

SHORT REPORT

Post-task modulation of resting state EEG differentiates MCI patients from controls

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Abstract

Background: Early identification of cognitive decline is critical for identifying individuals for inclusion in clinical trials and for eventual care planning.

Methods: A sample (ages 60–90 years) of consensus-diagnosed, community-dwelling Blacks (61 cognitively typical [HC], 28 amnesic mild cognitive impairment [aMCI], and 14 nonamnesic MCI [naMCI]) were recruited from the Michigan Alzheimer's Disease Research Center and the Wayne State University Institute of Gerontology. Participants received two resting state electroencephalograms (rsEEG, eyes closed) between which they engaged in a visual motion direction discrimination task. rsEEG %change current source densities across all frequency bands and regions of interest were calculated.

Results: EEG current density was not different across groups for pre-task resting state. However, compared to HC, aMCI showed significantly greater declines at temporal and central cortical sites, while naMCI showed significant parietal declines.

Conclusion: This novel approach of post-pre/cognitive challenge rsEEG successfully discriminated older persons with MCI from those without was sensitive to cognitive decline.

KEYWORDS

EEG markers, mild cognitive impairment, neuropsychology, resting-state electroencephalography (rsEEG)

1 | INTRODUCTION

Developing assessments that are sensitive to pre-clinical cognitive decline and neural dysfunction, before frank Alzheimer's disease (AD) pathology, is critical for the study of neurodegenerative mechanisms and interventions to promote cognitive resiliency. The present study investigates if cognitive engagement changes resting state electroencephalogram (rsEEG) cortical activity and if the pattern of change may identify persons with mild cognitive impairment (MCI), a pre-clinical stage to AD.

There is constant formation and dissolution of resting-state patterns involved in functional and coherent network configurations around a more deterministic anatomical structure.¹ With respect to diagnosing MCI, the rsEEG research mainly focuses on abnormalities in the frequency and topographical features of rsEEG rhythms to unveil neural dysfunctions.² MCI patients show global "slowing" of the baseline EEG (i.e., EEG containing more low-frequency power; for review see Babiloni et al.² and Vecchio et al.³). Other studies^{4,5} showed a reduced level of functional connectivity in patients with MCI. We also reported that older Blacks with MCI had reduced functional

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TABLE 1 Demographic information and computer anxiety scores of our participants

Demographic	Controls		aMCI		naMCI		P
	M	SD	M	SD	M	SD	
Sex							
Females (N, %)	53 (91%)		23 (81%)		12 (92%)		
Males (N, %)	5 (9%)		5 (19%)		1 (8%)		
Age	71.10	6.18	74.04	6.89	73.08	8.03	.22
Education	15.26	2.34	14.22	2.36	14.85	2.64	.18
GDS	.81	1.19	.65	.78	1.11	1.54	.59

Abbreviations: aMCI, amnesic mild cognitive impairment; GDS, Geriatric Depression Scale; naMCI, non-amnesic mild cognitive impairment; SD, standard deviation.

HIGHLIGHTS

- Resting state electroencephalographic (rsEEG) measures offer promise as biomarkers.
- We compared rsEEG density immediately prior to and after a cognitive task.
- Post-pre rsEEG% changes discriminate controls from amnesic and non-amnesic mild cognitive impairment (MCI).
- Within a Black community, this provided reliable MCI characterization.

RESEARCH IN CONTEXT

1. **Systematic review:** Early recognition of cognitive changes is critical in identifying those persons at risk for mild cognitive impairment (MCI) and dementia. Resting state electroencephalographic (rsEEG) measures offer promise as potential neurophysiological biomarkers for Alzheimer's disease (AD).
2. **Interpretation:** Comparison of rsEEG cortical activity immediately prior to and after a challenging cognitive task may effectively identify persons with MCI, a pre-clinical stage of AD. Compared to cognitively normal participants, persons with MCI showed significantly greater post-pre rsEEG % changes in specific cortical areas important for memory and problem solving.
3. **Future directions:** This novel approach of post-pre rsEEG comparisons after cognitive challenge may discriminate older Blacks at heightened risk for MCI from those with still preserved cognition. Approaches such as this that are portable and readily accepted may provide important information for community-based recruitment in clinical trials or better target those persons needing more invasive testing or imaging.

