Investigating the effects of noise exposure on self-report, behavioral and electrophysiological indices of hearing damage in musicians with normal audiometric thresholds

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Abstract

Musicians are at risk of hearing loss due to prolonged noise exposure, but they may also be at risk of early sub-clinical hearing damage, such as cochlear synaptopathy. In the current study, we investigated the effects of noise exposure on electrophysiological, behavioral and self-report correlates of hearing damage in young adult (age range = 18-27 years) musicians and non-musicians with normal audiometric thresholds. Early-career musicians (n = 76) and non-musicians (n = 47) completed a test battery including the Noise Exposure Structured Interview, pure-tone audiometry (PTA; .25 – 8 kHz), extended high-frequency (EHF; 12 and 16 kHz) thresholds, otoacoustic emissions (OAEs), auditory brainstem responses (ABRs), speech perception in noise (SPiN), and self-reported tinnitus, hyperacusis and hearing in noise difficulties. Total lifetime noise exposure was similar between musicians and non-musicians, the majority of which could be accounted for by recreational activities. Musicians showed significantly greater ABR wave I/V ratios than non-musicians and were also more likely to report experience of - and/or more severe - tinnitus, hyperacusis and hearing in noise difficulties, irrespective of noise exposure. A secondary analysis revealed that individuals with the highest levels of noise exposure had reduced outer hair cell function compared to individuals with the lowest levels of noise exposure, as measured by OAEs. OAE level was also related to PTA and EHF thresholds. High levels of noise exposure were also associated with a significant increase in ABR wave V latency, but only for males, and a higher prevalence and severity of hyperacusis. These findings suggest that there may be sub-clinical effects of noise exposure on various hearing metrics even at a relatively young age, but do not support a link between lifetime noise exposure and proxy measures of cochlear synaptopathy such as ABR wave amplitudes and SPiN. Closely monitoring OAEs, PTA and EHF thresholds when conventional PTA is within the clinically 'normal' range could provide a useful early metric of noise-induced hearing damage. This may be particularly relevant to early-career musicians as they progress through a period of intensive musical training, and thus interventions to protect hearing longevity may be vital.

Keywords

Noise exposure; musicianship; cochlear synaptopathy; speech perception in noise; otoacoustic emissions; tinnitus; hyperacusis

Highlights

- Noise exposure was not related to measures of cochlear synaptopathy
- Speech perception in noise was similar for musicians and non-musicians
- Noise exposure affected outer hair cell function despite normal hearing thresholds
- Noise exposure was related to increased ABR wave V latency for males only
- Musicians self-reported more hearing difficulties, irrespective of noise exposure

1. Introduction

Musicians are at risk of noise-induced hearing loss due to prolonged exposure to loud music (Greasley et al., 2018; Sataloff, 1991; Schink et al., 2014; Zhao et al., 2010). Hearing loss is particularly problematic for musicians as it affects their abilities to perform and limits employment opportunities, as well as affecting general wellbeing and quality of life. However, musicians' risk of hearing loss is not well understood since i) there is large between-individual variability in the susceptibility and extent of hearing loss (Abreu-Silva et al., 2011; Agrawal et al., 2008; Henderson et al., 1993; Toppila et al., 2000), ii) musicians may be reluctant to get their hearing tested because of concerns about the potential outcomes of getting a test (Greasley et al., 2018; Jansen et al., 2009), iii) musical training may enhance auditory and cognitive skills, which could moderate the detrimental effects of noise-induced hearing damage (Beach, 2018; Valderrama et al., 2018; Yeend et al., 2017), and iv) early signs of hearing loss can be very subtle and may not be easily detected using conventional clinical tests (e.g. pure tone audiometry). The aim of the study presented here was to determine whether early-career musicians are more likely to show symptoms of sub-clinical hearing damage as a result of noise exposure compared to non-musicians.

Although sub-clinical hearing deficits may have a number of different pathologies, cochlear synaptopathy - a loss of synapses between inner hair cells (IHCs) and spiral ganglion neurons (SGNs) -has received much attention recently (for reviews see; Hickox et al., 2017; Kujawa and Liberman, 2015; Liberman and Kujawa, 2017). Numerous studies in rodents (e.g. Kujawa and Liberman, 2009), guinea pigs (e.g. Furman et al., 2013; Lin et al., 2011) and rhesus monkeys (e.g. Valero et al., 2017) have shown that short-term exposure to medium-to-high intensity noise can cause a loss of synapses in basal cochlear regions without widespread hair cell damage. While cochlear thresholds were only temporarily increased and appeared to recover post exposure, wave I of the auditory brainstem response (ABR), which reflects auditory nerve (AN) activity, was reduced at suprathreshold levels and did not recover. High intensity noise exposure appeared to preferentially

affect synapses connecting IHCs to high-threshold, low spontaneous rate (low-SR) AN fibers which are responsible for coding suprathreshold sounds (Furman et al., 2013). This could explain why hearing sensitivity remains intact, but hearing in noise is impaired, and so has also been referred to as 'hidden hearing loss' (Kujawa and Liberman, 2015; Plack et al., 2014; Schaette and McAlpine, 2011).

It is not uncommon for people to report having difficulty with speech perception in noise (SPiN), and yet have audiometrically normal hearing (Brattico et al., 2005; Davis, 1989; Kujala et al., 2004; Pienkowski, 2017; Soalheiro et al., 2012); indeed it has recently been estimated that the proportion of adults who do so was ~10% of individuals aged 18-80 years (Parthasarathy et al., 2020). This has motivated numerous research groups to investigate noise-induced cochlear synaptopathy in humans (for recent reviews see; Bramhall et al., 2019; Kobel et al., 2017; Le Prell, 2019). A number of studies purport to show altered electrophysiology of hearing as a function of noise exposure. For example, Stamper and Johnson (2015a, 2015b) found a significant negative correlation between noise exposure and ABR wave I amplitude in normal-hearing females. More recently, Bramhall et al. (2017) showed reduced wave I amplitudes in military veterans and non-veterans with a history of firearm use compared to those with lower levels of noise exposure. Similarly, Liberman et al. (2016) demonstrated that the pre-synaptic summating potential (SP) generated by hair cells, and their ratio to the action potential (AP; wave I), was increased in individuals deemed as being at high-risk of noise-induced hearing damage compared to low-risk. These finding were proposed to be consistent with selective neural loss, although the effect was driven mainly by an increase in SP rather than by a decrease in AP. The high-risk group in this study also showed significantly poorer word recognition in noise performance, though at a low sound level (35 dB HL) not consistent with loss of high-threshold AN fibers.

By contrast, other studies have found no clear evidence of noise-induced cochlear synaptopathy. For example, Prendergast and colleagues (2017a) completed a structured interview to obtain a detailed

measure of lifetime noise in a large normal-hearing cohort (n = 126), and found no clear relationship with ABR wave amplitudes. Furthermore, lifetime noise exposure showed little-to-no effect on a wide range of behavioral tasks, and there were no strong correlations between behavioral and electrophysiological measures (Prendergast et al., 2017b). Guest and colleagues similarly demonstrated that noise exposure was not associated with ABR measures (Guest et al., 2017), and that SPiN was not associated with these electrophysiological measures, nor with noise exposure (Guest et al., 2018c). Other research groups have used alternative metrics of noise exposure, such as the preceding 12 months' exposure (Fulbright et al., 2017) and exposure to a single noisy event (Grinn et al., 2017), and have also found no association between ABR wave I amplitude and noise exposure. Recently, Grose et al. (2017) attempted to account for individual differences that affect wave I amplitudes (e.g. age, sex, head size) by normalising to wave V amplitudes, demonstrating that a history of loud music exposure was associated with a modest reduction in wave I/V ratios, consistent with cochlear synaptopathy (see also Gu et al., 2012; Schaette and McAlpine, 2011). However, there were no other electrophysiological or behavioral effects, nor any correlations with noise exposure, suggesting that even if noise-induced cochlear synaptopathy does occur in humans, the implications for hearing abilities are negligible. Le Prell et al. (2018) used a variety of individual and composite measures of noise exposure including preferred listening level, years of music player use, number of reported exposures, previous impulse noise exposure, and self-reported noise-induced changes to hearing after loud exposures, showing that there were no significant associations with functional measures of hearing.

Research investigating cochlear synaptopathy in musicians has also produced mixed findings, although few studies explicitly investigated musicianship as a factor, and instead targeted musicians as a high exposure/high-risk group. For example, 70% of the participants in the high-risk group of the study by Liberman et al. (2016) were studying music performance in local music colleges and conservatoires, and 71% of the high noise exposure group in the study by Grose et al. (2017) self-reported as musicians. Skoe and Tufts (2018) also demonstrated that ABR wave latencies were

increased (although ABR wave amplitudes were not reduced) for participants with high levels of noise exposure, most of whom were students taking part in music ensembles. In this study, noise exposure was measured using week-long dosimetry, revealing that musicians experienced significantly higher average daily exposure levels than non-musicians, with 47% of the musicians' days exceeding the recommended exposure limits specified by National Institute for Occupational Safety and Health (e.g. 3-dB exchange rate based on the doubling of sound intensity for each 3 dB increased above 85 dB, for 8-hours per day; NIOSH, 1998), compared with just 10% of the non-musicians' days (see Tufts and Skoe, 2018). However, the effects of noise exposure and musicianship on perceptual abilities were not measured, plus the effects of exposure on ABR wave latency may be attributable to IHC damage or AN demyelination, rather than cochlear synaptopathy per se (Skoe and Tufts, 2018).

Only a small number of studies have specifically accounted for the effects of musical training and noise exposure on auditory abilities. Yeend et al. (2017) conducted a comprehensive test battery to assess audiometric functioning, temporal and spectral processing, SPiN, cognitive abilities and musical training in adults with a range of lifetime noise exposures. The majority of the participants with the highest exposures were musicians with high levels of musical training. There were no effects of noise exposure on any of the auditory tests, suggesting no clear evidence for noise-induced synaptopathy. In addition, there was no relationship between musical training and SPiN, despite a positive relationship between musical training and temporal processing abilities. A follow-up electrophysiology study using the high noise exposure participants from this previous study (i.e. mostly musicians), plus newly recruited participants with low exposure, showed a moderate negative correlation between noise exposure and wave I amplitude (Valderrama et al., 2018). However, the results may reflect outer hair-cell dysfunction rather than synaptopathy since audiometric thresholds were not controlled for. Moreover, the null effect of noise exposure on SPiN persisted. It is also worth highlighting that musical training was not controlled for in this electrophysiology study. Prendergast et al. (2017b) included a measure of musical experience (total

years spent regularly playing a musical instrument) in their analysis, showing that musical experience was predictive of superior performance on a number of behavioural and SPiN measures, despite years of musical experience also being associated with increased noise exposure. Still, musical experience was not accounted for in the electrophysiological analysis with this participant cohort (Prendergast et al., 2017a).

In combination, the findings from Yeend et al. (2017), Prendergast et al. (2017b) and Grose et al. (2017) suggest that there may be very few consequences of noise-induced cochlear synaptopathy on SPiN in humans. Instead, noise-induced synaptopathy may be just one of many factors, including cognitive and auditory skills, that play a role in SPiN (Beach, 2018; Prendergast et al., 2017b; Valderrama et al., 2018; Yeend et al., 2017). Moreover, cognitive and auditory processing skills may be enhanced by musical training, which may improve SPiN, and thus lessen the negative effects of noise-induced cochlear synaptopathy on SPiN (Grose et al., 2017; Valderrama et al., 2018). Or from a reverse perspective, it has been proposed that noise damage may suppress the positive association between musical training and SPiN abilities (Skoe et al., 2018). Irrespective of noise exposure and hearing damage, there are numerous studies that advocate enhanced SPiN abilities in musicians, which may also be related to cognitive factors such as auditory working memory and selective attention (for a review see; Coffey et al., 2017). Additionally, musical training could promote neural plasticity in the auditory system, which might modify auditory processing, even at the subcortical level (i.e. auditory brainstem), and could also explain altered SPiN abilities (Bidelman et al., 2014; Kraus and Chandrasekaran, 2010; Musacchia et al., 2008, 2007; Parbery-Clark et al., 2011, 2009a; Sanju and Kumar, 2016).

It has been suggested that cochlear synaptopathy may also underlie the presence of tinnitus and hyperacusis in normal-hearing listeners. Several studies have shown that individuals with tinnitus and a normal audiogram have reduced wave I amplitudes, but wave V amplitudes are unchanged or increased (Bramhall et al., 2019b, 2018; Gu et al., 2012; Schaette and McAlpine, 2011; Valderrama et

al., 2018), though others found no effect on wave I amplitude (Guest et al., 2017). Similarly, Hickox and Liberman (2014) demonstrated that noise exposed mice had a heightened startle response associated with hyperacusis, as well as reduced wave I amplitudes and unchanged wave V amplitudes. It has been hypothesized that deafferentation of low-SR fibers causes a reduction of input to the central auditory system, which leads to a compensatory increase of neural activity to normalise input to higher levels of the ascending auditory pathway. This increased 'central gain' might lead to an amplification of spontaneous activity and neuronal hyperactivity, resulting in tinnitus and/or hyperacusis, while hearing thresholds remain unaffected (Hickox and Liberman, 2014; Schaette and McAlpine, 2011). Jansen et al. (2009) showed that 51% of musicians in their study complained of tinnitus compared with approximately 10-15% of the general population (Henry et al., 2005; Hoffman and Reed, 2004), and 79% of musicians complained of hyperacusis compared with approximately 9-15% of the general population (Andersson et al., 2002; Fabijanska et al., 1999). More recently, Camera et al. (2019) demonstrated that normal-hearing young adults with high levels of noise exposure, most of whom were musicians, had a reduced tolerance for background noise when listening to speech compared to those with low levels of noise exposure. Accordingly, another useful metric of cochlear synaptopathy in musicians may be the presence and severity of tinnitus and/or hyperacusis, which may also be less confounded by enhanced cognitive and auditory processing skills than SPiN.

