

1 **Effects of noise exposure on young adults with normal audiograms II: Behavioral measures**

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4 Garreth Prendergast^{a,*}, Rebecca E. Millman^{a,b}, Hannah Guest^a, Kevin J. Munro^{a,b}, Karolina Kluk^{a,b},

5 Rebecca S. Dewey^{c,d,e}, Deborah A. Hall^{d,e}, Michael G. Heinz^f, Christopher J. Plack^{a,b,g}.

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7 a, Manchester Centre for Audiology and Deafness, University of Manchester, Manchester Academic
8 Health Science Centre, M13 9PL, UK.

9 b, NIHR Manchester Biomedical Research Centre, Central Manchester University Hospitals NHS
10 Foundation Trust, Manchester Academic Health Science Centre, Manchester, M13 9WL, UK.

11 c, Sir Peter Mansfield Imaging Centre, School of Physics and Astronomy, University of Nottingham
12 Nottingham, NG7 2RD, UK.

13 d, National Institute for Health Research (NIHR) Nottingham Biomedical Research Centre,
14 Nottingham, NG1 5DU, UK.

15 e, Otology and Hearing Group, Division of Clinical Neuroscience, School of Medicine, University
16 of Nottingham, Nottingham, NG7 2UH, UK.

17 f, Department of Speech, Language, & Hearing Sciences and Biomedical Engineering, Purdue
18 University, West Lafayette, IN 47907, USA.

19 g, Department of Psychology, Lancaster University, Lancaster, LA1 4YF, UK.

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28 Abstract

29 An estimate of lifetime noise exposure was used as the primary predictor of performance on a range
30 of behavioral tasks: frequency and intensity difference limens, amplitude modulation detection,
31 interaural phase discrimination, the digit triplet speech test, the co-ordinate response speech
32 measure, an auditory localization task, a musical consonance task and a subjective report of hearing
33 ability. One hundred and thirty-eight participants (81 females) aged 18-36 years were tested, with a
34 wide range of self-reported noise exposure. All had normal pure-tone audiograms up to 8 kHz. It
35 was predicted that increased lifetime noise exposure, which we assume to be concordant with noise-
36 induced cochlear synaptopathy, would elevate behavioral thresholds, in particular for stimuli with
37 high levels in a high spectral region. However, the results showed little effect of noise exposure on
38 performance. There were a number of weak relations with noise exposure across the test battery,
39 although many of these were in the opposite direction to the predictions, and none were statistically
40 significant after correction for multiple comparisons. There were also no strong correlations
41 between electrophysiological measures of synaptopathy published previously and the behavioral
42 measures reported here. Consistent with our previous electrophysiological results, the present
43 results provide no evidence that noise exposure is related to significant perceptual deficits in young
44 listeners with normal audiometric hearing. It is possible that the effects of noise-induced cochlear
45 synaptopathy are only measurable in humans with extreme noise exposures, and that these effects
46 always co-occur with a loss of audiometric sensitivity.

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48 Keywords

49 Cochlear synaptopathy

50 Hidden hearing loss

51 Noise-induced hearing loss

52 Speech-in-noise

53 Psychophysics

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56 1. Introduction

57 Cochlear synaptopathy due to noise exposure (often referred to as “hidden hearing loss”) was
58 demonstrated in a mouse model by Kujawa and Liberman (2009). In the base of the cochlea, 50%
59 of synapses were lost between inner hair cells (IHCs) and auditory nerve (AN) fibers after a 2-hour
60 exposure to 100 dB SPL noise (8-16 kHz). Post-exposure, measures of absolute auditory sensitivity
61 were unaffected but a permanent decrease in the amplitude of wave I of the auditory brainstem
62 response (ABR), reflecting decreased auditory nerve activity, was seen in response to moderate- and
63 high-intensity stimuli. The synaptic loss was subsequently found to preferentially affect the high-
64 threshold, low spontaneous-rate (SR) AN fibers (Furman et al., 2013).

65

66 A loss of cochlear synapses due to noise exposure, in the presence of almost unaffected threshold
67 sensitivity, has also been demonstrated in a range of other rodents (e.g. guinea pig, Lin et al., 2011;
68 chinchilla, Hickox et al., 2015; 2017; rat, Möhrle et al., 2016). However, pure synaptopathy may be
69 more difficult to produce in primates. In the macaque model, high noise exposures (108 dB SPL or
70 greater) may be required for around 4 hours to produce supra-threshold reductions in the amplitude
71 of wave I of the ABR (Valero et al., 2017). A number of review articles give a thorough account of
72 the progression from the initial seminal work in the mouse, to the current understanding in the field
73 (Kobel et al., 2017; Liberman and Kujawa, 2017; Plack et al., 2014; 2016). However, the initial
74 account of cochlear synaptopathy as described in the mouse model may not be translated into an
75 analogous human pathology in a straightforward way (Hickox et al., 2017).

76

77 The evidence for noise-induced cochlear synaptopathy in human listeners is somewhat sparse and

78 inconsistent. Stamper and Johnson (2015a) first provided evidence for reductions in ABR wave I
79 amplitude with greater noise exposure in audiometrically normal human listeners. However,
80 audiograms were only measured up to 8 kHz, and it is possible that high-frequency hair cell loss
81 affected wave I amplitudes in the more exposed listeners (Don and Eggermont, 1978). Furthermore,
82 there was a confound of sex in that the most noise exposed listeners in the cohort were male, and
83 males also tend to show smaller ABR amplitudes due to factors such as skull thickness and head
84 size (Picton et al., 1981; Jerger and Hall, 1980). In a subsequent letter, Stamper and Johnson
85 (2015b) analyzed their data for the highest click level (90 dB nHL) for the two sexes independently.
86 The relation between wave I amplitude and a 12-month noise exposure estimate persisted for
87 females, but not males.

88

89 Recently, Bramhall et al. (2017) reported that non-veteran firearm users and veterans with high
90 levels of noise exposure have reduced wave I amplitudes relative to lower noise exposed veterans
91 and non-veterans without a history of firearm use. All groups had similar otoacoustic emissions and
92 normal audiograms up to 8 kHz, although noise-exposed veterans showed an average elevation of
93 audiometric threshold (averaged across 2000, 3000 and 4000 Hz) of 7.3 dB HL compared to non-
94 veterans. High-frequency audiometric testing (> 8 kHz) was only performed on 59% of participants,
95 and so the contribution of high-frequency hearing loss is uncertain.

96

97 In contrast to these findings of lower wave I amplitudes with greater noise exposure, we conducted
98 a large-scale study (N=126) of young, normal-hearing adults and found no significant relation
99 between lifetime noise exposure and ABR wave I amplitude for either males or females
100 (Prendergast et al., 2017). These findings were replicated in a subsequent study from the same
101 laboratory (Guest et al., 2017). Such negative findings are concordant with Liberman et al. (2016),
102 who reported no significant difference in wave I amplitude between their high- and low-exposure

103 groups. However, they did find a difference in the ratio of the summing potential (SP) relative to
104 the action potential (AP; effectively wave I). A larger SP/AP ratio was found for the high-noise
105 group, due mainly to a higher SP for that group. Since the SP is thought to be generated by the hair
106 cells (Kiang and Peake, 1960), a high SP/AP ratio is consistent with synaptopathy. However, it
107 remains unclear how a loss of cochlear synapses would lead to enhancement of the SP, or how the
108 SP would be affected by the substantial high-frequency audiometric deficit observed in the high-
109 noise group.

110

111 Although the electrophysiological results in humans are mixed, with a number of studies showing
112 discordant findings, it is possible that ABR measures are relatively insensitive to cochlear
113 synaptopathy (Bourien et al. 2014; Prendergast et al., 2017) and that behavioral measures of
114 auditory coding are more sensitive. Furthermore, important questions remain regarding the
115 behavioral consequences of cochlear synaptopathy, and, more generally, regarding whether or not
116 noise exposure is related to behavioral deficits in humans in the presence of normal audiometric
117 thresholds.

118

119 There is existing evidence that noise exposure leads to impaired performance on a range of
120 behavioral auditory tasks for listeners with normal audiograms, although some of these studies are
121 confounded by age and audiometric differences between the groups. Alvord et al. (1983) measured
122 identification scores for words in background noise presented at 60 dB HL. Noise-exposed listeners
123 performed on average 10% more poorly than non-noise-exposed listeners but the groups were not
124 sex-, age- or audiogram-matched. Kujala et al. (2004) used a task of deviant syllable detection and
125 for age- and audiogram-matched groups, found a decrease in performance for noise-exposed
126 listeners. Kumar et al. (2012) compared noise-exposed train drivers with age-matched controls and
127 found that the noise-exposed group had deficits in amplitude modulation detection and speech
128 recognition in background babble using stimuli presented at 80 dB SPL. However, it is not clear

129 from the paper if the groups were audiometrically matched and thus the observed differences may
130 be explained by a more standard, and measurable, loss of audiometric sensitivity. Hope et al. (2013)
131 compared noise-exposed Air Force pilots with non-exposed Air Force administrators and found a
132 deficit in speech-in-noise (vowel-consonant-vowel stimuli) perception thresholds for the noise-
133 exposed group. There was no difference between the groups on other auditory tasks including
134 simultaneous masking, backward masking and frequency discrimination. Though the groups were
135 audiometrically matched to within 2.2 dB, this only included frequencies up to and including 4000
136 Hz.

137

138 Stone et al. (2008) used a task in which normal-hearing listeners were required to discriminate
139 envelopes with different noise statistics at low sensation levels. Noise-exposed listeners showed a
140 deficit in performance compared to non-noise-exposed controls, though these differences were
141 observed at low sensation levels and therefore would not be dependent primarily on low-SR fibers.
142 This evidence is therefore difficult to reconcile with the animal model of noise-induced
143 synaptopathy. Liberman et al. (2016) demonstrated performance deficits on a speech-in-noise task
144 for noise-exposed listeners relative to less exposed controls. However, stimuli were presented at 35
145 dB HL, again suggesting minimal contributions of low-SR fibers to performance as at this sound
146 intensity the high-SR fibers are unlikely to be saturated and thus efficient coding is not primarily
147 dependent on low-SR fibers. Le Prell and Lobarinas (2016) examined measures of recreational
148 noise exposure for groups of audiometrically normal young people, divided based on performance
149 on a measure of word recognition in noise. The groups did not differ significantly in preferred
150 listening level, nor in number of sources of high-level noise they were exposed to. Additionally, no
151 reliable relation was observed between perceptual performance and the reported incidence of
152 temporary threshold shift. Finally, Yeend et al. (2017) report results from a cohort of 30-60 year-old
153 listeners. The primary aim was to characterize the perceptual deficits associated with increased
154 noise exposure. The authors reported no link between lifetime noise exposure and performance on

155 any of the psychophysical or speech tasks. High-frequency hearing thresholds were predictive of
156 speech-in-noise performance.

