant levels of noise exposure, such as at concerts and nightclubs, which is the current focus of research in our lab (Hearing in Teens study: <https://osf.io/ghd97>)

Another limitation relates to the semi-exploratory nature of this study and the non-correction for familywise error rate. We chose not to apply any controls (e.g., Bonferroni correction) so that potential changes to hearing in relation to noise exposure could be identified and discussed (i.e., hypothesis generating). However, this could mean that some of the reported findings may have occurred due to chance. If we apply a Bonferroni correction across the twelve linear effects models that measured the effects of noise exposure on hearing (i.e., Tables S4-15), the corrected $\alpha = .05/12 = .004$. Therefore, this would make the main effect of time on ABR wave V amplitude ($p = .015$) and wave I latency ($p = .021$) non-significant after correction, and would also make the interaction between time and musical experience for wave I latency ($p = .027$) and the three-way interaction between time, noise exposure and musicianship on the spatially offset condition of the CRM ($p = .037$) non-significant after correction. Accordingly, these findings should be interpreted with caution and further studies are required to support or refute these effects.

Further concerns relate to the high degree of heterogeneity in musicianship for both musician and non-musician groups. Zhang et al. (2018) define a musician as someone with at least six years of musical expertise. For the non-musician group in the longitudinal analysis, 10 participants had at least 6 years of musical experience at T0. Musical training in childhood has been shown to improve cognitive abilities (Bailey and Penhune, 2012; Bergman Nutley et al., 2014; Forgeard et al., 2008), which could persist into adulthood (Miendlarzewska and Trost, 2014). Accordingly, there may have been overlap between musician and non-musician groups in terms of auditory and cognitive skills, resulting in similar task performance between these groups. This is less of a concern for this study since we examined musicianship on a continuous scale (i.e., years of musical experience) across the whole participant sample. However, years of playing an instrument does not necessarily equate to high musical skill or ability. Future studies should include a range of tests to assess musical, auditory and

cognitive skills which could influence listening performance (as per Yeend et al., 2017), or which could be used to differentiate musician and non-musician groups more definitively.

An additional concern relates to volunteer bias, such that musicians may be more aware that they have some degree of noise-related hearing problem (either clinical or sub-clinical), which could make them more reluctant to take part in studies that might confirm their fears. This could explain why there was no difference in hearing function between musicians and non-musicians, and also why there was no differences in noise exposure, whereby musicians with low levels of noise exposure, and thus fewer hearing problems, were more willing to volunteer to take part in the study. Alternatively, these musicians with fewer hearing problems may be less concerned or aware of noise-induced hearing problems than musicians with hearing problems, and so they might underestimate the noise levels that they experience. This could also explain why lifetime noise exposure was similar between musicians and non-musicians in the current study.

5. Conclusions

To our knowledge, this is the first study to report the cumulative effects of noise exposure on proxy measures of cochlear synaptopathy in normal hearing adult humans. Although there were some subtle changes to hearing across the study period, these were largely unrelated to noise exposure, and some showed an improvement, which may be attributable to a practice/test-retest effect. Nevertheless, outer hair cell function was consistently found to be worse in those with higher levels of noise exposure at each time point. In addition, changes in tinnitus symptoms (i.e., improvements or worsening) may be related to levels of noise exposure and hearing protection use habits. Future longitudinal studies should monitor hearing health in relation to noise exposure more closely by taking more regular measurements, especially after periods of intensive noise exposure, and over a longer period of time. For musicians, this would involve assessing hearing at the point that musical training begins (i.e., childhood), and then again at regular intervals across the career span. This could form part of a hearing conservation programme to ensure hearing and career longevity for musicians.

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Figure captions

Figure 1. Estimated linear change in total noise exposure (NESI units) across the study period (T0-T2) based on fixed effects β values in the conditional growth model (Table S3) for musicians with low noise exposure (triangle symbols), musicians with high noise exposure (circle symbols), non-musicians with low noise exposure (plus symbols), and non-musicians with high noise exposure (square symbols) (groups determined at T0; Couth et al., 2020). Grey shaded errors represent 95% confidence bands of the estimate for each time point.

Figure 2. Estimated linear change in hearing thresholds across the study period (T0-T2) based on fixed effects β values in the conditional growth model for a) average low frequency PTA (.25 – 1 kHz; Table S4), b) average high frequency PTA (2-8 kHz; Table S5), and c) average EHF thresholds (12 and 16 kHz; Table S6). A negative slope indicates a worsening in thresholds. Linear change for a participant with average cumulative noise exposure is represented by circle symbols. Low cumulative noise exposure corresponds to 2 SD below the mean average yearly change in noise exposure (-8 NESI units), which is represented by square symbols. High cumulative noise exposure corresponds to 2 SD above the mean average yearly change in noise exposure (+8 NESI units), which is represented by triangle symbols. Grey shaded errors represent 95% confidence bands of the estimate for each time point.

Figure 3. Estimated linear change in average DPOAE level (dB SPL) across the study period (T0-T2) based on fixed effects β values in the conditional growth model (Table S7) for musicians with low noise exposure (triangle symbols), musicians with high noise exposure (circle symbols), non-musicians with low noise exposure (plus symbols), and non-musicians with high noise exposure (square symbols) (groups determined at T0; Couth et al., 2020). Grey shaded errors represent 95% confidence bands of the estimate for each time point.

Figure 4. Estimated linear change in SNRs for the CRM task across the study period (T0-T2) based on fixed effects β values in the conditional growth models for the offset condition (Table S8) and central condition (Table S9). SNRs for the offset condition are shown in the top panels for a) low musical experience (corresponding to 2 SD below the average years of musical experience = -12 years), and b) high musical experience (corresponding to 2 SD above the average years of musical experience = +12 years). SNRs for the central condition are shown in the bottom panels for c) low musical experience and d) high musical experience. A negative slope indicates an improvement in performance on the CRM task. Linear change for a participant with average cumulative noise exposure is represented by circle symbols. Low cumulative noise exposure corresponds to 2 SD

below the mean average yearly change in noise exposure (-8 NESI units), which is represented by square symbols. High cumulative noise exposure corresponds to 2 SD above the mean average yearly change in noise exposure (+8 NESI units), which is represented by triangle symbols. Grey shaded errors represent 95% confidence bands of the estimate for each time point.

Figure 5. Estimated linear change in ABR a) wave I amplitude (Table S10), b) wave V amplitude (Table S11), c) wave I latency (Table S13), and d) wave V latency (Table S14), across the study period (T0-T2) based on fixed effects β values in the conditional growth models. A positive slope indicates an increase in wave amplitudes/latencies across the study period. Linear change for a participant with average musical experience is represented by circle symbols. Low musical experience corresponds to 2 SD below the mean average yearly change in noise exposure (-12 years), which is represented by square symbols. High musical experience corresponds to 2 SD above the mean average yearly change in noise exposure (+12 years), which is represented by triangle symbols. Grey shaded errors represent 95% confidence bands of the estimate for each time point.

Figure 6. Pie charts showing change in tinnitus experience from T0-T1 (left panel) and T1-T2 (right panel). The top pie charts correspond to whether participants experienced a change in their experience of tinnitus since the previous testing session. For those who indicated a change, the middle pie charts correspond to whether participants indicated an improvement or worsening of their experience of tinnitus. The bottom pie charts correspond to the reasons why participants indicated an improvement or worsening of their experience of tinnitus.

Figure 7. Spearman's rho correlations between total noise exposure and average DPOAE level (a and b), and average wave V latency (c and d), at time T1 (left panels) and T2 (right panels). * Indicates significant correlation ($p < .05$).