While cochlear synaptopathy has received considerable attention, there are a number of other noise-induced hearing impairments that could constitute 'hidden hearing loss', and which could also explain the sequelae of cochlear synaptopathy, such as reduced ABRs and SPiN difficulties. For example, sub-clinical outer hair cell (OHC) dysfunction, as measured by otoacoustic emissions (OAEs), is common in young normal-hearing listeners with high levels of noise exposure (Hamdan et al., 2008; Job et al., 2009; Lapsley Miller et al., 2006; Lucertini et al., 2002; Mansfield et al., 1999). OHCs provide level-dependent amplification of the cochlear response (Brownell et al., 1985; Lu et al., 2006; Neely and Kim, 1986), and OHCs directly contribute to the input to IHCs (Dallos et al.,

2006). This could affect electrophysiological and perceptual measures and could explain the differences between high and low noise exposure groups observed in previous studies, rather than being due to cochlear synaptopathy.

Variability in hearing thresholds within the range considered to be 'normal' (-10 to 20 dB HL) could also account for differences in proxy measures of cochlear synaptopathy in previous studies (e.g. Bramhall et al., 2017; Stamper and Johnson, 2015a; Valderrama et al., 2018). In particular, high frequency (i.e. basal) cochlear regions provide the greatest contribution to ABR wave I generation (Abdala and Folsom, 1995; Don and Eggermont, 1978), especially at high stimulus levels (Eggermont and Don, 1980). Therefore, high frequency hearing deficits could also have contributed to reduced ABR amplitudes in previous studies (Gu et al., 2012; Liberman et al., 2016; Schaette and McAlpine, 2011). Furthermore, there is growing evidence that extended high frequency (EHF; > 8 kHz) hearing is linked to SPiN performance (Badri et al., 2011; Middlebrooks, 2015; Monson et al., 2019; Motlagh Zadeh et al., 2019; Yeend et al., 2017), and could be a sensitive early indicator of hearing deficits (Le Prell et al., 2013; Mehrparvar et al., 2011; Moore et al., 2017), irrespective of noise exposure. Therefore, it is essential that variability within the normal audiometric range (-10 to 20 dB), and at EHFs (>8 kHz), is accounted for when assessing variability in behavioural and electrophysiological measures of hearing.

In sum, there could be two opposing effects that influence SPiN abilities in musicians. First, musicians are exposed to more noise and so may be at greater risk of sub-clinical hearing damage including, but not limited to, cochlear synaptopathy. Second, musical training could improve SPiN and mask some of the negative effects of noise-induced hearing damage. What remains to be assessed are the effects of both lifetime noise exposure *and* musicianship on electrophysiological *and* perceptual measures of sub-clinical hearing impairments. In addition, it is possible that cochlear synaptopathy may contribute to the greater prevalence of tinnitus and/or hyperacusis in musicians. In the current study, we completed a comprehensive test battery to assess synaptopathic and non-

synaptopathic hearing damage, including PTA and EHF thresholds, OAEs, a SPiN test, ABRs, and self-report measures of tinnitus, hyperacusis and hearing in noise difficulties, in a large cohort of musicians and non-musicians with a wide range of lifetime noise exposures. In particular, 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.

2. Methods

2.1 Participants

Eighty-five early-career musicians and 52 non-musicians were recruited to the study. 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, across all classifications of instrument; strings (n = 24), wind (n = 7), brass (n = 13), keys (n = 15), percussion (n = 1), voice (n = 20), pop/amplified instruments (n = 5). Non-musicians were recruited via the University of Manchester Research Volunteering website and mostly consisted of students and staff members. Five participants (two musicians) did not pass the otoscope examination due to wax occlusion, eight participants (six musicians) did not pass the tympanometric screening, and one musician had moderately severe unilateral hearing loss (55-70 dB HL), and were all excluded from subsequent tests (see 2.2 Procedures). Consequently, 76 musicians (age range = 18-26; female n = 40) and 47 non-musicians (age range = 18-27; female n = 26) completed the full test battery and were included in the analysis. Musicians had an average of 13.3 years of musical experience (range = 8-20 years), started playing music at an average age of 7.1 years (range = 2-13.5 years), and were practicing their instrument an average of 15.1 hours per week at the time of testing (range = 1-36 hours). Twenty-eight non-musicians reported having at least some musical experience, with these participants reporting an average of 8.0 years of musical experience (range = 1-22 years), having started playing music at an average age of 13.5 years (range = 4-23

years), and were practicing their instrument an average of 0.8 hours per week at the time of testing (range = 0-7 hours). Across all non-musicians, participants reported an average of 4.8 years of musical experience and were practicing an average of 0.4 hours per week. All participants were native English speakers or were highly fluent in English as second language speakers (n = 9; 7 musicians).

2.2 Procedures

Participants completed all tests at the University of Manchester in a single three-hour session or split across two sessions. 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.

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 Tympstar diagnostic middle-ear analyser. Tympanic membrane compliances between 0.3-1.6 ml and peak pressures in the range -150 to +50 daPa were considered normal (Clark et al., 2007). Nine participants (four musicians) had slightly elevated (up to 1.8 ml) or low (0.2 ml) compliance; however all had present otoacoustic emissions and met the study hearing level criteria and so were included in the analysis.

2.2.3 Noise exposure

A comprehensive estimate of lifetime noise exposure was obtained via an early version of the Noise Exposure Structured Interview (NESI; Guest et al., 2018a; see also Lutman et al., 2008). The NESI

prompts participants to identify noisy occupational and recreational activities (above ~ 80 dB A) that they have engaged in throughout their lifetime. For each activity, the participant identified periods in their life in which exposure habits were relatively stable (e.g. attending nightclubs aged 18-21), and provided an estimate of the frequency and duration of exposure for each life period. Noise exposure level was estimated based on vocal effort required to hold a conversation or, for personal listening devices, typical volume control setting. Participants were also asked to report usage and type of hearing protection devices, if any. For each activity; frequency, duration, level and protector attenuation were combined to generate units of noise exposure based on the equal energy principle:

$$U = 10^{(L-A-90)/10} \times Y \times W \times D \times H/2080$$

where *U* is cumulative noise exposure, *L* is estimated noise exposure in dB A, *A* is level of attenuation provided by hearing protection in dB, *Y* is years of exposure, *W* is weeks of exposure per year, *D* is days of exposure per week, *H* is hours of exposure per day, and 2080 corresponds to the number of hours in a working year.

For the quantification of firearm exposure, the NESI uses the metric developed by Goley et al. (2011) that adjusts the A-weighted equivalent continuous exposure level for the greater kurtosis (peakedness) of impulsive noise (see Guest et al., 2018a for further details). Firearm exposure was incorporated with recreational and occupational noise exposure to obtain a measure of total lifetime noise exposure. Note that firearm use is relatively less common in the UK, with only 12 participants in the current sample reporting having ever fired a gun, and accounted for less than 1 unit of noise exposure for 9 of these participants. As such, there was no further examination of firearm exposure in the current experiment.

One noise exposure unit is equivalent to one working year of exposure at a daily level of 90 dB A (hence *L* – 90 in the above equation). Units for occupation and recreational activities, and the combined total (including any firearm exposure), were log transformed to produce a normal

distribution (for further details on the NESI see; Guest et al., 2018a). As per Prendergast et al. (2018), participants with at least one log unit of total noise exposure (i.e., equivalent to 10 working years of exposure to 90 dB A) were classified as high noise exposure, and those with less than one log unit were classified as low noise exposure.

2.2.4 Hearing thresholds

Pure-tone air-conduction thresholds were measured at 0.25-8 kHz according to British Society of Audiology (2018) recommended procedures, 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. Four participants (two musicians) had a mild unilateral hearing loss (25-40 dB HL) which was restricted to 8 kHz for three participants, and at .5, 4 and 8 kHz for one other participant. All participants with a mild hearing loss were included in the analysis since hearing thresholds were accounted for as a predictor variable in exploratory linear regression models (see section 3.7.2 Individual differences).

Extended high frequency (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 centered 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 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 initially given a short practice to familiarise themselves with the procedure. Participants were seated in a double-walled sound-attenuating booth for both PTA and EHF procedures.

2.2.5 Otoacoustic emissions

Outer hair cell (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. The cubic distortion product ($2f_1$-f_2) amplitude was used as a measure of the DPOAE level. Ninety-six sweeps were measured for each frequency. DPOAEs were classified as present if the signal-to-noise (SNR) ratio was ≥3 dB. Only a small percentage (3%) of all DPOAEs were absent. On these occasions, the DPOAE level was set to the estimated system distortion level.

2.2.6 Speech-in-noise perception

The coordinate response measure (CRM; Bolia et al., 2000) task was used to measure SPiN abilities. The CRM incorporates meaningful speech stimuli, high overall sound levels, distracting talkers, and spatial cues. The latter three attributes of this test are pertinent to cochlear synaptopathy, since the loss of low-SR fibers may be detrimental to the temporal and level cues required for spatial processing, especially at high sound levels (Plack et al., 2014). Spatial cues are also pertinent to extended high frequency hearing (> 8 kHz), whereby sound localisation of meaningful speech cues may be reliant on high frequency energy (e.g. Middlebrooks, 2015; Monson et al., 2019). The task also uses a closed-set of stimuli with a simple vocabulary, meaning that the CRM is appropriate for non-native English speaking participants. All speech stimuli were spoken by native British-English talkers (Kitterick et al., 2010).

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 1-4. Participants were instructed to

listen for the speech utterance, which contained the call sign "*Baron"*, and to identify the color and number spoken by that talker. Responses were made by selecting the identified color and number from a 4x4 grid presented on a computer screen. The gender and identity of the target talker was randomly selected on each trial from four male and four female talkers. Two masker utterances always contained two different call signs, although the color or number could match that of the target talker. 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.

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 degrees 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 ten subsequent turnpoints (2 dB step size). Thresholds were calculated as the average SNR of the final 10 turnpoints. Central and offset spatial 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. Participants were given a short practice consisting of separate blocks of central and offset conditions to familiarise themselves with the procedure.

2.2.7 Self-report measures

2.2.7.1 Tinnitus

Participants were asked whether they had ever experienced tinnitus ("*Yes/No*"), 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 five minutes*". Participants who answered "*Yes*" were asked whether they have tinnitus all the time, what caused them to experience tinnitus, and were

asked to complete the Tinnitus Handicap Inventory (THI; Newman et al., 1996). Participants who reported having experienced tinnitus previously, but for whom it was not present at the time of testing, were asked to reflect back on occasions when they had experienced tinnitus when completing the THI questionnaire.

2.2.7.2 Hyperacusis

Participants were asked whether they had ever experienced hyperacusis ("*Yes/No*"), 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*." Participants who answered "*Yes*" were also asked whether this was constant and what caused them to experience hyperacusis. All participants completed the Modified Khalfa Hyperacusis Questionnaire (HQ; Khalfa et al., 2002) to quantify hyperacusis severity.

2.2.7.3 Hearing in noise difficulties

Participants were asked whether they "*find it difficult to follow a conversation if there is background noise, such as TV, radio, children playing*", answering with a "*Yes/No*" response. All participants were asked to complete a subset of questions from the Self-efficacy for Situational Communication Management Questionnaire (SESMQ; Jennings et al., 2014). Participants were asked to rate on a seven-point Likert scale how well they could hear in 6 challenging noisy situations (see Supplementary Materials 1). An average score was calculated for each participant to obtain a measure of the severity of hearing in noise difficulties.

2.2.8 Electrophysiology

2.2.8.1 Recording procedure

ABRs were recorded using an ICS Chartr EP 200 (Otometrics-Natus) clinical system using ER3 insert earphones. 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. The reference and ground electrodes were switched for left and right ear stimulation. Electrode impedances were 2 kΩ or less in the majority of cases, and never more than 5 kΩ, to allow good data quality in all participants. Participants lay in a comfortable supine position and 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.

Click stimuli were 100 μs 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, in four separate blocks of trials. Responses were amplified with a gain of 50k and band-pass filtered between 0.1 and 1.5 kHz. Data were collected over a 20 ms epoch, with recording beginning 2 ms before stimulus onset. A minimum of 6000 sweeps were conducted for each ear and each stimulus level, with additional repetitions added for each rejection until a total of 6000 sweeps were obtained. An average of the 6000 sweeps was taken to form an average waveform for each ear and stimulus level.

2.2.8.2 Response identification

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.). Time windows for wave I and V peaks and troughs were specified based on the grand average waveforms for each stimulus intensity across all participants. The time windows for identifying the peak of wave I were 1.5 to 2.6 ms and 1.1 to 2.1 ms post stimulus onset for 60 and 80 dB nHL click intensities, respectively. Wave I troughs were 0.2 to 1.2 ms post wave I peak for both 60 and 80 dB nHL click intensities. Wave V peaks were 5.2 to 6.6 ms and 4.9 to 6.2 ms post stimulus onset for 60 and 80 dB nHL click intensities, respectively. On occasions, more than one peak or inflection was identified within the time window for wave V peaks which corresponded to the identification of wave IV or a wave IV/V blended morphology. The automated procedure identified Wave V peak as the later occurring peak/inflection within the time window. Wave V troughs were 0.5 to 1.5 ms and

0.4 to 1.8 ms post wave V peak for 60 and 80 dB nHL click intensities, respectively. Each participant's average waveform for each ear and click intensity was visually inspected to verify that the algorithm had correctly identified peaks and troughs. Wave I and V amplitudes were calculated from peak-to-trough, and used to determine wave I/V amplitude ratio for each participant. Wave I growth was calculated by subtracting the average wave I amplitude for the 60 dB nHL click from the average wave I amplitude for the 80 dB nHL click, for each participant.