157

158 In this article we describe a series of behavioral measures that we collected concurrent with the
159 electrophysiological data presented in Prendergast et al. (2017). We consider whether an estimate of
160 lifetime noise exposure is able to predict performance on a range of behavioral tasks for young
161 listeners with normal audiograms. By doing so we hoped to determine which, if any, behavioral
162 tasks may be affected by synaptopathy, based on the assumption that greater lifetime noise exposure
163 is a proxy for increased cochlear synaptopathy. As well as psychophysical tasks used to examine the
164 coding fidelity of a listener's auditory system, we included tasks more representative of real-world
165 listening ability, including speech-in-noise tasks, an auditory localization task, and a musical
166 consonance task. Finally, we included the Speech, Spatial and Qualities of Hearing Scale (SSQ;
167 Gatehouse and Noble, 2004) questionnaire to measure self-reported listening ability. Listeners with
168 normal audiograms often report that they have listening difficulties (e.g. Davies, 1989), and it may
169 be important to capture aspects of more general listening ability, beyond specific laboratory tasks.

170

171 The rationales for the tasks and stimuli chosen are based in part on what is known about noise-
172 induced synaptopathy in the animal model. A compelling overview of how this may express itself in
173 humans is provided by Bharadwaj et al. (2014), in which the authors predict that a loss of low-SR
174 fibers will lead to a reduction in temporal coding, with poorer representations of acoustic signals in
175 the auditory nerve. This would then lead to a reduction in the ability to discriminate subtle timing
176 differences, for example in a frequency discrimination or inter-aural phase discrimination task.
177 Bharadwaj et al. (2015) demonstrated that subcortical EEG measures, the ability of a listener to
178 detect differences in the phase of a stimulus between ears, and amplitude modulation detection
179 performance all co-vary seemingly due to individual differences in temporal coding. Although the
180 study contained a crude measure of noise exposure history, which suggested that noise-exposed

181 participants have weaker evoked responses and elevated behavioral thresholds, the authors were
182 cautious in concluding that noise-induced synaptopathy was the primary factor. In this study we
183 included comparable tasks to ascertain if temporal coding varies as a function of lifetime noise
184 exposure.

185

186 The effects of noise exposure were predicted to be most readily observed in response to high-level
187 stimuli, as these would lead to saturation of high-SR fibers and therefore any differences in residual
188 coding would be carried by the population of low-SR fibers. This approach is based on the low-SR
189 hypothesis, supported by data from Furman et al. (2013), and assumes that this fiber group is
190 critically important to the encoding of high-intensity sounds. Hence, for most tasks we used both
191 low- and high-level conditions in order to provide a differential measure of the effects of
192 synaptopathy. Note that this approach is insensitive to synaptopathy if medium- and high-SR fibers
193 are able to encode high-level sounds by modulating their firing patterns (Young and Sachs, 1979).
194 In addition, noise-induced audiometric hearing loss, caused mainly by damage to the outer hair cells
195 (OHCs), typically manifests in the 3000-6000 Hz region (Toynbee, 1860; McBride and Williams,
196 2001). Therefore, for a number of tasks, stimuli with frequency components in two spectral regions
197 were used, with the assumption that synaptopathy is most likely to occur in the same frequency
198 range as noise-induced outer hair cell dysfunction. Hence, for the psychophysical tasks, we
199 measured performance at 255 Hz and 4000 Hz to provide another differential measure. Differential
200 measures may help to control for the effects of variability between individuals due to factors
201 unrelated to synaptopathy (Plack et al., 2016).

202

203 Finally, musical training is related to enhanced performance on some auditory tasks (Parbery-Clark
204 et al., 2009; Zendel and Alain, 2009). Yeend et al. (2017) reported that sensitivity to temporal fine
205 structure and amplitude modulation was enhanced in musically trained listeners. In order to control
206 for the effects of musical experience in our cohort, we included an estimate of the number of years

207 during which a musical instrument was played regularly.

208

209 **2. Methods**

210 **2.1. Participants**

211 One hundred and thirty-eight participants (82 females), with a wide range of noise exposures, were
212 tested, 123 of whom were also tested as part of an electrophysiological study of noise-induced
213 synaptopathy (Prendergast et al., 2017). Participants were recruited mainly via a publicly available
214 University of Manchester website listing active research projects. Advertisements were also placed
215 in a number of bars and music venues in Manchester city center. All participants exhibited clinically
216 normal audiometric thresholds (see section 2.3). Males had a mean age of 23.3 years (range, 18-36)
217 and females had a mean age of 23.1 years (range, 18-36). The procedures were approved by the
218 University of Manchester Research Ethics Committee and all participants gave informed consent
219 (project number 14163).

220

221 **2.2. Noise exposure**

222 Lifetime noise exposure was estimated using a structured interview developed to assess the
223 effectiveness of the UK noise at work regulations (Lutman et al., 2008). The specific
224 implementation used is described fully by Prendergast et al. (2017). In summary, participants are
225 asked to consider any high-noise (above ~ 85 dBA) environments/activities to which they have
226 exposed themselves with a degree of repeatability over the course of their lifetime. The duration,
227 frequency and level of exposure is estimated from discussion with the participant (including any
228 attenuation from hearing protection used) and entered into the following formula:

229

$$230 \quad U = 10^{(L-A-90)/10} \times Y \times W \times D \times H / 2080,$$

231

232 where U is cumulative noise exposure, L is estimated noise exposure level in dBA, A is attenuation

233 provided by hearing protection in dB, Y is years of exposure, W is weeks of exposure per year, D is
234 days of exposure per week, H is hours of exposure per day, and 2080 corresponds to the number of
235 hours in a working year. One noise exposure unit is equivalent to exposure for 1 year to a working
236 daily level of 90 dBA. For our purposes, we used the raw noise immission units and these were log
237 transformed to produce a normal distribution. Each such logarithmic unit is a factor of 10 in terms
238 of lifetime exposure energy.

239

240

241 **2.3. Pure tone audiometry**

242 Pure tone audiometry was performed for each ear separately at octave frequencies between 250 and
243 8000 Hz in accordance with the British Society of Audiology (2011) recommended procedure.

244 Thresholds were measured using VIASYS GSI-Arrow audiometers coupled to TDH-39P supra-
245 aural headphones, with MX41 cushions. The audiometric criterion for inclusion in the study was
246 audiometric thresholds < 25 dB HL in both ears at all test frequencies.

247

248 High-frequency audiometry was also performed at 16 kHz using a Creative E-MU 0202 USB
249 soundcard. Sounds were played over Sennheiser HDA 200 circum-aural headphones designed for
250 high-frequency audiometry. The sound stimulus was a quarter-octave wide band of noise centered at
251 16 kHz and converted from digital to analog at a sample rate of 48 kHz using a 24-bit depth. Stimuli
252 were 220 ms in duration (including 10-ms raised-cosine ramps) ramps and there was an inter-
253 stimulus interval of 500 ms. A three-alternative forced-choice procedure was used, with a two-
254 down, one-up staircase adaptively setting the stimulus level. Stimulus level was varied
255 arithmetically using a step size of 4 dB for the first four reversals and 2 dB for the following 10
256 reversals. Thresholds were calculated by averaging the levels of the final 10 reversals from a single
257 run.

258

259 Participants were asked if they suffered from tinnitus. If a positive response was given, participants
260 were asked further questions to determine if this constituted prolonged tinnitus and when it was last
261 perceived. If participants reported this percept regularly (at least every month), they were recorded
262 as having tinnitus.

263

264 **2.4. Behavioral tasks**

265 All sounds were presented using a Creative E-MU 0202 USB soundcard and Sennheiser HD650
266 circum-aural headphones. All stimuli were presented diotically, except for the interaural phase
267 difference (IPD) task and the localization task. Many of the behavioral tasks were performed in both
268 a low- and high-frequency region (255 Hz and 4000 Hz respectively, denoted “L” and “H”) and also
269 at a low and high sound intensity (40 and 80 dB SPL, denoted “40” and “80”). This was done to test
270 the specific hypothesis that high-threshold, high-frequency fibers are preferentially affected by
271 lifetime noise exposure. Unless specified, a two-down, one-up adaptive track was used, and the first
272 four reversals were made using one step size and the final 10 using a smaller step size. Thresholds
273 were calculated from the average of the tested parameter values at the final 10 reversals. Each of the
274 four conditions was completed once in a random order in each block of trials. Three blocks were
275 presented for each task. The mean threshold across the three blocks was taken as the final mean for
276 each condition. Where geometric tracking was used, a geometric average of the means was
277 calculated. 10-ms ramps were used to gate the onset and offset of all stimuli, unless otherwise
278 specified, and these are included in all stimulus durations reported.

279

280 For three of the psychophysical discrimination tasks a two-alternative forced-choice paradigm was
281 used in which the listener was asked to detect which of two observation intervals, each consisting of
282 four stimuli (AAAA vs ABAB), contained non-identical stimuli (Hopkins and Moore, 2010). This
283 paradigm has been shown to minimize practice effects (e.g. King et al., 2013). For these tasks, there

284 was a 50-ms silent period between stimuli within one of the two observation intervals, and a 500-ms
285 silent period between observation intervals. Minimal training was given, with the experimenter
286 confirming that the participant understood the task via a brief discussion after hearing the stimuli
287 and by observing two correct responses on a brief practice run. The numbered intervals were
288 visually cued with white lights on the screen for the duration of each stimulus and feedback was
289 given in the form of a red or green light for incorrect and correct responses, respectively.
290 Participants made their responses using numeric buttons on the keyboard. The participant could take
291 a break between adaptive tracks and did not commence another test sequence until they indicated
292 they were ready. Specific details for each of the tasks performed are given in the following sections.