Figure 1

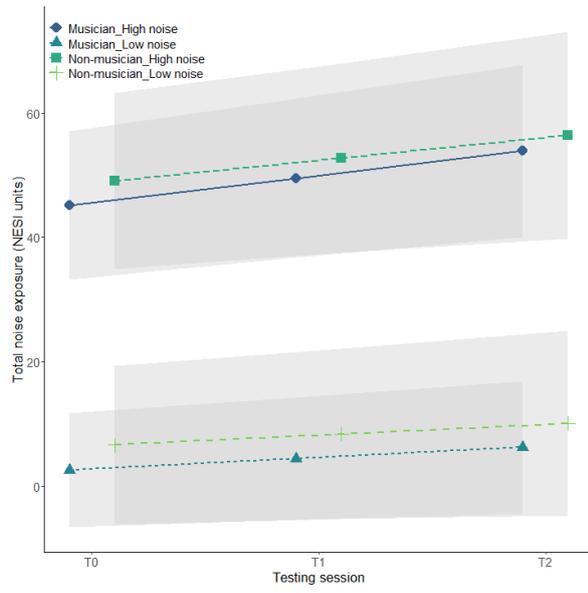


Figure 2

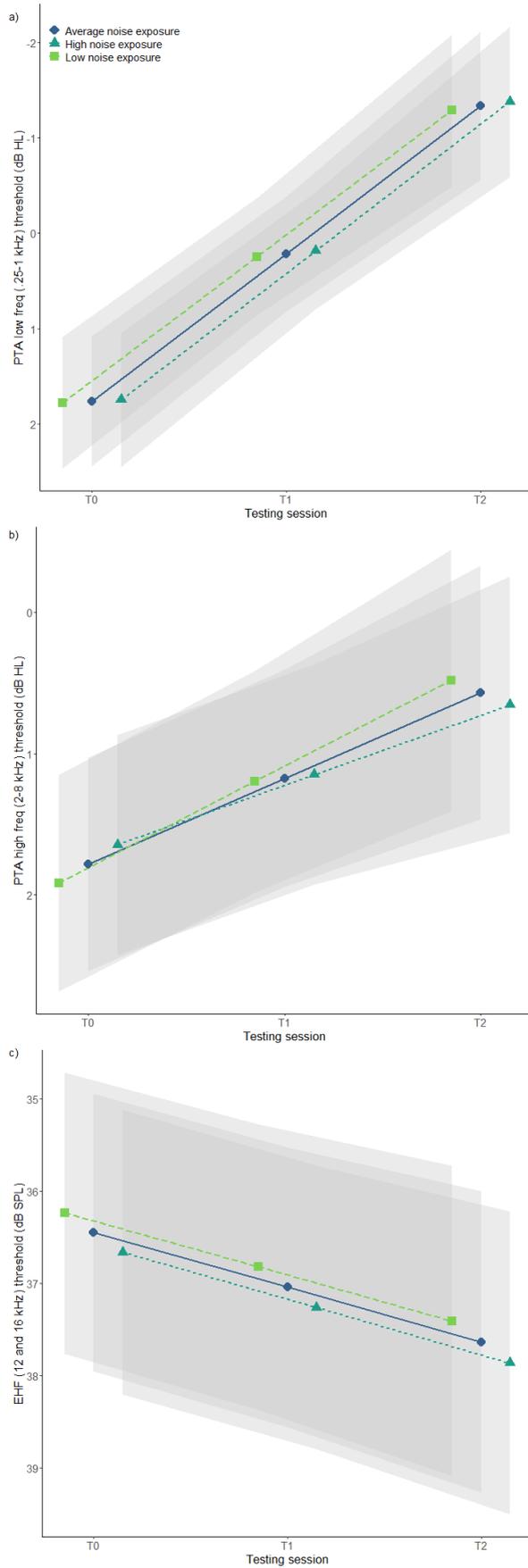


Figure 3

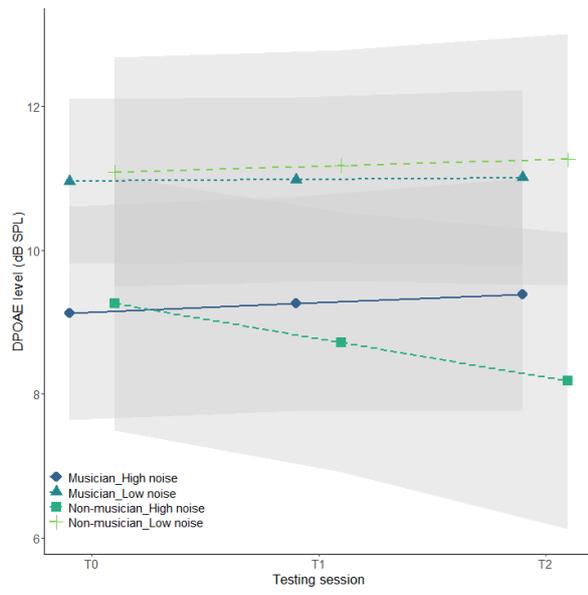


Figure 4

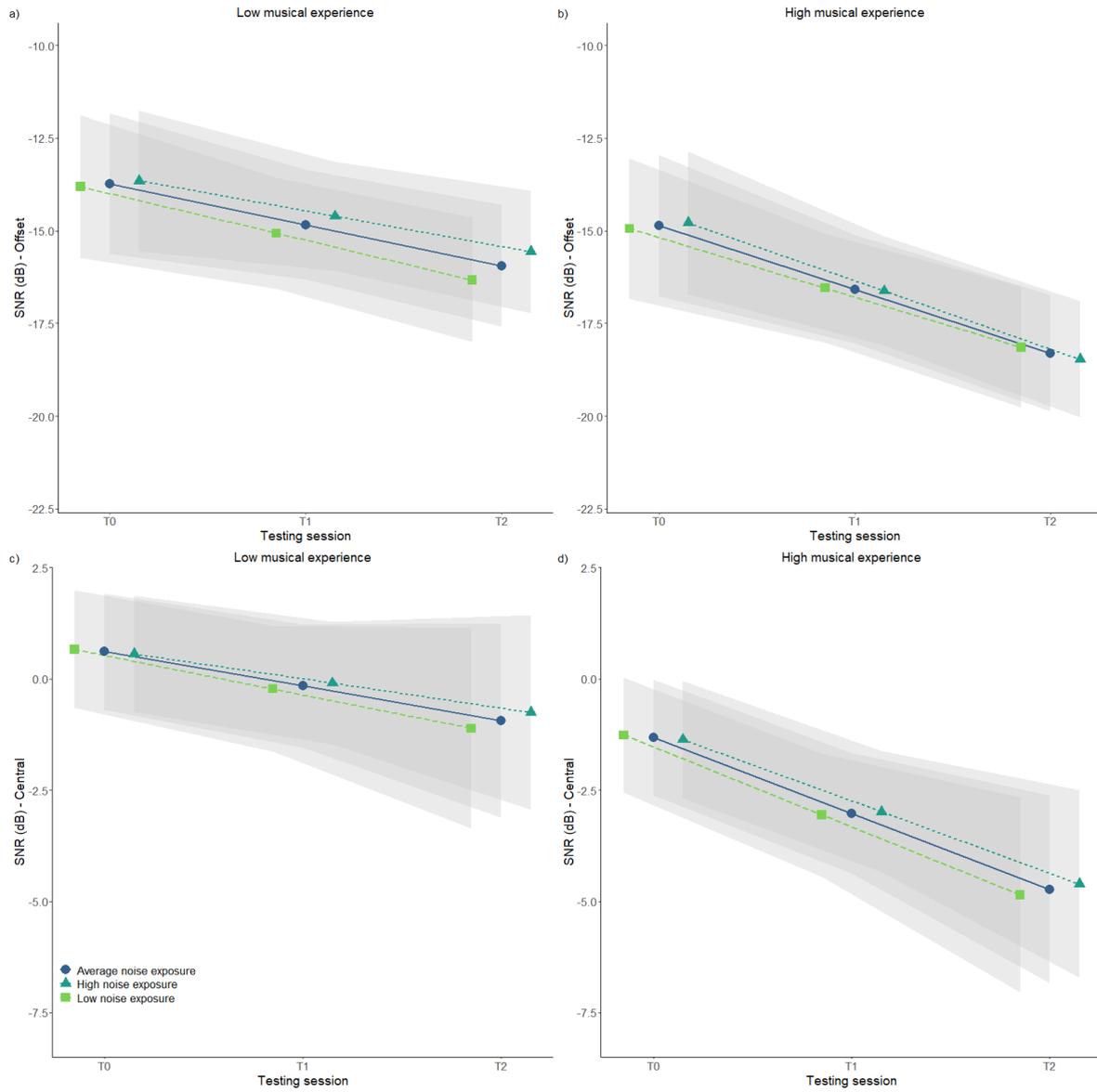


Figure 5

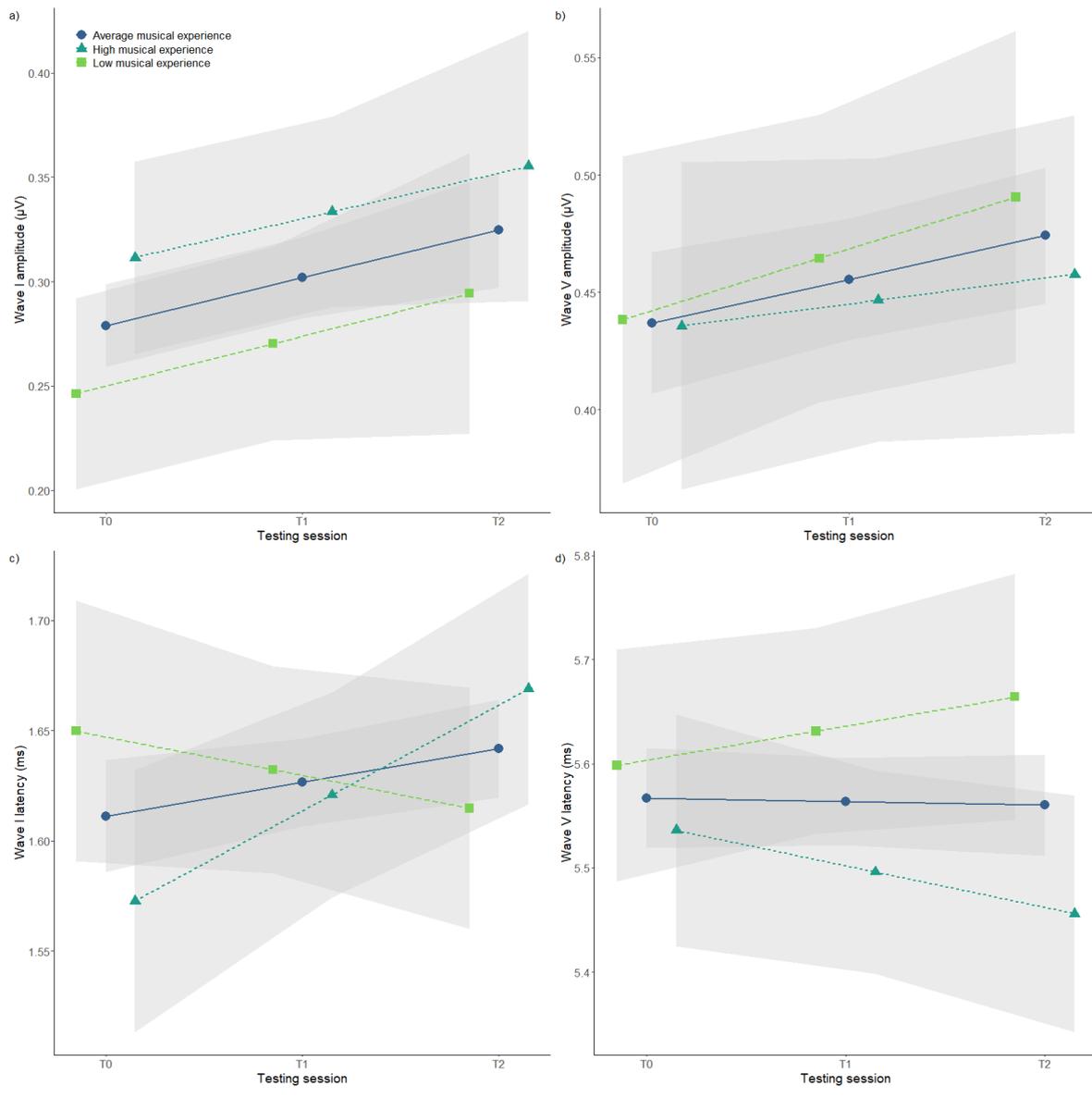


Figure 6

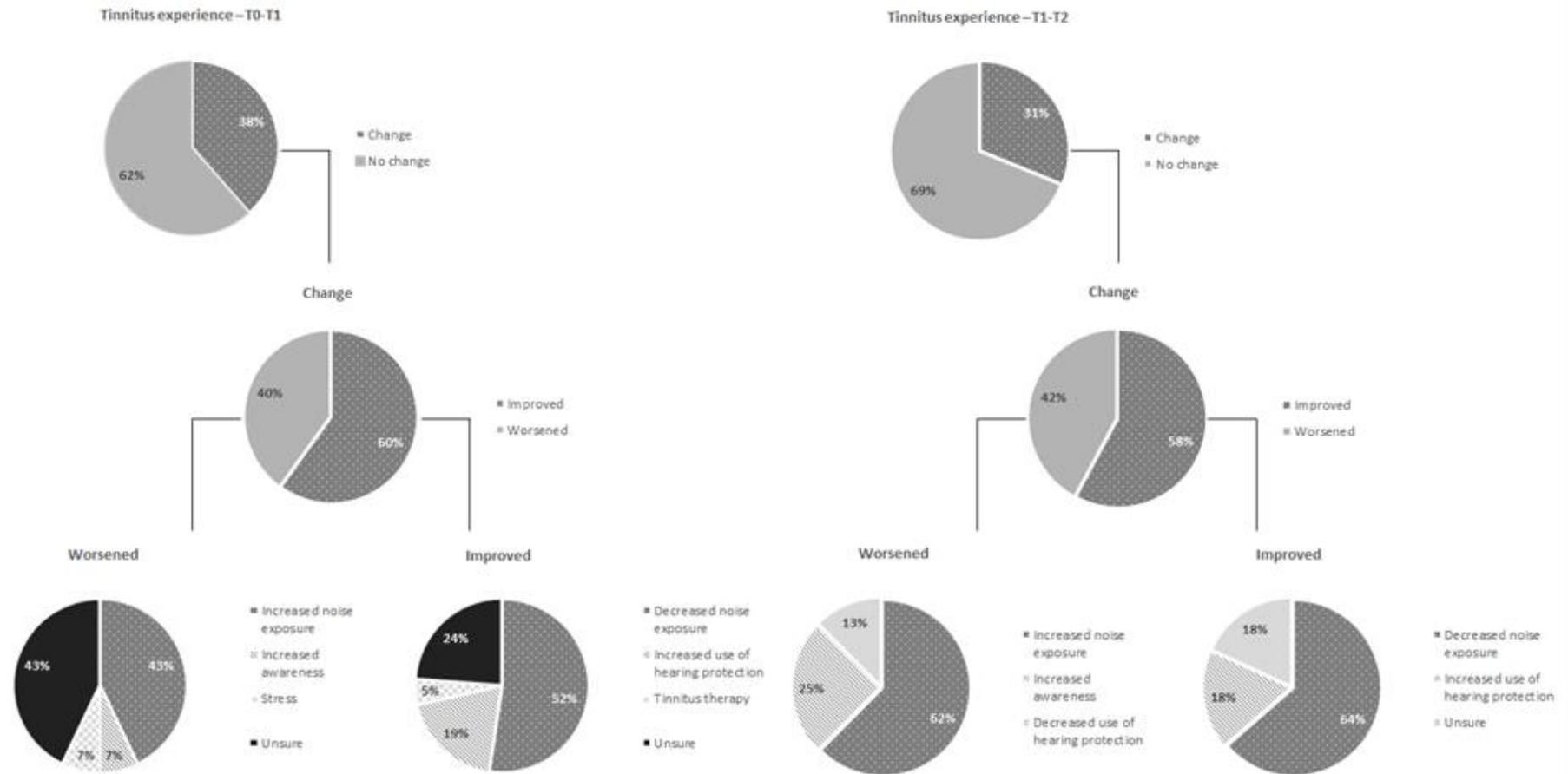


Figure 7

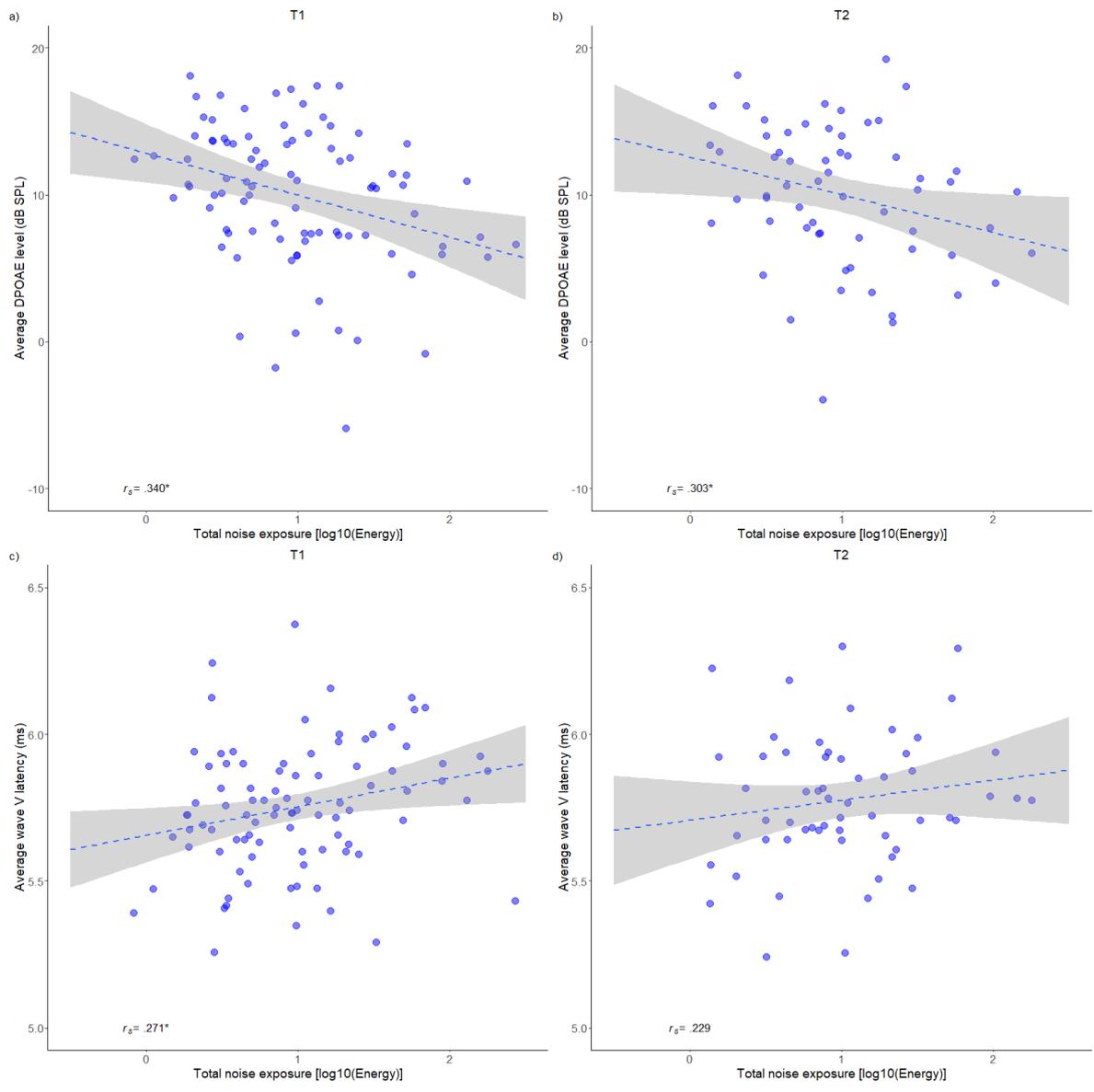


Table 1

Table 1 Intraclass correlation coefficients between measures at T0-T1 and T1-T2. Interpretation of ICC3 values according to Cicchetti, 1994.

Measure	T0-T1		T1-T2	
	ICC3	Interpretation	ICC3	Interpretation
PTA low (.25-1 kHz)	0.668	Good	0.507	Fair
PTA high (2-8kHz)	0.753	Excellent	0.870	Excellent
EHF thresholds (12 and 16 kHz)	0.887	Excellent	0.881	Excellent
Average DPOAE (1-6 kHz)	0.929	Excellent	0.942	Excellent
CRM offset	0.571	Fair	0.704	Good
CRM central	0.350	Poor	0.585	Fair
ABR wI amplitude (80dB nHL)	0.497	Fair	0.921	Excellent
ABR wI latency (80dB nHL)	0.575	Fair	0.776	Excellent
ABR wV amplitude (80dB nHL)	0.651	Good	0.807	Excellent
ABR wV latency (80dB nHL)	0.690	Good	0.720	Good

Author statement

This study was approved by the University of Manchester Research Ethics Committee (approval reference – 16388 – 29/09/2016) in accordance with the Declaration of Helsinki 2013. All participants provided informed consent.

Supplementary materials 1

Due to a product recall of the ICS Chartr EP 200 system in June 2019, we switched to using an Interacoustics Eclipse clinical system. This meant that at T1, two participants (both non-musicians) had ABRs recorded using the Eclipse system, and at T2, 26 participants (20 musicians) had ABRs recorded using the Eclipse system. Identical montage configurations and recording parameters were used for both systems. Nevertheless, it was possible that ABR wave amplitudes and latencies may have differed between the two systems, which could have falsely indicated a change to ABRs between these two testing sessions.

To test this possibility, we compared ABRs for 25 participants³ (20 musicians) who had ABRs recorded using the Interacoustics Eclipse system at T2, to 37 participants (28 musicians) who had ABRs recorded using the ICS Chartr EP 200 system at T2. Average wave I and average wave V amplitudes and latencies were taken across left and right ears, at both 60 dB nHL and 80 dB nHL levels separately. Independent samples t-tests indicated significant differences between the recording systems for average wave I and average wave V latencies at both 60 dB nHL and 80 dB nHL intensities (all $p < .001$), and wave I-V interpeak latency at 80 dB nHL ($p = .004$) (Table S1). Wave I and wave V amplitudes did not significantly differ between the two systems (all $p > .05$), but approached significance for wave I at 80 dB nHL ($p = .051$) and for wave I/V ratio at 80 dB nHL ($p = .052$) (Table S1). Independent samples t-tests also showed that there were no significant differences in PTA thresholds at T2 and levels of noise exposure reported at T2 between the two groups (both $p > .05$; Table S1).

