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CONCISE REPORT

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Musical experience partially counteracts temporal speech processing deficits in putative mild cognitive impairment

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Abstract

Mild cognitive impairment (MCI) commonly results in more rapid cognitive and behavioral declines than typical aging. Individuals with MCI can exhibit impaired receptive speech abilities that may reflect neurophysiological changes in auditory-sensory processing prior to usual cognitive deficits. Benefits from current interventions targeting communication difficulties in MCI are limited. Yet, neuroplasticity associated with musical experience has been implicated in improving neural representations of speech and offsetting age-related declines in perception. Here, we asked whether these experience-dependent effects of musical experience might extend to aberrant aging and offer some degree of cognitive protection against MCI. During a vowel categorization task, we recorded single-channel electroencephalograms (EEGs) in older adults with putative MCI to evaluate speech encoding across subcortical and cortical levels of the auditory system. Critically, listeners varied in their duration of formal musical experience (0–21 years). Musical experience sharpened temporal precision in auditory cortical responses, suggesting that musical experience produces more efficient processing of acoustic features by counteracting age-related neural delays. Additionally, robustness of brainstem responses predicted the severity of cognitive decline, suggesting that early speech representations are sensitive to preclinical stages of cognitive impairment. Our results extend prior studies by demonstrating positive benefits of musical experience in older adults with emergent cognitive impairments.

KEYWORDS
auditory event-related potentials (ERPs), cognitive aging, experience-dependent plasticity, frequency-following response (FFR), music-to-language transfer effects

INTRODUCTION

Cognitive aging results in structural and functional changes throughout the central auditory pathway,[1–3] as well as declines in associated auditory processing skills necessary for effective speech comprehension.[4,5] Older adults demonstrate less precise and altered neural encoding of speech which has been attributed to slowed neural conduction times, poor neural synchrony, and reduced inhibitory processes.[6–8] Senescent neural changes parallel perceptual difficulties commonly reported by aging adults in clinical settings. Older adults often exhibit deficits in temporal processing, which is vital for accurate speech perception particularly in challenging listening conditions (e.g., noisy or reverberant environments).[9]

Aging effects are further exacerbated in those with mild cognitive impairment (MCI),[10] a form of atypical cognitive aging causing memory lapses in short-term recall and/or impaired critical thinking and decision-making skills.[11] Because individuals with MCI are also at higher risk of progressive cognitive deficits,[11] early identification and intervention is vital. While most current diagnostic assessments focus on aspects of cognitive function, recent studies reveal that MCI alters

early sensory processing by compounding auditory perceptual deficits due to typical aging.[12–14] Thus, in addition to more commonly noted cognitive issues, there is emerging evidence that MCI is associated with early declines in *auditory-sensory* processing, including complex receptive language processes[15,16] and even how accurately the brain encodes speech itself.[12,17] In this vein, neuroimaging techniques and complex speech perception tasks may aid in the early detection of neurocognitive changes associated with MCI.

Current MCI treatments do little to slow disease progression and primarily address associated symptoms or focus on improving quality of life.[18] Interventions targeting communication difficulties in this population provide limited benefit for improving language or cognitive skills.[10,15] However, studies show that musical experience may counteract age-related declines by robustly enhancing the neural encoding of speech across subcortical and cortical levels of the central auditory system.[19–21] Individuals with musical experience demonstrate stronger cognitive abilities (i.e., working memory, long-term memory, and attentional control),[22–24] higher-fidelity neural encoding of acoustic features,[25] and improved neural timing precision;[19,26] all of which may contribute to enhanced speech perception. Nevertheless, while musical experience might help counteract normal age-related declines in speech processing,[20,27] it remains unclear whether such experience-dependent advantages transfer to populations with suspected cognitive impairments. A critical demonstration of music's benefits on the aging brain is to confirm whether similar neural enhancements occur in at-risk populations or those with borderline neurocognitive performance (e.g., putative MCI). This would provide novel evidence that music engagement might bolster communication skills at early stages and decelerate the progression of cognitive aging.

To this end, the current study investigated the impact of musical experience on speech encoding in individuals with putative MCI. We measured frequency-following responses (FFRs) and cortical event-related potentials (ERPs) in older adults with varying degrees of musical experience who were identified as being at risk for MCI based on cutoff-level performance on neurocognitive screening assessments. Our findings reveal that abnormally large FFRs predict the severity of cognitive impairment and may serve as an objective marker for MCI severity. We further show that musical experience mitigates some age-related delays in neural speech encoding in this at-risk population, paving the way for future translational music interventions in older populations.