2.3 Data analysis

All statistical analyses were performed using SPSS (IBM Corp.). Repeated measures ANOVAs with two between-subject factors – musicians vs. non-musicians, and low vs. high noise exposure – were used to assess hearing thresholds, otoacoustic emissions, SPiN abilities from the CRM task, and electrophysiological measures. Greenhouse-Geisser corrected values are reported for violations of sphericity. Main effects were assessed using pairwise comparisons with α automatically adjusted for multiple comparisons using Bonferroni corrections (i.e. α = .05). Interactions were assessed using post-hoc *t*-tests with Bonferroni corrections for multiple comparisons applied manually. Bonferroni corrections are very conservative, which increases the risk of Type-II error when many comparisons are conducted, and it may not be appropriate to apply corrections over a range of different measures when a number of different statistical procedures are used (Armstrong, 2014). Accordingly, post-hoc Bonferroni corrections were applied on a measure-by-measure basis with corrected α-values reported for each individual measure (McDonald, 2014).

Chi-square tests were used to determine whether the proportions of participants assigned to different groups based on self-report (e.g. tinnitus vs. no-tinnitus) varied between musicians and non-musicians, and low and high noise exposure groups.

For dependent variables with a single measure, such as levels of noise exposure, spatial release from masking on the CRM, THI scores, HQ scores, and average hearing in noise difficulty; scores were

assessed using independent samples *t*-tests to determine differences between musicians and non-musicians, and low and high noise-exposure groups. Differences between musicians and non-musicians with low and high noise exposures (i.e. four group comparison) were assessed using a one-way ANOVA. Mann-Whitney *U*-tests and Kruskal-Wallis tests were used on occasions where data was non-normally distributed.

Note that due to occasional technical errors during measurement recording, a small amount of data was lost for certain tests, hence there is slight variation in degrees of freedom between different measures.

3. Results

3.1 Noise exposure

Log-transformed occupational noise exposure scores were significantly higher for musicians (median = -.09; min = -1.31, max = 1.60) compared to non-musicians (median = -.64; min = -3.00, max = 1.39) [Mann-Whitney Test: $U = 807$, $p < .001$, $r = .459$]. However, the proportion of participants classified as low and high noise exposure was not significantly different between musicians (low n = 50; 65.8%) and non-musicians (low n = 30; 63.8%) [$\chi^2(1) = .049$, $p = .825$, $V = .020$]. Recreational noise exposure scores were not significantly different between musicians (mean = .62; SD = .70) and non-musicians (mean = 0.85; SD = .73) [$t(121) = -1.694$, $p = .093$, $d = .313$]. Total noise exposure was also not significantly different between musicians (mean = .81; SD = .79) and non-musicians (mean = .90; SD = .70) [$t(121) = -.775$, $p = .440$, $d = .141$]. Note that the biggest contribution to total noise exposure for both musicians and non-musicians was from recreational activities, hence the similarity of total noise exposure across the two groups. The mean log-transformed total noise-exposure score for the low noise exposure group was .46 (SD = .37; min = -.65; max = .98) and for the high noise exposure group was 1.55 (SD = .42; min = 1.00; max = 2.65).

3.2 Hearing thresholds

Mean PTA and EHF thresholds split by musicianship and noise exposure groups are shown in Figure 1. For standard PTA thresholds (.25-8 kHz), the repeated measures ANOVA showed no significant main effects of musicianship or noise exposure, and no significant interactions (all $p > .05$).

For EHF thresholds (12 and 16 kHz), the repeated measures ANOVA showed no significant main effects of musicianship or noise exposure (both $p > .05$), but there was a significant interaction between musicianship and noise exposure [$F(1, 119) = 4.206$, $p = .042$, $\eta_p^2 = .034$]. Post-hoc *t*-tests showed that musicians with high noise exposure (mean = 35.22 dB SPL, SD = 6.63) had better thresholds than non-musicians with high noise exposure (mean = 40.07 dB SPL, SD = 7.88) [$t(41) = -2.174$, $p = .035$, $d = .665$], however this effect was not significant after controlling for multiple comparisons (Bonferroni correction; $\alpha = .05/6 = .008$). There were no other significant differences between high and low noise exposed musicians and non-musicians. No other significant interactions were observed (all $p > .05$).

[Figure 1.]

3.3 Otoacoustic emissions

Mean DPOAE levels for 1-6 kHz split by musicianship and noise exposure groups are shown in Figure 2. A repeated measures ANOVA revealed no significant main effects of musicianship or noise exposure, and no significant interactions (all $p > .05$).

[Figure 2.]

3.4 Speech-in-noise perception

Figure 3 shows the mean SNR values for central and offset conditions of the CRM task, split by musicianship and noise exposure groups. A repeated measures ANOVA showed a significant main effect of masker position [$F(1, 119) = 1012.183$, $p < .001$, $\eta_p^2 = .895$] with better performance in the

spatially offset condition (mean = -13.96 dB, SD = 4.32) compared to the central condition (mean = -.22 dB, SD = 2.72). No other significant main effects or interactions were observed (all $p > .05$).

For the spatial release from masking measure (SRFM; i.e. central-offset difference), independent samples *t*-tests showed no significant difference between musicians and non-musicians [$t(121) = .063$, $p = .950$, $d = .011$] and no significant difference between high and low noise exposure groups [$t(121) = -.666$, $p = .507$, $d = .128$]. A one-way ANOVA with four groups (musicians low exposure, musicians high exposure, non-musicians low exposure, non-musicians high exposure) was also not significant [$F(3, 119) = .679$, $p = .567$, $\eta^2 = .017$].

[Figure 3.]

3.5 ABR wave amplitudes

Grand average ABR waveforms split by musicianship and noise exposure group are shown in Figure 4. Mean amplitudes for waves I and V, and wave I/V ratios, are shown in Figure 5.

[Figure 4.]

3.5.1 Wave I amplitude

A repeated measures ANOVA revealed a main effect of stimulus level [$F(1, 98) = 407.676$, $p < .001$, $\eta_p^2 = .806$] with significantly smaller wave I amplitudes for 60 dB nHL clicks (mean = .08 µV, SD = .04) compared to 80 dB nHL clicks (mean = .28 µV, SD = .10). The main effects of musicianship and noise exposure were non-significant, and there were no significant interactions (all $p > .05$).

The difference between wave I amplitudes for 60 and 80 dB nHL clicks was calculated to determine a measure of wave I growth, showing no significant main effects of musicianship or noise exposure, and no interactions (all $p > .05$).

3.5.2 Wave I/V ratio

The repeated measures ANOVA revealed a main effect of stimulus level [$F(1,98) = 193.155$, $p < .001$, $\eta_p^2 = .663$] with significantly smaller wave I/V ratios for the 60 dB nHL clicks (mean = .27 μV, SD = .19) compared to 80 dB nHL clicks (mean = .67 μV, SD = .28). There was also a main effect of musicianship [$F(1,98) = 4.940$, $p = .029$, $\eta_p^2 = .048$] with significantly greater wave I/V ratio for musicians (mean = .51 μV, SD = .23) compared to non-musicians (mean = .42 μV, SD = .12). 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 $p > .05$).

[Figure 5.]

3.6 Self-report measures

Differences in mean THI scores, HQ scores and SIN difficulty ratings between musicians and non-musicians, split by low and high noise exposure groups, are shown in Figure 6. Note that tinnitus and hyperacusis severity appears to be higher (i.e. worse) for musicians compared to non-musicians, irrespective of noise exposure.

[Figure 6.]

3.6.1 Tinnitus

Eighty-eight participants (71.5%) reported having experienced tinnitus (constant tinnitus n = 4; 3.3%). Of these 88 participants, 70 (79.5%) reported that tinnitus had occurred abruptly following exposure to a loud event (e.g. nightclub, concert etc.), 4 (4.5%) reported that it occurred gradually, and 14 (15.9%) reported that the cause was unknown. Chi-square tests confirmed that the proportion of participants who reported having experienced tinnitus was not significantly different between musicians (n = 56; 73.7%) and non-musicians (n = 32; 68.1%) [$\chi^2(1) = .447$, $p = .504$, $V = .060$], and was not significantly different between low noise exposure (n = 55; 68.8%) and high noise exposure groups (n = 33; 76.7%) [$\chi^2(1) = .878$, $p = .349$, $V = .084$]. The proportion of participants who

reported having experienced tinnitus was not significantly different between musicians and non-musicians with low and high levels of noise exposure [$\chi^2(3) = 6.144$, $p = .105$, $V = .223$].

For those who reported having experienced tinnitus, THI scores were significantly higher (i.e. more severe tinnitus) for musicians (median score = 12) than non-musicians (median score = 8) (Mann-Whitney Test: $U = 623$, $p = .017$, $r = .254$). There were no significant differences in THI scores between low and high noise exposure groups (Mann-Whitney Test: $U = 903.5$, $p = .972$, $r = .004$), and there were no significant differences between low and high noise exposed musicians and non-musicians [Kruskal-Wallis Test: $\chi^2(3) = 6.152$, $p = .104$, $\eta^2 = .038$].

3.6.2 Hyperacusis

Twenty-nine participants (23.6%) reported experiencing hyperacusis (constant hyperacusis n =2; 1.6%). Of these, 28 (96.6%) reported that the cause was unknown, and one (3.4%) reported that it occurred gradually. Chi-square tests showed that the proportion of participants who reported having experienced hyperacusis was not significantly different between musicians (n = 20; 26.3%) and non-musicians (n = 9; 19.2%) [$\chi^2(1) = .828$, $p = .363$, $V = .082$]. The proportion of participants who reported having experienced hyperacusis was significantly higher in the high noise exposure group (n = 16; 37.2%) compared to the low noise exposure group (n = 13; 16.3%) [$\chi^2(1) = 6.819$, $p = .009$, $V = .235$]. Non-musicians with low noise exposure had the smallest proportion of participants reporting having experienced hyperacusis (n = 3; 10.0%) compared to non-musicians with high noise exposure (n = 6; 35.3%), musicians with high noise exposure (n = 10; 38.5%) and musicians with low noise exposure (n = 10; 20.0%) [$\chi^2(3) = 7.916$, $p = .048$, $V = .254$].

For the hyperacusis questionnaire, there were significantly higher HQ scores (i.e. more severe hyperacusis) for musicians (median score = 26) compared to non-musicians (median score = 18) (Mann-Whitney Test: $U = 1301.5$, $p = .012$, $r = .227$). There were no significant differences in HQ scores between low and high noise exposure groups (Mann-Whitney Test: $U = 1466$, $p = .178$, $r =$

.122), but there was a significant difference in HQ scores between musicians and non-musicians with high and low noise exposure [Kruskal-Wallis Test: $\chi^2(3) = 9.290$, $p = .026$, $\eta^2 = .053$]. HQ scores were lower for non-musicians with low noise exposure (median score = 13) compared to musicians with low noise exposure (median score = 25) (Mann-Whitney Test: $U = 487.5$, $p = .009$, $r = .292$) and musicians with high noise exposure (median score = 33.5) (Mann-Whitney Test: $U = 230$, $p = .009$, $r = .351$), although these differences were not significant after controlling for multiple comparisons (Bonferroni correction; $\alpha = 0.05/6 = .008$). There were no other significant differences between groups (all $p > .008$).

3.6.3 Hearing in noise difficulties

The proportion of participants who reported hearing in noise difficulties was significantly higher for musicians (n = 27; 35.5%) compared to non-musicians (n = 7; 14.9%) [$\chi^2(1) = 6.181$, $p = .013$, $V = .224$], but there were no significant differences between low noise exposure (n = 25; 31.3%) and high noise exposure (n = 9; 20.9%) groups [$\chi^2(1) = 1.489$, $p = .222$, $V = .110$]. Musicians with low noise exposure had the highest proportion of self-reported hearing in noise problems (n = 21; 42.0%) compared to musicians with high noise exposure (n = 6; 23.1%), non-musicians with low noise exposure (n = 4; 13.3%) and non-musicians with high noise exposure (n = 3; 17.7%) [$\chi^2(3) = 9.344$, $p = .025$, $V = .276$].

When asked to rate how well they could hear in different noisy situations, musicians reported worse hearing in noise abilities (median score = 5.17) compared to non-musicians (median score = 5.50) (Mann-Whitney Test: $U = 1326.50$, $p = .017$, $r = .216$). There were no significant differences in self-reported hearing in noise abilities between high and low noise exposure groups (Mann-Whitney Test: $U = 1648.50$, $p = .704$, $r = .034$), and there were no significant differences between musicians and non-musicians with high and low levels of noise exposure [Kruskal-Wallis Test: $\chi^2(3) = 7.599$, $p = .055$, $\eta^2 = .039$].