Given these differences between the two ABR systems, we chose to apply a correction factor to the Interacoustics Eclipse data to bring this in line with the Chartr EP data. To do this, we chose participants who had ABRs recorded using the Eclipse system at T2, reported less than one unit of total noise exposure between visits T1 and T2, and had <5 dB difference in average PTA thresholds between T1 and T2. Thirteen participants (10 musicians) fit these criteria. To confirm a difference between the ABRs recorded using the Chartr EP system at T1 and Eclipse system at T2 in these participants, we conducted paired samples t-tests for average wave I and wave V amplitudes and latencies. Significant differences between testing sessions were found for wave I and wave V latencies for both stimulus intensities (all $p < .001$), wave I-V interpeak latencies at 80 dB nHL ($p < .001$), and wave I amplitudes at 80 dB nHL ($p = .022$) (Table S2). Accordingly, we considered that correction factors were warranted for all wave amplitudes and latencies, for both stimulus intensities.

³ Note one non-musician did not complete ABRs at T1, and so was not included in this correction factor analysis. Correction values were applied to this participants' data for inclusion in the linear mixed models analysis.

Correction factors were calculated in two ways. First by calculating *absolute* differences in ABR measures between T1 and T2 for each of the 13 selected participants, and then calculating the mean difference across these participants for each of the ABR measures (as per Table S2). These mean absolute differences were then applied to all 25 participants' ABR measures that were recorded using the Eclipse system. The second method for calculating the correction factors was to take the *ratio* between ABR measures recorded at T1 and those recorded at T2 for each of the 13 selected participants, and then calculate the mean ratio across these participants for each of the ABR measures. These mean ratios were then applied to all 25 participants' ABR measures that were recorded using the Eclipse system.

Table S 1 Independent samples t-tests comparing ABR measures for the ICS Chartr EP 200 system to the Interacoustics Eclipse system at T2 prior to applying any correction factors.