MATERIALS AND METHODS

Participants

A sample of 17 older adults ($M \pm SD$, 71.1 ± 8.0 years; range: 52–86 years) was selected post hoc from a larger database of studies conducted at the Rotman Research Institute based on two inclusion criteria. First, we selected individuals who might be considered at risk for MCI and early cognitive decline based on lower-than-normal scores on cognitive screening assessments. Cognitive status was assessed by the Montreal Cognitive Assessment (MoCA; normative cutoff for normal cognitive function = 26 points).[28] Participants scoring near or below the normative cutoff for MCI (≤27 points; 25 ± 2.3; range: 20–27) were identified as being at risk or having putative MCI, respectively. We included individuals with borderline scores (= 27) to (1) assess older adults at risk for MCI and (2) ensure better post hoc matching of groups along other confounding variables (e.g., hearing status, age, education, etc.). While these scores fall within or near the normative range for MCI (19–25.2), they remain above the MoCA normative range for Alzheimer's disease or more severe dementia (11.4–21).[28] Underlying etiology was unknown.

From those identified with at-risk MoCA scores, we then divided the sample into two groups based on whether listeners had any prior musical experience. Those with musical experience (Ms; $n = 8$; two females) were defined as amateur instrumentalists who received at least 1 year of private instruction on their principal instrument (8.4 ± 6.4 years) prior to age 13 (9.9 ± 2.7 years). Participants with no musical experience (NMs; $n = 9$; five females) reported no previous musical experience (0.0 ± 0.0 years) of any kind in their lifetime. While this classification is perhaps more lax than typical delineations of musical experience, we aimed to assess a broader range. Treating musical experience as a graded variable allowed us to assess whether any musical experience, however minimal, might offer protective effects against MCI.

Air conduction hearing thresholds were measured at octave frequencies between 250 and 8000 Hz. Both groups had normal pure-tone averages (PTAs) (≤25 dB HL; average of 500, 1000, and 2000 Hz) bilaterally, which did not differ between groups ($t_{15} = -0.71$, $p = 0.49$). Moreover, although both groups showed mild-to-moderate high-frequency hearing loss characteristic of normal age-related presbycusis, critically, the audiometric profile was identical between groups from 250 to 8000 Hz (*t*-test at each audiometric frequency: $t_{15} = 0.23$, $p = 0.82$). Both groups were also balanced in age ($t_{15} = -0.56$, $p = 0.58$), sex (Fisher's exact test, $p = 0.33$), and cognitive function as assessed by the MoCA ($t_{15} = -1.79$, $p = 0.09$).[a] Ms reported more years of formal education on average (M: 19.1 ± 3.0 years; NM: 14.8 ± 3.6 years; $t_{15} = -2.67$, $p = 0.02$); therefore, education was included as a covariate in all subsequent analyses. All participants were right-handed,[29] native English speakers who reported no history of neuropsychiatric disorders, and were not receiving any treatment related to cognitive decline.

Stimuli

A synthetic five-step vowel continuum ("vw 1–5") was constructed so that each 100 ms token would differ minimally acoustically, yet be

[a] Post hoc power calculations indicated the group difference in MoCA scores with an effect size of d=0.91 achieved 42% power (*t*-test, 2-tailed, $\alpha = 0.05$, G*Power 3.1), which is likely underpowered given our small sample. $n = 20$/group would be needed to detect a difference in MoCA scores between groups at 80% power. However, had groups differed in cognitive status, this would have confounded interpretations on whether differences in FFR/ERPs were attributable to cognitive status or musical experience. Thus, the matching of our groups with on-the-cusp MoCA scores helps rule out this possibility.

perceived categorically.[30] The first formant (F1) frequency was varied parametrically over five equal steps between 430 and 730 Hz, resulting in a stimulus set that spanned a perceptual phonetic continuum from /u/ to /a/. All other stimulus attributes (e.g., fundamental frequency and higher formants) were identical between tokens. For further details, see Ref. 30 and Figure S1.