connectivity and brain topology as compared to cognitive typical adults.⁶ These investigations highlight the use of rsEEG to detect differences in functional architecture between MCI and controls; however, greater sensitivity to detect pre-clinical cognitive decline may be evident in functional changes after cognitive engagement. Lingering brain activity after a task is expected to interfere with subsequent task accuracy and speed, as suggested by dual-task interference,⁶ and cognitive deficits in MCI may in part be related to slowed disengagement and return to baseline function.

Based upon the reviewed literature, we hypothesized that MCI will show slower return to baseline compared to controls, that is, greater relative percent change in post-cognitive engagement rsEEG spectral current source density (CSD). In line with the defining diagnostic feature of amnesic complaints, we expected to observe greater differences in percent change of rsEEG CSD between persons with amnesic MCI (aMCI) and controls in the temporal cortices. Deficits related to non-amnesic MCI (naMCI) may be more variable, and we expected differences across regions outside of the temporal cortex. We test these hypotheses in a community sample of older Blacks.

2 | METHODS

2.1 | Subjects

We recruited 87 Black participants from the greater Detroit area (see Table 1 for demographics of the participants) who endorsed a change in

memory or other cognitive functions over the past year. All participants underwent the National Alzheimer's Disease Coordinating Centers (NACC) Uniform Data Set (UDS) assessment battery, including standardized medical, neurological, and neuropsychological components. Consensus conference diagnosis followed NACC criteria (https://www.alz.washington.edu/WEB/researcher_home.html; UDS Form D1: Clinician Diagnosis), diagnosing 58 participants as normally functioning (HC), and 41 MCI. UDS further characterizes both aMCI (N = 28) and naMCI (N = 13) based on established criteria⁷ and reflecting whether a MCI participant meets UDS criteria for an amnesic disorder in addition to whatever other cognitive domains were affected within the MCI diagnostic pattern. All participants provided written informed consent and procedures were approved by university institutional review boards.

2.2 | EEG recordings

Brain vision (Brain Vision, Inc.) equipment was used to record scalp EEG activity with high-density Acti Cap (64 active electrodes), modified according to the International 10-20 System. The recording locations included the FCz electrode as an online reference and the AFz electrode at midline location as a ground. Filter settings for low and high pass were 70 Hz and 0.1 Hz, respectively. For these filters the cut-off frequencies were set at 3 dB down; the rolloff was 12 dB per octave at both sides. We maintained impedances below 10 k Ω for each channel and within a 5 k Ω range balance across all channels. The sampling rate was 500 Hz with 32 bit resolution.

We recorded baseline EEG twice: (1) at the beginning of the EEG session, and (2) at the end of EEG session, immediately after a participant performed motion direction discrimination task. After proper placing of the electrode cap with 64 electrodes and obtaining satisfactory impedances, the participant was seated behind the desk in a comfortable chair, adjusted for height, in a dimly lit room. Initially, we recorded rsEEG for at least 3 minutes with eyes closed, followed by 3 minutes with eyes open. Here we present spectral analyses obtained only with eye-closed baseline EEG recordings before and after motion direction discrimination task to be as close as possible to the true baseline.⁸ Barry et al.⁹ showed that during the eyes-open, as compared to eyes-closed, mean activity in the delta, theta, alpha, and beta bands are reduced across sites.