ABR measure		Levene's Test for Equality of Variances		t-test for Equality of Means				
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference
wI latency_60 dB nHL	Equal variances assumed	1.747	0.191	-9.348	60	0.000*	-0.384	0.041
	Equal variances not assumed			-9.778	58.375	0.000	-0.384	0.039
wI amplitude_60 dB nHL	Equal variances assumed	6.000	0.017	-1.196	60	0.236	-0.014	0.011
	Equal variances not assumed			-1.277	59.851	0.206	-0.014	0.011
wV latency_60 dB nHL	Equal variances assumed	2.210	0.142	-6.179	60	0.000*	-0.403	0.065
	Equal variances not assumed			-6.605	59.876	0.000	-0.403	0.061
wV amplitude_60 dB nHL	Equal variances assumed	0.107	0.745	-0.676	60	0.501	-0.018	0.027
	Equal variances not assumed			-0.686	54.181	0.495	-0.018	0.027
w I/V ratio_60 dB nHL	Equal variances assumed	0.531	0.469	-0.705	60	0.484	-0.027	0.038
	Equal variances not assumed			-0.762	59.981	0.449	-0.027	0.036
wI-V IPL_60 dB nHL	Equal variances assumed	2.991	0.089	-0.331	60	0.742	-0.018	0.055
	Equal variances not assumed			-0.356	59.994	0.723	-0.018	0.051
wI latency_80 dB nHL	Equal variances assumed	0.798	0.375	-12.969	60	0.000*	-0.349	0.027
	Equal variances not assumed			-12.995	52.023	0.000	-0.349	0.027
wI amplitude_80 dB nHL	Equal variances assumed	5.431	0.023	-1.826	60	0.073	-0.056	0.031
	Equal variances not assumed			-1.991	59.695	0.051	-0.056	0.028
wV latency_80 dB nHL	Equal variances assumed	0.076	0.783	-8.697	60	0.000*	-0.504	0.058
	Equal variances not assumed			-8.729	52.321	0.000	-0.504	0.058
wV amplitude_80 dB nHL	Equal variances assumed	1.748	0.191	0.073	60	0.942	0.003	0.035
	Equal variances not assumed			0.075	56.444	0.941	0.003	0.034
w I/V ratio_80 dB nHL	Equal variances assumed	2.547	0.116	-1.985	60	0.052	-0.130	0.065
	Equal variances not assumed			-2.104	59.498	0.040	-0.130	0.062
wI-V IPL_80 dB nHL	Equal variances assumed	0.002	0.961	-3.024	60	0.000*	-0.155	0.051
	Equal variances not assumed			-3.029	51.977	0.004	-0.155	0.051
PTA_T2	Equal variances assumed	0.153	0.697	1.783	60	0.080	1.464	0.821
	Equal variances not assumed			1.711	44.149	0.094	1.464	0.855
Total noise exposure_T2	Equal variances assumed	1.700	0.197	0.532	60	0.597	0.409	0.769
	Equal variances not assumed			0.5	40.463	0.620	0.409	0.818

* indicates a significant difference

Table S 2 Paired samples t-tests comparing T1 ABR measures recorded with the ICS Chartr EP 200 system to T2 ABR measures recorded with the Interacoustics Eclipse system prior to applying any corrections factors, for 13 participants with < 1 unit of total noise exposure between T1 and T2 and <5 dB difference in PTA thresholds between T1 and T2.