3.7 Exploratory analysis

3.7.1 ABR wave latencies

Previous research has suggested that ABR wave latency could be a useful metric of noise-induced hearing damage (Skoe and Tufts, 2018), and has been proposed to be a more sensitive measure of cochlear synaptopathy than ABR wave amplitudes (Mehraei et al., 2016). We therefore analyzed the latency of wave I and wave V peaks, and the wave I-V interpeak latency, extracted using the peak-picking procedure in MATLAB (Mathworks, Inc.). Mean peak latencies for waves I and V, and the wave I-V interpeak latency, are shown in Figure 7.

[Figure 7]

3.7.1.1 Wave I peak latency

A repeated measures ANOVA revealed a significant main effect of stimulus level only [$F(1,114) = 800.931$, $p < .001$, $\eta_p^2 = .875$] with significantly delayed wave I peaks for 60 dB nHL clicks (mean = 2.03 ms, SD = .20) compared to 80 dB nHL clicks (mean = 1.61 ms, SD = .13). The main effects of musicianship and noise exposure were not significant, and there were no significant interactions (all $p > .05$).

3.7.1.2 Wave V peak latency

Similar to wave I peak latency, the repeated measures ANOVA showed a significant main effect of stimulus level on wave V peak latency [$F(1,115) = 550.443$, $p < .001$, $\eta_p^2 = .827$] with significantly delayed wave V peaks for 60 dB nHL clicks (mean = 6.01 ms, SD = .23) compared to 80 dB nHL clicks (mean = 5.57 ms, SD = .23). The main effect of noise exposure was also found to be significant [$F(1,115) = 7.977$, $p = .006$, $\eta_p^2 = .065$] with delayed wave V latencies for the high noise exposure group (mean = 5.86 ms, SD = .19) compared to the low noise exposure group (mean = 5.76 ms, SD =

.22). The main effect of musicianship was not significant, and there were no significant interactions (all $p > .05$).

3.7.1.3 Wave I-V interpeak latency

The repeated measures ANOVA revealed a significant main effect of noise exposure only [$F(1,114) = 4.214$, $p = .042$, $\eta_p^2 = .036$] with a larger interpeak latency for the high noise exposure group (mean = 4.02 ms, SD = .19) compared to the low noise exposure group (mean = 3.95 ms, SD = .20). There were no other significant main effects or interactions (all $p > .05$).

3.7.2 Highest and lowest noise exposures

The average total noise exposure for the participant sample was 0.84 (SD = .64) log units of energy. Given the normal distribution of total noise exposure in this participant sample, this means that a large proportion of participants were close to the cut-off criteria for low and high noise exposure groups (i.e. 1 log-unit of total noise exposure), which could explain why few effects of noise exposure were observed. To address this issue, we re-ran the analysis with the top and bottom 20% of participants from the total noise exposure distribution (musicians n = 32, non-musicians n = 20). The mean log-transformed noise exposure score for the low noise exposure group was 0.05 (SD = 0.25; min = -0.65; max = 0.30) and for the high noise exposure group was 1.79 (SD = 0.35; min = 1.31; max = 2.65).

The results from this sub-sample of participants closely followed those from the entire participant sample (see Supplementary materials 2). Novel findings from this sub-sample included the main effect of noise exposure on DPOAE levels [$F(1,48) = 11.702$, $p = .001$, $\eta_p^2 = .196$], where levels were significantly lower for the high noise exposure group (mean = 8.79 dB SPL, SD = 3.02) compared to the low noise exposure group (mean = 12.05 dB SPL, SD = 3.18). For completeness, DPOAE noise levels and SNRs were also analyzed, showing no effect of noise exposure on the DPOAE noise level [$F(1,48) = .168$, $p = .684$, $\eta_p^2 = .003$], but there was an effect of noise exposure on the SNR [$F(1,48) =$

8.199, p = .006, η_p^2 = .146]. Accordingly, the main effect of noise exposure on DPOAE levels was not due to the high noise exposure group having different DPOAE noise levels.

Mean HQ scores were also found to be significantly higher for the high noise exposure group (mean = 30.81, SD = 20.57) compared to the low noise exposure group (mean = 19.19, SD = 11.83) [t(39.909) = -2.496, p = .017, d = .692].

3.7.3 Individual differences

Twenty-eight non-musicians reported having some level of musical experience, which could explain why there were very few differences found between musicians and non-musicians. To address this potential confound, musicianship and noise exposure were assessed on a continuous scale (as per Yeend et al., 2017), with Spearman's rho correlations conducted between years of musical experience, total noise exposure, THI scores, HQ scores, hearing in noise difficulty scores, mean tympanic membrane compliance and mean tympanic peak pressure to account for middle-ear function, average low PTA (.25-1 kHz), average high PTA (2-4 kHz), average EHF thresholds (12 and 16 kHz), average DPOAE levels (1-6 kHz), 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, for the whole participant sample. The correlation matrix is shown in Figure 8a. Bonferroni corrections for multiple comparisons would result in $\alpha = .05/120 = 4.17 \times 10^{-4}$, which could inflate Type II error rate. Therefore, to correct for multiple comparisons we applied the false discovery rate (FDR) method with a p-value of .05 and a q-value of .10 (McDonald, 2014).

Years of musical experience was not significantly related to any measures after applying FDR corrections. Noise exposure was significantly correlated with average ABR wave V latency (r_s = .293, n = 120, p = .001; Figure 8b) and average DPOAE level (r_s = -.259, n = 123, p = .004; Figure 8c). Average DPOAE level was also significantly correlated with average high PTA (r_s = -.254, n = 123, p =

.005) and average EHF thresholds (r_s = -.338, n = 123, p < .001). Significant correlations were also observed between all self-report measures; THI, HQ and hearing in noise difficulties (all $p \leq .001$).

Note that average tympanometric compliance was significantly correlated with average ABR wave V latency (r_s = .315, n = 120, p < .001) and average high PTA thresholds (r_s = .296, n = 123, p = .001). We therefore conducted partial correlations between total lifetime noise exposure and average ABR wave V latency, and average DPOAE levels and average high PTA thresholds, while controlling for average tympanometric compliance. The levels of significance were unchanged when controlling for tympanometric compliance.

[Figure 8.]

Separate multiple linear regression analyses were also conducted for proxy measures of cochlear synaptopathy, including SPiN abilities (SRFM from the CRM task), wave I amplitude and wave I/V ratio of the ABR for the 80 dB nHL click, average ABR wave V latency, THI score, HQ score, and self-reported hearing in noise difficulty. Predictor variables included gender, mean tympanic membrane compliance, mean tympanic peak pressure, average low PTA thresholds, average high PTA thresholds, average EHF thresholds, average DPOAE levels, years of musical experience, total lifetime noise exposure, and wave I amplitude of the ABR for the 80 dB nHL click (except for the regression models where dependent variables were electrophysiological measures). As with the correlation matrix, we applied the false discovery rate (FDR) correction with a p-value of .05 and a q-value of .10 for each regression model.

The regression model including wave V latency as the dependent variable was found to be significant [F(9,110) = 4.813, p < .001, R^2 = .283], with gender, tympanometric pressure and lifetime noise exposure as significant predictor variables after accounting for FDR (all p < .05 and q < .01; see Supplementary Materials 3). To examine the effect of gender, an independent samples t-tests showed that males had significantly delayed ABR wave V latencies (mean = 5.89 ms, SD = .18)

compared to females (mean = 5.71 ms, SD = .20) [$t(118) = -5.100$, $p < .001$, $d = .939$]. Males also showed significantly higher levels of noise exposure (mean = .98, SD = .67) compared to females (mean = .73, SD = .59) [$t(118) = -2.196$, $p = .030$, $d = .400$]. Spearman's rho correlations were also conducted between total log noise exposure and ABR wave V latency for males and females separately (see Figure 8c), revealing a significant correlation for males ($r_s = .352$, n = 55, $p = .008$), but not for females ($r_s = .147$, n = 65, $p = .243$).

The regression model including hyperacusis score as the dependent variable was also found to be significant [$F(10,108) = 2.032$, $p = .037$, $R^2 = .158$]. The only significant predictor after FDR correction was average high frequency (2-8 kHz) PTA thresholds ($\beta = -.305$, $t = -2.793$, $p = .006$).

4. Discussion

The aim of the current study was to explore the effects of noise exposure on self-report, behavioral and electrophysiological measures of hearing damage, including cochlear synaptopathy, in early-career musicians and non-musicians. Our results support previous findings from our laboratory which provide little evidence of noise-induced cochlear synaptopathy in young normal hearing adults (Guest et al., 2019, 2018c, 2018b, 2017; Prendergast et al., 2019, 2018, 2017b, 2017a).Despite musicians displaying some minor differences from non-musicians in the ABR, musicians do not show altered SPiN compared to non-musicians, and there were few significant interactions with noise exposure. These null effects of musicianship may be due to similar levels of total lifetime noise exposure between musicians and non-musicians. Similar performance between musicians and non-musicians was observed even when comparing participants with the highest and lowest levels of noise exposure, and when analysing associations between noise exposure, years of musical experience, and proxy measures of cochlear synaptopathy. Nevertheless, musicians were more likely to report experience of- or more severe- tinnitus, hyperacusis and/or hearing in noise difficulties compared to non-musicians, irrespective of noise exposure. In addition, the exploratory analysis showed that high levels of noise exposure - the majority of which is from recreational activities -

negatively affected OHC function, as measured by DPOAEs. Furthermore, increased ABR wave V latency was associated with higher levels of noise exposure, for males only.

4.1 No effect of noise exposure on proxy measures of cochlear synaptopathy

Several studies from other research groups have also failed to support the hypothesis that cochlear synaptopathy is linked to lifetime noise exposure in normal hearing young adults (Fulbright et al., 2017; Grinn et al., 2017; Yeend et al., 2017). However, animal studies are more consistent at demonstrating the effects of noise exposure on auditory nerve function and anatomy (e.g. Kujawa and Liberman, 2009), and there are some studies in young adults that purportedly show the effects of noise exposure on proxy measures of cochlear synaptopathy (Bramhall et al., 2017; Grose et al., 2017; Liberman et al., 2016). These inconsistent findings have been discussed in depth in previous research and review articles (Bramhall et al., 2019a; Hickox et al., 2017; Le Prell, 2019), which provide several possible explanations for our null findings, as summarised below.

4.1.1 Young adults are less susceptible to noise-induced cochlear synaptopathy

Recent evidence suggests that primates are more resilient to cochlear synaptopathy compared to rodents (Valero et al., 2017), which may be due to a combination of genetic, environmental, and anatomical differences (Abreu-Silva et al., 2011; Bramhall et al., 2019a). Dobie and Humes (2017) surmised that the noise levels required to induce cochlear synaptopathy in mice (e.g. ~100 dB SPL octave band noise for 2 hours; Kujawa and Liberman, 2009) would exceed OSHA limits when correcting for inter-species audiometric differences. Specifically, it was estimated that humans require ~114 dB SPL continuous exposure for 2 hours in order to induce cochlear synaptopathy. Most individuals are unlikely to be exposed to these high sound levels through common recreational and occupational activities (and at least not without sufficient hearing protection for the latter; Control of Noise at Work Regulations, 2005). For classical musicians - as per the majority of musicians in the current study - typical average noise equivalent exposures are 75 to 98 dB A

(McBride et al., 1992; Pawlaczyk-Łuszczyńska et al., 2011; Royster et al., 1991; Schmidt et al., 2011). On the contrary, the military veterans included in the study by Bramhall et al. (2017) were exposed to high peak-intensity military noise and firearms, which could explain the observed reduction in ABR wave I amplitude compared to non-veterans.

A second possibility is that cochlear synaptopathy does occur in humans, but this may be more related to an aging auditory system than to noise exposure. Studies that have included a younger cohort of participants, including the study presented here, have found little evidence of a link between noise exposure and proxy measures of cochlear synaptopathy (e.g. Prendergast et al., 2017a, 2017b), whereas studies that have found a relation between these measures tended to be slightly older (Gu et al., 2012; Schaette and McAlpine, 2011; Valderrama et al., 2018). It is difficult to disentangle the effects of aging and noise exposure, since the two are closely related (Prendergast et al., 2019). Nevertheless, Johannesen et al. (2019) recently showed that decreasing wave I growth was associated with increasing age, but not with increasing noise exposure. Similarly, some of the weak effects of noise exposure on electrophysiological measures found by Prendergast et al. (2017a) were not significant after controlling for age.

4.1.2 Young adults are resilient to the effects noise-induced cochlear synaptopathy

Even if cochlear synaptopathy does occur in young normal hearing adults, it has been proposed that the effects on perceptual abilities, such as SPiN, may be negligible (Grose et al., 2017; Oxenham, 2016; Valderrama et al., 2018). Instead, it has been shown that superior auditory skills such as amplitude modulation detection and temporal fine structure processing, and superior cognitive abilities such as working memory, attention and language skills, may be more predictive of SPiN abilities (Valderrama et al., 2018; Yeend et al., 2017). It is possible that superior cognitive and/or auditory processing skills in humans may offset, or even supersede, the detrimental effects of noise-induced cochlear synaptopathy (Beach, 2018; Kamerer et al., 2019).

4.1.3 The effects of cochlear synaptopathy are difficult to detect in young normal hearing adults

An alternative explanation for the null findings is that noise-induced cochlear synaptopathy is difficult to detect due to the high levels of variability between individuals (Prendergast et al., 2019). Compared to precisely controlled animal studies, human participant samples are much more genetically and anatomically diverse, and have much more varied environments (e.g. noise exposure, exposure to ototoxic chemicals, health and lifestyle factors etc.). Additionally, the pattern of noise exposure in humans is much more irregular and impossible to accurately monitor across the lifespan. With the additional prospect of synaptic repair in humans (Kaas, 2001; Martino et al., 2011; Rosa and Bonfanti, 2018), and uncertainty as to whether repeated noise exposure has an additive or non-linear cumulative effect, it is challenging to anticipate the health of the auditory nerve in relation to noise exposure in humans.