EEG recording and preprocessing

Data acquisition and response evaluation were similar to previous reports from our laboratory.[19] Stimuli (alternating polarity) were presented binaurally at 83 dB SPL via insert earphones (ER-3A, Etymotic Research) with extended acoustic tubing (50 cm; transducers within room) to reduce stimulus artifact. Adding the responses to alternating stimulus polarities further minimizes stimulus artifact from contaminating the FFR.[31,32] During ERP recording, participants performed a categorical perception task. Using a forced-choice procedure, participants heard 200 randomly ordered vowel tokens and indicated whether they perceived "u" or "a" via a button press on the keyboard. Following participants' behavioral response, the interstimulus interval (ISI) was jittered randomly between 400 and 600 ms (20-ms steps, rectangular distribution) to avoid alpha entrainment of the EEG (see p. 168 of Ref. 33) and listeners' anticipation of their behavioral response. We then collected an additional 2000 passive trials (ISI = 150 ms) to measure the sub-microvolt FFR.[34] During the passive block, participants watched a self-selected movie with subtitles to facilitate a calm yet wakeful state throughout recording.

Continuous EEGs were recorded differentially between an electrode placed on the high forehead at the hairline referenced to linked mastoids. A third electrode on the mid-forehead (~Fpz) served as the common ground. This montage is optimal for recording evoked responses of both subcortical and cortical origins.[30,35] Because scalp-recorded EEG reflects the aggregate response from multiple neural generators, the current literature debates the predominate generators of the FFR for stimuli containing low frequencies (<100 Hz).[36–40] Although there is some evidence that FFRs contain cortical contributions at these low frequencies,[36,40] the response is dominated by midbrain (and more peripheral) auditory sources when recorded via EEG.[37,38] Electrode impedances were $\leq$3 kΩ. EEGs were digitized at 20 kHz and bandpass filtered online between 0.05 and 3500 Hz (second-order Butterworth IIR; 12 dB/oct) using SynAmps2 amplifiers (24 bit A/D resolution; >110 dB common mode rejection; Compumedics NeuroScan). Traces were then segmented (cortical ERP: −100 to 600 ms; brainstem FFR: −40 to 210 ms), baselined to the prestimulus interval, and subsequently averaged in the time domain to obtain ERPs for each condition.[41] Trials exceeding a ±50 μV threshold were rejected as blinks prior to averaging. Grand average evoked responses were then bandpass filtered (20th-order elliptic filter; 80 dB stop band attenuation, 0.1 dB passband ripple) in different frequency bands to emphasize subcortical (80–2500 Hz) and cortical (1–30 Hz) activity.[35,42]

Behavioral data analysis

Psychometric identification functions were created by calculating the proportion of trials identified as a single vowel class (i.e., /a/) for each token (i.e., vw 1–5). Comparing the slope of the psychometric functions between groups allowed us to assess possible differences in the "steepness" (i.e., rate of change) of the categorical speech boundary as a function of musical experience. Behavioral speech labeling speeds, that is, reaction times (RTs), were computed separately for each participant as the mean response latency across trials for a given speech token. Following our previous reports on categorical speech perception,[19,30] RTs shorter than 200 ms or exceeding 5500 ms were regarded as implausibly fast and lapses of attention, respectively, and were excluded from the analysis.

Electrophysiological data analysis

To provide comparable measures of response amplitude between FFRs and cortical ERPs, we measured (1) the root mean square (RMS) amplitude of the steady state portion of FFRs (50–150 ms window) and (2) N1–P2 magnitude of cortical ERPs averaged across speech tokens (i.e., vw 1–5). N1 was identified as the peak negativity 70–120 ms and P2 as the peak positivity 150–250 ms.[43] The overall magnitude of the N1–P2 complex, computed as the voltage difference between the two individual peaks, was used as a singular index of the total cortical activation across vowel tokens. While other FFR/ERP measures are available in the literature,[33,44] FFR RMS and cortical N1–P2 metrics are advantageous because they both provide a description of the overall amplitude of the evoked response at subcortical and cortical levels using an isomorphic metric. We also evaluated the latencies of the P1 (peak positivity 45–65 ms), N1, and P2 components of the cortical response as well as the N1–P2 interpeak latency (IPL). Latencies reflect neural processing efficiency with prolonged latencies suggesting less efficient neural encoding. As MCI is known to compromise processing speed and efficiency,[45,46] we wanted to evaluate whether musical experience influenced processing efficiency in at-risk individuals.