ABR measure	Paired Differences		t	df	Sig. (2-tailed)
	Mean	Std. Deviation			
T1 wl latency_60 dB nHL - T2 wl latency_60 dB nHL	0.388	0.094	14.959	12	0.000*
T1 wl amplitude_60 dB nHL - T2 wl amplitude_60 dB nHL	0.005	0.032	0.522	12	0.611
T1 wV latency_60 dB nHL - T2 wV latency_60 dB nHL	0.423	0.058	26.137	12	0.000*
T1 wV amplitude_60 dB nHL - T2 wV amplitude_60 dB nHL	0.029	0.056	1.886	12	0.084
T1 wl/V ratio_60 dB nHL - T2 wl/V ratio_60 dB nHL	-0.002	0.100	-0.072	12	0.943
T1 wl-V IPL_60 dB nHL - T2 wl-V IPL_60 dB nHL	0.033	0.114	1.042	12	0.318
T1 wl latency_80 dB nHL - T2 wl latency_80 dB nHL	0.350	0.030	41.486	12	0.000*
T1 wl amplitude_80 dB nHL - T2 wl amplitude_80 dB nHL	0.031	0.043	2.628	12	0.022*
T1 wV latency_80 dB nHL - T2 wV latency_80 dB nHL	0.555	0.088	22.671	12	0.000*
T1 wV amplitude_80 dB nHL - T2 wV amplitude_80 dB nHL	0.002	0.081	0.078	12	0.939
T1 wl/V ratio_80 dB nHL - T2 wl/V ratio_80 dB nHL	0.082	0.151	1.962	12	0.073
T1 wl-V IPL_80 dB nHL - T2 wl-V IPL_80 dB nHL	0.205	0.097	7.622	12	0.000*

* indicates a significant difference

After applying these correction factors, independent samples t-tests were repeated to compare ABRs for the 25 participants who had ABRs recorded using the Eclipse system at T2, to the 37 participants who had ABRs recorded using the Chartr EP system at T2. For both correction methods (absolute and ratio), there were no significant differences between any of the corrected Eclipse system ABR measures and the Chart EP system measures (all $p > .05$; Table S3 and Table S4).

For all wave latency measures, mean differences between the Chartr EP system and the Eclipse system were smaller for the absolute correction values (Table S3) compared to the ratio correction values (Table S4). Conversely, for nearly all wave amplitude measures (except wave V amplitude at 60 dB nHL), mean differences between the Chartr EP system and the Eclipse system were smaller for the ratio correction values (Table S4) compared to the absolute correction values (Table S4). Accordingly, for the linear mixed effects models, wave latency values for the Eclipse system were corrected using the mean absolute differences, whilst the wave amplitude values were corrected using the mean ratios.

Table S 3 Independent samples t-tests comparing ABR measures for the ICS Chartr EP 200 system to the Interacoustics Eclipse system at T2 after correcting using mean absolute differences.

ABR measure		Levene's Test for Equality of Variances		t-test for Equality of Means				
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference
wI latency_60 dB nHL	Equal variances assumed	1.747	0.191	0.111	60	0.912	0.005	0.041
	Equal variances not assumed			0.116	58.375	0.908	0.005	0.039
wI amplitude_60 dB nHL	Equal variances assumed	6.000	0.017	-0.784	60	0.436	-0.009	0.011
	Equal variances not assumed			-0.837	59.851	0.406	-0.009	0.011
wV latency_60 dB nHL	Equal variances assumed	2.210	0.142	0.306	60	0.760	0.020	0.065
	Equal variances not assumed			0.328	59.876	0.744	0.020	0.061
wV amplitude_60 dB nHL	Equal variances assumed	0.103	0.749	0.402	60	0.689	0.011	0.027
	Equal variances not assumed			0.408	54.184	0.685	0.011	0.027
w I/V ratio_60 dB nHL	Equal variances assumed	0.530	0.469	-0.757	60	0.452	-0.029	0.038
	Equal variances not assumed			-0.818	59.981	0.416	-0.029	0.036
wI-V IPL_60 dB nHL	Equal variances assumed	2.991	0.089	0.265	60	0.792	0.015	0.055
	Equal variances not assumed			0.285	59.994	0.777	0.015	0.051
wI latency_80 dB nHL	Equal variances assumed	0.798	0.375	0.021	60	0.983	0.001	0.027
	Equal variances not assumed			0.021	52.023	0.983	0.001	0.027
wI amplitude_80 dB nHL	Equal variances assumed	5.431	0.023	-0.805	60	0.424	-0.025	0.031
	Equal variances not assumed			-0.878	59.695	0.384	-0.025	0.028
wV latency_80 dB nHL	Equal variances assumed	0.076	0.783	0.875	60	0.385	0.051	0.058
	Equal variances not assumed			0.879	52.321	0.384	0.051	0.058
wV amplitude_80 dB nHL	Equal variances assumed	1.748	0.191	0.122	60	0.903	0.004	0.035
	Equal variances not assumed			0.126	56.444	0.900	0.004	0.034
w I/V ratio_80 dB nHL	Equal variances assumed	2.553	0.115	-0.725	60	0.471	-0.047	0.065
	Equal variances not assumed			-0.769	59.505	0.445	-0.047	0.062
wI-V IPL_80 dB nHL	Equal variances assumed	0.003	0.958	0.977	60	0.332	0.050	0.051
	Equal variances not assumed			0.979	51.957	0.332	0.050	0.051

Table S 4 Independent samples t-tests comparing ABR measures for the ICS Chartr EP 200 system to the Interacoustics Eclipse system at T2 after correcting using mean ratios.

ABR measure		Levene's Test for Equality of Variances		t-test for Equality of Means				
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference
wl latency_60 dB nHL	Equal variances assumed	0.045	0.833	0.500	60	0.619	0.022	0.044
	Equal variances not assumed			0.501	52.071	0.618	0.022	0.044
wl amplitude_60 dB nHL	Equal variances assumed	2.525	0.117	-0.101	60	0.920	-0.001	0.012
	Equal variances not assumed			-0.105	57.768	0.917	-0.001	0.011
wV latency_60 dB nHL	Equal variances assumed	1.373	0.246	0.378	60	0.707	0.025	0.067
	Equal variances not assumed			0.399	59.147	0.692	0.025	0.063
wV amplitude_60 dB nHL	Equal variances assumed	0.043	0.836	0.488	60	0.627	0.014	0.028
	Equal variances not assumed			0.486	50.958	0.629	0.014	0.028
w I/V ratio_60 dB nHL	Equal variances assumed	0.183	0.67	-0.064	60	0.949	-0.003	0.039
	Equal variances not assumed			-0.068	59.684	0.946	-0.003	0.037
wl-V IPL_60 dB nHL	Equal variances assumed	2.861	0.096	0.314	60	0.755	0.017	0.055
	Equal variances not assumed			0.337	59.979	0.737	0.017	0.051
wl latency_80 dB nHL	Equal variances assumed	0.245	0.623	0.368	60	0.714	0.011	0.030
	Equal variances not assumed			0.351	43.356	0.727	0.011	0.032
wl amplitude_80 dB nHL	Equal variances assumed	3.224	0.078	-0.745	60	0.459	-0.024	0.032
	Equal variances not assumed			-0.798	59.909	0.428	-0.024	0.029
wV latency_80 dB nHL	Equal variances assumed	0.649	0.424	1.026	60	0.309	0.062	0.061
	Equal variances not assumed			1.008	48.541	0.318	0.062	0.062
wV amplitude_80 dB nHL	Equal variances assumed	1.705	0.197	0.115	60	0.909	0.004	0.035
	Equal variances not assumed			0.118	56.357	0.907	0.004	0.034
w I/V ratio_80 dB nHL	Equal variances assumed	0.748	0.391	-0.500	60	0.619	-0.034	0.068
	Equal variances not assumed			-0.516	56.691	0.608	-0.034	0.066
wl-V IPL_80 dB nHL	Equal variances assumed	0.098	0.755	1.051	60	0.298	0.055	0.052
	Equal variances not assumed			1.042	50.056	0.303	0.055	0.053