2.3 | Experimental procedures

We performed computerized testing and EEG recordings in a community center at the University of Michigan (UM) Detroit Center. We evaluated several available spaces with a Gauss meter prior to EEG recording to find the area with the least external noise (preferably <.3 mG) to obtain acceptable EEG signal. Active electrodes also were used to additionally isolate external noise, to minimize cable movement artefacts, and to keep impedances at bellow 10k Ω .¹⁰

2.4 | EEG data analyses

Analyzer 2 (Brain Vision, Inc.) was used for pre-processing of the baseline EEG data following the recommendations from the OHBM COBIDAS MEEG committee.¹¹ Off-line was used for inspection to identify and remove segments of EEG contaminating either excessive noise, saturation, or lack of EEG activity. We segmented cleaned EEG data in consecutive epochs of 2 seconds and analyzed off-line (1024 data points; 0.488 Hz resolution; Hanning window). An automatic computerized procedure using a rejection criterion of +/- 100 mV on any channel affected by artifacts (muscular, instrumental) was used to identify acceptable epochs. The artifact-free segments were additionally detrended and baseline corrected before averaging. On average we obtained 90 (range, 64–115) 2-second artifact-free segments per subject to perform fast Fourier transform (FFT) analyses to evaluate power in major frequency bands: delta (2–4 Hz), theta (4–8 Hz), alpha (8–

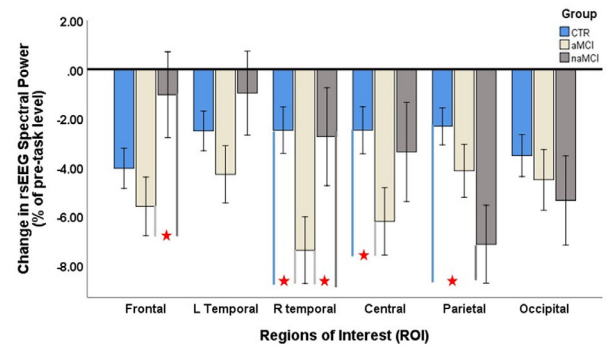


FIGURE 1 Overall effects of motion direction discrimination task on post as compared pre resting state electroencephalogram (rsEEG). Effect estimates are post versus pre % change in EEG current source density for six different cortical regions separate for controls (HC), amnesic mild cognitive impairment (aMCI), and nonamnesic MCI (naMCI). ★ indicates statistically significant pairwise comparison

12 Hz), and beta (12–30 Hz). Frequencies > 30 Hz were not included in power analyses due to contamination of pericranial musculature electrical activity of scalp-recorded neuroelectric activity.¹² To reduce the number of recording sites for further analysis we designated a total of six regions of interest (ROIs; Figure 1): frontal—F (Fp1, Fp2, AF3, AF7, AF4, AF8, F7, F5, F3, F1, Fz, F2, F4, F6, F8, Fc5, FC3, FC1, FC2, FC4, FC6), left temporal—LT (FT9, FT7, T7, TP7, TP9) and right temporal—RT (FT10, FT8, T8, TP8, TP10), central—C (C5, C3, C1, Cz, C2, C4, C6, CP5, CP3, CP1, CPz, CP2, CP4, CP6), parietal—P (P7, P5, P3, P1, Pz, P2, P4, P6, P8, PO7, PO3, POz, PO4, PO8), and occipital—Occ (PO9, O1, Oz, O2, PO10). Before calculating absolute power for each ROI, EEG epochs were transformed into the reference-free CSD distribution, which removes nearly all volume conduction effects.¹³

2.5 | STATISTICAL PROCEDURES

SPSS for Windows (release 22.0.0) was used for data analyses. Initially, all the measures were evaluated for normality using a Shapiro–Wilk test. As expected, all the EEG spectral power measures were skewed and a log transformation was applied. The primary hypothesis was tested as a 6 (ROI) \times 4 (Band) \times 3 (Group) repeated-measures general linear model that tested MCI-related difference in spectral power. The analysis was made of measures taken prior to the task, as well as in percentage change $([(\text{post} - \text{pre}) / \text{pre}] \times 100)$ in rsEEG after cognitive engagement. Age, years of education, and sex were included as covariates in the models. Significant omnibus effects were evaluated with paired comparisons between groups by region, adjusted for covariates.

3 | RESULTS

3.1 | Demographic data

Demographic information and computer anxiety scores of our participants are presented in Table 1. As expected, there was no difference between HC, aMCI, and naMCI subjects in terms of age and education, nor Geriatric Depression Scale (GDS).