Brain–behavior relationships

Pairwise Pearson's correlations were used to investigate correspondences between subcortical and cortical speech representations (subcortical: RMS amplitude; cortical: P1, N1, and P2 latencies, N1–P2 magnitude, and IPL), musical experience, and cognitive status. False discovery rate corrections were applied to multiple correlations,[47] and adjusted *p*-values are reported.

Statistical analyses

We used a linear model for each dependent variable (*lme4*; R),[48,49] with group (two levels: M and NM) serving as the between-subjects

factor and education, PTA, and age serving as continuous covariates. All variables were normally distributed as assessed via residual and QQ plots. Although groups did not differ in hearing sensitivity, age-related hearing loss is known to alter brainstem and cortical auditory evoked potentials.[1,6] Therefore, we included PTA as a covariate to control for potential hearing-related differences in neural responses. Because cognitive function declines with age,[4,46] age was also included as a covariate. Calculated variance inflation factors for these covariates were all less than 2, indicating they were not significantly correlated within our sample. An initial MANOVA assessed vowel token effects across all dependent variables. No main nor interaction effects containing vowel were significant (all Pillai's trace *p*s > 0.80). Thus, responses were averaged across tokens to reduce the dimensionality of the data. Tukey–Kramer corrections were used to control Type I error inflation for multiple comparisons. Effect sizes for significant results are given as partial eta-squared (η_p^2).

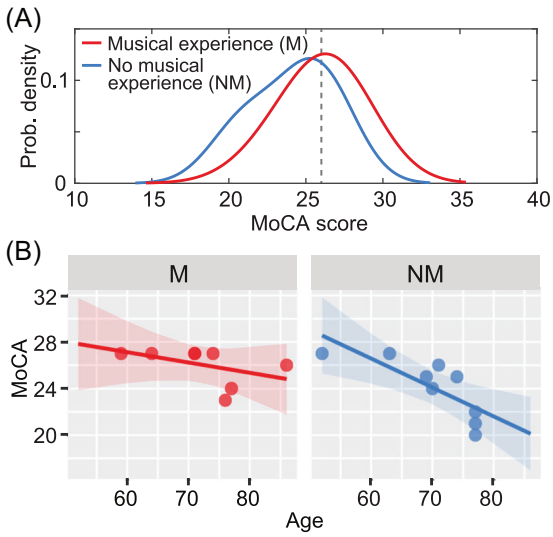

FIGURE 1 Age-related declines in cognitive function in older adults with (M) and without (NM) prior musical experience. (A) Probability density functions for the distributions of MoCA scores per group. Dotted line is the cutoff score for normal cognitive functioning.[28] Older adults with prior musical experience had marginally higher cognitive function than those with no experience. (B) Cognitive function declines with age for both groups but appears less precipitous in Ms. Shading = 95% CI

RESULTS

To evaluate whether auditory processing benefits associated with prior musical experience extends to individuals at risk for atypical cognitive aging, we assessed the neural encoding of speech across subcortical (FFR) and cortical (ERP) levels during a vowel categorization task. Because individuals with MCI demonstrate overexaggerated neural responses and impaired processing speeds, we evaluated response amplitudes and latencies to investigate the impact of musical experience on each, respectively. Importantly, we also evaluated how these neural measures relate to the extent of musical experience and cognitive status. While cognitive function and behavioral categorization was similar between groups, those with prior musical experience demonstrated more efficient processing (i.e., decreased response latencies) during encoding of acoustic features than their NM peers. We also found more robust FFR amplitudes related to poorer cognitive function (i.e., lower MoCA scores), suggesting that preclinical changes in cognitive status may be detected in early stages of auditory processing.

Cognitive function

Figure 1 depicts the level of cognitive function for each group as measured by probability densities of MoCA scores. Groups did not differ in MoCA after controlling for age, hearing sensitivity, and education ($F_{1,11} = 1.24$, $p = 0.29$; Figure 1A). Yet, cognitive function moderately declined with increasing age in both groups ($F_{1,11} = 8.80$, $p = 0.01$, $\eta_p^2 = 0.44$; Figure 1B).