Supplementary materials 2

Table S2 Linear mixed effects model for occupational noise exposure

	Unconditional means model			Unconditional linear growth model			Conditional linear growth model		
	β	SE	p	β	SE	p	β	SE	p
Fixed effects									
Intercept	2.660	0.629	<.001	1.937	0.533	<.001	1.981	0.540	<.001
Time				0.936	0.333	0.006	0.882	0.350	0.013
Musicianship group (vs. Musicians)							-1.269	0.525	0.018
Noise exposure group (vs. Low noise)							1.816	0.509	0.001
Time*Musicianship group (vs. Musicians)							-0.739	0.349	0.037
Time*Noise exposure group (vs. Low noise)							0.639	0.347	0.069
Time*Musicianship group (vs. Musicians)*Noise exposure group (vs. Low noise)							-0.256	0.327	0.436
Random effects									
Residual	4.669	0.536	<.001	0.198	0.038	<.001	0.198	0.038	<.001
<i>Intercept + Time (Individual level)</i>									
Intercept	35.370	5.446	<.001	26.478	3.888	<.001	22.257	3.272	<.001
Intercept*Slope				7.400	1.872	<.001	5.280	1.622	0.001
Slope				10.201	1.546	<.001	9.182	1.408	<.001

Table S3 Linear mixed effects model for recreational noise exposure

	Unconditional means model			Unconditional linear growth model			Conditional linear growth model		
	β	SE	p	β	SE	p	β	SE	p
Fixed effects									
Intercept	18.541	3.902	<.001	17.151	3.819	<.001	23.755	3.609	<.001
Time				1.716	0.359	<.001	2.048	0.393	<.001
Musicianship group (vs. Musicians)							3.335	3.507	0.344
Noise exposure group (vs. Low noise)							19.529	3.403	<.001
Time*Musicianship group (vs. Musicians)							0.563	0.392	0.153
Time*Noise exposure group (vs. Low noise)							0.633	0.391	0.108
Time*Musicianship group (vs. Musicians)*Noise exposure group (vs. Low noise)							0.390	0.376	0.302
Random effects									
Residual	14.676	1.684	<.001	2.579	0.431	<.001	2.579	0.431	<.001
<i>Intercept + Time (Individual level)</i>									
Intercept	1425.3	208.83	<.001	1368.8	199.99	<.001	999.82	146.18	<.001
Intercept*Slope				44.588	14.411	0.002	33.388	11.962	0.005

Slope	9.737	1.707	<.001	9.164	1.620	<.001
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Table S4 Linear mixed effects model for total noise exposure

	Unconditional means model			Unconditional linear growth model			Conditional linear growth model		
	β	SE	p	β	SE	p	β	SE	p
Fixed effects									
Intercept	21.364	4.039	<.001	19.254	3.892	<.001	25.859	3.604	<.001
Time				2.645	0.505	<.001	2.902	0.549	<.001
Musicianship group (vs. Musicians)							2.009	3.503	0.568
Noise exposure group (vs. Low noise)							21.246	3.398	<.001
Time*Musicianship group (vs. Musicians)							-0.198	0.547	0.718
Time*Noise exposure group (vs. Low noise)							1.121	0.544	0.042
Time*Musicianship group (vs. Musicians)*Noise exposure group (vs. Low noise)							-0.166	0.505	0.743
Random effects									
Residual	24.135	2.769	<.001	3.124	0.597	<.001	3.106	0.591	<.001
<i>Intercept + Time (Individual level)</i>									
Intercept	1523.6	223.76	<.001	1421.1	207.71	<.001	996.75	145.81	<.001
Intercept*Slope				84.234	21.213	<.001	61.619	17.089	<.001
Slope				21.010	3.704	<.001	19.814	3.527	<.001

Table S4 Linear mixed effects model for PTA low (.25-1 kHz)

	Unconditional means model			Unconditional linear growth model			Conditional linear growth model		
	β	SE	p	β	SE	p	β	SE	p
Fixed effects									
Intercept	0.475	0.303	0.120	1.768	0.345	<.001	1.762	0.340	<.001
Time				-1.546	0.208	<.001	-1.547	0.207	<.001
Musical experience (Years at T0)							0.095	0.060	0.113
Total noise exposure							-0.002	0.009	0.841
Time*Musical experience (Years at T0)							0.011	0.037	0.768
Time*Total noise exposure							-0.002	0.006	0.737
Time*Musical experience (Years at T0)* Total noise exposure							-4.160	0.001	0.581
							$\times 10^{-4}$		
Random effects									
Residual	7.876	0.904	<.001	5.119	0.833	<.001	5.203	0.856	<.001
<i>Intercept + Time (Individual level)</i>									
Intercept	5.532	1.308	<.001	6.770	1.757	<.001	6.399	1.721	<.001
Intercept*Slope				-0.280	0.849	0.742	-0.255	0.833	0.760
Slope				0.541	0.689	0.432	0.429	0.692	0.535

Table S5 Linear mixed effects model for PTA high (2-8 kHz)

	Unconditional means model			Unconditional linear growth model			Conditional linear growth model		
	β	SE	p	β	SE	p	β	SE	p
Fixed effects									
Intercept	1.626	0.371	<.001	1.905	0.383	<.001	1.897	0.374	<.001
Time				-0.521	0.155	0.001	-0.495	0.149	0.001
Musical experience (Years at T0)							0.086	0.065	0.191
Total noise exposure							-0.017	0.010	0.083
Time*Musical experience (Years at T0)							-0.010	0.027	0.703
Time*Total noise exposure							0.014	0.004	0.002
Time*Musical experience (Years at T0)* Total noise exposure							-4.630 x 10 ⁻⁴	0.001	0.496
Repeated measures ^a									
UN (1,1)	13.976	2.059	<.001	13.853	2.022	<.001	13.153	1.920	<.001
UN (2,1)	11.480	2.039	<.001	11.744	2.038	<.001	11.633	2.007	<.001
UN (2,2)	18.221	2.745	<.001	17.896	2.655	<.001	17.963	2.683	<.001
UN (3,1)	10.693	1.986	<.001	11.090	1.976	<.001	11.168	1.944	<.001
UN (3,2)	14.952	2.502	<.001	14.013	2.348	<.001	14.201	2.369	<.001
UN (3,3)	16.126	2.649	<.001	15.306	2.479	<.001	15.389	2.491	<.001

Table S6 Linear mixed effects model for EHF thresholds (12-16 kHz)

	Unconditional means model			Unconditional linear growth model			Conditional linear growth model		
	β	SE	p	β	SE	p	β	SE	p
Fixed effects									
Intercept	36.659	0.755	<.001	36.408	0.756	<.001	36.452	0.746	<.001
Time				0.661	0.175	<.001	0.593	0.181	0.002
Musical experience (Years at T0)							0.053	0.130	0.683
Total noise exposure							0.026	0.018	0.152
Time*Musical experience (Years at T0)							-0.004	0.032	0.890
Time*Total noise exposure							0.001	0.005	0.848
Time*Musical experience (Years at T0)* Total noise exposure							0.001	0.001	0.528
Repeated measures ^a									
UN (1,1)	54.848	8.011	<.001	54.782	7.991	<.001	52.928	7.742	<.001
UN (2,1)	50.716	7.912	<.001	50.824	7.908	<.001	49.600	7.752	<.001
UN (2,2)	60.105	8.821	<.001	59.917	8.768	<.001	59.567	8.766	<.001
UN (3,1)	52.653	8.359	<.001	53.696	8.363	<.001	51.817	8.120	<.001
UN (3,2)	53.925	8.696	<.001	54.578	8.601	<.001	53.644	8.502	<.001
UN (3,3)	60.068	9.951	<.001	60.729	9.686	<.001	58.849	9.431	<.001

^a The random effects within-subject error covariance structure was deemed to have a redundant Slope parameter and so the test statistic and variance could not be computed. Accordingly, the LMM was re-run using an unstructured (UN) residual covariance structure in which the variance between time points is not constant and correlations between time points differ across time.