The specificity and sensitivity of various proxy measures of cochlear synaptopathy have also been scrutinised. For example, wave I amplitudes of the ABR might not be determined by low-SR auditory nerve fibers (Bourien et al., 2014), which may instead have a role in controlling the efferent system (Carney, 2018). Caution should also be exercised in the interpretation of wave I/V ratios in relation to tinnitus/hyperacusis (Schaette and McAlpine, 2011), since the central gain hypothesis has not been fully validated. There is also an ongoing argument as to the which ABR montage configuration yields the most robust and reliable ABR wave amplitudes for assessing auditory nerve function, where it has been suggested that a horizontal montage (Laughlin et al., 1999) and/or electrode placement on- or near to- the tympanic membrane using a 'tiptrode' produces a larger wave I recording (Ferguson;, 1989; Minaya and Atcherson, 2015; Prendergast et al., 2018). However, Prendergast et al. (2018) demonstrated only very slight increases in reliability for wave I amplitude when using tiptrodes compared to electrodes placed on the mastoid (as in the current experiment), with both vertical ABR montage configurations producing excellent intraclass correlation

coefficients. Given that the canal tiptrodes are more costly and can cause some discomfort, it is not clear that such equipment is necessary for the recording of ABR waves I or V.

In relation to SPiN measures, Prendergast et al., (2019, 2017b) suggest that the association between these measures and noise exposure might only be observed for certain listening tasks with highly-challenging listening conditions (e.g. time compressed and reverberated speech; Liberman et al., 2016). This seems less likely for the CRM task used in the current study, since the condition with spatially congruent target and distracting speakers produced high SNRs (i.e. high difficulty), but was not related to noise exposure.

4.1.4. Measures of lifetime noise exposure are imperfect

As with other retrospective measures of noise exposure, the NESI may have provided erroneous measures of lifetime noise exposure. However, there is a stark difference between low and high exposure groups in terms of lifetime energy of exposure, with the high noise exposure group having ~15 times more exposure (not log transformed) than the low noise exposure group. This difference is even greater when considering participants in the top and bottom 20% of noise exposures across the distribution; equating to ~70 times more exposure (not log-transformed) in the high noise exposure group compared to low. As such, it seems unlikely that the effects of lifetime noise exposure would be eradicated by some imprecision in self-reported noise exposure. Furthermore, we were able to demonstrate some effects of noise exposure in the current experiment, such as ABR wave V latency, DPOAE levels, and the prevalence and severity of hyperacusis (see section 4.2). Similarly, previous experiments from our laboratory have demonstrated correlations with EHF thresholds (Prendergast et al., 2017a) and with tinnitus prevalence (Guest et al., 2017), suggesting that the NESI is a valid and reliable measure of noise exposure.

There has also been criticism that the NESI does not capture information relating to TTS and temporary tinnitus following noise exposure, which may be a prerequisite for noise-induced damage

(Kujawa and Liberman, 2009). In the current experiment, the majority of participants who experienced temporary tinnitus reported that this was due to exposure to a loud event (70/88; ~80%). Yet, experience of temporary tinnitus was not different between low and high noise exposure groups, and there were no significant correlations between tinnitus severity and noise exposure, ABR amplitudes or SPiN abilities. Likewise, Yeend et al. (2017) demonstrated that neither self-reported TTS following noise exposure nor tinnitus were predictive of SPiN abilities (see also Le Prell et al., 2018). It should be noted, however, that our definition of tinnitus (i.e. having experienced a ringing/buzzing in the ears for at least 5 minutes at any point in the participants' lives) could be too broad, which could explain the high prevalence of tinnitus in this participant sample, and could also lead to a high degree of individual variability in terms of symptom severity, and hence the lack of an effect of noise exposure on tinnitus measures. It may have been beneficial to use stricter criteria for tinnitus, such as reporting frequent tinnitus (i.e. at least once a week) or constant tinnitus (although this was only 4 participants in our sample).

4.2 Potential noise-induced hearing damage

4.2.1 The effects of noise exposure on ABR wave latency

Similar to Skoe and Tufts (2018), we showed that high levels of noise exposure did not have a significant impact on ABR wave amplitudes, but rather noise exposure may affect ABR wave latencies. On the contrary, we only found an effect of noise exposure on wave V latency (and thus wave I-V interpeak latency), whereas Skoe and Tufts (2018) showed that both wave I and V latencies were affected by previous noise exposure. Delays to ABR wave latencies as a result of peripheral damage (e.g. IHC loss) might be expected to affect all waves of the ABR (Burkard et al., 1997). Instead, our findings suggest a delayed propagation of the action potential along the auditory brainstem pathway which occurs central to the auditory nerve. While this finding seems unlikely to be due to cochlear synaptopathy, since wave I amplitudes and latencies were unaffected, it is difficult to ascertain the exact mechanism of this effect. Prendergast et al. (2017a) also found a

significant correlation between noise exposure and ABR wave V latency, although this effect was non-significant after controlling for the effect of age. Since participants in the current experiment were relatively young and the age-range was narrow, the effect of aging is less likely to explain delayed central propagation, and indeed the correlation between noise exposure and ABR wave V latency persists after controlling for age [$r(117) = .259$, $p = .004$].

Stressing the auditory system by increasing ABR stimulus presentation rate (J. H. Kim et al., 2013; Lasky, 1997; Shi et al., 2013; Skoe and Tufts, 2018) or presenting the ABR stimulus in various levels of background noise (Mehraei et al., 2016) may help to tease out the effects of noise exposure on synaptic function and neural conduction at different stages of the auditory pathway. For example, Skoe and Tufts (2018) showed a correlation between noise exposure and rate-dependent changes to wave I-V interpeak latency, theorizing that noise exposure could lead to demyelination of the central auditory system without affecting audiometric thresholds (see S. E. Kim et al., 2013). However, measuring ABR wave latencies for multiple stimulus presentation rates, and/or in multiple background noise levels, is probably not a feasible method for assessing sub-clinical hearing loss in a clinical setting.

Interestingly, the correlation between noise exposure and wave V latency was only significant for males in the current experiment. This may be in part due to males reporting slightly higher levels of noise exposure than females, and so the effects of noise exposure on ABR wave latency were more pronounced for this group.

4.2.2 The effects of noise exposure on outer hair cell function

Further non-synaptopathic effects of noise exposure were found for OHC function. In particular, the exploratory analysis showed that participants with the highest levels of noise exposure had poorer DPOAEs compared to participants with the lowest levels of noise exposure. This finding is further supported by the significant negative correlation between noise exposure and average DPOAE level.

Note that DPOAE measures indicated clinically normal OHC function for all participants in the current experiment i.e. DPOAE SNRs ≥ 3 dB for the majority of frequencies tested. Sub-clinical OHC dysfunction is more common in young normal hearing listeners with high levels of noise exposure compared to those with low levels of noise exposure (Hamdan et al., 2008; Job et al., 2009; Lapsley Miller et al., 2006; Lucertini et al., 2002; Mansfield et al., 1999).

Our results also revealed a significant negative correlation between average DPOAE level and average high-frequency (2-8 kHz) PTA and EHF (12 and 16 kHz) thresholds. Although hearing thresholds were not linked to noise exposure, these data support well-established evidence that OHC function is fundamental to hearing thresholds (Stebbins et al., 1979).

Reduced OHC function and elevated PTA and EHF thresholds did not have any effect on ABRs or SPiN performance in the current experiment. However, elevated PTA and EHF thresholds could potentially explain some of the electrophysiological and behavioural findings which have been attributed to cochlear synaptopathy in previous studies. For example, Bramhall et al. (2017) found elevated PTA thresholds between 2-6 kHz in military veterans with high levels of noise exposure compared to non-veterans with lower levels of noise exposure, which could explain the observed reduction in ABR wave I amplitudes in veterans compared to non-veterans (although wave I amplitudes were adjusted for OAE levels in this experiment). Therefore, elevated hearing thresholds and/or OHC dysfunction within the clinically 'normal' range might make it problematic to use ABR wave I reduction as a proxy measure of cochlear synaptopathy, and should be accounted for in the analysis. These findings also offer clinical utility for assessing hearing function below 25 dB HL and greater than 8 kHz as an early marker of hearing deficits (Le Prell et al., 2013; Mehrparvar et al., 2011; Moore et al., 2017), which is not necessarily related to noise-induced damage.

4.2.3 The effects of noise exposure on self-reported hyperacusis

Another significant effect of noise exposure was found for hyperacusis, whereby the high noise exposure group were more than twice as likely to report having experienced hyperacusis compared to the low noise exposure group. The exploratory analysis also revealed that participants with the highest levels of noise exposure reported more severe hyperacusis than those with the lowest noise exposures. However, there was no significant correlation between HQ scores and noise exposure, nor with any of the electrophysiological measures included in the test battery. As such, this effect is unlikely to support the central-gain hypothesis (Hickox and Liberman, 2014), and the cause is uncertain.

4.3 No effects of musicianship on hearing measures

In addition to the null effects of noise exposure on SPiN in the current study, there were also no differences between musicians and non-musicians, and no interactions between noise exposure and musicianship. This could be due to the finding that total lifetime noise exposure was not significantly different between musicians and non-musicians. Based on previous research, it was hypothesized that musical training may be able to compensate for the detrimental effects of noise exposure on SPiN (Grose et al., 2017; Valderrama et al., 2018; Yeend et al., 2017), or that noise exposure may reduce musicians' advantage for SPiN (Skoe et al., 2018). Since total noise exposure was similar between musicians and non-musicians, it would be reasonable to predict enhanced SPiN for musicians in the current study.

While there are many studies that advocate enhanced SPiN in musicians, regardless of noise exposure (Bidelman et al., 2014; Clayton et al., 2016; Parbery-Clark et al., 2011, 2009b, 2009a; Slater et al., 2015; Strait et al., 2010; Swaminathan et al., 2015), there are others that show little-to-no effect of musicianship (Boebinger et al., 2015; Couth et al., 2019; Madsen et al., 2019, 2017; Ruggles et al., 2014; Yeend et al., 2017). There are several possible explanations for these discrepant findings. First, it has been pointed out that musicians' benefit for SPiN over non-musicians may be very small in some studies (e.g. < 1 dB SNR; Parbery-Clark et al., 2009b) and so might not be a

particularly robust finding (Madsen et al., 2019). Speech-in-noise tests also vary between studies in terms of task demands, target speech stimuli, number and type of maskers, characteristics of the masking noise (e.g. informational vs. energetic masking), and the spatial separation between the target and masking speakers (e.g. centrally aligned vs. offset; Madsen et al., 2019; Swaminathan et al., 2015).

The CRM task used in the current experiment was comparable to the SPiN task used by Swaminathan et al. (2015), using both spatially and offset distracting speakers whose speech utterances were highly similar to the target speaker (i.e. high informational masking). Swaminathan et al. (2015) also showed equal performance for musicians and non-musicians in centrally aligned conditions, but in contrast showed better performance for musicians in spatially offset conditions (i.e. greater SRFM). In Swaminathan et al. (2015), distracting stimuli were spatially offset by 15 degrees azimuth on either side of centre, compared to 60 degrees in the current study. Musicians' mean SNRs for the spatially offset condition in the current study (SNR = -14.2 dB) were comparable to the musicians in Swaminathan et al. (SNR = -15.1 dB), whereas non-musicians' SNRs for the spatially offset condition were much higher in the current study (SNR = -13.6 dB) than the non-musicians in Swaminathan et al. (SNR = -8.5 dB). Accordingly, musicians may reach maximum SRFM benefit by 15 degrees masker separation, whereas non-musicians may require up to 60 degrees of masker separation to achieve the same level of SRFM benefit. By testing finer degrees of separation between target and distractor speakers, it may be possible to ascertain the effects of musicianship on SRFM.

Similar to there being a high degree of inter-individual variability in noise exposure, there could also be a high degree of heterogeneity in musicianship for both musician and non-musician groups, which could explain the null effects in the current study (Levitin, 2012; Ruggles et al., 2014). We did not place any strict criteria on the definition of a 'musician' or 'musicianship', choosing to focus on a group of individuals who were in the early stages of pursuing a career in music, and so instrument

class, years of playing, hours of practice, and the age that musical training began was variable within this group. Zhang et al. (2018) have recently tried to establish a general consensus for the term "musician", suggesting that the most robust definition is someone who has at least six years of musical expertise. For the non-musician group in the current experiment, there were 28 participants who had learned to play a musical instrument at some point during their lives, and 17 of these had at least 6 years of musical experience. 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 SPiN performance between these groups. To address this limitation, we examined musicianship on a continuous scale (i.e. years of musical experience) across the whole participant sample, showing no significant correlations with any measures. However, years of playing an instrument does not necessarily equate to high musical skill or ability. It would have been beneficial to include a range of tests to assess musical, auditory and cognitive skills in the current test battery (as per Yeend et al., 2017).