Behavioral speech categorization

Ms and NMs demonstrated similar vowel categorization across the continuum (identification function slope: $t_{15} = -0.90$, $p = 0.38$; Figure 2A). The stair-stepped shape of the curves demonstrates that tokens were perceived categorically as /a/ or /u/, respectively. Likewise, similar patterns in RTs were observed between the groups ($F_{1,60} = 2.03$, $p = 0.16$; Figure 2B). However, decision speeds varied across vowels, with stimulus token having a moderate effect on responses ($F_{4,60} = 15.41$, $p < 0.0001$, $\eta_p^2 = 0.51$). As expected, both groups categorized the prototypical endpoint tokens (i.e., vw 1, 2, 4, and 5) more quickly than the ambiguous midpoint token (i.e., vw 3) (M: $t_{23.5} = -8.97$, $p < 0.001$; NM: $t_{23.5} = -11.32$, $p < 0.001$; Figure 2B), indicating the hallmark slowed decision speed for category-ambiguous speech sounds.[50]

Electrophysiological data

FFRs

Figure 3A shows average FFRs and response spectra for Ms and NMs for the prototypical /u/ and /a/ tokens (i.e., vw 1 and vw 5). The strength of subcortical speech encoding, as measured via FFR RMS amplitude averaged across tokens, was similar across groups ($F_{1,11} = 0.65$, $p = 0.44$). To test whether high harmonics (timbre) encoding differed between groups, we assessed the average amplitudes of the third through the sixth harmonics (H3–H6). Like FFR RMS amplitude, strength of upper harmonics encoding was similar between groups ($F_{1,11} = 2.89$, $p = 0.12$). However, FFR RMS amplitudes varied with age such that older individuals demonstrated larger responses than their younger peers ($F_{1,11} = 8.44$, $p = 0.01$, $\eta_p^2 = 0.43$; Figure 3B) regardless of musical experience.

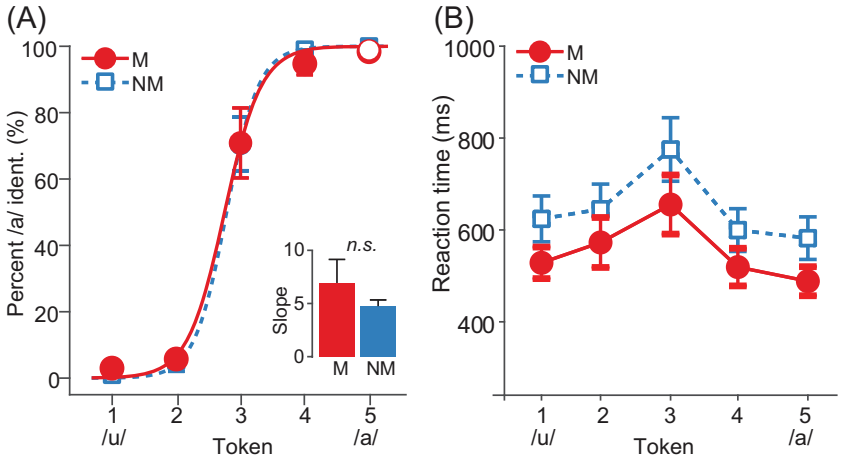

FIGURE 2 Group performance in speech categorization. Both groups demonstrated similar (A) psychometric function slopes and (B) reaction times when identifying speech sounds along a vowel gradient. More prototypical tokens (those near the extremes of the continuum) were categorized faster than those at the midpoint (i.e., vw 3), indicating slower decisions for category-ambiguous speech sounds. Error bars = ±1 s.e.m.