293

294 **2.4.1. Frequency difference limens (FDLs)**

295 The AAAA vs. ABAB paradigm was used. Tones were 200 ms in duration. Stimulus levels of 40
296 and 80 dB SPL were used for each frequency. The low-frequency standard stimulus (A) was a 255-
297 Hz pure tone. For the high-frequency condition, the stimulus was a transposed tone, consisting of a
298 4000-Hz carrier modulated by a half-wave rectified, and low-pass filtered, pure tone (using a
299 fourth-order Butterworth filter with a cutoff frequency of 2040 Hz). Transposed tones were used as
300 they are designed to produce equivalent neural temporal firing patterns in high-frequency spectral
301 regions of the cochlea as occur in low-frequency spectral regions in response to a pure tone
302 (Bernstein and Trahiotis, 2002). For the standard stimulus (A) the frequency of the pure tone
303 modulator was 255 Hz. Note that for the high-frequency condition, the task was modulation
304 frequency discrimination. In both cases, the frequency of the pure tone for the comparison stimulus
305 (B) was higher than that of the standard, and was varied adaptively. The starting difference in
306 frequency was 10% and the frequency of the comparison stimulus was varied geometrically with an
307 initial step size of a factor of 2 and a subsequent step size of $\sqrt{2}$. For the high-frequency conditions,
308 low-pass pink noise was added in order to mask combination tones. The cut-off frequency of the
309 noise band was 2500 Hz and the spectrum level at 1000 Hz was 40 dB below the signal level. Based

310 on estimates of distortion product level by Oxenham et al. (2009), the noise should have masked
311 any combination tones below 2500 Hz.

312

313 **2.4.2. Intensity difference limens (IDLs)**

314 The AAAA vs. ABAB paradigm was used. Tones were 200 ms in duration. Stimuli were pure tones
315 presented at 255 or 4000 Hz, and at two levels (40 and 80 dB SPL for the standard, A, stimuli). The
316 comparison stimulus (B) was higher in level than the standard. The starting Weber fraction was 10
317 dB and was varied arithmetically with an initial step size of 4 dB and a second step size of 2 dB.

318

319 **2.4.3. Interaural phase difference discrimination (IPD)**

320 The AAAA vs. ABAB paradigm was used. Tones were 300 ms in duration (including 50 ms ramps).
321 The low-frequency stimulus was a 255-Hz pure tone, the starting phase of which was varied
322 adaptively for the target tones. For the high-frequency condition, the stimulus was a transposed
323 tone, consisting of a 4000-Hz tonal carrier modulated by a half-wave rectified, and low-pass
324 filtered, 255-Hz pure tone. Stimulus levels of 40 and 80 dB SPL were used for each frequency. For
325 the comparison stimulus (B) the phase of the pure tone or pure-tone modulator was varied
326 adaptively. The starting difference was a positive shift of 30 degrees for stimulus B (stimulus A
327 always had a starting phase of 0) and the phase was varied geometrically using an initial step size of
328 a factor of 1.56 and a second step size of 1.25. The maximum difference between the phase of the
329 reference and target was restricted to 90 degrees. If the maximum difference was reached, the
330 difference remained fixed until two correct responses were given consecutively. For the high-
331 frequency conditions, low-pass pink noise was added in order to mask combination tones. The cut-
332 off frequency of the noise band was 2500 Hz and the spectrum level at 1000 Hz was 40 dB below
333 the signal level.

334

335 **2.4.4. Amplitude modulation detection (AMD)**

336 A three-alternative forced-choice paradigm was used. Stimuli were 200 ms in duration. Carriers
337 were 255-Hz and 4000-Hz pure tones for the low- and high-frequency stimuli respectively, and the
338 target stimulus was a carrier sinusoidally amplitude modulated at 25 Hz. Carrier levels of 40 and 80
339 dB SPL were used for each frequency. The RMS energy was equated across intervals. The starting
340 modulation depth was 50% and this was then geometrically varied according to a two-down one-up
341 track with an initial step size factor of 1.56 and a final step size factor of 1.25. There was a 500-ms
342 inter-stimulus interval between each of the three tones.

343

344 **2.4.5. Digit triplet test (DTT)**

345 In the DTT, the participant is required to identify three spoken digits presented sequentially in a
346 background noise (Smits et al., 2004). The digits were in the range 1-9 and the correct identification
347 of all three was required for a correct response. The digits were voiced recordings from a single
348 speaker taken from McShefferty et al. (2013). The noise was speech-shaped and fixed at each of
349 two levels (40 and 80 dB SPL) while the sound level of the spoken digits was varied. A method of
350 constant stimuli was used, with six repetitions at each of eight signal-to-noise ratios (SNRs). Each
351 SNR / level combination was presented once, in a random order, in each block of trials. Three
352 blocks were presented, and the overall percent correct responses calculated for each condition. The
353 SNRs used were -24 to -3 in steps of 3 dB. The stimulus began with 200 ms of noise before the first
354 digit was presented. There was a 50-ms interval between each of the spoken digits and the noise
355 was continuous. Participants made their response by selecting three tick-boxes covering the range 1-
356 9 using a computer mouse and then confirming their selection. Visual feedback was given in the
357 form of a green (correct) or red (incorrect) light. For each individual, a cumulative Gaussian was
358 fitted to the data to model the distribution and to allow the SNR to be estimated for a range of
359 response rates. The results section uses 25%, 50% and 75% correct points to give an overview of

360 the psychometric function.

361

362 **2.4.6. Co-ordinate response measure (CRM)**

363 In the CRM speech task (Bolia, 2000), the participant is presented with a number of speech
364 utterances of the structure “*Ready <call sign> go to <color> <number> now*”, in which there are
365 eight unique callsigns, four different colors (Blue, Red, White, Green) and the number is in the
366 range 1-4. The participant’s callsign was always “Baron” and they were instructed to listen for the
367 speaker who says “*Ready Baron*” and identify the color and number spoken by that speaker. The
368 gender and identity of the target was changed on each trial and there were four male and four
369 female speakers. Two maskers were presented simultaneously, which were always different
370 speakers and different callsigns, although the color and number could match that of the target. All
371 stimuli were spoken by native British-English speakers (Kitterick et al., 2010).

372

373 The CRM was performed at two sound levels (40 and 80 dB SPL) which defined the level of the
374 combined masker stimuli, and in two different masker configurations; one where the maskers were
375 presented centrally (CRM_c) and one in which they were offset by 60 degrees azimuth on either side
376 of the mid-line (CRM_o). This was achieved by multiplying the acoustic stimuli by head-related
377 impulse responses from the CIPIC database (Algazi et al., 2001). In each trial the target sentence
378 was presented centrally at a sound level which varied trial-by-trial and the maskers were presented
379 at a fixed sound level. A method of constant stimuli was used, with six repetitions at each of eight
380 SNRs. Each offset / level / SNR combination was presented once, in a random order, in each block
381 of trials. Three blocks were presented, and the overall percent correct responses calculated for each
382 condition. The SNRs used were -6 dB to +8 dB in steps of 2 dB for the central condition and -14 dB
383 to 0 dB in steps of 2 dB for the offset condition. Participants indicated their response by clicking on
384 one of 16 buttons on the computer display, arranged in four color-coded columns with each row
385 identified as a separate number. Visual feedback was given in the form of a green (correct) or red

386 (incorrect) light. For each individual, a cumulative Gaussian was fitted to the data to model the
387 distribution and to allow the SNR to be interpolated for a range of response rates. The results
388 section uses 25%, 50% and 75% correct points to give an overview of the psychometric function.

389

390

391 **2.4.7. Localization task (LOC)**

392 The auditory localization task was performed for levels of 40 and 80 dB SPL. A single spoken word
393 (“Tiger” taken from the CRM corpus) was heard in quiet presented over headphones after being
394 multiplied by one of 17 head-related impulse responses (Algazi et al., 2001), intended to make the
395 percept originate from one of the following virtual azimuths: +/- 80, 65, 55, 45, 35, 25, 15, 5 and 0
396 degrees. Each speech token was presented with zero degrees elevation. Participants indicated their
397 response by clicking one of 17 boxes on the computer display laid out schematically in a semi-
398 circle, as if looking down on the participant perceiving the sound source. Each location was
399 presented six times in a single run, and three runs were completed for each sound level. Both the
400 order of the runs and of the stimuli within a run were randomized. No feedback was provided.

401

402 **2.4.8. Musical consonance task (CON)**

403 There is evidence that ratings of the perceived pleasantness of chords are related to the strength of
404 neural temporal coding (Bones et al., 2014), and temporal coding has been linked to synaptopathy
405 (Bharadwaj et al., 2014). Hence a consonance preference task may be effective at identifying
406 temporal coding deficits due to synaptopathy. The stimuli and methodology were based on Bones
407 and Plack (2015). Two-note chords (dyads) were created by combining each of eight complex tones
408 (fundamental frequencies, F0s, of 293.66, 311.12, 329.62, 349.22, 370, 392, 415.3, 440 Hz) with
409 each of 11 higher-F0 tones. Each complex tone contained 20 equal-amplitude harmonics. Each dyad
410 is named after the musical interval between the F0s of the high and low notes (ratios of 1.06, 1.12,
411 1.19, 1.26, 1.33, 1.41, 1.50, 1.59, 1.68, 1.78, 1.89). Dyads were 2 s in duration, including 10 ms

412 raised-cosine onset and offset ramps. Each dyad was preceded by Gaussian noise with the same
413 duration and filtering (low-pass filtered at 6000 Hz). A 500-ms silence separated the noise and the
414 dyad. The purpose of the noise was to prevent trials being influenced by the preceding stimulus and
415 thus biasing the pleasantness judgement of the current trial (McDermott et al., 2010). Listeners were
416 asked to rate how pleasant or unpleasant they found the chord using a seven-point Likert scale (-3 to
417 +3). The harmonics of each note had the same amplitude, and the overall level of each dyad was 80
418 dB SPL.

419

420 **2.4.9. Self-report assessment of hearing ability**

421 The SSQ was used to allow listeners to report their hearing ability in several domains, which are
422 split into three scales; speech, spatial and qualities of hearing. The questionnaire consists of 49
423 questions which describe a listening situation and ask people to rate their listening ability in that
424 situation from 0-10, with a higher number indicating better performance and an improved sense of
425 hearing ability. The SSQ is designed to provide a comprehensive assessment of an individual's
426 perceived ability to hear in the real world. The Speech scale (consisting of 14 items) covers an
427 extensive range of realistic speech contexts that vary in their assumed difficulty. The items cover
428 conditions of competing sound, the number of speakers, and selective attention (attending to one
429 speech stream in a background of many), in an attempt to identify specific listening environments in
430 which the ability to hear speech may be affected. The Spatial hearing scale (consisting of 17 items)
431 addresses direction, distance, and movement discrimination abilities. The Qualities scale (consisting
432 of 18 items) addresses issues related to the ability to segregate sounds, the clarity of sounds, and the
433 demand of listening effort. For each individual, the mean score across all of the items on a scale was
434 taken, which allows all three scales to be plotted on the same axis. This average score for each scale
435 is used as a summary metric for each listener and can be compared to that listeners' noise exposure
436 score to ascertain if there is a relation between the two. A negative relation is predicted, with
437 increasing noise exposure expected to be associated with a decreasing score, which would indicate a

438 decrease in the listener's perceived hearing ability.