TABLE 2 Resting state eyes-closed EEG CSD spectral powers for pre- and post-cognitive engagement for controls (HC), amnesic MCI (aMCI), and nonamnesic MCI (naMCI)

Group ROI	Controls		aMCI		No-aMCI	
	Pre (M, SD)	Po (M, SD)	Pre (M, SD)	Po (M, SD)	Pre (M, SD)	Po (M, SD)
<i>Frontal</i>						
δ	4.86 (0.63)	4.60 (0.64)	4.91 (0.66)	4.55 (0.56)	4.77 (0.57)	4.71 (0.74)
θ	4.48 (0.83)	4.00 (0.77)	4.71 (0.72)	4.06 (0.60)	4.32 (0.74)	4.11 (0.77)
α	4.13 (1.02)	4.06 (1.04)	4.18 (0.70)	4.13 (0.68)	4.16 (1.03)	4.06 (0.60)
β	4.77 (0.81)	4.77 (0.76)	4.79 (0.61)	4.69 (0.65)	4.85 (0.77)	4.92 (0.75)
<i>L temporal</i>						
δ	5.04 (0.78)	4.82 (0.76)	5.10 (0.73)	4.80 (0.58)	5.08 (0.76)	5.09 (0.85)
θ	4.77 (0.92)	4.41 (0.85)	4.99 (0.74)	4.50 (0.66)	4.75 (0.70)	4.56 (0.76)
α	4.76 (1.02)	4.76 (0.98)	4.74 (0.74)	4.72 (0.73)	4.67 (0.91)	4.66 (0.93)
β	4.97 (0.87)	5.02 (0.78)	5.06 (0.62)	4.98 (0.73)	5.11 (0.92)	5.15 (0.83)
<i>R temporal</i>						
δ	5.12 (0.70)	4.95 (0.68)	5.21 (0.72)	4.79 (0.62)	4.87 (0.75)	4.67 (0.79)
θ	4.83 (0.89)	4.46 (0.82)	5.05 (0.77)	4.39 (0.68)	4.46 (0.75)	4.21 (0.79)
α	4.71 (0.98)	4.71 (0.97)	4.76 (0.75)	4.57 (0.84)	4.47 (0.93)	4.43 (0.95)
β	5.03 (0.80)	5.00 (0.75)	5.04 (0.67)	4.74 (0.87)	4.93 (0.70)	4.89 (0.59)
<i>Central</i>						
δ	4.66 (0.83)	4.49 (0.83)	4.76 (0.88)	4.34 (0.75)	4.61 (0.77)	4.41 (0.70)
θ	4.45 (0.98)	4.11 (0.95)	4.65 (0.85)	4.21 (0.80)	4.32 (0.93)	4.02 (0.85)
α	4.50 (1.07)	4.49 (1.09)	4.74 (0.93)	4.60 (1.02)	4.47 (1.27)	4.37 (1.19)
β	4.84 (0.90)	4.83 (0.89)	4.88 (0.68)	4.65 (0.76)	4.89 (0.91)	4.74 (0.80)
<i>Parietal</i>						
δ	4.71 (0.74)	4.57 (0.72)	4.75 (0.75)	4.45 (0.70)	4.75 (0.65)	4.40 (0.72)
θ	4.60 (0.86)	4.38 (0.85)	4.67 (0.81)	4.49 (0.86)	4.60 (0.77)	4.21 (0.88)
α	5.14 (1.02)	5.09 (1.05)	5.20 (1.09)	5.10 (1.15)	4.96 (1.22)	4.71 (1.36)
β	5.06 (0.80)	4.97 (0.81)	5.04 (0.73)	4.80 (0.93)	5.17 (0.79)	4.77 (0.79)
<i>Occipital</i>						
δ	4.74 (0.71)	4.57 (0.75)	4.70 (0.76)	4.43 (0.66)	4.80 (0.82)	4.59 (0.86)
θ	4.63 (0.77)	4.39 (0.82)	4.64 (0.78)	4.43 (0.77)	4.72 (0.81)	4.39 (1.01)
α	4.96 (0.92)	4.82 (0.97)	4.95 (0.95)	4.80 (0.99)	4.93 (1.19)	4.71 (1.30)
β	5.31 (0.83)	5.12 (0.82)	5.40 (0.76)	5.10 (0.95)	5.37 (0.79)	5.11 (0.84)

Abbreviations: ROI, region of interest; SD, standard deviation.