It is also important to emphasise that the cross-sectional design of the current study can only provide a snapshot of hearing function in relation to musicianship and noise exposure. Indeed, it was surprising that total noise exposure was similar for musicians and non-musicians, despite musicians showing higher levels of occupational noise exposure, and it was not our intention to match noise exposure between these groups. This finding was due to the relatively high levels of recreational noise exposure compared to occupational noise exposure, for both musicians and non-musicians, where both groups were at an age (e.g. 18-25 years) and period in life (e.g. university undergraduate) where they were starting to experience high levels of recreational exposure (e.g. concerts and nightclubs). Crucially, this finding highlights the importance of estimating both occupational *and* recreational noise dose when investigating the effects of lifetime noise exposure, rather than assuming that differences in occupational exposure alone will ensure distinct levels of

total noise exposure. Moreover, the data presented here represents the first of three participant visits, where the test battery is being repeated once a year as musicians progress through an intensive period of musical training at the start of their careers. This longitudinal study design will provide a better insight into the accumulative effects of musical experience, noise exposure, and inter-individual risk of noise-induced damage, where it is anticipated that lifetime noise exposure will steadily increase for musicians (especially occupational exposure), but may start to decelerate for non-musicians.

4.4 Musicians report more hearing complaints

Consistent with previous research, musicians were more likely to self-report experience of - and/or more severe - hearing problems such as tinnitus, hyperacusis and hearing in noise difficulties (Camera et al., 2019; Couth et al., 2019; Greasley et al., 2018; Jansen et al., 2009; Kähäri et al., 2003; Laitinen, 2005; Laitinen and Poulsen, 2008). These findings were not related to noise exposure, and musicians with *low* noise exposure were actually *more* likely to report hearing in noise difficulties.

For tinnitus and hyperacusis severity measures, higher scores for musicians is unlikely to be explained by the central gain hypothesis since there was no correlation between these measures and wave I/V ratios, plus musicians had significantly greater wave I/V ratios compared to non-musicians, which would suggest a reduction in central gain for musicians. There are several studies that have demonstrated heightened brainstem auditory evoked potentials in musicians compared to non-musicians, in particular the frequency following response, whereby musical training fine-tunes how sound is processed subcortically, leading to enhancement of acoustic features (for a review see Sanju and Kumar, 2016). However, altered electrophysiological functioning for musicians did not translate into enhanced SPiN abilities in the current study. Including measures of midbrain and cortical auditory-evoked potentials in future studies could be useful for determining the effects of musicianship - and noise exposure - on auditory processing, where central gain could occur at later stages of the auditory pathway (Chambers et al., 2016).

The most likely explanation for worse self-reported hearing problems in musicians is due to musicians being more aware and/or concerned about hearing problems, and thus being more likely to report hearing problems compared to non-musicians (Chesky et al., 2009; Greasley et al., 2018; Laitinen, 2005; Laitinen and Poulsen, 2008). This could also explain the observed correlations between self-report measures, whereby those who are more aware/concerned will consistently score more highly across these different measures of hearing problems.

4.5 Conclusions

Lifetime noise exposure does not appear to be related to ABR proxy measures of cochlear synaptopathy in normal-hearing young adults. That is not to say that noise exposure is not a concern, since we did observe a decrease in OHC function, increased ABR wave V latency, and a higher prevalence and severity of hyperacusis with higher levels of noise exposure, most of which was from recreational activities. These indices of noise-induced damage would otherwise be 'hidden' by having a normal audiogram. Closely monitoring OHC function when hearing thresholds are clinically normal could provide a timely measure of noise-induced hearing damage, especially for individuals with high levels of noise exposure, such as musicians. Furthermore, by examining the effects of noise exposure on hearing function longitudinally, it may be possible to determine whether musicians are more susceptible to hearing problems, and thus develop interventions to protect hearing longevity.

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Figure captions

Figure 1. Mean PTA thresholds and EHF thresholds for musicians (a and b) and non-musicians (c and d). Low noise exposure groups are represented by square symbols with solid lines, and high noise exposure groups are represented by triangle symbols with broken lines. Error bars represent 95% confidence intervals. Color version available online.

Figure 2. Mean DPOAE levels (line graphs) and noise floor (shaded areas) for a) musicians and b) non-musicians. DPOAE level for low noise exposure groups are represented by square symbols with solid lines, and high noise exposure groups are represented by triangle symbols with broken lines. DPOAE noise floors for low exposure groups are represented by shaded areas with a solid line and high noise exposure groups are represented by shaded areas with a broken line. Error bars represent 95% confidence intervals for DPOAE levels. Color version available online.

Figure 3. Mean SNRs for the CRM task for low noise exposed musicians (purple bars), high noise exposed musicians (green bars), low noise exposed non-musicians (blue bars) and high noise exposed non-musicians (orange bars). SNRs for centrally aligned masking conditions are represented by solid shading and spatially offset masking conditions are represented by broken shading. More negative values indicate better performance. Error bars represent 95% confidence intervals. Color version available online.

Figure 4. Grand average ABR waveforms for 60 dB nHL click stimuli (top panels; a and b) and 80 dB nHL click stimuli (bottom panels; c and d). Musicians' ABR waveforms are shown in the left panels (a and c), with low noise exposed musicians represented by solid purple lines and high noise exposed musicians represented by broken green lines. Non-musicians' ABR waveforms are shown in the right panels (b and d), with low noise exposed musicians represented by solid blue lines and high noise exposed musicians represented by broken orange lines. Shaded areas represent 95%

confidence intervals. Wave I and V amplitudes were measured from peak to subsequent trough, as demonstrated in panel c). Color version available online.

Figure 5. Mean ABR amplitudes for a) wave I, b) wave V, and c) the wave I/V ratio for low noise exposed musicians (purple bars), high noise exposed musicians (green bars), low noise exposed non-musicians (blue bars) and high noise exposed non-musicians (orange bars). Wave amplitudes for the 60 dB nHL click stimulus are represented by broken shading and 80 dB nHL click stimulus are represented by solid shading. Error bars represent 95% confidence intervals. Color version available online.

Figure 6. Mean scores on self-report measures a) Tinnitus Handicap Inventory, b) Hyperacusis Questionnaire, and c) Hearing in noise difficulties for low noise exposed musicians (purple bars), high noise exposed musicians (green bars), low noise exposed non-musicians (blue bars) and high noise exposed non-musicians (orange bars). For a) and b) higher scores indicate more severe tinnitus/hyperacusis, for c) lower scores indicate more severe hearing in noise difficulties. Error bars represent 95% confidence intervals. Color version available online.

Figure 7. Mean ABR peak latencies for a) wave I, b) wave V, and c) the wave I-V interpeak latency for low noise exposed musicians (purple bars), high noise exposed musicians (green bars), low noise exposed non-musicians (blue bars) and high noise exposed non-musicians (orange bars). Wave latencies for the 60 dB nHL click stimulus are represented by broken shading and 80 dB nHL click stimulus are represented by solid shading. Error bars represent 95% confidence intervals. Color version available online.

Figure 8. a) Correlation matrix between test battery measures for the whole participant sample. Red squares indicate positive correlations and blue squares indicate negative correlations. Significant correlations are indicated by * ($p < .05$ and $q < .10$). Scatter plots for total lifetime noise exposure against b) average wave V latency and c) DPOAE level. Data is shown for males (blue

squares/blue dotted line) and females (pink crosses/pink dashed line). The overall regression line is shown in black. R values in the inset figure legends represent Spearman's rho correlations, with * indicating a significant correlation ($p < .05$ and $q < .10$). Color version available online.

Figure 1

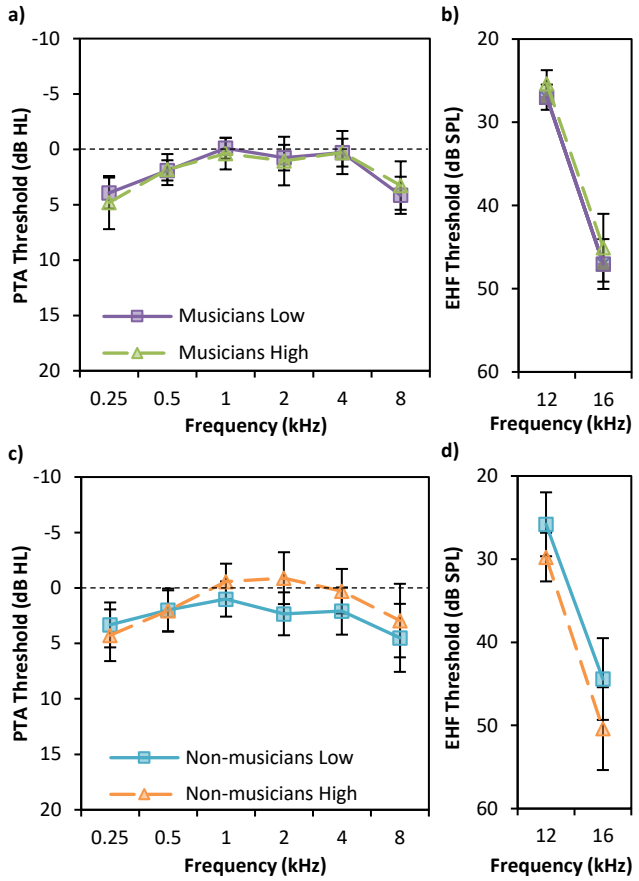

a)
b)
c)
d)
PTA Threshold (dB HL)
EHF Threshold (dB SPL)
Frequency (kHz)
Musicians Low
Musicians High
Non-musicians Low
Non-musicians High
-10
-5
0
5
10
15
20
30
40
50
60
0.25
0.5
1
2
4
8
12
16

Figure 2

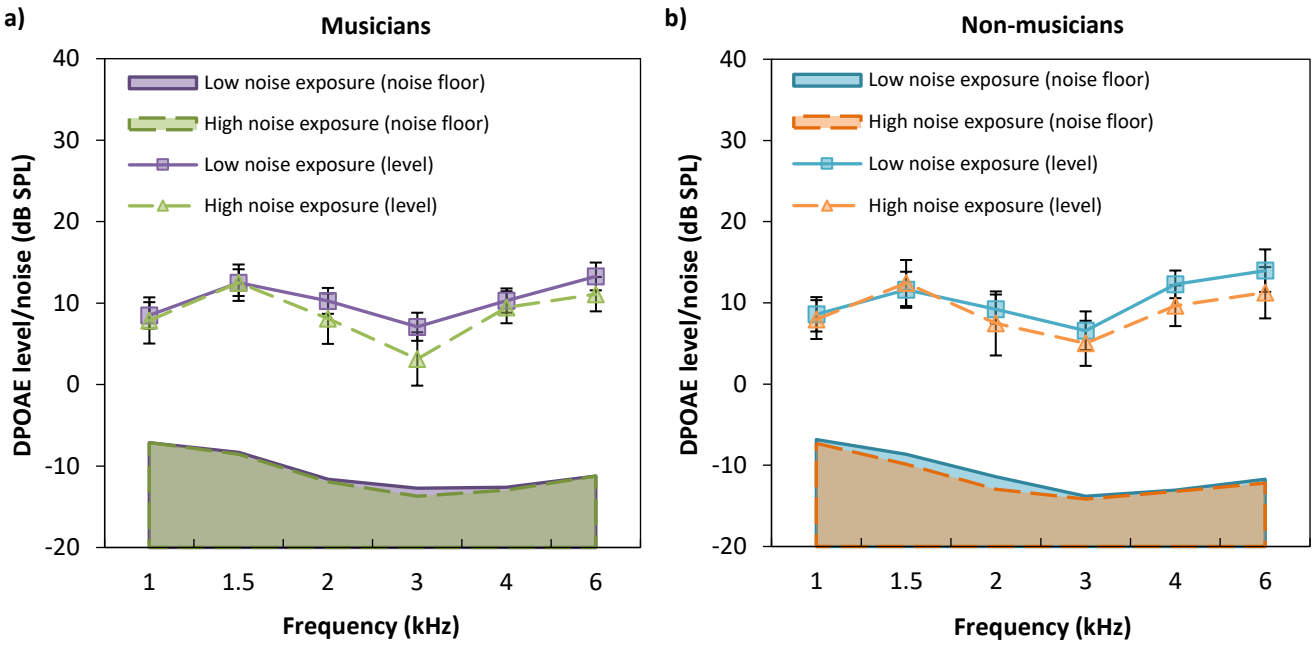

a)
Musicians
Low noise exposure (noise floor)
High noise exposure (noise floor)
Low noise exposure (level)
High noise exposure (level)
DPOAE level/noise (dB SPL)
40
30
20
10
0
-10
-20
1
1.5
2
3
4
6
Frequency (kHz)
b)
Non-musicians
Low noise exposure (noise floor)
High noise exposure (noise floor)
Low noise exposure (level)
High noise exposure (level)
DPOAE level/noise (dB SPL)
40
30
20
10
0
-10
-20
1
1.5
2
3
4
6
Frequency (kHz)

Figure 3

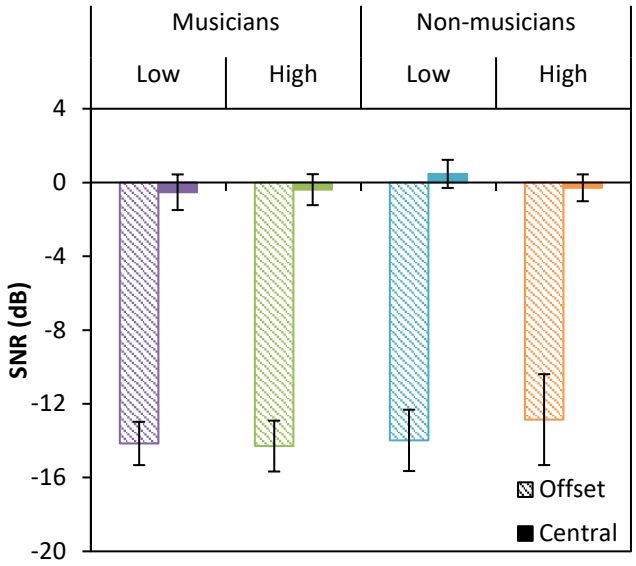

Musicians
Non-musicians
Low
High
Low
High
4
0
-4
-8
-12
-16
-20
SNR (dB)
Offset
Central

