1 **Title:** Musical experience partially counteracts temporal speech processing deficits in mild cognitive
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44 **ABSTRACT**

45 Mild cognitive impairment (MCI) commonly impacts older adults resulting in more rapid cognitive and

46 behavioral declines than typical aging. Individuals with MCI can exhibit impaired receptive speech

47 abilities that may reflect neurophysiological changes in auditory-sensory processing prior to usual

48 cognitive deficits. Benefits from current interventions targeting communication difficulties in MCI are

49 limited. Yet, neuroplasticity associated with musical experience has been implicated in improving neural

50 representations of speech and offsetting age-related declines in perception. Here, we asked whether these

51 experience-dependent effects of musicianship might extend to aberrant aging and offer some degree of

52 cognitive protection against MCI. During a vowel categorization task, we recorded single-channel EEGs

53 in older adults with putative MCI to evaluate speech encoding across subcortical and cortical levels of the

54 auditory system. Critically, listeners varied in their duration of formal musical training experience (0-21

55 years). Older musicians exhibited sharpened temporal precision in auditory cortical responses suggesting

56 musical experience produces more efficient processing of acoustic features by offsetting age-related

57 neural delays. Additionally, we found robustness of brainstem responses predicted severity of cognitive

58 decline suggesting early speech representations are sensitive to pre-clinical stages of cognitive

59 impairment. Our preliminary results extend prior studies by demonstrating positive benefits of musical

60 experience in older adults with emergent cognitive impairments.

61

62 **Keywords:** auditory event-related potentials (ERPs); brainstem frequency-following response (FFR);

63 categorical perception (CP); experience-dependent plasticity; cognitive aging; music-to-language transfer

64 effects

65 **INTRODUCTION**

66   Cognitive aging results in both structural and functional changes throughout the central auditory

67 pathway[1-3] as well as declines in associated auditory processing skills necessary for effective speech

68 comprehension[4,5]. Older adults demonstrate less precise and altered neural encoding of speech which has

69 been attributed to slowed neural conduction times, poor neural synchrony, and reduced inhibitory

70 processes[6-8]. Senescent neural changes parallel perceptual difficulties commonly reported by aging adults

71 in clinical settings. Older adults often exhibit deficits in temporal processing which is vital for accurate

72 speech perception particularly in challenging listening conditions such as noisy or reverberant

73 environments[5,9].

74   Aging effects are further exacerbated in those with mild cognitive impairment (MCI)[10], a form of

75 atypical cognitive aging causing memory lapses in short-term recall and/or impaired critical thinking and

76 decision-making skills[11]. Because individuals with MCI are also at higher risk of progressive cognitive

77 deficits with potential conversion from MCI to Alzheimer's disease or other forms of dementia[11], early

78 identification and intervention is vital. While most current diagnostic and intervention techniques focus

79 on aspects of cognitive function, recent studies reveal MCI alters early sensory processing by

80 compounding the auditory perceptual deficits due to typical aging[12-14]. Thus, in addition to the more

81 commonly noted cognitive issues, there is emerging evidence that MCI is associated with early declines

82 in *auditory-sensory* processing including complex expressive and receptive language processing[15] and

83 even how accurately the brain encodes speech itself[12,16]. In this vein, neuroimaging techniques and

84 complex speech perception tasks may aid in the early detection of neurocognitive changes associated with

85 MCI.

86   Current MCI treatments do little to slow disease progression and primarily address associated

87 symptoms or focus on improving quality of life[11,17]. Interventions targeting communication difficulties in

88 this population provide limited benefit for improving language or cognitive skills[15]. However, studies

89 show musical experience may counteract age-related declines by robustly enhancing the neural encoding

90 of speech at both subcortical and cortical levels of the central auditory system[18-20]. Individuals with

91 musical experience demonstrate stronger cognitive abilities (i.e., working memory and attentional

92 control[21,22]), higher fidelity neural encoding of acoustic features[23,24], and improved neural timing

93 precision[18,25], all of which contribute to enhanced speech perception abilities. Nevertheless, while

94 musicianship might help counteract normal age-related declines in speech processing[19,26], it remains

95 unclear whether such experience-dependent advantages transfer to populations with suspected cognitive

96 impairments. A critical demonstration of music's benefits on the aging brain is to confirm whether similar

97 neural enhancements occur in at risk populations or those with borderline neurocognitive performance

98   (e.g., MCI). This would provide novel evidence that music engagement might bolster communication

99   skills at early stages and decelerate the progression of cognitive aging.