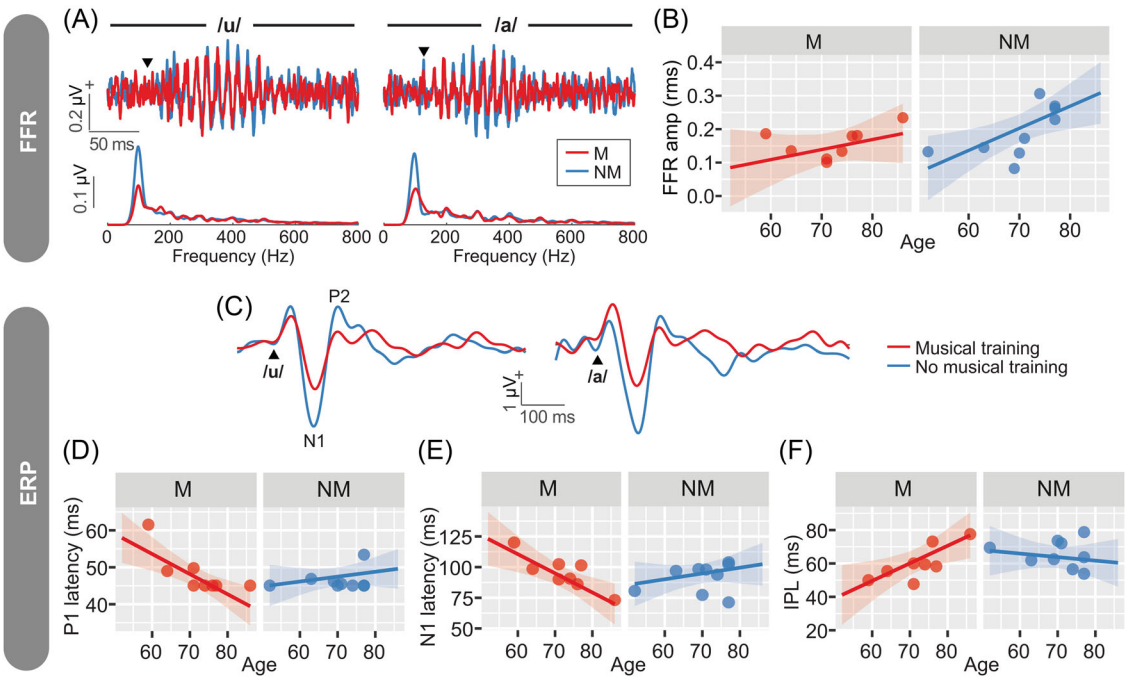

FIGURE 3 FFRs and cortical ERPs are modulated by age and musical experience. Average (A) FFR waveforms and spectra and (C) ERP responses for M and NMs are plotted for the prototypical tokens (i.e., /u/, /a/). (B) FFR amplitudes increase with age but are invariant between groups. (D–E) Cortical P1 and N1 latencies are modulated by age and musical experience. Ms demonstrate more efficient neural encoding with advancing age than their NM peers. (F) NMs showed little change in IPLs with increasing age, while Ms demonstrate a gradual increase in IPL. Shading = 95% CI

Cortical ERPs

Figure 3C shows the average cortical ERPs for Ms and NMs for prototypical /u/ and /a/. Although there appear to be group differences in N1 amplitude for these tokens, N1–P2 amplitude was invariant across groups after collapsing across tokens ($F_{1,11} = 0.13$, $p = 0.72$). However, stark group and group × age differences were observed for response latencies, demonstrating medium effect sizes across analyses. Ms demonstrated shorter latencies on average than NMs for P1 and N1 (P1: $F_{1,11} = 12.30$, $p = 0.005$, $\eta_p^2 = 0.53$, M = 47.59 ms, NM = 47.61 ms; N1: $F_{1,11} = 10.31$, $p = 0.01$, $\eta_p^2 = 0.48$, M = 93.3 ms, NM = 95.3 ms). Group × age interactions revealed decreased P1 and N1 latencies in Ms with increasing age, while NMs demonstrated little age-related change (P1: $F_{1,11} = 12.22$, $p = 0.005$, $\eta_p^2 = 0.53$, Figure 3D; N1: $F_{1,11} = 10.53$, $p = 0.01$, $\eta_p^2 = 0.49$, Figure 3E; see also partial correlations in Supporting Information 2). A small effect of group was also noted in N1–P2 IPL ($F_{1,11} = 6.42$, $p = 0.03$, $\eta_p^2 = 0.37$, Figure 3F), with responses being more prolonged in NMs than Ms. However, the group × age interaction was also significant ($F_{1,11} = 6.05$, $p = 0.03$, $\eta_p^2 = 0.35$; Figure 3F), whereas IPLs increased with age in Ms, NMs' ERPs were relatively invariant (though they were longer overall). Prolonged IPLs in Ms were driven by age-related decreases in N1 latency as neither group nor age