Table S7 Linear mixed effects model for average DPOAEs (1-6 kHz)

	Unconditional means model			Unconditional linear growth model			Conditional linear growth model		
	β	SE	p	β	SE	p	β	SE	p
Fixed effects									
Intercept	10.332	0.413	<.001	10.330	0.417	<.001	10.106	0.450	<.001
Time				0.006	0.106	0.958	-0.074	0.122	0.546
Musicianship group (vs. Musicians)							0.066	0.438	0.880
Noise exposure group (vs. Low noise)							-0.915	0.424	0.034
Time*Musicianship group (vs. Musicians)							-0.152	0.121	0.214
Time*Noise exposure group (vs. Low noise)							-0.131	0.121	0.283
Time*Musicianship group (vs. Musicians)*Noise exposure group (vs. Low noise)							-0.182	0.120	0.132
Random effects									
Residual	1.323	0.152	<.001	1.081	0.186	<.001	1.056	0.181	<.001
<i>Intercept + Time (Individual level)</i>									
Intercept	15.539	2.343	<.001	15.417	2.389	<.001	14.669	2.277	<.001
Intercept*Slope				0.034	0.433	0.937	-0.030	0.425	0.943
Slope				0.267	0.186	0.152	0.264	0.181	0.145

Table S8 Linear mixed effects model for the offset condition of the CRM task

	Unconditional means model			Unconditional linear growth model			Conditional linear growth model		
	β	SE	p	β	SE	p	β	SE	p
Fixed effects									
Intercept	-15.495	0.335	<.001	-14.287	0.409	<.001	-14.297	0.410	<.001
Time				-1.435	0.202	<.001	-1.415	0.207	<.001
Musical experience (Years at T0)							-0.047	0.072	0.513
Total noise exposure							0.010	0.011	0.361
Time*Musical experience (Years at T0)							-0.026	0.037	0.483
Time*Total noise exposure							0.002	0.006	0.752
Time*Musical experience (Years at T0)* Total noise exposure							-0.001	0.001	0.037
Random effects									
Residual	7.780	0.890	<.001	5.385	0.822	<.001	5.228	0.807	<.001
<i>Intercept + Time (Individual level)</i>									
Intercept	7.479	1.567	<.001	11.140	2.338	<.001	11.279	2.398	<.001
Intercept*Slope				-2.051	1.035	0.047	-2.375	1.080	0.028
Slope				0.378	0.675	0.576	0.595	0.699	0.395

Table S9 Linear mixed effects model for the central condition of the CRM task

	Unconditional means model			Unconditional linear growth model			Conditional linear growth model		
	β	SE	<i>p</i>	β	SE	<i>p</i>	β	SE	<i>p</i>
Fixed effects									
Intercept	-1.401	0.285	<.001	-0.336	0.284	0.241	-0.350	0.280	0.215
Time				-1.262	0.241	<.001	-1.241	0.239	<.001
Musical experience (Years at T0)							-0.080	0.049	0.105
Total noise exposure							-0.006	0.008	0.395
Time*Musical experience (Years at T0)							-0.039	0.043	0.368
Time*Total noise exposure							0.013	0.007	0.063
Time*Musical experience (Years at T0)* Total noise exposure							-1.450 x 10 ⁻⁴	0.001	0.869
Random effects									
Residual	8.522	0.975	<.001	6.015	0.993	<.001	5.830	0.960	<.001
<i>Intercept + Time (Individual level)</i>									
Intercept	4.314	1.172	<.001	2.407	1.445	0.096	2.335	1.403	0.096
Intercept*Slope				1.134	0.998	0.256	0.964	0.984	0.327
Slope				1.140	0.942	0.226	1.135	0.918	0.216

Table S10 Linear mixed effects model for ABR wave I amplitudes

	Unconditional means model			Unconditional linear growth model			Conditional linear growth model		
	β	SE	<i>p</i>	β	SE	<i>p</i>	β	SE	<i>p</i>
Fixed effects									
Intercept	0.297	0.010	<.001	0.279	0.010	<.001	0.279	0.010	<.001
Time				0.023	0.007	0.001	0.023	0.007	0.001
Musical experience (Years at T0)							0.003	0.002	0.120
Total noise exposure							-5.837 x 10 ⁻⁵	2.600 x 10 ⁻⁴	0.823
Time*Musical experience (Years at T0)							-9.054 x 10 ⁻⁵	0.001	0.943
Time*Total noise exposure							-7.657 x 10 ⁻⁵	1.900 x 10 ⁻⁴	0.688
Time*Musical experience (Years at T0)* Total noise exposure							-1.328 x 10 ⁻⁵	2.436 x 10 ⁻⁵	0.587
Random effects									
Residual	0.005	0.001	<.001	0.003	4.900 x 10 ⁻⁴	<.001	0.003	4.850 x 10 ⁻⁴	<.001
<i>Intercept + Time (Individual level)</i>									
Intercept	0.007	0.001	<.001	0.007	0.001	<.001	0.007	0.001	<.001
Intercept*Slope				-0.001	0.001	0.488	-0.001	0.001	0.439
Slope				0.002	0.001	0.002	0.002	0.001	0.002

Table S11 Linear mixed effects model for ABR wave V amplitudes

	Unconditional means model			Unconditional linear growth model			Conditional linear growth model		
	β	SE	p	β	SE	p	β	SE	p
Fixed effects									
Intercept	0.452	0.013	<.001	0.438	0.015	<.001	0.437	0.015	<.001
Time				0.018	0.007	0.015	0.019	0.007	0.010
Musical experience (Years at T0)							-1.120 $\times 10^{-4}$	0.003	0.966
Total noise exposure							-1.180 $\times 10^{-4}$	3.930 $\times 10^{-4}$	0.766
Time*Musical experience (Years at T0)							-0.001	0.001	0.621
Time*Total noise exposure							-5.393 $\times 10^{-5}$	2.000 $\times 10^{-4}$	0.788
Time*Musical experience (Years at T0)* Total noise exposure							-3.106 $\times 10^{-5}$	2.537 $\times 10^{-5}$	0.223
Random effects									
Residual	0.007	0.001	<.001	0.006	0.001	<.001	0.006	0.001	<.001
<i>Intercept + Time (Individual level)</i>									
Intercept	0.013	0.002	<.001	0.016	0.003	<.001	0.016	0.003	<.001
Intercept*Slope				-0.001	0.001	0.276	-0.001	0.001	0.252
Slope				3.520 $\times 10^{-4}$	0.001	0.707	2.550 $\times 10^{-4}$	0.001	0.788

Table S12 Linear mixed effects model for ABR wave I/V ratio

	Unconditional means model			Unconditional linear growth model			Conditional linear growth model		
	β	SE	p	β	SE	p	β	SE	p
Fixed effects									
Intercept	0.698	0.024	<.001	0.686	0.028	<.001	0.662	0.030	<.001
Time				0.015	0.016	0.348	0.031	0.018	0.085
Musical experience (Years at T0)							-0.078	0.029	0.009
Total noise exposure							0.015	0.028	0.590
Time*Musical experience (Years at T0)							0.033	0.018	0.066
Time*Total noise exposure							0.006	0.017	0.745
Time*Musical experience (Years at T0)* Total noise exposure							-0.004	0.015	0.777
Random effects									
Residual	0.028	0.003	<.001	0.023	0.004	<.001	0.023	0.004	<.001
<i>Intercept + Time (Individual level)</i>									
Intercept	0.044	0.008	<.001	0.055	0.012	<.001	0.049	0.011	<.001
Intercept*Slope				-0.009	0.006	0.157	-0.006	0.006	0.290
Slope				0.006	0.005	0.198	0.005	0.005	0.304

Table S13 Linear mixed effects model for ABR wave I latency

	Unconditional means model			Unconditional linear growth model			Conditional linear growth model		
	β	SE	p	β	SE	p	β	SE	p
Fixed effects									
Intercept	1.623	0.010	<.001	1.6111	0.013	<.001	1.611	0.013	<.001
Time				0.016	0.007	0.021	0.015	0.007	0.026
Musical experience (Years at T0)							-0.003	0.002	0.153
Total noise exposure							1.980	3.330	0.553
Time*Musical experience (Years at T0)							$\times 10^{-4}$	$\times 10^{-4}$	
Time*Total noise exposure							0.003	0.001	0.027
Time*Musical experience (Years at T0)* Total noise exposure							-1.480	1.850	0.427
							$\times 10^{-4}$	$\times 10^{-4}$	
							4.797	2.080	0.818
							$\times 10^{-6}$	$\times 10^{-5}$	
Random effects									
Residual	0.005	0.001	<.001	0.004	0.001	<.001	0.004	0.001	<.001
<i>Intercept + Time (Individual level)</i>									
Intercept	0.008	0.001	<.001	0.012	0.003	<.001	0.012	0.002	<.001
Intercept*Slope				-0.003	0.001	0.031	-0.003	0.001	0.038
Slope				0.002	0.001	0.167	0.001	0.001	0.198

Table S14 Linear mixed effects model for ABR wave V latency

	Unconditional means model			Unconditional linear growth model			Conditional linear growth model		
	β	SE	p	β	SE	p	β	SE	p
Fixed effects									
Intercept	5.563	0.021	<.001	5.5668	0.024	<.001	5.567	0.024	<.001
Time				0.004	0.012	0.705	-0.004	0.012	0.770
Musical experience (Years at T0)							-0.003	0.004	0.536
Total noise exposure							8.818	0.001	0.889
Time*Musical experience (Years at T0)							$\times 10^{-5}$		
Time*Total noise exposure							-0.003	0.002	0.164
Time*Musical experience (Years at T0)* Total noise exposure							3.636	3.350	0.914
							$\times 10^{-5}$	$\times 10^{-4}$	
							4.129	4.173	0.324
							$\times 10^{-5}$	$\times 10^{-5}$	
Random effects									
Residual	0.016	0.002	<.001	0.014	0.003	<.001	0.013	0.002	<.001
<i>Intercept + Time (Individual level)</i>									
Intercept	0.036	0.006	<.001	0.042	0.008	<.001	0.043	0.008	<.001
Intercept*Slope				-0.004	0.003	0.246	-0.005	0.004	0.132
Slope				0.002	0.003	0.333	0.003	0.003	0.189