100   To this end, the current study aimed to investigate the impact of musical experience on speech

101   encoding in individuals with putative MCI. We measured brainstem frequency-following responses

102   (FFRs) and cortical event-related potential (ERPs) in older adults with varying degrees of musical

103   experience who were identified as being at risk for MCI based on cutoff-level performance on

104   neurocognitive assessments. Our findings reveal that abnormally large brainstem FFRs predict severity of

105   cognitive impairment and may serve as an objective marker for MCI severity. We further show musical

106   experience mitigates some age-related delays in neural speech encoding in this at-risk population, paving

107   the way for future translational music interventions in older populations.

RESULTS

109   We evaluated a sample of seventeen older adults ($M \pm SD$, 71.1 ± 8.0 yrs; range: 52-86 yrs) who

110   demonstrated borderline to slight degrees of cognitive impairment as assessed by the Montreal Cognitive

111   Assessment (MoCA). We separated participants into groups based on their extent of musical experience.

112   Individuals with no prior musical training were included in the nonmusician group (NM; $n = 8$) while

113   those with *any* prior musical training were included in the musician group (M; $n = 9$). Participants

114   performed a categorical perception task in which they were presented with tokens from a synthetic five-

115   step vowel continuum spanning from /u/ to /a/ (hereafter "vw 1-5"). Using a forced choice task,

116   participants identified each vowel token as /u/ or /a/ while we recorded subcortical FFRs and cortical

117   ERPs simultaneously using a single-channel montage.

Cognitive function

119   Figure 1 depicts the level of cognitive function for each group as measured by the MoCA. The

120   groups did not differ in MoCA after controlling for age, hearing sensitivity, and education ($F_{1,11} = 1.24$, p

121    $= 0.29$; Fig. 1a). Yet, cognitive function declined with increasing age in both groups ($F_{1,11} = 8.80$, $p =$

122   0.01, $\eta_p^2 = 0.44$; Fig. 1b). Though Ms appeared to have a slower rate of age-related cognitive decline, the

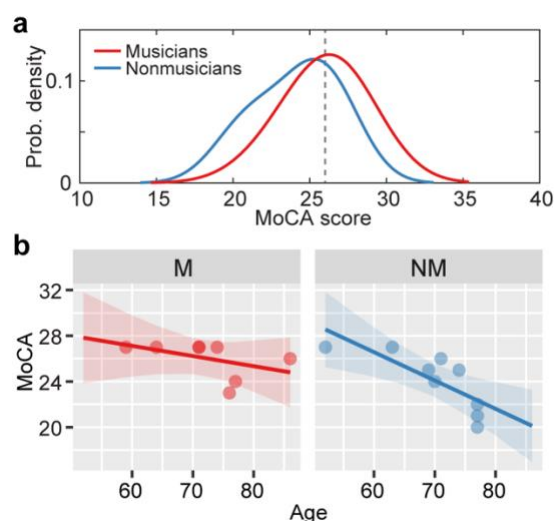123   group x age interaction did not reach significance ($F_{1,11} = 1.95$, $p = 0.19$).

Figure 1. Age-related declines in cognitive function in older musicians and nonmusicians. (a) Distribution of MOCA scores per group. Dotted line, cutoff score for normal cognitive functioning[27]. Older musicians had marginally higher cognitive function than nonmuscians. (b) Cognitive function declines with age for both groups but appears less precipitous in musicians. shading = 95% CI

Behavioral speech categorization

Ms and NMs demonstrated similar vowel categorization across the continuum (identification function slope: $t_{15} = -0.90$, $p = 0.38$; Fig. 2a). The stair-stepped shape of the curves demonstrate that tokens were perceived categorically as /a/ or /u/, respectively. Likewise, similar patterns in reaction times (RTs) were observed between the groups ($F_{1,60} = 2.03$, $p = 0.16$; Fig. 2b). However, decision speeds varied across vowels ($F_{4,60} = 15.41$, $p < 0.0001$, $\eta_p^2 = 0.51$). As expected, both groups categorized the prototypical endpoint tokens (i.e., vw 1, 2, 4, 5) more quickly than the ambiguous midpoint token (i.e., vw 3) (M: $t_{23.5} = -8.97$, $p < 0.001$; NM: $t_{23.5} = -11.32$, $p < 0.001$; Fig. 2b), indicating the hallmark slowing of decision speed for category-ambiguous speech sounds[28].
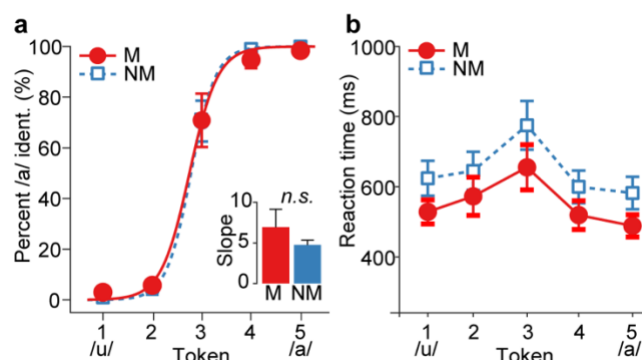

Figure 2. Group performance in speech categorization. Both groups demonstrated similar (a) psychometric function slopes and (b) reaction times when identifying speech sounds along a vowel gradient. More prototypical tokens (those near the extremes of the continuum) were categorized faster

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142   than those at the midpoint (i.e., vw 3) indicating slower decisions for category-ambiguous speech sounds.

