Periodic And Aperiodic Electroencephalographic Components During The Transition From Quiet Wakefulness To Light Sleep In Alzheimer's Disease Patients With Mild Cognitive Impairment

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Background

Alzheimer's disease dementia (**ADD**) is characterized by prominent disruptions in the periodic component of resting-state eyes-closed electroencephalographic (**rsEEG**) rhythms, particularly within the alpha band (8–12 Hz), which is closely linked to cortical vigilance networks[1]. In contrast, early evidence suggests that the aperiodic, non-oscillatory rsEEG component[2] (**Figure 1**), reflecting overall cortical arousal, remains relatively unaffected[3].

This exploratory study aimed to determine whether similar spectral alterations are already detectable in the prodromal phase of Alzheimer's disease, namely in patients with mild cognitive impairment due to AD (**ADMCI**), and whether such changes can be observed during vigilance transitions from quiet wakefulness to light sleep in daytime EEG recordings[4].

Periodic and Aperiodic EEG Components

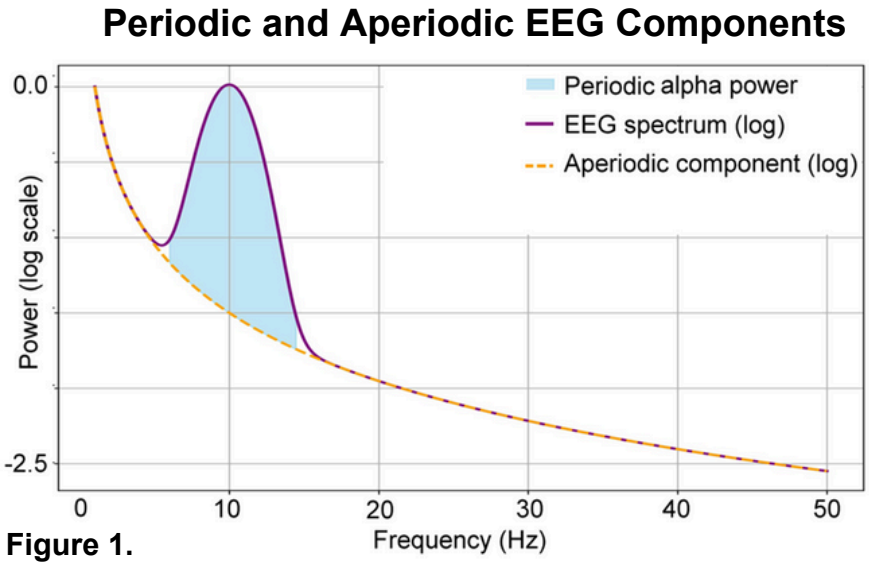

Figure 1.

Methods

As part of the **PREDICT-NEURODEGEN** project, clinical, demographic, and prolonged (~30 minutes) resting-state EEG (rsEEG) recordings were obtained from 19 ADMCI patients (mean age: 69.8 ± 1.3, 10 Females) and 18 age- and sex-matched cognitively unimpaired older adults (Nold; mean age: 65.5 ± 1.6, 10 Females). As reported in **Table 1**.

Demographic and clinical data in ADMCI and Nold participants (mean ± SE)

	ADMCI (N = 19)	Nold (N = 18)	Statistical test
Age	69.8 ± 1.3	65.5 ± 1.6	T-test: *n.s.*
Sex (F/M)	10/9	10/8	χ^2: *n.s.*
Education (years)	13.1 ± 1.1	15.4 ± 0.6	T-test: *n.s.*
MMSE	26.4 ± 0.7	29.4 ± 0.3	Mann-Whitney U test: $p < .001$
GDS	2.9 ± 0.6	2.6 ± 0.7	T-test: *n.s.*

Table 1. Demographic and clinical characteristics (mean ± standard error of the mean) of the patients with mild cognitive impairment due to Alzheimer's disease (ADMCI) and the cognitively unimpaired older (Nold) control participants. Legend. ADMCI: mild cognitive impairment due to Alzheimer's disease; Nold: cognitively unimpaired older adults; GDS: 30-item Geriatric Depression Scale; MMSE: Mini-Mental State Examination.

Vigilance stages were visually scored across the 30-minute rsEEG recordings, identifying EEG epochs dominated by alpha activity (wakefulness) and by theta-dominant light sleep (ripples), based on a previously validated procedure. EEG signals were processed using the EEGLAB toolbox in MATLAB, and periodic and aperiodic components were estimated via the FOOOF algorithm.

Results

Compared to Nold controls, ADMCI participants exhibited significantly reduced reactivity of periodic alpha power between wakefulness and ripples across the entire scalp (Group × Stage interaction, $p < .001$), as well as lower alpha power during wakefulness (**Figure 2**). Conversely, the aperiodic slope increased significantly from wakefulness to ripples in both groups (Stage main effect, $p < .001$), with no significant differences by group or scalp region. Differential scalp maps of periodic alpha power (**Figure 3a**) and aperiodic component (**Figure 3b**) are reported below.