439

440 **2.4.10 Musical experience**

441 To estimate the degree of musical experience, we asked those participants who reported having
442 learnt an instrument: "Between what ages did you regularly play?" The total number of years of
443 playing a musical instrument was taken as the metric of musical experience. A subset of participants
444 worked in the music industry as sound engineers/technicians and these participants scored highly on
445 this metric.

446

447 **3. Results**

448

449 Many of the behavioral thresholds were found to be non-normally distributed and so in these cases
450 Spearman's rho was used in order to evaluate the extent to which lifetime noise exposure predicted
451 performance. Due to attrition, the number of participants varies slightly for each task and so the
452 number of participants included is noted for each task. Note that the primary focus here is on the
453 relation of behavioral performance to noise exposure, not on the relations between behavioral
454 measures. Due to the exploratory nature of this study, a large number of comparisons are performed.
455 This approach comes with a multiple comparisons penalty, to the extent that potentially genuine,
456 albeit weak, relations may be discarded. Therefore, no correction for multiple comparisons has been
457 performed and discussion of the results considers any relation which reaches an alpha of 0.05, albeit
458 with appropriate caveats.

459

460 **3.1. Noise exposures**

461 Estimated lifetime noise exposure scores varied with respect to $\log(\text{energy})$ from 0 (a listener with
462 effectively no exposure to sounds with levels estimated to be above 85 dBA) to 2.54. In terms of
463 energy, there was a difference of a factor of 300 between the lowest and the highest noise exposed

464 participants. There was no significant difference between noise exposure scores for males
465 (mean=1.37, s.d.=0.54) and females (mean=1.22, s.d.=0.51): $t(136)=1.63$, $p=0.10$. Therefore, the
466 remaining results for male and female listeners were pooled. Noise exposure is used as the primary
467 predictor variable in the analyses. Fig. 1 shows the distribution of noise exposures for the cohort as
468 a function of age.

469

470 In addition to considering the entire cohort in correlational analyses, it is instructive to examine
471 groups with extreme low and high noise exposure. This division into sub-groups may increase the
472 likelihood of observing the effects of synaptopathy, and provides a concise and clear visual
473 indication of the sensitivity of each measure to noise exposure. Hence, in the figures that follow,
474 we present data for the 25% of the cohort (34 individuals in each group) with the lowest (green
475 open squares) and highest (black filled squares) noise exposure scores. These groups had mean
476 exposures (expressed on a logarithmic scale) of 0.63 (range; 0-0.95) and 1.95 (range; 1.60-2.54),
477 respectively. Across the different tasks, the number of individuals included sometimes changed
478 slightly due to attrition, though the mean exposures were always close to those presented here.

479

480 **3.2. Audiometric data and tinnitus**

481 Fig. 2 shows audiometric data (averaged across the ears) for all listeners, and for the low- and high-
482 exposure groups. There was very little effect of noise exposure on audiometric threshold for
483 frequencies up to 8 kHz, although there was a substantial difference between groups at 16 kHz, with
484 the high-exposure group having poorer hearing thresholds on average. The Pearson correlation
485 between 16-kHz audiometric thresholds and lifetime noise exposure was statistically significant
486 ($r=0.29$; $p<0.001$), as also reported by Prendergast et al. (2017) using a near-identical dataset.
487 Pearson correlation coefficients revealed no significant relation between audiometric threshold and
488 noise exposure at 2000 Hz ($r=0.09$; $p=0.27$), 4000 Hz ($r=0.10$; $p=0.24$), and 8000 Hz ($r=0.02$;
489 $p=0.82$). Musical experience showed no statistically significant relation with any of the audiometric

490 thresholds tested.

491

492 Ten participants reported experiencing prolonged tinnitus. Three of these participants were in the
493 lowest 25% of noise exposures and five in the highest 25%.

494

495 **3.3. Psychophysics (FDL, IDL, IPD, AMD)**

496

497 Fig. 3 shows the results for the four psychophysical experiments: FDL, IDL, IPD and AMD. In each
498 panel, results for all four conditions are shown for each of the two levels and frequencies (L40, L80,
499 H40, and H80, where “L” and “H” refer to low and high frequency, and the number refers to the
500 level in dB SPL). There were no marked differences between the two groups, and small confidence
501 intervals, which suggest that any effects of exposure were small.

502

503 Table 1 shows Spearman correlations for the whole group (with the N for each task indicated)
504 between the noise exposure scores and the thresholds for each of the conditions. Table 1 also shows
505 the correlation of noise exposure with two differential measures; one which contrasts different
506 sound levels in the same spectral region (4000 Hz) and one which contrasts different spectral
507 regions at the same sound level (80 dB SPL). It was predicted that cochlear synaptopathy would be
508 associated with a positive correlation (increasing threshold with increasing noise exposure) in each
509 case, based on the assumption that a positive correlation in the differential measure is caused by an
510 elevation of threshold for high-noise exposed listeners in the H80 condition and equivalence of
511 thresholds across exposure for the lower frequency/level condition. This assumption is premised on
512 the low-SR fibers being primarily affected by noise exposure. There were only weak correlations,
513 and these must be considered with caution as no correction for multiple comparisons was applied,
514 and none of the significant correlations would survive Bonferroni correction.

515

516 The strongest relations across all tasks were in the opposite direction to those predicted. For the
517 FDL task, increasing noise exposure was related to an improvement in performance. This appears to
518 be driven by high-noise participants outperforming low-noise participants in the high-frequency,
519 high-level (H80), condition but performing more poorly in the high-frequency, low-level (H40)
520 condition. For the IPD task there was a weak relation with noise exposure in the predicted direction
521 with the differential measure, computed using H80-L80. This relation was partly driven by higher
522 thresholds for the more exposed listeners in the H80 condition, as predicted, but it was driven more
523 strongly by the noise-exposed listeners outperforming the less exposed in the L80 condition. There
524 was also a weak effect for the IDL task, again for the differential measure computed using the two
525 high-level conditions. This negative relation with noise exposure was driven by the fact that high-
526 and low-noise exposed participants performed comparably in the H80 condition, but there was a
527 decrease in performance with increasing noise exposure for the L80 condition.

528

529 The strongest relation of interest in the tasks presented is that between lifetime noise exposure and
530 AMD, though it was in the opposite direction to that predicted. There was a negative relation
531 between lifetime noise exposure and AMD threshold in the H80 condition; i.e. performance
532 improved as noise exposure increased. The use of the differential frequency measure strengthened
533 the relation, as in the L80 condition there was a slight decrease in performance with increasing
534 noise exposure. The relations need to be validated in different cohorts to establish if they are in fact
535 genuine, weak effects related to lifetime noise exposure.

536

537 Audiometric thresholds at 4000 Hz were found to correlate significantly with performance on the
538 AMD high-frequency conditions, with high thresholds associated with better performance (H40, ρ
539 = -0.25; H80, ρ = -0.31; H80 -H40, ρ = -0.37; all $p < 0.01$). However, the correlations with noise
540 exposure for these conditions showed a similar strength to the original correlations once

541 audiometric sensitivity was controlled for (H40, $\rho = -0.25$; H80, $\rho = -0.31$; H80 -H40, $\rho =$
542 -0.37 ; all $p < 0.01$). No other behavioral measure (including those reported in sections 3.4., 3.5. and
543 3.6) varied significantly as a function of 4000 Hz audiometric threshold.

544

545 **3.4. Speech measures (DTT, CRM)**

546 Fig. 4 shows a summary of performance for each of the three speech tasks used; the DTT, the co-
547 ordinate response measure with central maskers (CRM_c), and the co-ordinate response measure
548 with maskers spatially offset (CRM_o). The SNRs at which 25%, 50% and 75% correct performance
549 was estimated to occur are plotted for the low- and high-noise exposed groups. In each case, the
550 differences between the groups are small.

551

552 Table 2 summarizes the relations between noise exposure and performance across the full group of
553 participants (with N specified for each task). The relations were weak: None of the significant
554 effects would survive correction for multiple comparisons and therefore must be interpreted with
555 caution. The DTT showed a relation between performance and lifetime noise exposure for the
556 differential measures taken at 25% and 50% correct on the psychometric function in the opposite
557 direction to that predicted, i.e. with improving performance (a decrease in SNR) as a function of
558 lifetime noise exposure. The effect was strongest at 25% correct, which was driven by highly noise-
559 exposed listeners performing more poorly than lower noise-exposed listeners at 40 dB SPL and
560 outperforming them at 80 dB SPL (a moderate but insignificant relation). A similar pattern was seen
561 for 50% correct on the psychometric function, although it was weaker and only reached significance
562 for the differential measure.

563

564 The CRM_c task, in which the maskers were presented from the same spatial location as the target,
565 revealed some weak trends. All three points on the psychometric function showed a qualitatively

566 similar trend, with high noise-exposed listeners outperforming the less noise exposed in the 40 dB
567 SPL condition and the groups being largely comparable for the 80 dB SPL condition.
568 There were no significant relations between noise exposure and performance on the CRMo task, in
569 which the maskers were spatially offset. The 25% and 50% values for the CRMo task were
570 extrapolated downwards from the range of SNRs tested (0 to -14 dB) and this extrapolation likely
571 contributed in part to the increased confidence intervals for these values.

572

573 **3.5. Localization task (LOC)**

574 Fig. 5 shows the average localization error in both conditions for the 25% of listeners with the
575 lowest and highest levels of noise exposure (total N = 126; 31 participants in each of the two
576 exposure groups). The results were averaged across the midline, such that each point is the average
577 absolute error for both positive and negative azimuths. A summary error score was calculated for
578 each participant by summing the mean absolute errors for each of the azimuths in order to correlate
579 performance with noise exposure. Spearman's rho indicated no significant relation between noise
580 exposure and localization error for either the 40 dB SPL ($\rho=0.11$, $p>0.05$) or 80 dB SPL
581 ($\rho=0.04$, $p>0.05$) condition, nor was there a relation for the differential measure: the ratio between
582 average errors at 80 and 40 dB SPL ($\rho=-0.02$, $p>0.05$).

583

584 **3.6. Musical consonance (CON)**

585 Fig. 6 shows the average rating for each of the 11 two-note chords for the 25% of highest and
586 lowest noise-exposed participants (total N=125, 31 participants in each of the exposure groups).
587 Using a technique described by Bones and Plack (2015), a consonance preference score was
588 calculated by taking the average z-score for the five most consonant chords and subtracting the
589 average z-score for the five most dissonant chords for each participant. Spearman's rho indicated
590 that this consonance preference score did not vary significantly with noise exposure ($\rho=0.11$, p

591 >0.05). The predicted direction was a reduction in consonance preference as a function of
592 increasing lifetime noise exposure, due to a loss of temporal coding precision, which would mimic
593 that observed in older listeners (Bones and Plack, 2015).