143   errorbars = ±1 s.e.m.

Electrophysiological data

145   To provide comparable measures of response amplitude between brainstem and cortical ERPs, we

146   measured (i) the root mean square (RMS) amplitude of the steady state portion of brainstem FFRs (50 -

147   150 ms window) and (ii) N1-P2 magnitude of cortical ERPs averaged across speech tokens (i.e., vw 1-5).

148   We also evaluated the latencies of ERP components (i.e., P1, N1, P2) and the N1-P2 interpeak latency

149   (IPL). Latencies reflect neural processing efficiency with prolonged latencies suggesting less efficient

150   neural encoding. As MCI is known to compromise processing speed and efficiency[29,30], we wanted to

151   evaluate whether musical experience influenced processing efficiency in at-risk individuals.

152   **Brainstem FFRs.** Figure 3a shows average FFRs and response spectra for Ms and NMs for the

153   prototypical /u/ and /a/ tokens (i.e., vw 1 and vw 5). The strength of brainstem speech encoding, as

154   measured via FFR RMS amplitude, was similar across groups ($F_{1, 11} = 0.65$, $p = 0.44$). However, FFR

155   amplitudes varied with age such that older individuals demonstrated more robust brainstem responses

156   than their younger peers ($F_{1, 11} = 8.44$, $p = 0.01$, $\eta_p^2 = 0.43$; Fig. 3b). Unusually large speech FFRs in

157   cognitively at-risk individuals corroborates previous studies demonstrating overexaggerated brainstem

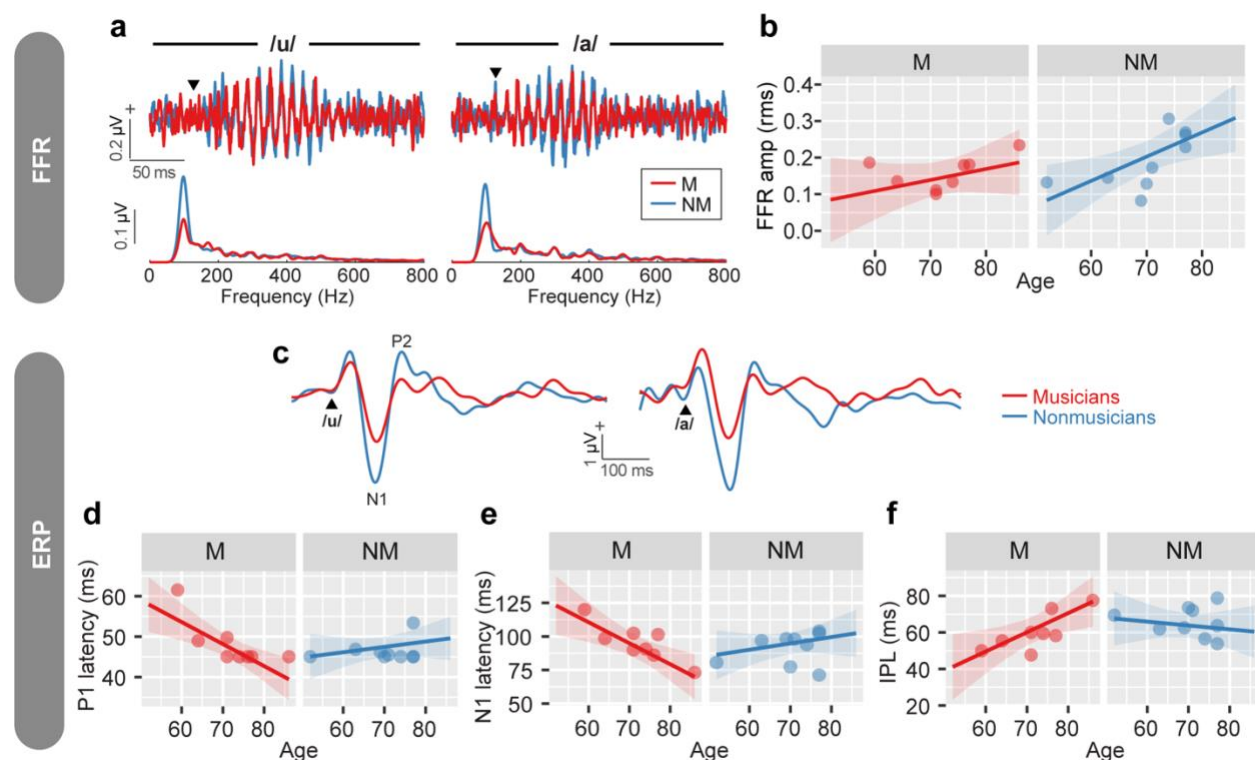158   responses in MCI[12].