Figure 4

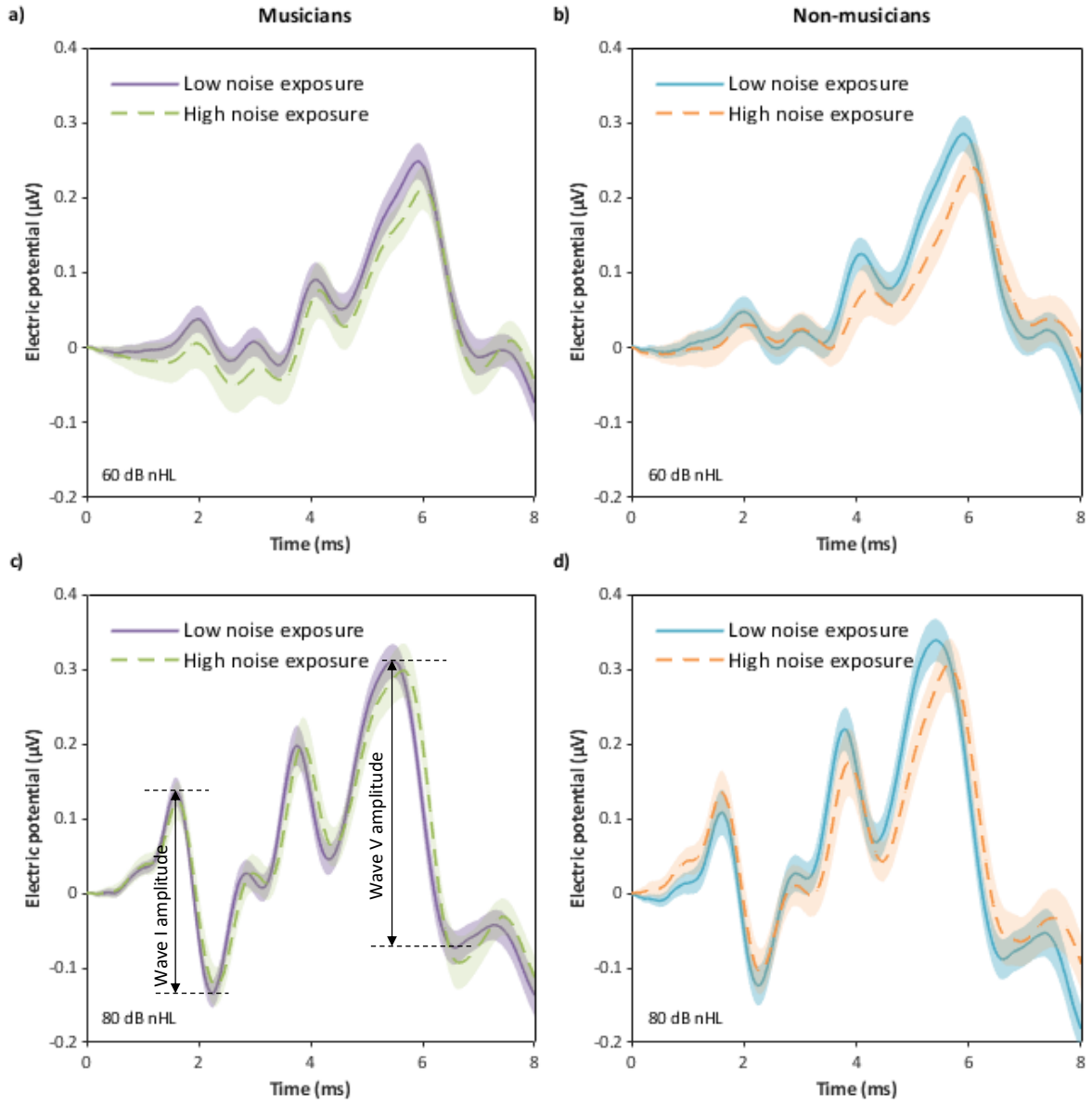

a)
Musicians
b)
Non-musicians
c)
d)
Low noise exposure
High noise exposure
Electric potential (µV)
Time (ms)
60 dB nHL
80 dB nHL
Wave I amplitude
Wave V amplitude

Figure 5

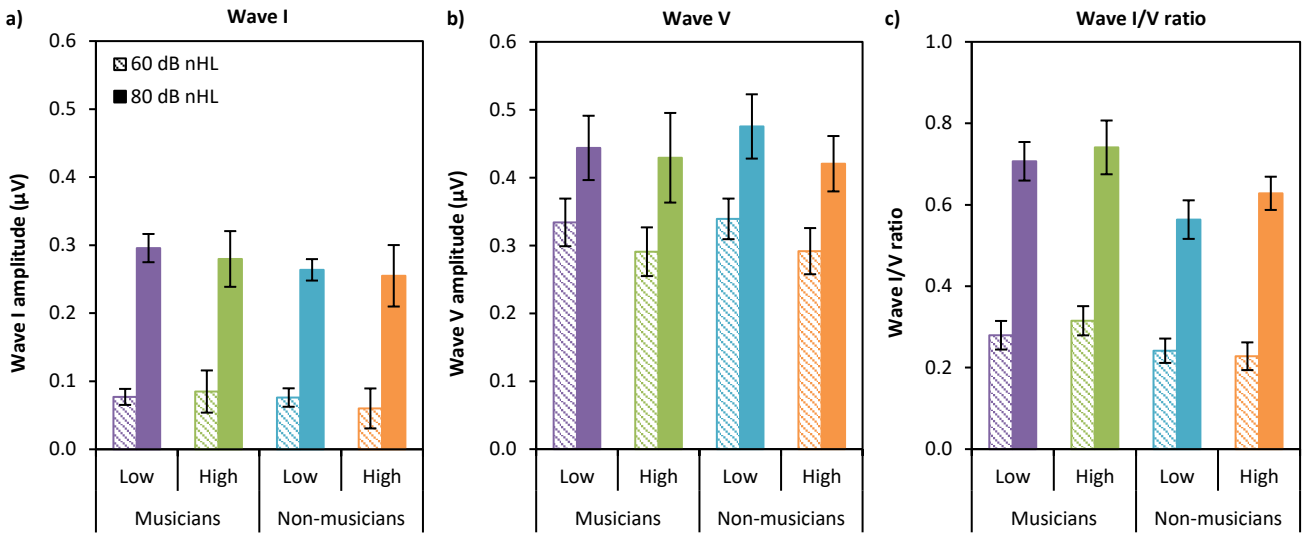

a)
Wave I
60 dB nHL
80 dB nHL
Wave I amplitude (µV)
0.6
0.5
0.4
0.3
0.2
0.1
0.0
Low
High
Low
High
Musicians
Non-musicians
b)
Wave V
Wave V amplitude (µV)
0.6
0.5
0.4
0.3
0.2
0.1
0.0
Low
High
Low
High
Musicians
Non-musicians
c)
Wave I/V ratio
Wave I/V ratio
1.0
0.8
0.6
0.4
0.2
0.0
Low
High
Low
High
Musicians
Non-musicians

Figure 6

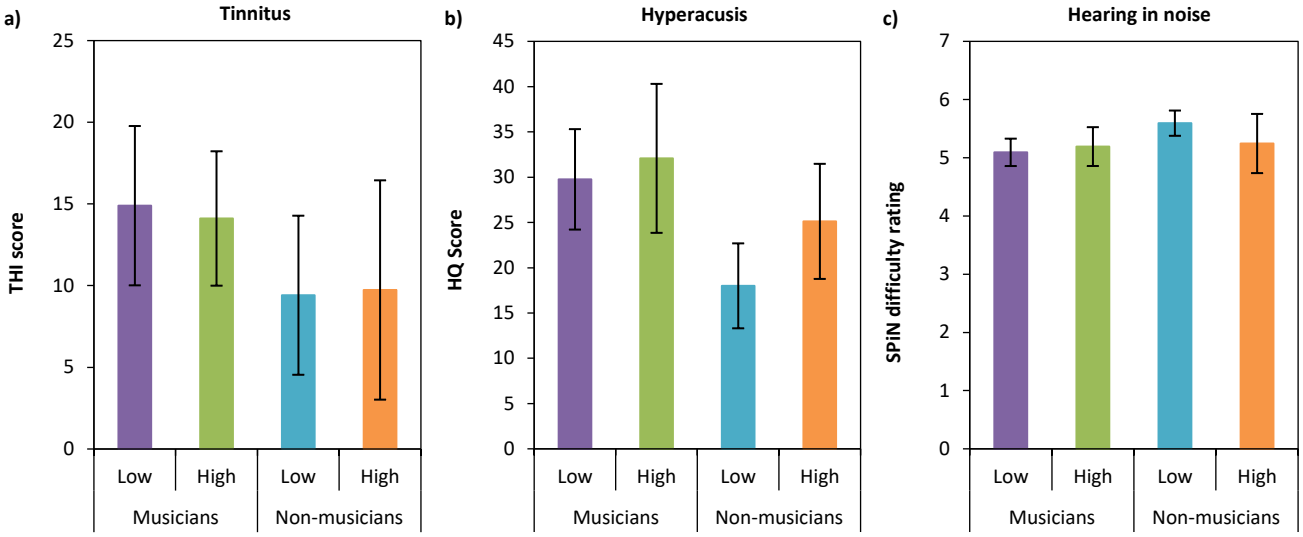

a)
Tinnitus
THI score
25
20
15
10
5
0
Low
High
Low
High
Musicians
Non-musicians
b)
Hyperacusis
HQ Score
45
40
35
30
25
20
15
10
5
0
Low
High
Low
High
Musicians
Non-musicians
c)
Hearing in noise
SPiN difficulty rating
7
6
5
4
3
2
1
0
Low
High
Low
High
Musicians
Non-musicians

Figure 7

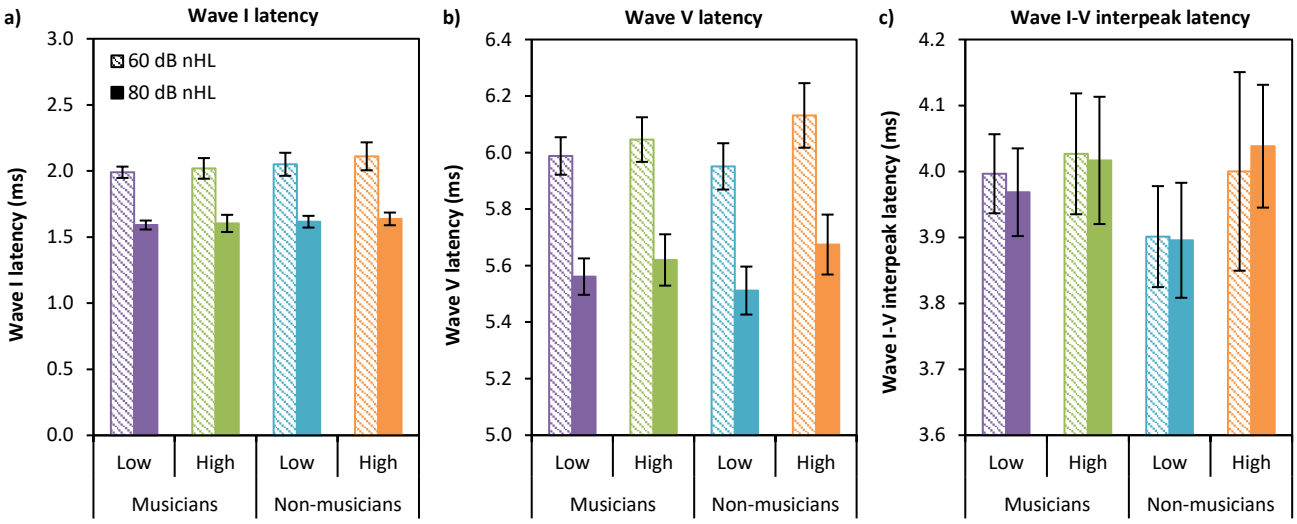
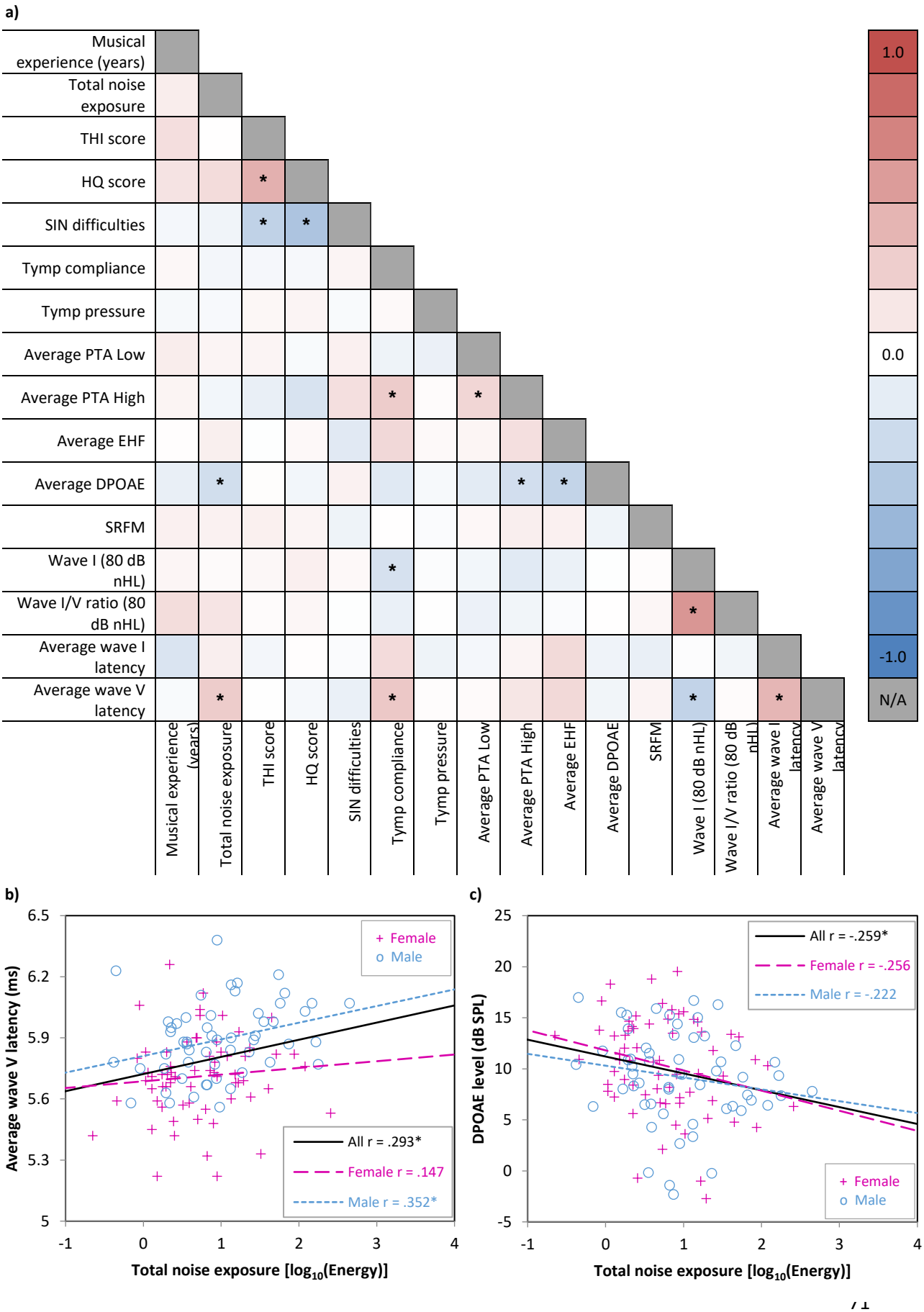