3.2 | Resting state EEG spectral power before cognitive engagement

Resting state EEG spectral power measurements before and after cognitive engagement are presented in Table 2 separately for normal controls, aMCI, and naMCI.

Resting state—eyes closed—EEG spectral powers were entered into mixed $6 \times 4 \times 3$ mixed analysis of covariance with ROI (6 levels—F, LT, RT, C, P, Occ), Band (4 levels—delta, theta, alpha, beta) as within-subjects variables and Group (controls, aMCI, naMCI) as between-subjects variable. Sex, age, and education were included as covariates. Analyses did not reveal any group differences ($F [2,93] = .24$,

$P = .787$), or between-group \times ROI interaction (Wilks' $\lambda [10,178] = .62$, $p = .792$), Group \times Band interaction (Wilks' $\lambda [6,182] = .96$, $P = .451$), or Group \times ROI \times Band interaction (Wilks' $\lambda [30,158] = 1.20$, $P = .238$) in rsEEG before cognitive engagement.

3.3 | Change in resting state EEG spectral power after cognitive engagement

However, significant group differences were evident in the percent change in rsEEG after cognitive engagement. The results of the multivariate analysis showed the main effect of group ($F [2,92] = 3.45$,

TABLE 3 Pre versus post task % change of resting state EEG spectral power for controls, aMCI, and naMCI and the between-group comparisons

ROI	Group means						Between-group comparisons					
	HC		aMCI		naMCI		HC vs. aMCI		HC vs. naMCI		aMCI vs. naMCI	
	M	SD	M	SD	M	SD	t-test	d	t-test	d	t-test	d
Frontal	-4.05	6.10	-5.60	7.14	-1.04	5.40	1.05	.23	-1.64	.46	-2.05	.72
L temporal	-2.52	5.34	-4.30	7.37	-0.97	5.31	1.22	.28	-0.88	.28	-1.45	.52
R temporal	-2.49	7.49	-7.39	7.66	-2.76	4.37	2.82	.65	0.12	.04	-2.03	.74
Central	-2.49	7.39	-6.22	7.26	-3.38	6.68	2.20	.51	0.40	.13	-1.19	.41
Parietal	-2.33	5.16	-4.16	5.13	-7.15	8.80	1.54	.36	2.64	.67	1.38	.38
Occipital	-3.52	6.73	-4.27	6.05	-5.36	6.87	0.66	.12	0.88	.27	0.40	.17

Note: Group means and between-group comparisons are reported, including an estimate of effect size (Cohen's *d*). Significant between-group differences ($P < 0.05$) are in bold.

Abbreviations: aMCI, amnesic mild cognitive impairment; EEG, electroencephalogram; HC, healthy control; naMCI, nonamnesic mild cognitive impairment; ROI, region of interest; SD, standard deviation.

$P = .036$) and significant interaction effect group \times ROI (Wilks' λ [10,176] = 2.45, $P = .009$) to indicate groups differed overall and the magnitude of the difference varied across cortical regions. There was no effect of band (Wilks' λ [3,90] = 1.64, $P = .186$) or an interaction with group (Wilks' λ [6,180] = .41, $P = .871$) or region (Wilks' λ [15,78] = .96, $P = .503$), therefore further post hoc analysis evaluated between-group differences by region in the average signal across bands. Post hoc analyses demonstrated differences between MCI and HC across ROIs (see Table 3 and Figure 1). Patients with aMCI showed significantly greater decrease of rsEEG in right temporal and the central ROIs as compared to HC ($t = 2.82$, and $t = 2.20$, respectively). Patients with naMCI showed significantly greater decrease of rsEEG in the parietal region compared to HC ($t = 2.64$). There was also statistically significant decrease of rsEEG in the frontal and right temporal ROIs in aMCI compared to naMCI ($t = 2.05$, and $t = 2.03$, respectively).