160   **Figure 3. Brainstem FFRs and cortical ERPs are modulated by age and musical experience.**

161   Average (a) FFR waveforms and spectra and (c) ERP responses for M and NMs are plotted for the

162   prototypical tokens (i.e., /u/, /a/). (b) FFR amplitudes increase with age but are invariant between groups.

163   (d-e) Cortical P1 and N1 latencies are modulated by age and musical experience. Ms demonstrate more

164   efficient neural encoding with advancing age than their NM peers. (f) NMs showed little change in IPLs

165   with increasing age while Ms demonstrate a gradual increase in IPL. shading = 95% CI

166   **Cortical ERPs.** Figure 3c shows the average cortical ERPs for Ms and NMs for prototypical /u/

167   and /a/. N1-P2 amplitude was invariant across groups ($F_{1, 11} = 0.13$, $p = 0.72$). However, stark group and

168   group x age differences were observed for response latencies. Ms demonstrated shorter latencies on

169   average than the NM group for the P1 and N1 waves (P1: $F_{1, 11} = 12.30$, $p = 0.005$, $\eta_p^2 = 0.53$, Fig. 3d; N1:

170    $F_{1, 11} = 10.31$, $p = 0.01$, $\eta_p^2 = 0.48$, Fig. 3e). Group × age interactions revealed Ms demonstrated decreased

171   P1 and N1 latencies with increasing age while NMs demonstrated little age-related change (P1: $F_{1, 11} =$

172   12.22, $p = 0.005$, $\eta_p^2 = 0.53$, Fig. 3d; N1: $F_{1, 11} = 10.53$, $p = 0.01$, $\eta_p^2 = 0.49$, Fig. 3e). Group differences

173   were also noted in N1-P2 IPL ($F_{1, 11} = 6.42$, $p = 0.03$, $\eta_p^2 = 0.37$, Fig. 3f) with responses being more

174   prolonged in NMs than Ms. However, the group × age interaction was also significant ($F_{1, 11} = 6.05$, $p =$

175   0.03, $\eta_p^2 = 0.35$; Fig. 3f); whereas IPLs increased with age in Ms, NMs' ERPs were relatively invariant

176   (though they were longer overall). The prolonged IPL in Ms was driven by the age-related speeding up of

177   N1 latency as neither group nor age differences were noted for P2 latency (group: $F_{1, 11} = 2.15$, $p = 0.17$;

178   age: $F_{1, 11} = 0.27$, $p = 0.62$). Thus, because Ms' N1 latency decreased with age and no change was

179   observed for P2, the time elapsed between the occurrence of N1 and P2 (i.e., N1-P2 IPL) was prolonged.

180   This is further suggested by the negative correlation between N1 latency and IPL ($r = -0.83$, $p_{adj} = 0.001$).

181   No other significant effects or interactions were found.

182   ***Relations between neural measures, musical experience, and cognitive status***

183   Pairwise Pearson correlations allowed us to determine the degree to which musical experience

184   influenced brainstem and/or cortical speech encoding in individuals at risk for MCI. The analyses

185   between neural measures, musical experience, and MoCA scores revealed cognitive status was predicted

186   by brainstem response amplitude (Fig. 4a); larger speech FFRs were associated with lower (poor) MoCA

187   scores ($r = -0.60$, $p_{adj} = 0.05$). Critically, we found that listeners' duration of musical experience related to

188   auditory cortical response latency and amplitudes (Fig. 4c, d). More extended musical experience was

189   associated with later P1 latency ($r = 0.69$, $p_{adj} = 0.02$; Fig. 4c). Additionally, musical experience was

190   inversely related to N1-P2 amplitude ($r = -0.62$, $p_{adj} = 0.05$; Fig. 4d) suggesting listeners with less musical

191   experience have abnormally robust cortical speech responses, as observed in more severe forms of MCI

192   and cognitive aging[12]. Though trending in the predicted direction, musical experience did not predict