594

595 **3.7. Speech, Spatial, and Qualities of Hearing Scale (SSQ)**

596 Fig. 7 shows the average subjective rating on the three scales of the SSQ for the 25% of lowest and
597 highest noise-exposed participants (total N=135). A high SSQ score indicates good self-perceived
598 hearing abilities. Contrary to the prediction, self-report hearing ability increased slightly with
599 lifetime noise exposure for the Spatial (Spearman's $\rho=0.17$; $p<0.05$) and Qualities scales
600 ($\rho=0.23$; $p<0.01$). No such relation was observed for the Speech scale of the questionnaire. The
601 relation between lifetime noise exposure and SSQ score was statistically significant for two of the
602 three scales. However, this was achieved by virtue of a large sample size and low variability across
603 ratings. The mean difference between the groups was <1 , which is the unit of granularity in the
604 measure. Therefore, although these relations may indicate an underlying difference in perceived
605 hearing ability that is of interest and potentially important to characterize, these differences are not
606 of clinical relevance.

607

608 **3.8. Relation of behavioral measures to musical experience**

609 Musical experience correlated positively with noise exposure ($\rho=0.38$; $p<0.001$). Hence musical
610 experience could have been a confound, with the deleterious effects of noise exposure compensated
611 by the performance benefits associated with musical experience (Parbery-Clark et al., 2009; Zendel
612 and Alain, 2009; Yeend et al, 2017). Table 1 reports two further correlational analyses for the
613 psychophysical measures: one in which partial correlations were performed between performance
614 and lifetime noise exposure, with musical experience controlled. The second analysis is for the
615 correlation of performance and musical experience.

616

617 Of the six correlations with noise exposure, all but one remained of a similar strength after
618 controlling for musical experience. The relation between AMD H80 and noise exposure was
619 markedly reduced, but four of the remaining five AMD conditions remained significant at the 0.05
620 level. The correlations indicate that AMD and FDL performance improved significantly with
621 increased musical training. However, none of the differential measures were significantly correlated
622 with musical experience.

623
624 A similar pattern was seen for the speech tasks (Table 2), with the differential measures for the DTT
625 and CRMc tasks having the strongest correlations with noise exposure once musical experience was
626 controlled. The correlation with musical experience reached significance for the 40 dB SPL
627 condition of the CRMc task (at 25% and 50% correct) but the differential measures did not. The
628 differential measure for 50% correct on the DTT showed a significant correlation with musical
629 experience.

630
631 Scores for the localization and musical consonance tasks showed no significant correlations either
632 with musical experience controlled, or with musical experience on its own. For the SSQ, scores for
633 the three scales were not significantly correlated with musical experience. However, the partial
634 correlations between noise exposure and SSQ score, controlling for musical experience, were 0.12,
635 0.19 (both $p < 0.05$) and 0.23 ($p < 0.01$) for the Speech, Spatial and Qualities components,
636 respectively. This suggests that the relation initially shown between subjective report of hearing
637 ability and noise exposure is not related to the degree of musical experience reported by the listener.

638

639 **3.9. Relation of behavioral measures to electrophysiological measures of synaptopathy**

640 One core assumption of this study was that increased lifetime noise exposure is a proxy for
641 increased levels of synaptopathy. Prendergast et al. (2017) used a largely identical dataset and found
642 no relation between lifetime noise exposure and objective physiological measures of synaptopathy.

643 One reason for this, which was discussed in that paper, is that supra-threshold ABR measures may
644 not be sensitive enough to detect subtle changes in auditory processing. However, it could be argued
645 that better estimates of synaptopathy can be obtained by using the electrophysiological measures,
646 with the assumption that a weaker evoked response is indicative of greater underlying synaptopathy.
647 To address this issue, we looked at how our battery of measures related to two core differential
648 measures of synaptopathy, the wave I/V amplitude ratio and the FFR responses reported in
649 Prendergast et al. (2017). The FFR (expressed in dB SNR) generated in response to a 255-Hz pure
650 tone was used to assess the relation with low-frequency psychophysical conditions. The envelope
651 FFR (expressed in dB SNR), generated in response to the modulated waveform (255 Hz
652 modulation) of a 4000 Hz carrier was used to assess the relation with the high-frequency
653 psychophysical conditions. The differential FFR measure was obtained by subtracting the SNR for
654 the low-frequency FFR from the SNR for the envelope FFR, and this was used to assess any
655 relation with differential behavioral measures. The differential FFR was also used to investigate
656 whether there was any association with performance on the speech, musical consonance,
657 localization, and SSQ measures.

658
659 The wave I/V ratio at 100 dB peSPL showed no significant relation with scores for any of the
660 psychophysical, speech, musical consonance, or localization tasks, or the SSQ measures ($p > 0.05$ for
661 all tests). The correlations of the FFR measures with behavioral performance are reported in Table
662 3. There were no significant relations between the differential FFR measure and the speech, musical
663 consonance or localization tasks, nor the SSQ measure. The correlations with the psychophysical
664 thresholds were generally weak. For all four psychophysical tasks, performance on the L40
665 condition showed a negative correlation with the FFR SNR in response to a 255-Hz pure tone. This
666 association was strongest, and reached significance, for the AMD task. This AMD condition
667 previously showed no relation with noise exposure and the effects of cochlear synaptopathy were

668 expected to be observed in the high-frequency envelope FFR, rather than the low-frequency FFR.
669 None of the differential behavioral measures showed a significant relation with the differential FFR
670 measure.

671

672 **3.10. Relation of behavioral measures to 16-kHz audiometric thresholds**

673 Liberman et al. (2016) suggested that high-frequency audiometry may be a marker for cochlear
674 synaptopathy at lower frequencies. To test this prediction, Spearman's rho correlations were
675 computed between 16-kHz audiometric thresholds and scores for each of the behavioral tasks.

676

677 For the psychophysical tasks all the individual and differential measures were used. The 80 dB SPL
678 condition was used for the speech, localization, and musical consonance tasks. The only task whose
679 scores showed a significant relation with 16 kHz thresholds was AMD. In the H80 condition,
680 performance improved with increasing 16-kHz thresholds ($\rho = -0.25$; $p < 0.01$), although this
681 relation was markedly reduced when a partial correlation was performed which controlled for the
682 audiometric pure tone average at 2000, 4000 and 8000 Hz ($\rho = -0.15$; $p > 0.05$). For the H80 – L80
683 differential measure, the relation was similar, performance improving with reduced 16-kHz
684 audiometric sensitivity ($\rho = -0.26$; $p < 0.01$) and for this condition the relation persisted after
685 controlling for low-frequency audiometric thresholds ($\rho = -0.18$; $p < 0.05$). These relations were in
686 the opposite direction to that predicted on the basis of synaptopathy, as the expected effect of
687 greater noise exposure (and therefore potentially greater synaptopathy) would be to reduce the
688 fidelity of temporal coding and elevate behavioral thresholds. However, it is known that
689 sensorineural hearing loss is often associated with improved AMD thresholds (e.g. Füllgrabe et al.,
690 2003). From this perspective, relating to OHC dysfunction in participants with a high-frequency
691 audiometric loss, the correlations were in the predicted direction. The H80 – H40 differential
692 measure showed a comparable trend, with performance improving with increasing 16-kHz threshold

693 ($\rho = -0.27$; $p < 0.01$) and this trend persisted after correcting for low-frequency audiometric
694 thresholds ($\rho = -0.23$; $p < 0.01$). The H80, and both differential conditions, were the only conditions
695 in which performance on the AMD task varied with high-frequency thresholds. The speech,
696 localization, and musical consonance tasks, in addition to IPD, FDL, IDL, and SSQ, did not show
697 any significant relation with 16-kHz thresholds ($p > 0.05$). The pure tone average of the 2000, 4000
698 and 8000 Hz audiometric thresholds was positively related to audiometric sensitivity at 16 kHz
699 ($r = 0.31$; $p < 0.01$).

700

701 **4. Discussion**

702 The main aim of this study was to establish whether performance on a range of behavioral tasks
703 varies as a function of lifetime noise exposure for young listeners with normal audiograms. Overall,
704 there was no strong evidence that performance is affected by noise exposure. There were some
705 weak trends which may be of interest for further study, but these did not survive correction for
706 multiple comparisons. This study provides further evidence that any effects of cochlear
707 synaptopathy are difficult to observe in young human listeners with normal audiograms.

708

709 **4.1. Psychophysical results**

710

711 The IDL, FDL and AMD thresholds are consistent with those in the literature for normal-hearing
712 listeners (e.g. Viemeister and Bacon, 1988; He et al., 1979; Füllgrabe et al., 2003; Moore and Ernst,
713 2012). Whilst the IPD thresholds for the transposed stimuli are larger than those reported by
714 Bernstein and Trahiotis (2002), they are comparable with IPD thresholds reported by Bharadwaj et
715 al. (2015).

716

717 The basic psychophysical results indicate some weak relations of potential interest, although these
718 are difficult to explain, as performance improved as a function of noise exposure for some

719 conditions and declined for others. These contradictions were found both across conditions of the
720 same task and across the different tasks. The strongest effects occurred for the differential measures,
721 which attempt to account for some of the inherent variability across different listeners. Therefore,
722 future investigations of sub-clinical hearing deficits, which are not readily identified from
723 audiometric testing, may benefit from differential measures in order to reduce the impact of
724 individual differences on performance (Plack et al., 2016). It must be noted however, that the
725 differential measure based on level assumes that synaptopathy affects one condition (H80, in the
726 context of the current study) and does not affect the lower-level condition (H40), which can then act
727 as a within-subject control. Such an assumption is based on the evidence that low-SR fibers are
728 primarily affected by noise exposure (Furman et al., 2013). If a specific fiber group is not targeted
729 in this way in humans, then the results obtained with a differential measure based on level become
730 more difficult to interpret.