4 | DISCUSSION

Our results provide exploratory evidence for changes in cortical modulation as indexed by changes in rsEEG after cognitive engagement in HC, aMCI, and naMCI older Blacks. Results showed significant decrease in the power of rsEEG after a short motion direction discrimination task, as compared to rsEEG before the perceptual task. After the cognitive task, older adults with aMCI compared to HC displayed greater decreases in rsEEG in right temporal and central cortical regions, while naMCI showed a similar pattern only in parietal cortical region. Older adults with aMCI compared to naMCI showed greater change in rsEEG measured over the frontal and right temporal regions.

At present, we can only speculate as to the origin of the greater decrease in rsEEG at post-task in MCI compared to HC. To the extent that the task engaged the cerebral cortex, greater decreases in rsEEG after the task may indicate MCI patients are slower to return to a baseline, or default, state. The observed pattern may be consistent with the hypothesis that cognitive deficits in MCI are in part attributed to lin-

gering functional activation, which diminishes cognitive efficiency and increases the likelihood for error. The differentiation of brain regions is consistent with typical clinical features of MCI: it is plausible that variability in post-task activation within the temporal cortices would contribute to declarative memory deficits in aMCI. The differentiation of the naMCI from HC regarding significant decrease of rsEEG over the parietal region is quite perplexing. The heterogeneous patterns of brain atrophy and brain activity in naMCI compared to aMCI and controls has been well established and further supported by our findings: the regional pre-post changes of rsEEG for naMCI are not as uniform as for aMCI and controls: naMCI showed the peak of % change at parietal ROI while the minimal change was over the frontal temporal sites. To the best of our speculation we assume that in naMCI slowed return rsEEG to baseline at parietal ROI is due to impaired cortical activity resulted from parietal cortical atrophy, white matter lesions, and increased AD neuropathology at this region. The sources of the variability in post-task rsEEG will require further study. For example, slowed return to baseline state may be due to persistent activation of task-based functional networks, slowed re-activation of resting state networks, or possibly new cognitive engagement related to self-appraisal of past task performance.

Somewhat surprisingly, the results did not show significant difference among the three groups for pre-task rsEEG. This finding is contrary to several reports of MCI patients displaying decreased EEG spectral power compared to HC, primarily in lower frequency bands (for review see Babiloni et al.¹). However, pre-clinical functional deficit and AD-related neuropathology may be more evident after the challenge of a task, and the recovery to baseline rsEEG appears to be a sensitive index to differentiate MCI from HC. This initial report warrants additional study with a larger, independent sample to determine whether changes in rsEEG CSD in specific cortical regions after cognitive engagement may be a sensitive biomarker to identify and classify persons with aMCI and naMCI. After further validation, this approach may be useful for clinical screening and community-based research.

In sum, the results of the present study demonstrate that repeated short rsEEG recording could be an effective EEG marker of cognitive deficit in older adults. Our present study indicates that the task-related alterations in rsEEG can discriminate those older Blacks with MCI from those with age-typical cognition. Furthermore, this study also attests to the feasibility of combined rsEEG as a sensitive recording technique for probing cognitive-induced changes in brain activity. Future research using this technique in conjunction with systematic manipulation of specific cognitive engagement dose and modality parameters will no doubt lead to an increased understanding of the complex rsEEG dynamics. Future studies combining both behavioral and rsEEG outcome measures aiming to probe the effects of cognitive engagement on cortical network activity will no doubt aid scientific understanding as well as clinical relevance to use pre-post task EEG modulation approach.

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CONFLICTS OF INTEREST

The authors declare no conflict of interest with respect to this research, authorship, and/or the publication of this article.

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