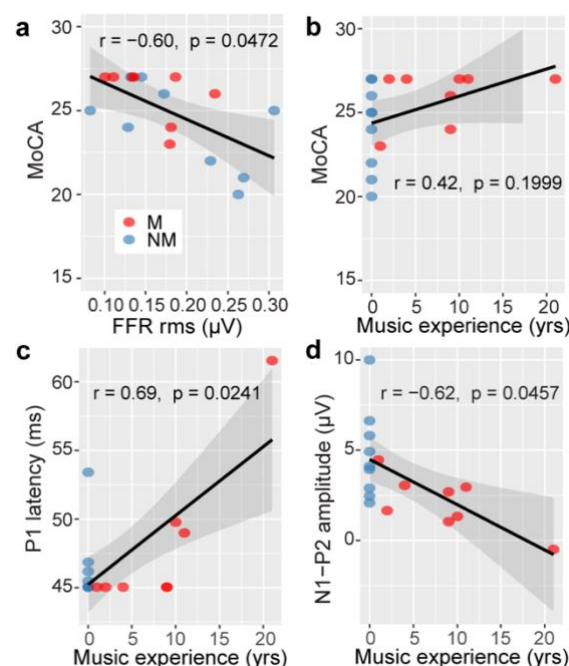193   cognitive scores ($r = 0.42$, $p_{adj} = 0.20$; Fig. 4b).

Figure 4. Correlations depicting relationships between MOCA scores, musical experience, and neural measures. (a) Cognitive status is predicted via brainstem FFRs; larger FFRs are indicative of lower (poorer) MOCA scores. (b) Extent of musical experience was not associated with degree of cognitive impairment, *per se.* (c-d) Longer duration of musical experience is associated with later P1 latencies and smaller N1-P2 amplitudes. *p* values reflect FDR adjustments for multiple comparisons. shading = 95% CI

DISCUSSION

Typical aging degrades neural representations of speech producing weaker encoding and timing delays in auditory brain processing that are further exacerbated by MCI[6-8,12]. By recording speech-evoked potentials via single-channel EEG, we sought to evaluate the neuroplastic effects of prior music experience in older adults with putative MCI. Our data reveal (i) overly robust brainstem FFRs predict severity of cognitive impairment (MoCA scores) and (ii) musical experience offsets neural processing delays in older adults with atypical cognitive aging.

In our sample of individuals with borderline MCI, cognitive function correlated with FFR amplitudes but not cortical responses. Similarly, Bidelman, et al. [12] reported that brainstem measures better distinguish older adults with and without MCI than cortical responses alone. More specifically, we found those with poorer MoCA scores—indicative of more severe cognitive impairment—demonstrated overexaggerated FFRs. These findings corroborate previous work suggesting individuals with MCI demonstrate hypersensitive auditory sensory responses across both subcortical and cortical levels of processing[12,13,31,32]. Hypersensitive encoding in MCI could be due to disinhibition in feedback from higher-order brain areas (e.g., prefrontal cortices) which normally regulate early auditory encoding via

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216   sensory gating mechanisms[31-33]. Yet, altered feedback from prefrontal cortices would likely influence

217   neural representations within both subcortical and cortical levels which is not observed here. Changes

218   local to brainstem have also been identified in preclinical stages of Alzheimer's disease[34]. Thus, the

219   overly robust subcortical responses we observed in those with more severe cognitive impairment most

220   likely result from localized changes within the brainstem that precede more obvious stages of cognitive

221   decline. The lack of correlations between cortical ERPs and cognitive function might be due to the mild

222   severity of cognitive deficits in our sample. It is possible that cognitive decline was not yet egregious

223   enough to influence the robustness of auditory processing at the cortical level. Indeed, primary and

224   secondary auditory cortices are not directly influenced by neuropathological changes in cognition until

225   more advanced stages of the disease[35]. Yet, that subcortical enhancements scale with MoCA scores even

226   in a borderline clinical population suggests that brainstem FFRs are highly sensitive to pre-clinical

227   cognitive deficits and may serve as an early biomarker to index cognitive decline prior to traditional

228   neuropsychological tests[12,16].

229   Our results also suggest that musicianship might mitigate the normal decline in temporal

230   processing commonly observed in aging adults. Aging results in poorer neural precision and synchrony

231   which is thought to contribute to perceptual temporal processing deficits in older adults, including poor

232   speech-in-noise understanding[5,6,8,25,36,37]; furthermore, cognitive impairment exacerbates these typical age-

233   related declines in processing efficiency[10,13,15]. Here, we show that older adults at risk for MCI but with

234   prior musical experience have faster cortical responses to speech with advancing age than their

235   nonmusician peers who are similarly susceptible to cognitive aging. Critically, the differential effect of

236   age in older musicians vs. nonmusicians is promising in that it demonstrates music engagement later in

237   life might help slow the progression of cognitive decline, particularly in those most at risk. Still, while the

238   correlational nature of our data is provocative, larger scale and/or longitudinal training studies are needed

239   to confirm whether music enrichment programs might truly offer an effective intervention for MCI.