731

732 The condition with the strongest relation with noise exposure was AMD for the high carrier
733 frequency and high carrier level. However, this relation was counter to the predicted direction, as
734 performance improved with increasing noise exposure. Similar effects have been reported in the
735 literature when quantifying the modulation detection sensitivity of hearing-impaired listeners with a
736 sensorineural hearing loss. Moore et al. (1996) reported that listeners with unilateral sensorineural
737 hearing loss perceived enhanced envelope fluctuations in the impaired ear relative to the near-
738 normal ear, possibly due to the loss of cochlear compression associated with OHC dysfunction.
739 Also, Kale and Heinz (2010) reported enhanced envelope coding in auditory-nerve-fiber responses
740 from noise-exposed chinchillas with permanent sensorineural hearing loss. Therefore, the relation
741 between AMD performance and noise exposure may actually be driven by subtle differences in
742 OHC function, an interpretation supported by the fact that 16-kHz thresholds were also related to
743 AMD performance. Elevated thresholds at 16 kHz may be an early marker for sub-clinical OHC

744 dysfunction in the standard audiometric range which is not detectable using pure-tone audiometry,
745 although there was no effect of exposure on transient-evoked otoacoustic emission amplitudes,
746 measured up to 4000 Hz, in the present cohort (Prendergast et al., 2017). Such an explanation would
747 highlight the need to reconsider how we define “normal” hearing for the purposes of research
748 studies and may have interesting implications for future investigations of sub-clinical processing
749 deficits, but would contribute little to our understanding of noise-induced cochlear synaptopathy.

750

751 **4.2. Speech measures and self-report**

752 The DTT and the CRMc results both revealed weak relations with noise exposure that were again
753 non-significant after correction. However, for the CRMc task, the relation with noise exposure was
754 primarily observed for the 40 dB SPL condition, showing an improvement in performance with
755 increasing noise exposure. This is opposite to the effect reported by Liberman et al. (2016) for their
756 low-level speech task. The DTT showed different effects across the two sound levels, with
757 increasing noise exposure relating to decreasing performance at 40 dB SPL and increasing
758 performance at 80 dB SPL. The effects for the DTT occurred at 25% and 50% correct on the
759 psychometric function. It is possible that the effects of synaptopathy are more apparent in difficult
760 listening conditions, which would be concordant with Liberman et al. (2016), who used both time
761 compression and reverberation to increase the difficulty of the task and exacerbate the differences
762 between low and high noise exposed individuals. This notion is also supported by recent behavioral
763 data collected in rats. Lobarinas et al. (2017) found a reduction in the ability to detect a narrowband
764 of noise presented in an ongoing background noise after exposure to intense (109 dB SPL) noise.
765 This was associated with a supra-threshold decrease in wave I amplitude, consistent with a loss of
766 cochlear synapses. However, the behavioral reduction in sensitivity was only observed for the most
767 challenging condition tested (20 dB SNR).

768

769 **4.3. Effects of musical experience**

770 FDL and AMD thresholds were found to vary strongly with musical experience. However, this was
771 only seen when looking at individual conditions, and none of the differential measures showed such
772 a relation. The partial correlations, which controlled for musical experience, resulted in a weaker
773 relation between noise exposure and performance for a number of the individual psychophysical
774 tasks. However, the differential measures resulted in more robust correlations, as the partial
775 correlations controlling for musical experience were comparable in magnitude to the initial
776 correlation with noise exposure.

777

778 Performance on the speech tasks was not clearly related to musical experience, with only the 40 dB
779 SPL CRM task with central maskers showing a clear relation between years of musical training and
780 performance. In a pattern similar to that seen for the psychophysical tasks, when controlling for
781 musical experience in a partial correlation, the coefficients decreased in magnitude but the
782 differential measures remained largely unaltered, and still showed a weak, but significant
783 correlation. For the SSQ estimate of hearing ability, the correlations with noise exposure were
784 unchanged or increased after musical experience was controlled.

785

786 To summarize, the data presented here are consistent with recent work by Yeend et al. (2017) in that
787 a participant's degree of musical training is predictive of their performance on a number of
788 psychophysical and speech-in-noise tasks. This adds further complexity to a series of parameters
789 which are already difficult to delineate; those with high-degrees of noise exposure tend to be older,
790 possibly have poorer high-frequency hearing, and are also more likely to have musical training
791 which leads to enhanced performance on a number of auditory tasks. The data presented in the
792 current manuscript highlight the value of using a differential measure of performance, as it is these
793 measures which are largely unchanged after controlling for musical experience. Using a differential
794 estimate of performance in an individual may control for musical experience and allow a more
795 direct measure of the effects of noise exposure.

797 **4.4 Relation of behavioral measures to electrophysiological measures**

798 Prendergast et al. (2017) reported, in a dataset largely overlapping with the current cohort, no clear
799 changes in ABR or FFR as a function of lifetime noise exposure. The estimate of lifetime noise
800 exposure is sub-optimal, but does appear to accurately differentiate those with high levels of noise
801 exposure from those with much lower exposure. We approached the current study with the
802 hypothesis that noise-induced synaptopathy may be too subtle to detect using auditory evoked
803 potentials, and that behavioral changes may be more readily observed. Therefore, we maintained the
804 assumption that greater lifetime noise exposure is a legitimate proxy for an underlying loss of
805 cochlear synapses. A counter-argument would be that electrophysiological measures of auditory
806 function are a better proxy for underlying cochlear synaptopathy. Such an approach would posit that
807 those with weaker ABRs and FFRs have sustained a loss of cochlear synapses which accounts for
808 this altered response and thus they should also exhibit poorer behavioral performance. However, the
809 wave I/V ratio was found not to be predictive of performance on any of the tasks used in this study.
810 The strength of these correlations was generally weaker than those for performance versus noise
811 exposure. The FFR was found to be weakly predictive of performance when single conditions were
812 considered separately and this was for the low-frequency FFR and not the envelope FFR for the
813 high frequency region. The differential FFR was not predictive for the differential behavioral
814 conditions. Hence, using the electrophysiological metrics as a marker for synaptopathy did not
815 provide any further insight into the relation of synaptopathy to behavioral measures.

816

817 It has been reported previously that the strength of auditory evoked potentials in an individual is
818 predictive of performance on psychophysical tasks for normal-hearing listeners (e.g., Bones et al.,
819 2014; Bharadwaj et al., 2015). Such a relation, with stronger evoked responses being concordant
820 with better behavioral performance, is consistent with temporal coding precision being crucial for
821 accurate auditory perception. However, these results were not replicated in the present study.

822 Furthermore, if such measures are to be used to better understand noise-induced cochlear
823 synaptopathy, they must in some way be linked to the noise exposure history of the individual. The
824 present approach assumes a simple relation between noise exposure and behavioral thresholds. The
825 interpretation is complicated if different listeners have different degrees of susceptibility to
826 suffering physiological damage from acoustic trauma. It may also be the case that an acoustic event
827 is more damaging depending on when in the lifetime it occurs. It is currently unknown whether
828 such factors affect the manifestation of cochlear synaptopathy in humans.

829

830 **4.5. Can noise-induced synaptopathy in humans with normal audiograms be disregarded?**

831 The lack of an effect of noise exposure on behavioral performance is consistent with Prendergast et
832 al. (2017) and Guest et al. (2017), who found no systematic changes in the ABR or the frequency-
833 following response as a function of noise exposure. The current study, using a wide range of
834 behavioral measures, further supports the idea that the amount of cumulative lifetime exposure to
835 high intensity sounds is not related to meaningful changes in auditory perception in young,
836 audiometrically normal adults. However, it is possible that behavioral performance is relatively
837 insensitive to synaptopathy. Oxenham (2016) applied a theoretical model based on signal detection
838 theory to demonstrate that a 50% loss of synapses would lead to a decrease in d-prime on a typical
839 psychophysical task by a factor of $\sqrt{2}$, which is close to the limits of test sensitivity, and well within
840 the range of expected variability across audiometrically normal young adults. This analysis suggests
841 that, even if substantial synaptopathy occurs, it may be difficult to measure its effects on perception.
842 There are also some potential limitations in our methodology that should be considered. One
843 possible limitation is that the stimuli for the four psychophysical tasks were narrowband, and hence
844 for the high-level conditions, off-frequency listening (particularly on the high-frequency side of the
845 excitation pattern) may have contributed to performance. This may have reduced the impact of low-
846 SR fiber loss by recruiting unsaturated high-SR fibers. Another possibility, discussed by Prendergast

847 et al. (2017), is that our retrospective self-report measure of noise exposure is too unreliable to
848 distinguish individuals in terms of potential synaptopathy. However, as we argued previously, the
849 differences in estimated exposure between the lowest and highest exposed were so great that it is
850 unlikely that meaningful effects were washed out by imprecision in the estimates. In addition,
851 essentially the same noise measure was significantly predictive of tinnitus in a recent study (Guest
852 et al., 2017), even though the range of exposures and number of participants were smaller than in
853 the present study. This suggests that the measure is sufficiently sensitive to distinguish between
854 participants in terms of exposure.

855

856 Despite these caveats, our findings across the three studies from our laboratory to date are
857 consistent with the hypothesis that noise-induced cochlear synaptopathy is insignificant in young
858 humans with normal audiograms. In animal models, it is possible to titrate the noise exposure so as
859 to deliver the maximum intensity possible without permanent threshold shift. The exposures
860 encountered by humans are not so precise, and it may be that exposures sufficient to significantly
861 reduce the number of cochlear synapses are also likely to lead to a loss of OHC function and an
862 elevation of audiometric thresholds, particularly at high frequencies. Dobie and Humes (2017)
863 discussed the difficulties involved in extrapolating the exposure levels used in the animal work to
864 the human listener. They used historical evidence from human studies, in which very intense
865 laboratory exposures were used, and the degree of temporary threshold shift as a proxy for damage
866 to the auditory system. They argued that human listeners require much higher exposures than
867 rodents to produce equivalent damage. The studies that have reported a decrease in wave-I ABR
868 amplitude with noise exposure (Bramhall et al., 2017) and an increase in SP/AP ratio (Liberman et
869 al., 2016) also reported audiometric differences between the groups. As discussed previously, there
870 are differences in sensitivity in the 3000-6000 Hz range in the Bramhall et al. (2017) study.
871 Liberman et al. (2016) reported significant differences in audiometric sensitivity between low and

872 high exposure groups at 10 kHz and above, and a non-significant difference between the two groups
873 at 8 kHz.

874

875 There are two competing hypotheses regarding the relation of audiometric loss to the differences in
876 ABR waveforms between the exposure groups observed in some studies. The first is that high-
877 frequency threshold elevations, and perhaps mild low-frequency (<8 kHz) threshold elevations, are
878 markers for synaptopathy, and that the electrophysiological effects are a direct result of noise-
879 induced synaptopathy. A second hypothesis is that the electrophysiological effects and the high-
880 frequency audiometric deficits share the same cause: basal hair cell dysfunction, as opposed to
881 cochlear synaptopathy at lower frequencies. As Liberman et al. (2016) suggest, the use of high-
882 frequency masking noise to remove the contribution from basal regions when making ABR
883 recordings may help to differentiate between these hypotheses.