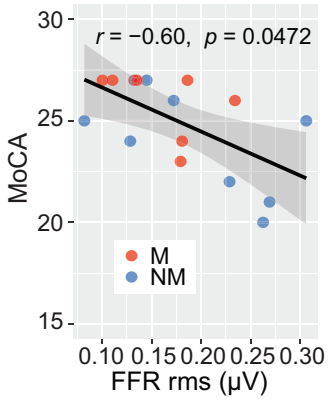

FIGURE 4 Correlation depicting the relationship between MoCA scores and FFR amplitude. Cognitive status is predicted via FFRs; larger FFRs are indicative of lower (poorer) MoCA scores. Shading = 95% CI

differences were noted for P2 latency (group: $F_{1,11} = 2.15$, $p = 0.17$; age: $F_{1,11} = 0.27$, $p = 0.62$). Thus, because Ms' N1 latency decreased with age and no change was observed for P2, the time elapsed between the occurrence of N1 and P2 (i.e., N1–P2 IPL) was prolonged. This is further supported by a negative correlation between N1 latency and IPL ($r = -0.83$, $p_{adj} = 0.001$). No other significant effects or interactions were found.

Relations between neural measures, musical experience, and cognitive status

The analyses between neural measures, musical experience, and MoCA scores revealed that cognitive status relates to subcortical response amplitude (Figure 4); larger speech FFRs were associated with poorer MoCA scores ($r = -0.60$, $p_{adj} = 0.05$). Though trending in the predicted direction, musical experience did not predict cognitive scores ($r = 0.42$, $p_{adj} = 0.20$). No other correlations remained significant following exclusion of an observation identified as an outlier and influential point (using studentized residuals and Cook's distance) and correction for multiple comparisons.

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 that (1) overly robust FFRs predict the severity of cognitive impairment (MoCA scores) and (2) musical experience counteracts declines in temporal processing in older adults at risk for atypical cognitive aging.

In our sample of individuals with borderline MCI, cognitive function correlated with FFR amplitudes but not cortical responses. Previous work suggests that individuals with MCI demonstrate hypersensitive auditory responses across both subcortical and cortical levels of processing when compared to age-matched controls[43,51] and that FFR hypersensitivity strengthens with age.[12] In the current study evaluating those at risk for MCI (i.e., individuals who likely demonstrate larger than normal FFRs), those with lower MoCA scores—indicative of poorer cognitive function—demonstrated strengthened FFRs. Moreover, FFR amplitude increased with age, consistent with prior reports that the influence of MCI on auditory-sensory processing is age dependent. Hypersensitive encoding in this population could be due to disinhibition in feedback from higher-order brain areas (e.g., prefrontal cortices) that normally regulate early auditory encoding via sensory gating mechanisms.[43,51–53] Changes local to brainstem have also been identified in preclinical stages of Alzheimer's disease.[54] Thus, the overly robust subcortical responses observed in those with more severe cognitive impairment may result either from reduced top-down regulation or localized changes within the brainstem that precede more obvious stages of cognitive decline. These changes may arise from altered corticofugal signaling within the brainstem-cortical pathway as previously reported in older adults during challenging listening conditions.[1,55] The lack of correlation between cortical ERPs and cognitive function might be due to the mild severity of cognitive deficits in our sample. It is possible that cognitive decline was not yet egregious enough to influence the robustness of auditory processing at the cortical level. Indeed, auditory cortices are not directly influenced by neuropathological changes in cognition until more advanced stages of the disease.[56] Alternatively, the lack of correlation may be attributed to the study being underpowered given our small sample size.

Our results also suggest that early musical experience might mitigate declines in temporal processing commonly observed in aging adults. Aging results in poorer neural precision and synchrony, which is thought to contribute to perceptual temporal processing deficits in older adults, including poor speech-in-noise understanding;[5,6,8,57] furthermore, cognitive impairment exacerbates these typical age-related declines in processing efficiency.[13] Here, we show that older adults at risk for MCI but with prior musical experience have faster cortical responses to speech with advancing age than nonmusically experienced peers. Critically, the differential effect of age in older adults with and without prior musical experience is promising in that it demonstrates even slight degrees of music engagement across the lifespan may slow progression of cognitive decline, particularly in those most at risk. Still, while the correlational nature of our data is provocative, larger scale and/or longitudinal training studies are needed to confirm whether music enrichment programs might truly offer an effective intervention for those at risk for MCI.