240   Prior work suggests musical experience enriches neural representations of speech across the

241   auditory hierarchy and improves auditory skills necessary for speech understanding[23,38,39]. The functional,

242   neuroplastic changes due to musical training experience, even when completed early in life, continue to

243   impact auditory processing and speech perception into adulthood[18-20,40]. Training benefits have been

244   shown to limit typical aging effects, resulting in higher fidelity (i.e., amplified and more temporally

245   precise) neural encoding[18,25]. Our results provide further support for notions that musical experience

246   counteracts normal age-related declines in temporal precision by extending those findings to atypical

247   cognitive aging. Furthermore, our findings provide new evidence to suggest brief to moderate musical

248   experience attenuates auditory neural aging effects as several of our participants received only minimal

249   experience (i.e., < 5 years). Recent studies confirm the benefits of short-term musical experience on the

250   neural encoding and perception of normal and even noise-degraded speech in older adults[26,41].

251   The earlier cortical ERP latencies in musicians suggest these individuals exhibit more efficient

252   sensory encoding of auditory inputs. P1 and N1 reflect the initial registration of acoustic stimuli within

253   early primary and secondary auditory cortices[42-44]. Because these components dominantly reflect

254   exogenous stimulus properties[43,44], shorter ERP latencies in musicians imply that musical experience

255   improves initial cortical processing efficiency for complex stimulus features. Seemingly in contrast, our

256   correlational analyses revealed P1 latency increases with degree of musical training. This relationship

257   appears to be driven by the participant with the greatest extent of experience (21 years) who also is the

258   youngest in the M group (Fig. 4c). This result further highlights the influence of age on auditory

259   processing efficiency in older musicians. The age-related increases in IPLs (N1-P2 latency) in those with

260   musical experience appear to result from reduced latencies mainly in the N1 (rather than P2). The lack of

261   group differences in P2 latency might be expected given this wave indexes stimulus classification[45,46] and

262   we did not find group differences in behavioral categorization. In sum, our ERP latency data suggest that

263   musicians processed speech sounds more efficiently than their nonmusician peers allowing more time for

264   the perceptual decision-making process. Still, RTs were similar (though in the correct direction) between

265   groups so the behavioral relevance of these neural effects remains speculative. For future studies, it would

266   be interesting to evaluate whether changes in cortical IPLs relate to perceived effort in speech perception

267   or participants' confidence in response selection. For example, musical experience might make speech

268   processing less demanding in cognitively at-risk older adults even if it does not yield strong advantages at

269   the behavioral level[47,48].

270   We found no group differences in cortical amplitude measures which suggests latencies may

271   serve as a more sensitive measure of neuroplastic effects in individuals at risk for MCI. This result is

272   consistent with previous studies showing differences in cortical responses between individuals with and

273   without musical experience in the later auditory potentials (i.e., > 100 ms)[23,49-51]. Despite the lack of a

274   group effect, we did find a strong association between degree of musical experience and ERP amplitudes:

275   individuals with more experience demonstrated less robust N1-P2 amplitudes. This result replicates

276   previous work revealing musical experience modulates N1-P2 amplitude[52,53]. Tasks requiring focused

277   attention tend to increase N1 amplitude[43,54]. Thus, decreased N1-P2 in Ms may indicate the task was less

278   cognitively taxing (i.e., requiring less attentional resources) for those with more musical experience. As

279   with the latencies, the reduction in N1-P2 amplitudes suggests that the task might have required less

280   neural activation to produce equivalent behavioral speech categorization and further advances the notion

281   of improved neural efficiency in those with more extensive musical background. That the impact of

282   musical experience is restricted to cortical responses replicates prior work suggesting musical training

283   does not influence the robustness of the steady-state FFR in older adult listeners[18,55]. Furthermore,

284   because age reduces FFR amplitude[6,36,37], it is possible the neuroplastic benefits of musical experience in

285   subcortical levels of processing, if present in this population, were not robust enough to be detected in our

286   small sample (M: $n = 8$).

287   Previous studies have separately focused on the impact of MCI or musical experience on auditory

288   processing in older adults. Here, we examined the combined effects of musical experience in individuals

289   at risk for MCI to directly assess the interaction between these positive (experience-dependent;

290   musicianship) and maladaptive (MCI) forms of plasticity. Our results bolster the novel utility of

291   brainstem speech-FFRs as an index of monitoring cognitive decline in individuals with borderline clinical

292   symptomology[12]. They further suggest neurological changes detected in auditory-sensory brainstem

293   processing may serve as an early biomarker for detecting cognitive deficits prior to traditional behavioral

294   metrics[12]. Additionally, our findings provide new evidence that musical engagement might offset typical

295   age-related delays in auditory cortical brain processing resulting in more efficient speech processing in

296   older adults with putative MCI. While our findings are preliminary and await replication in a larger

297   sample, our cross-sectional data opens the door for future training studies examining music intervention

298   as a protective solution for mitigating auditory-perceptual declines due to cognitive aging.