EEG Spectral Components

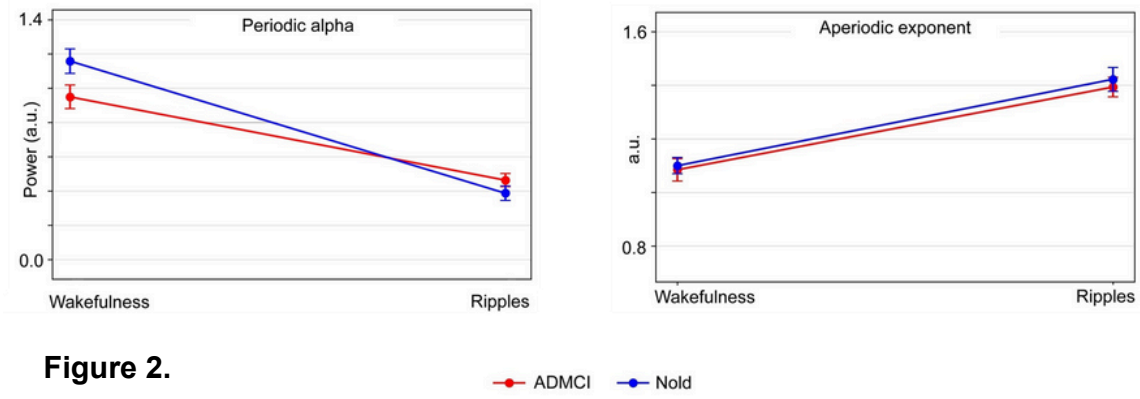

Figure 2.

Periodic Alpha Power Differential Maps

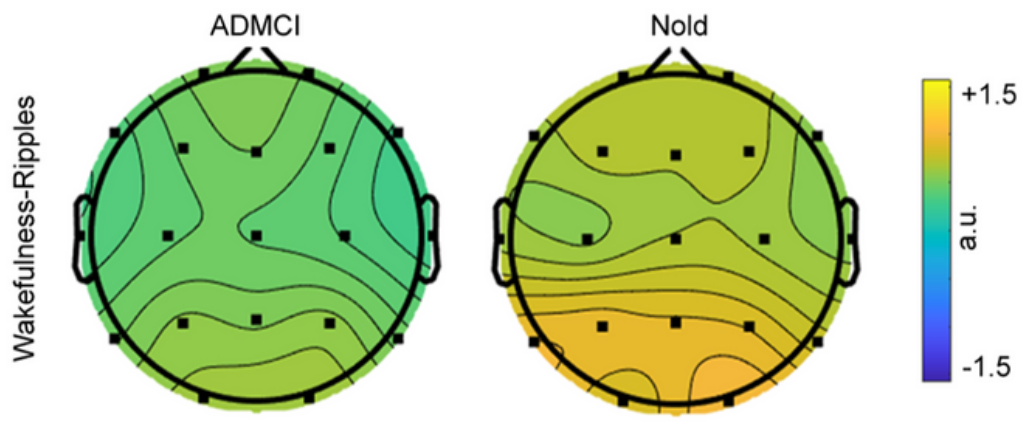

Figure 3a

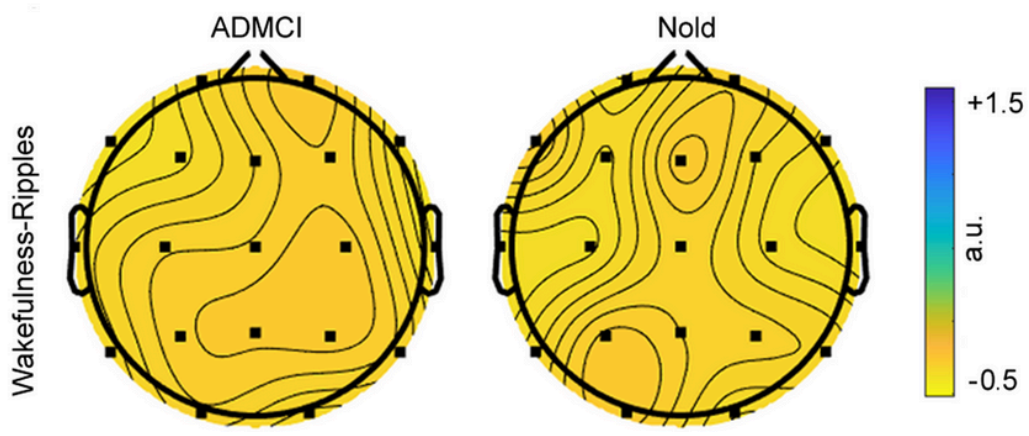

Figure 3b.

Conclusions

These preliminary findings indicate that periodic rsEEG alpha activity is already disrupted in the prodromal phase of Alzheimer's disease, suggesting early thalamocortical dysregulation. In contrast, aperiodic, non-oscillatory dynamics associated with global cortical arousal appear preserved across wake-sleep transitions in ADMCI.

References

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