Prior work suggests that musical experience enriches neural representations of speech across the auditory hierarchy and improves auditory skills necessary for speech understanding.[58–60] Musical experience benefits have been shown to limit typical aging effects, resulting in higher fidelity (i.e., amplified and more temporally precise)

neural encoding.[19,26] These functional, neuroplastic changes due to musical training, even when completed early in life, continue to impact auditory processing and speech perception into adulthood.[19–21,61,62] Our results provide further support for notions that musical experience counteracts normal age-related declines in temporal precision by extending those findings to atypical cognitive aging. Somewhat surprisingly, we found that processing efficiency improved with increasing age in those with prior musical experience. The effects of musical experience beginning early in life (prior to 13 years) may compound over time, resulting in more efficient processing of acoustic features. Indeed, White-Schwoch *et al.*[61] observed faster neural responses to speech in older adults with little (1–3 years) to moderate (4–14 years) musical experience when compared to individuals with no musical experience. Importantly, they found this enhanced processing efficiency persisted even without continued practice. These authors suggest that early music experience induces lasting neuroplastic changes that fine-tune the central auditory system for improved subsequent processing throughout the lifespan. With similar age of onset for musical experience in our sample, those more advanced in age would have amassed additional auditory experience allowing for more time to capitalize on the neuroplastic and functional benefits associated with musical experience. The improved processing efficiency with age cannot be attributed to older Ms simply having more experience than younger Ms because the participant with the most extensive experience (21 years) was also the youngest. Furthermore, our findings provide new evidence to suggest brief to moderate musical experience attenuates auditory neural aging effects, as several of our participants received only minimal experience (i.e., < 5 years). Recent studies confirm the benefits of short-term musical experience on neural encoding and perception of normal and even noise-degraded speech in older adults.[27,61,63]

The earlier cortical ERP latencies in older adults with prior musical experience suggest that these individuals exhibit more efficient sensory encoding of auditory inputs. P1 and N1 reflect the initial registration of acoustic stimuli within primary and secondary auditory cortices.[33,64,65] Because these components dominantly reflect exogenous stimulus properties,[33,65] shorter ERP latencies in Ms imply that musical experience improves initial cortical processing efficiency for complex stimulus features. The age-related increases in IPLs (N1–P2 latency) in those with musical experience appear to result from reduced latencies mainly in N1 (rather than P2). The lack of group differences in P2 latency might be expected given this wave indexes stimulus classification[30] and no group differences were noted in behavioral categorization. In sum, our ERP latency data suggest that older adults with some degree of musical experience processed speech sounds more efficiently than their peers with no prior musical experience, allowing more time for the perceptual decision-making process. Still, RTs were similar (though in the correct direction) between groups, so the behavioral relevance of these neural effects remains speculative. For future studies, it would be interesting to evaluate whether changes in cortical IPLs relate to perceived effort in speech perception or participants' confidence in response selection. For example, musical experience might make speech processing less demanding in cognitively at-risk older adults even if it does not yield strong advantages at the behavioral level.[66,67]

We found no group differences in cortical amplitude measures, which suggests that latencies may serve as a more sensitive measure of neuroplastic effects in individuals at risk for MCI. This result is consistent with previous studies showing differences in cortical responses between individuals with and without musical experience in later auditory potentials (i.e., >100 ms).[68–70] That the impact of musical experience is restricted to cortical responses replicates prior work, suggesting that musical training does not influence the robustness of the steady-state FFR in older adult listeners.[19,71] Alternatively, it is possible that these differences, if present, were not detected due to the study being underpowered.

Previous studies have separately focused on the impact of MCI or musical experience on auditory processing in older adults. Here, we examined the combined effects of musical experience in individuals at risk for MCI to directly assess the interaction between these positive (experience-dependent; music) and maladaptive (putative MCI) forms of plasticity. Our results bolster the novel utility of speech FFRs as an index of monitoring cognitive decline in individuals with borderline clinical symptomology.[12] Additionally, our findings provide new evidence that musical engagement might counteract typical age-related declines in temporal precision in auditory cortical processing, resulting in more efficient speech processing in older adults with putative MCI. While our findings await replication in a larger sample, our cross-sectional data are promising in that it opens the door for future training studies examining music intervention as a potential solution for mitigating auditory-perceptual declines due to cognitive aging.

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AUTHOR CONTRIBUTIONS

G.M.B. designed the experiment. C.N.P. and G.M.B. acquired and analyzed the data, interpreted the results, and contributed to the writing of the manuscript. Both authors accept responsibility for the integrity of the data analyzed in this study. Requests for materials should be addressed to G.M.B [gbidel@indiana.edu].

COMPETING INTERESTS

The authors declare no competing interests.

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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