299   **METHODS**

300   *Participants*

301   Seventeen older adults (*M±SD,* 71.1 ± 8.0 yrs; range: 52-86 yrs) participated in the study. The

302   sample was selected *post hoc* from a larger database of studies examining auditory-cognitive aging,

303   speech processing, and the brain[6,18] based on two inclusion criteria. First, we selected individuals who

304   might be considered at risk for MCI and early cognitive decline based on lower-than-normal scores on

305   cognitive screening. Cognitive status was assessed by the Montreal Cognitive Assessment (MoCA;

306   published normative cutoff score for normal cognitive function=26)[27]. Participants scoring near or below

307   the normative cutoff for MCI (≤ 27 points; 25 ± 2.3; range: 20-27) were identified as being at-risk or

308   having putative MCI. We included individuals with borderline scores (=27) to (i) assess older adults at

309   risk for MCI and (ii) ensure better *post hoc* matching of groups along other confounding variables (e.g.,

310   hearing status, age, education, etc.). While these scores fall within or near the normative range for mild

311   cognitive impairments (19-25.2), they remain above the MoCA normative range for Alzheimer's Disease

312   or more severe dementia (11.4-21)[27]. Underlying etiology was unknown.

313   From among those identified with at-risk MoCA scores, we then divided the sample into two

314   groups based on whether listeners had *any* prior musical training. Musicians (Ms; n = 8; 2 female) were

315   defined as amateur instrumentalists who had received at least 1 year of private instruction on their

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316   principal instrument. Ms had on average 8.4 ± 6.4 yrs of musical experience. Nonmusicians (NMs; n = 9;

317   5 female) had no previous musical experience (0.0 ± 0.0 yrs) of any kind in their lifetime. While this

318   definition is perhaps more lax than typical definitions of musicians, we aimed to assess musical

319   experience as a continuous rather than dichotomized trait[56] so as to not discard individual differences[57].

320   Given the preliminary nature of this study, treating musical experience as a graded variable also provided

321   us the opportunity to assess if musicianship of any kind, and however minimal, might offer protective

322   effects against MCI.

323   On average, the groups demonstrated clinically normal hearing (≤ 25 dB HL) based on pure-tone

324   average (PTA) thresholds (i.e., average of 500, 1000, 2000 Hz) bilaterally, and hearing sensitivity did not

325   differ between groups (t_{15} = -0.71, p = 0.49). Groups were also balanced in age (t_{15} = -0.56, p = 0.58) and

326   cognitive function as assessed by the MoCA (t_{15} = -1.79, p = 0.09). Ms reported more years of formal

327   education on average (M: 19.1 ± 3.0 yrs; NM: 14.8 ± 3.6 yrs; t_{15} = -2.67, p = 0.02); therefore, education

328   was included as a covariate in all subsequent analyses. All participants were right-handed[58] and native

329   English speakers who reported no history of neuropsychiatric disorders. Participants were compensated

330   for their time and provided written, informed consent in compliance with the Declaration of Helsinki for

331   experiments involving humans and a protocol approved by the Baycrest Centre Research Ethics

332   Committee.

333   ***Stimuli***

334   A synthetic five-step vowel continuum ("vw 1-5") was constructed so that each 100 ms token

335   would differ minimally acoustically, yet be perceived categorically[46,59]. The first formant (F1) frequency

336   was varied parametrically over five equal steps between 430 and 730 Hz resulting in a stimulus set that

337   spanned a perceptual phonetic continuum from /u/ to /a/. All other stimulus attributes (e.g., fundamental

338   frequency, higher formants) were identical between tokens. For further stimulus details, see Bidelman et

339   al.[46].

340   ***EEG recording procedures and preprocessing***

341   Data acquisition and response evaluation were similar to previous reports from our laboratory

342   (e.g., [6,18,46]). Stimuli were presented binaurally at 83 dB SPL via insert earphones (ER-3A, Etymotic

343   Research) with extended acoustic tubing (50 cm) to prevent electromagnetic stimulus artifact from

344   contaminating brainstem responses. During ERP recording, we used a forced-choice procedure in which

345   participants heard 200 randomly ordered tokens and were asked to indicate whether they perceived "u" or

346   "a" via a button press on the keyboard. Following participants' behavioral response, the interstimulus

347   interval (ISI) was jittered randomly between 400 and 600 ms (20-ms steps, rectangular distribution) to

348   avoid alpha entrainment of the EEG[44, p. 168] and listeners' anticipation of their behavioral response. We

349   then collected an additional 2000 passive trials (ISI = 150 ms) to measure the sub-microvolt FFR[60].

350   Brainstem responses are roughly an order of magnitude smaller than cortical activity, and a larger number

351   of trials is required to obtain a comparable signal to noise ratio for brainstem FFRs and cortical ERPs[61].