884

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891

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1117 **Figure Captions:**

1118 Fig. 1. Noise exposure scores as a function of age for 137 participants. The regression line is plotted
1119 with the Pearson correlation coefficient shown in the text (* = 0.05, **= p<0.01).

1120

1121 Fig. 2. Pure tone audiometric thresholds (averaged across ears and listeners) are shown, with 95%
1122 confidence intervals, for the whole group and for the 25% of participants with the highest and
1123 lowest noise exposures.

1124

1125 Fig. 3. The four panels show the results of the four psychophysical tasks: Frequency difference
1126 limens (FDL), intensity difference limens (IDL), interaural phase difference discrimination (IPD),

1127 and amplitude modulation detection (AMD). Mean thresholds and 95% confidence intervals are
1128 plotted for the 25% of participants with the lowest and highest lifetime noise exposures for the four
1129 conditions of each task.

1130

1131 Fig. 4. Mean thresholds (and 95% confidence intervals) are shown for the DTT, CRMc and CRMo
1132 speech tasks. The SNRs required for 25%, 50% and 75% correct on the psychometric function are
1133 plotted for the 25% of participants with the highest and lowest noise exposures in black (closed) and
1134 green (open) symbols, respectively.

1135

1136 Fig. 5. Mean localization error (and 95% confidence intervals) for the 25% of participants with the
1137 lowest and highest noise exposures (green and black lines, respectively).

1138

1139 Fig. 6. Mean pleasantness ratings are shown (along with 95% confidence intervals) for the 11 dyads
1140 in the consonance task. Results for the 25% of listeners with the lowest and highest lifetime noise
1141 exposures are plotted in green and black, respectively.

1142

1143 Fig. 7. Mean ratings (and 95% confidence intervals) for the Speech, Spatial, and Qualities scales of
1144 the SSQ. Results for the 25% of listeners with the lowest and highest lifetime noise exposures are
1145 shown by green open squares and black solid squares, respectively.

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1150 **Tables**

1151 **Table 1. Spearman's rho coefficients are shown for the relation between thresholds for each of**
 1152 **the four psychophysical tasks (and the two differential measures) and lifetime noise exposure,**
 1153 **lifetime noise exposure controlling for musical experience, and musical experience. Conditions**
 1154 **are labelled with the letter denoting frequency [(L)ow or (H)igh] and the numeric value**
 1155 **indicating sound level (40 or 80, respectively). Positive correlations indicate results in the**
 1156 **predicted direction (worse performance with increasing noise exposure) for the correlation**
 1157 **and partial correlation with noise exposure. For musical experience, negative correlations**
 1158 **indicate results in the predicted direction (better performance with increasing musical**
 1159 **training). * = $p < 0.05$; ** = $p < 0.01$ (uncorrected).**

1160

Task (N)	Condition					
	L40	L80	H40	H80	H80 – L80	H80 – H40
Correlation with noise exposure						
FDL (138)	-0.11	-0.09	0.14	-0.13	0.03	-0.23 **
IPD (138)	-0.05	-0.16	0.03	0.08	0.19 *	0.04
IDL (134)	-0.02	0.11	0.02	-0.06	-0.17 *	-0.09
AMD (133)	0.02	0.08	-0.03	-0.20 *	-0.24 **	-0.21 *
Correlation with noise exposure, controlling for musical experience						
FDL (138)	-0.01	0.15	0.06	-0.04	-0.03	-0.19*
IPD (138)	-0.04	0.07	-0.10	0.10	0.17*	0.04
IDL (134)	0.01	0.09	0.16	-0.03	-0.16	-0.09
AMD (133)	-0.11	0.04	0.16	-0.11	-0.21*	-0.18*
Correlation with musical experience						
FDL (138)	-0.26**	-0.36**	0.00	-0.22**	0.16	-0.16
IPD (138)	-0.05	-0.16	-0.08	-0.03	0.10	-0.00
IDL (134)	-0.08	-0.10	-0.19*	-0.23**	-0.07	-0.01
AMD (133)	-0.23**	-0.19*	-0.19*	-0.26**	-0.13	-0.12

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1163 **Table 2. Spearman's rho coefficients are shown for the relation between threshold on the speech tasks (and the differential measure) and**
 1164 **lifetime noise exposure, lifetime noise exposure controlling for musical experience, and musical experience. Otherwise as Table 1.**

Task (N)	Correlation with noise exposure			Correlation with noise exposure, controlling for musical experience			Correlation with musical experience		
	40 dB SPL	80 dB SPL	80 – 40 dB SPL	40 dB SPL	80 dB SPL	80 – 40 dB SPL	40 dB SPL	80 dB SPL	80 – 40 dB SPL
DTT (139)									
75%	0.12	-0.08	-0.11	0.10	-0.05	-0.08	0.08	-0.09	-0.10
50%	0.16	-0.12	-0.21 *	0.12	-0.09	-0.16	0.11	-0.09	-0.17 *
25%	0.17 *	-0.13	-0.24 **	0.14	-0.05	-0.20 *	0.10	-0.07	-0.14
CRMc (136)									
75%	-0.19 *	-0.01	0.09	-0.14	0.03	0.07	-0.15	0.10	0.04
50%	-0.18 *	0.02	0.21 *	-0.11	0.03	0.19 *	-0.20 *	-0.03	0.10
25%	-0.15	-0.06	0.21 *	-0.08	0.09	0.19 *	-0.19 *	-0.04	0.10
CRMo (136)									
75%	-0.06	-0.11	-0.09	-0.02	-0.08	-0.10	-0.10	-0.07	-0.01
50%	-0.11	-0.09	-0.12	-0.05	-0.09	0.15	-0.16	-0.02	0.05
25%	-0.11	-0.10	-0.09	-0.05	0.11	0.11	-0.15	0.03	0.03

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1170 **Table 3. Spearman's rho coefficients are shown for the relation between the FFR measures and threshold for each of the behavioral tasks.**
1171 **Conditions are labelled with the letter denoting frequency [(L)ow or (H)igh] and the numeric value indicating sound level (40 or 80 dB SPL,**
1172 **respectively). For the L40 and L80 conditions, correlations were with the 255-Hz pure-tone FFR. For the H40 and H80 conditions, correlations**
1173 **were with the envelope FFR for a 4000-Hz carrier amplitude modulated at 255 Hz. For the differential measures (H80-L80, and H80-H40),**
1174 **correlations were with the differential FFR measure (envelope FFR minus pure-tone FFR). In each case, the predicted relation between the**
1175 **FFR measure and performance is a negative one. Those with noise-induced synaptopathy are expected to have lower FFR scores and poorer**
1176 **(higher) psychophysical thresholds. * = $p < 0.05$; ** = $p < 0.01$ (uncorrected).**

1177

	Condition					
Task (N)	L40	L80	H40	H80	H80 – L80	H80 – H40
FDL (123)	-0.08	0.00	-0.06	0.08	-0.03	0.09
IPD (123)	-0.12	0.05	0.04	-0.06	-0.15	-0.13
IDL (119)	-0.08	-0.15	-0.09	0.04	-0.01	0.00
AMD (119)	-0.19 *	-0.06	0.04	0.15	0.12	0.11

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1184 Figure 1

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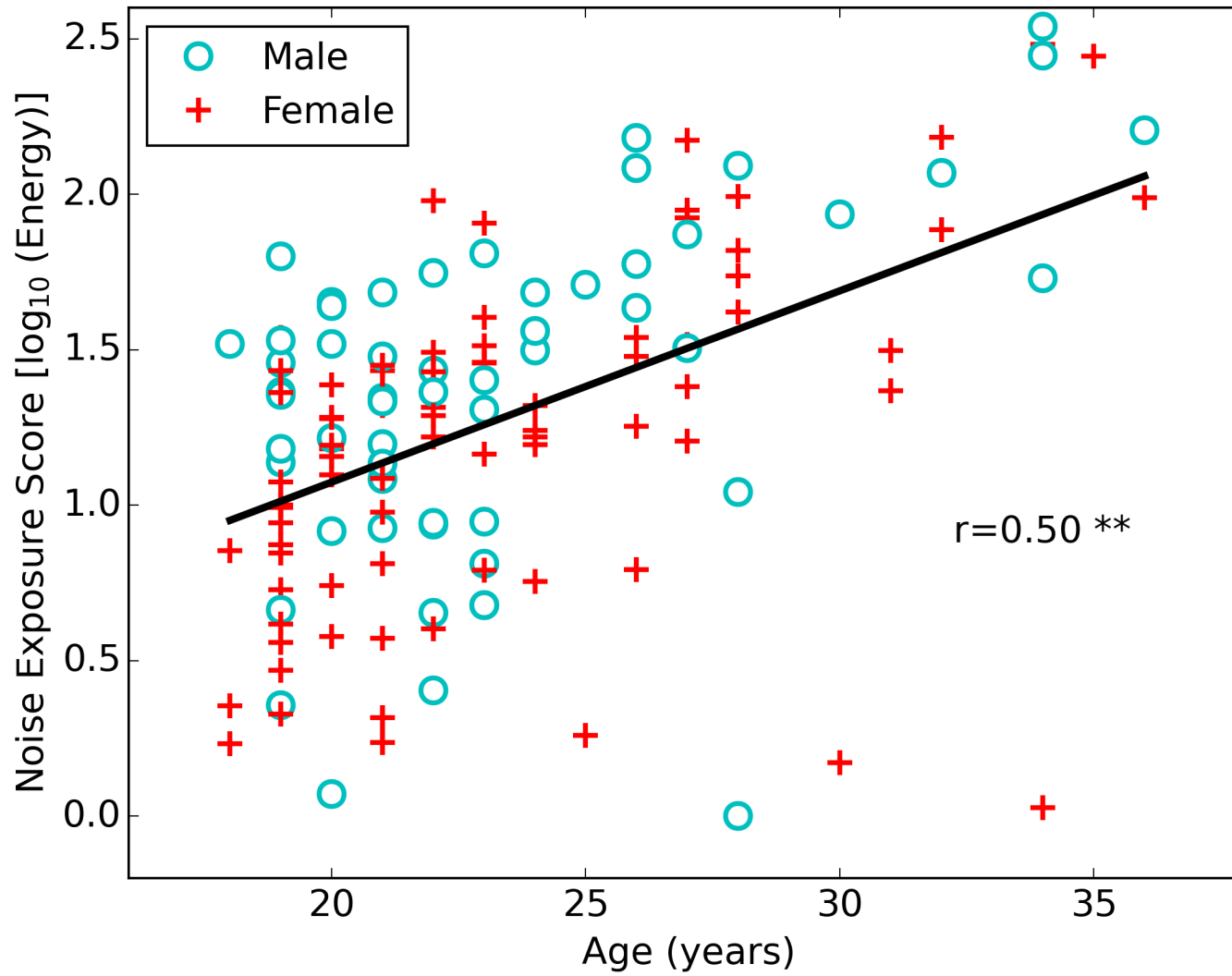
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1205 Figure 2