Table S15 Linear mixed effects model for ABR wave I-V interpeak latency

	Unconditional means model			Unconditional linear growth model			Conditional linear growth model		
	β	SE	p	β	SE	p	β	SE	p
Fixed effects									
Intercept	3.939	0.018	<.001	3.956	0.023	<.001	3.956	0.024	<.001
Time				-0.020	0.014	0.180	-0.018	0.015	0.225
Musical experience (Years at T0)							0.001	0.004	0.756
Total noise exposure							1.350 $\times 10^{-4}$	0.001	0.828
Time*Musical experience (Years at T0)							-0.007	0.003	0.016
Time*Total noise exposure							2.790 $\times 10^{-4}$	4.100 $\times 10^{-4}$	0.498
Time*Musical experience (Years at T0)* Total noise exposure							6.386 $\times 10^{-5}$	4.393 $\times 10^{-5}$	0.149
Random effects									
Residual	0.024	0.003	<.001	0.018	0.004	<.001	0.016	0.003	<.001
<i>Intercept + Time (Individual level)</i>									
Intercept	0.022	0.005	<.001	0.035	0.008	<.001	0.038	0.008	<.001
Intercept*Slope				-0.010	0.005	0.048	-0.012	0.005	0.014
Slope				0.006	0.004	0.173	0.008	0.005	0.067

Supplementary materials 3

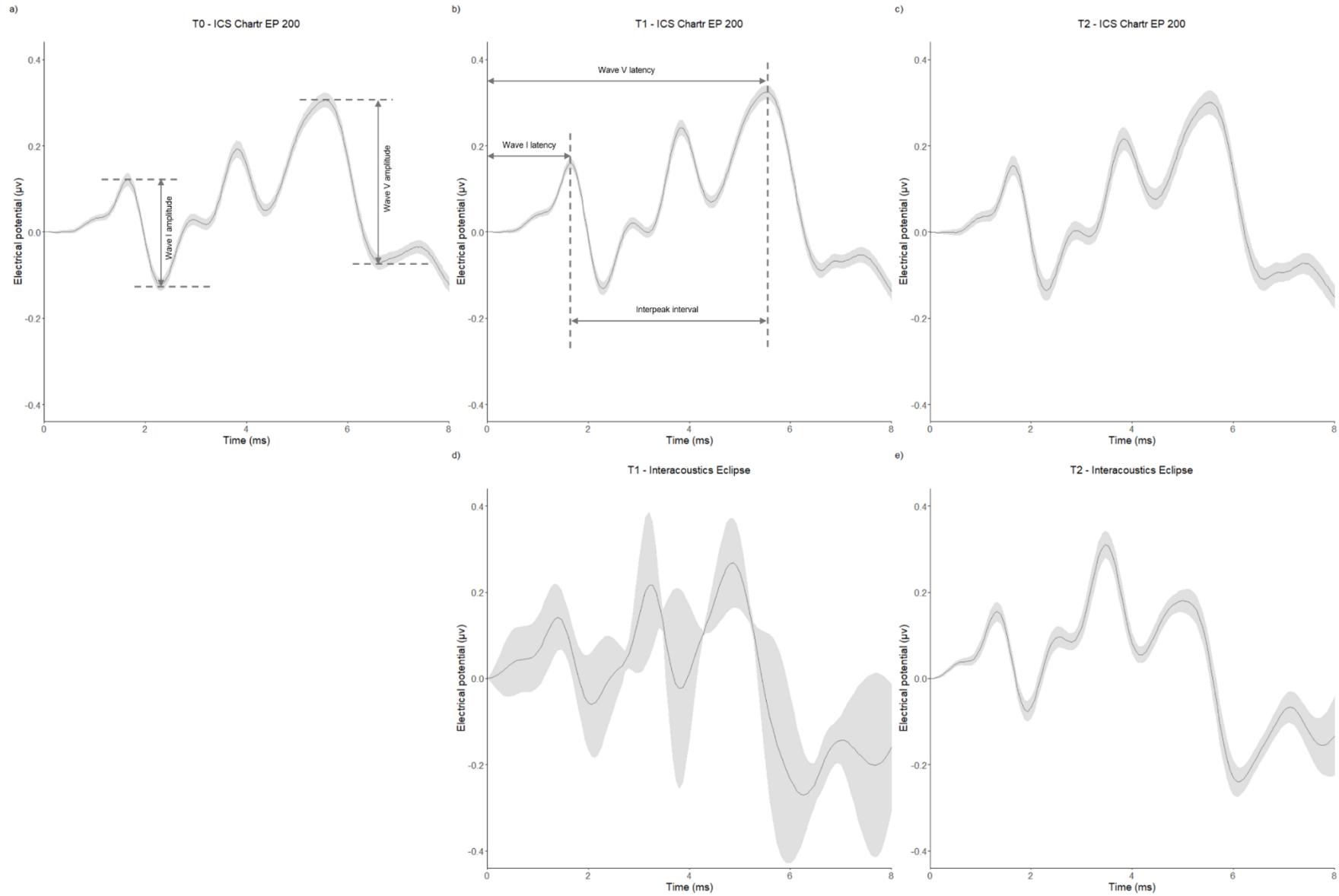


Figure S1. Grand average ABR waveforms for 80 dB nHL click stimuli captured using the ICS Chartr EP 200 clinical system (top panels) at a) T0, b) T1 and c) T2, and using the Interacoustics Eclipse system (bottom panels) at d) T1 and e) T2. Shaded areas represent 95% confidence intervals. Wave I and V amplitudes were measured from peak to subsequent trough, as demonstrated in panel a). Wave I and V latencies were measured from stimulus onset (0 ms) to the wave peak, and the interpeak interval was measured as the difference between wave I and V latencies, as demonstrated in panel b).

Supplementary materials 4

Table S1 Spearman's rho correlations between variable tested at T1.

	Average PTA low	Average PTA High	Average EHF	Average DPOAE	SRFM	Wave I amplitude (80 dB nHL)	Wave I/V ratio (80 db nHL)	Wave I latency (80 dB nHL)	Wave V latency (80 dB nHL)	Total noise exposure	Musical experience (years)
Average PTA High	.404*	-									
Average EHF	.068	.269*	-								
Average DPOAE	-.212	-.334*	-.324*	-							
SRFM	.078	.016	-.092	-.010	-						
Wave I amplitude (80 dB nHL)	-.071	-.285*	-.215	.167	.104	-					
Wave I/V ratio (80 db nHL)	.002	-.021	.068	-.018	.082	.549*	-				
Wave I latency (80 dB nHL)	-.107	.067	.173	-.086	-.095	-.208	.029	-			
Wave V latency (80 dB nHL)	.037	.130	.193	-.175	.011	-.302*	.147	.546*	-		
Total noise exposure	.168	.090	-.027	-.340*	.120	-.060	.000	.113	.271*	-	
Musical experience (years)	.086	.022	-.046	-.102	.130	.196	.225	-.010	-.112	.042	-

* Indicates significant value after FDR correction ($p < .05$ and $q < .10$)

Table S2 Spearman's rho correlations between variable tested at T2.

	Average PTA low	Average PTA High	Average EHF	Average DPOAE	SRFM	Wave I amplitude (80 dB nHL)	Wave I/V ratio (80 db nHL)	Wave I latency (80 dB nHL)	Wave V latency (80 dB nHL)	Total noise exposure	Musical experience (years)
Average PTA High	.460*	-									
Average EHF	.400*	.307*	-								
Average DPOAE	-.404*	-.416*	-.423*								
SRFM	.445*	.269	.010	-.034	-						
Wave I amplitude (80 dB nHL)	-.189	-.193	-.161	-.044	.018	-					
Wave I/V ratio (80 db nHL)	-.018	-.053	-.044	-.129	.147	.705*	-				
Wave I latency (80 dB nHL)	.063	.133	.273	-.338*	-.131	-.314*	-.199	-			
Wave V latency (80 dB nHL)	.206	.158	.102	-.230	-.096	-.488*	-.199	.575*	-		
Total noise exposure	.038	.081	.017	-.303*	-.136	.061	-.044	.229	.174	-	
Musical experience (years)	.231	.093	.120	-.361*	-.079	-.021	.163	.081	.045	.276	-

* Indicates significant value after FDR correction ($p < .05$ and $q < .10$)