352   During the passive block, participants watched a self-selected movie with subtitles to facilitate a calm yet

353   wakeful state throughout recording.

354   Continuous EEGs were recorded differentially between an electrode placed on the high forehead

355   at the hairline (~Fpz) referenced to linked mastoids. A third electrode on the mid-forehead served as the

356   common ground. This montage is optimal for recording evoked responses of both subcortical and cortical

357   origin[23,46,62]. Electrode impedances were ≤ 3 kΩ. EEGs were digitized at 20 kHz and bandpass filtered

358   online between 0.05 and 3500 Hz (SynAmps2, Compumedics NeuroScan). Traces were then segmented

359   (cortical ERP: -100-600 ms; brainstem FFR: -40-210 ms), baselined to the pre-stimulus interval, and

360   subsequently averaged in the time domain to obtain ERPs for each condition[63]. Trials exceeding a ± 50 μV

361   threshold were rejected as blinks prior to averaging. Grand average evoked responses were then bandpass

362   filtered in different frequency bands to emphasize brainstem (80-2500 Hz) and cortical (1-30 Hz) neural

363   activity, respectively[18,46,61,62].

Behavioral data analysis

365   Psychometric identification functions were created by calculating the proportion of trials

366   identified as a single vowel class (i.e., /a/) for each token (i.e., vw 1-5). Comparing the slope of the

367   psychometric functions between groups allowed us to assess possible differences in the "steepness" (i.e.,

368   rate of change) of the categorical speech boundary as a function of musical experience. Behavioral speech

369   labeling speeds, i.e., RTs, were computed separately for each participant as the mean response latency

370   across trials for a given speech token. Following our previous reports on categorical speech perception

371   (e.g., [18,46]), RTs shorter than 200 ms or exceeding 5500 ms were regarded as implausibly fast responses

372   and lapses of attention respectively and were excluded from analysis.

Electrophysiological data analysis

374   To provide comparable measures of response amplitude between brainstem and cortical ERPs, we

375   measured (i) the RMS amplitude of the steady state portion of brainstem FFRs (50 -150 ms window) and

376   (ii) N1-P2 magnitude of cortical ERPs averaged across speech tokens (i.e., vw 1-5). N1 was identified as

377   the peak negativity between 70 and 120 ms and P2 as the peak positivity between 150 and 250 ms (e.g.,

378   [6,32]). The overall magnitude of the N1-P2 complex, computed as the voltage difference between the two

379   individual waves, was used as a singular index of the total cortical activation across vowel tokens. While

380   other FFR/ERP measures are available in the literature[44,64], FFR RMS and cortical N1-P2 metrics are

381   advantageous because they both provide a description of the overall amplitude of the evoked response at

382   brainstem and cortical levels using an isomorphic metric. We also evaluated the latencies of the P1 (peak

13

383   positivity between 45 and 65 ms), N1, and P2 components of the cortical response as well as the N1-P2

384   IPL.

Brain-behavior relationships

386   Pairwise Pearson's correlations were used to investigate correspondences between subcortical and

387   cortical speech representations (brainstem: RMS amplitude; cortical: P1, N1, P2 latencies and N1-P2

388   magnitude and IPL), musical experience, and cognitive status. False discovery rate corrections were

389   applied to multiple correlations[65], and adjusted p-values are reported. This analysis allowed us to

390   determine the degree to which musical experience influenced brainstem and/or cortical speech encoding

391   in individuals at risk for MCI.

Statistical analyses

393   We used an analysis of covariance (ANCOVA) on each dependent variable (*lme4*; R)[66,67] with

394   group (2 levels: M, NM) serving as the between-subjects factor and education, PTA, and age serving as

395   continuous covariates. Following our previous work, responses were averaged across vowel tokens to

396   reduce the dimensionality of the data and directly assess musicianship effects on neural responses[12].

397   Although groups did not differ in hearing sensitivity, age-related hearing loss is known to alter brainstem

398   and cortical auditory evoked potentials[1,6]. Therefore, we included PTA as a covariate in analyses to

399   control for potential hearing-related differences in neural responses. Cognitive function declines with

400   age[4,30]; therefore, age was also included as a covariate in all analyses. When needed, Tukey-Kramer

401   corrections were used to control Type I error inflation for multiple comparisons. Effect sizes for

402   significant results are given as partial eta-squared (η_p^2).

Data Availability

404   The datasets generated and analyzed during the current study are available from the corresponding author

405   on reasonable request.

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566   **AUTHOR CONTRIBUTIONS**

567   G.M.B. designed the experiment; C.N.P. and G.M.B. acquired and analyzed the data, interpreted the

568   results, and contributed to the writing of the manuscript.

569   **ADDITIONAL INFORMATION**

570   *Competing interests*

571   The authors declare no competing interests.