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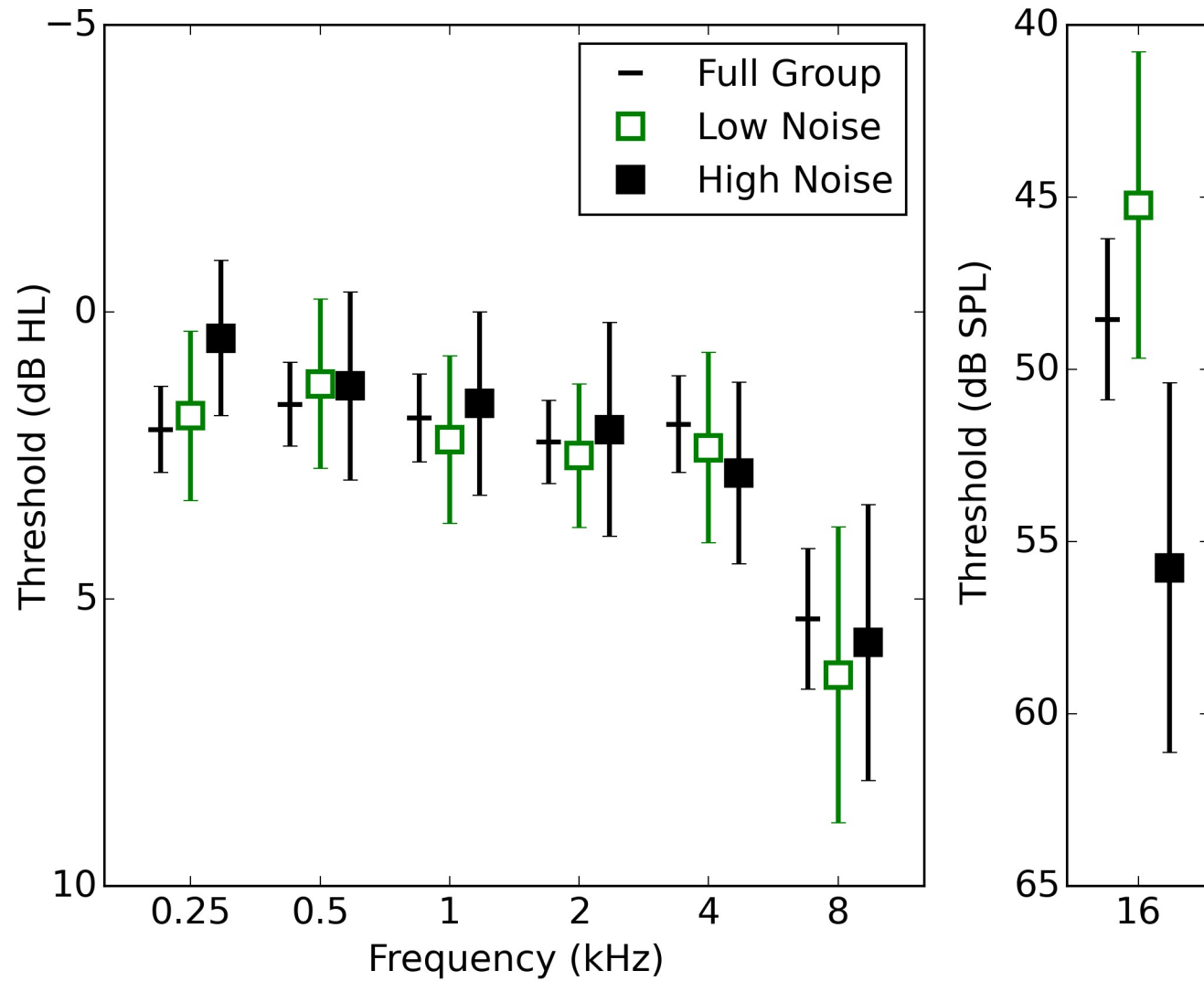
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1226 Figure 3

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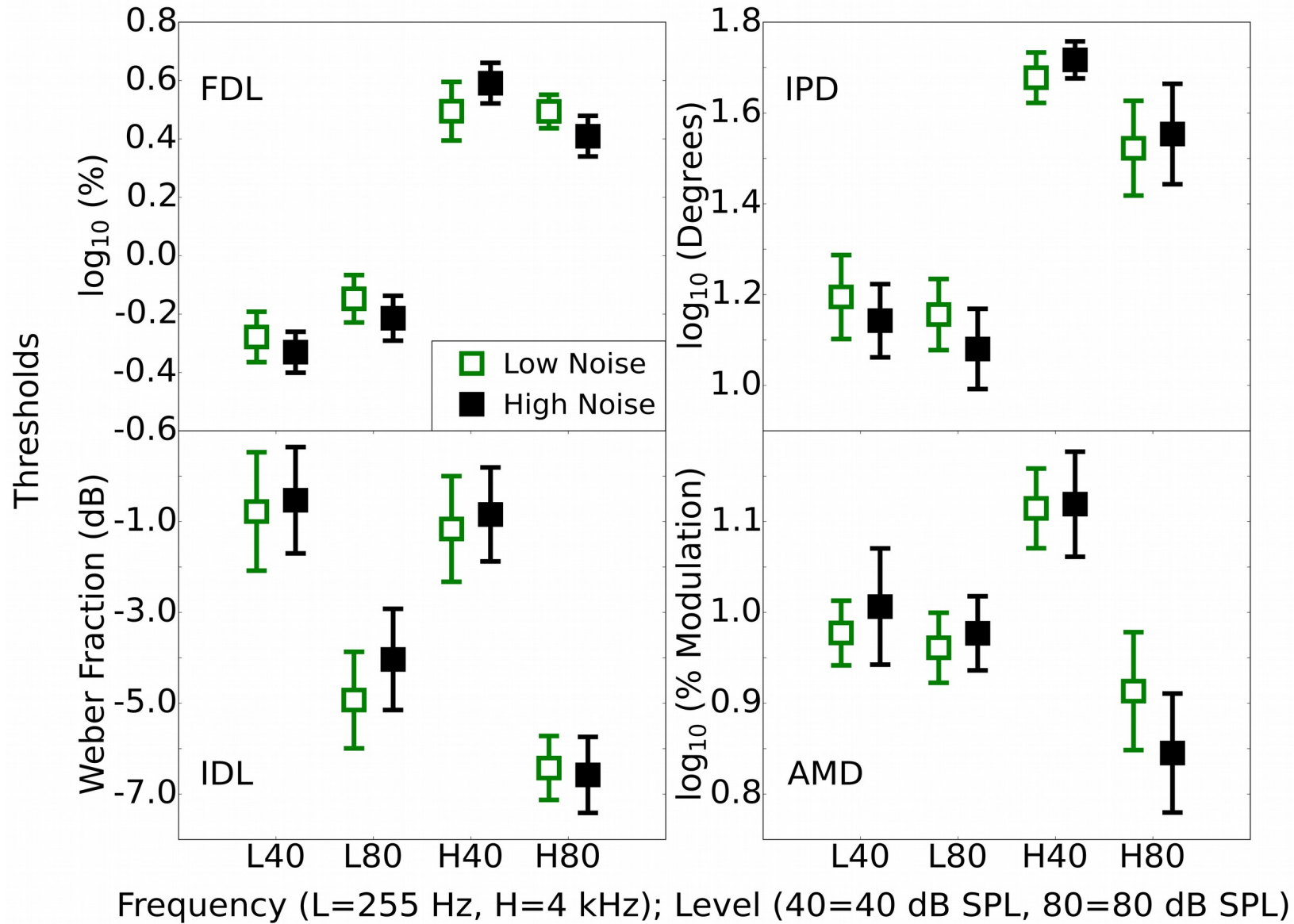
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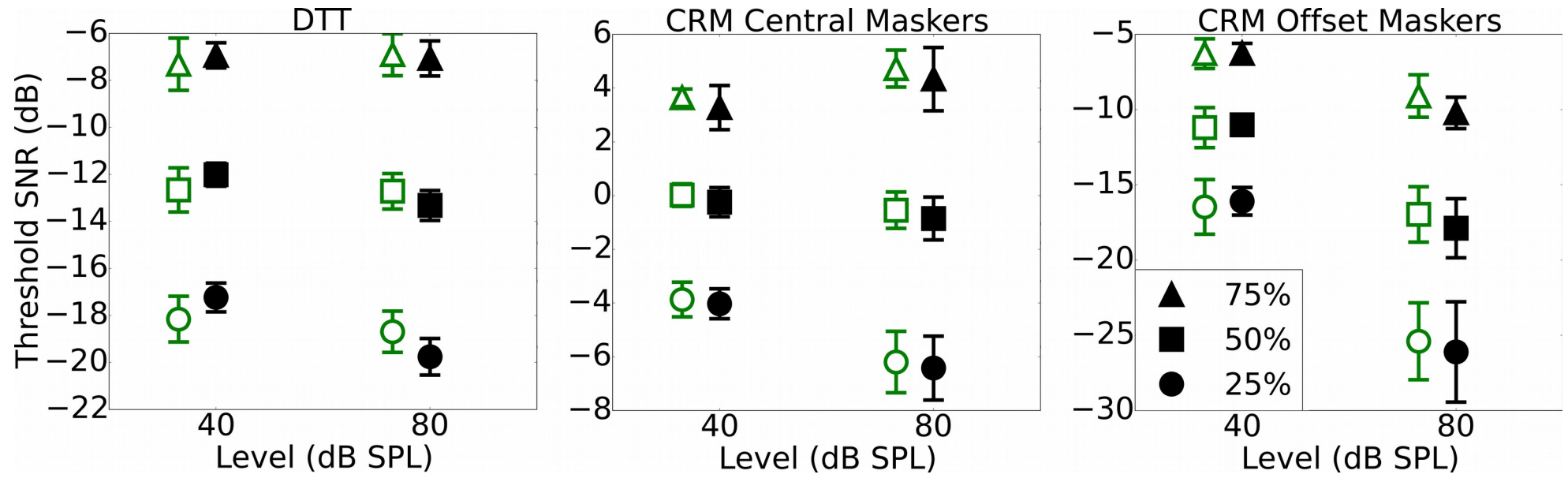
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1247 Figure 4

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1258 Figure 5

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