a)
Wave I latency
60 dB nHL
80 dB nHL
Wave I latency (ms)
3.0
2.5
2.0
1.5
1.0
0.5
0.0
Low
High
Low
High
Musicians
Non-musicians
b)
Wave V latency
Wave V latency (ms)
6.4
6.2
6.0
5.8
5.6
5.4
5.2
5.0
Low
High
Low
High
Musicians
Non-musicians
c)
Wave I-V interpeak latency
Wave I-V interpeak latency (ms)
4.2
4.1
4.0
3.9
3.8
3.7
3.6
Low
High
Low
High
Musicians
Non-musicians

Figure 8


a)
Musical experience (years)
Total noise exposure
THI score
HQ score
SIN difficulties
Tymp compliance
Tymp pressure
Average PTA Low
Average PTA High
Average EHF
Average DPOAE
SRFM
Wave I (80 dB nHL)
Wave I/V ratio (80 dB nHL)
Average wave I latency
Average wave V latency
1.0
0.0
-1.0
N/A
b)
Average wave V latency (ms)
Total noise exposure [log10(Energy)]
+ Female
o Male
All r = .293*
Female r = .147
Male r = .352*
c)
DPOAE level (dB SPL)
Total noise exposure [log10(Energy)]
All r = -.259*
Female r = -.256
Male r = -.222
+ Female
o Male

Supplementary Materials 1

Subset of the questions from the Self-efficacy for Situational Communication Management Questionnaire (SESMQ; Jennings 2005) used to assess self-perceived hearing in noise difficulties.

Please imagine yourself in each of the following situations and answer the questions.

You are having a family dinner in your home. There is more than one conversation occurring at a time.

How well can you hear in this situation?

Not well at all **1** **2** **3** **4** **5** **6** **7** *Very well*

You are at a wedding reception with 200 guests. Your friend/family member starts talking to you.

How well can you hear in this situation?

Not well at all **1** **2** **3** **4** **5** **6** **7** *Very well*

You are in a restaurant with a family member or friend. You are seated in a dim and noisy spot.

How well can you hear in this situation?

Not well at all **1** **2** **3** **4** **5** **6** **7** *Very well*

You are at home watching television with a family member. She/he turns and speaks to you.

How well can you hear in this situation?

Not well at all **1** **2** **3** **4** **5** **6** **7** *Very well*

You are waiting for a train/plane at a busy station. Your friend is sitting beside you and says something without looking at you.

How well can you hear in this situation?

Not well at all **1** **2** **3** **4** **5** **6** **7** *Very well*

You hold a party in your home. Someone you do not know very well starts up a conversation. She/he puts one hand over her/his mouth when they are speaking.

How well can you hear in this situation?

Not well at all **1** **2** **3** **4** **5** **6** **7** *Very well*

Supplementary Materials 2

Results for the exploratory analysis of the top and bottom 20% of participants from the total noise exposure distribution. Statistical analyses reflect those of the whole participant sample: repeated measures ANOVAs for PTA, EHF thresholds, DPOAE levels, ABR wave I amplitudes, ABR wave I/V ratios, ABR wave I and wave V latencies, and the CRM task (Table 1); independent samples *t*-tests and oneway ANOVAs for total log noise exposure, spatial release from masking, THI scores, HQ scores and SPiN scores (Table 2); chi-square tests for prevalence of self-reported tinnitus, hyperacusis and speech-in-noise difficulties (Table 3). For simplicity, only the effects of interest are presented; noise exposure, musicianship and the noise exposure by musicianship interaction. Analysis of further main effects and interactions for each measure are available upon request.

The findings closely match those of the whole participant sample. Novel findings from this sub-sample included the main effect of noise exposure on DPOAE levels [$F(1,48) = 11.702$, $p = .001$, $\eta_p^2 = .196$], where levels were significantly lower for the high noise exposure group (mean = 8.79 dB SPL, SD = 3.02) compared to the low noise exposure group (mean = 12.05 dB SPL, SD = 3.18). Mean HQ scores were also found to be significantly higher for the high noise exposure group (mean = 30.81, SD = 20.57) compared to the low noise exposure group (mean = 19.19, SD = 11.83) [$t(39.909) = -2.496$, $p = .017$, $d = .692$]. Non-musicians with high noise exposure also had the greatest proportion of participants reporting having experienced tinnitus (n = 10; 100%) compared to non-musicians with low noise exposure (n = 5; 50%), musicians with high noise exposure (n = 11; 68.75%) and musicians with low noise exposure (n = 14; 87.50%) [$\chi^2(3) = 8.694$, $p = .034$, $V = .409$].

Table 1 Repeated measures ANOVA tests for the exploratory analysis including main effects of noise exposure, musicianship and the noise exposure x musicianship interaction. Values in bold indicate a significant effect.

	Noise exposure			Musicianship			Noise exposure*Musicianship		
Measure	*F*	*p*	η_p^2	*F*	*p*	η_p^2	*F*	*p*	η_p^2
PTA threshold	0.559	0.458	0.012	0.594	0.445	0.012	0.678	0.414	0.014
EHF threshold	1.558	2.180	0.031	0.016	0.900	0.000	**8.647**	**0.005**	**0.153**
DPOAE level	**11.702**	**0.001**	**0.196**	0.989	0.325	0.020	1.589	0.214	0.032
ABR wave I amplitude	0.175	0.677	0.004	3.902	0.055	0.085	0.200	0.657	0.005
ABR wave I/V ratio	0.512	0.478	0.012	**5.036**	**0.030**	**0.107**	0.027	0.870	0.001
ABR wave I latency	3.052	0.088	0.068	1.854	0.181	0.042	0.008	0.930	0.000
ABR wave V latency	**8.566**	**0.005**	**0.166**	0.300	0.587	0.007	0.606	0.441	0.014
CRM task	0.001	0.811	0.000	0.468	0.497	0.010	0.058	0.811	0.001

Table 2 Independent samples *t*-tests between high and low noise exposure groups/ musicians and non-musicians, and one-way ANOVAs tests to explore the interaction between noise exposure and musicianship. These tests were conducted for variables with one level in the dependent variable of the exploratory analysis. Values in bold indicate a significant effect.

	Low vs. High noise exposure			Musicians vs. Non-musicians			Noise exposure*Musicianship		
Measure	*t*	*p*	*d*	*t*	*p*	*d*	*F*	*p*	η2
Total noise exposure	**-20.608**	**0.000**	**5.716**	-0.605	0.548	0.169	**155.841**	**0.000**	**0.907**
SRFM	-0.602	0.550	0.167	0.531	0.598	0.150	0.269	0.847	0.017
THI scores	0.145	0.885	0.046	0.592	0.558	0.185	0.172	0.915	0.014
HQ scores	**-2.496**	**0.017**	**0.692**	0.904	0.370	0.266	2.352	0.084	0.128
Hearing in noise score	1.288	0.204	0.357	-0.112	0.911	0.032	0.688	0.564	0.041

Table 3 Chi-square tests for the exploratory analysis including main effects of noise exposure, musicianship and the noise exposure x musicianship interaction. Values in bold indicate a significant effect.

	Low vs. High noise exposure			Musicians vs. Non-musicians			Noise exposure*Musicianship		
Measure	*χ2*	*p*	*V*	*χ2*	*p*	*V*	*χ2*	*p*	*V*
Tinnitus	0.433	0.510	0.091	0.068	0.795	0.036	**8.694**	**0.034**	**0.409**
Hyperacusis	**5.026**	**0.025**	**0.311**	0.000	1.000	0.000	5.067	0.167	0.312
Hearing in noise score	1.038	0.308	0.141	0.026	0.872	0.022	1.412	0.703	0.165

Supplementary Materials 3

Table 4 Exploratory linear regression models for self-report proxy measures of cochlear synaptopathy

	THI score			HQ score			Hearing in noise score		
	R^2 = .085, F(10,74) = .688, p = .732			R^2 = .158, F(10,108) = 2.032, p = .037			R^2 = .128, F(10,108) = 1.591, p = .119		
Variable	B	β	*t*	B	β	*t*	B	β	*t*
Gender	-.911	-.035	-.286	1.887	.051	.520	-.004	-.002	-.023
Tymp compliance	-.350	-.008	-.065	3.445	.059	.580	.108	.041	.392
Tymp pressure	.097	.096	.808	.158	.128	1.419	-.003	-.051	-.554
Low PTA	.878	.232	1.747	.191	.034	.346	.024	.095	.935
High PTA	-.936	-.288	-1.949	-1.407	-.305	-2.793**	.058	.274	2.470*
EHF	-.043	-.028	-.209	.133	.061	.580	-.020	-.205	-1.928
DPOAE level	-.283	-.100	-.752	-.576	-.142	-1.365	.025	.138	1.305
wI amplitude	7.247	.057	.473	21.307	.114	1.234	.263	.031	.330
Years of musicianship	.139	.060	.519	.677	.210	2.342*	-.025	-.171	-1.870
Total noise exposure	-1.969	-.100	-.813	2.403	.083	.869	-.020	-.015	-.154

* p < .05, although non-significant after FDR correction. ** p < .05 and q < .10

Table 5 Exploratory linear regression models for behavioural proxy measure of cochlear synaptopathy

	SRFM		
	R^2 = .047, F(10,108) = .528, p = .867		
Variable	B	β	*t*
Gender	-.692	-.078	-.747
Tymp compliance	.441	.032	.291
Tymp pressure	.022	.076	.788
Low PTA	-.014	-.011	-.101
High PTA	.131	.118	1.018
EHF	-.032	-.061	-.548
DPOAE level	.008	.009	.078
wI amplitude	2.576	.058	.585
Years of musicianship	.019	.025	.257
Total noise exposure	1.111	.160	1.575

Table 6 Exploratory linear regression models for electrophysiological proxy measure of cochlear synaptopathy

	wI amplitude			wI/V ratio			wV latency		
	R^2 = .094, F(9,109) = .1.256, p = .269			R^2 = .068, F(9,109) = .877, p = .548			R^2 = .283, F(9,110) = 4.813, p < .001		
Variable	B	β	*t*	B	β	*t*	B	β	*t*
Gender	-.017	-.084	-.837	-.008	-.015	-.149	.130	.306	3.430**
Tymp compliance	-.086	-.275	-2.692**	-.109	-.124	-1.200	.151	.227	2.495**
Tymp pressure	.000	-.028	-.303	-.001	-.061	-.648	-.001	-.044	-.539
Low PTA	-.002	-.056	-.544	-.007	-.080	-.766	-.002	-.033	-.357
High PTA	.001	.021	.187	.004	.062	.542	.005	.092	.926
EHF	9.623E-5	.008	.076	.001	.029	.268	.001	.057	.590
DPOAE level	-.001	-.055	-.512	.005	.081	.747	.006	.132	1.372
Years of musicianship	.001	.066	.712	.009	.178	1.893	-.002	-.066	-.803
Total noise exposure	.006	.039	.393	.049	.113	1.129	.080	.240	2.763**

** p < .05 and q < .10