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TREATMENT EFFECTS ON EVENT-RELATED EEG POTENTIALS AND OSCILLATIONS IN ALZHEIMER'S DISEASE

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ABBREVIATIONS

Aβ42: Amyloid beta 42 peptide

ACh: Acetylcholine

AChEIs: Acetylcholinesterase inhibitors

ADD: Alzheimer's disease dementia

ASSR: Auditory steady state responses

CSF: Cerebrospinal fluid

EEG: Electroencephalography

ERPs: Event-related potentials

EROs: Event-related oscillations

fMRI: Functional magnetic resonance imaging

FDG-PET: Fluorodeoxyglucose positron emission tomography

MEG: Magnetoencephalography

ADMCI: Mild cognitive impairment due to Alzheimer's disease

MRI: Magnetic resonance imaging

NMDA: N-methyl-D-aspartate

p-tau: Phospho-tau protein

TMS: Transcranial magnetic stimulation

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ABSTRACT

Alzheimer's disease dementia (ADD) is the most diffuse neurodegenerative disorder belonging to mild cognitive deficits (MCI) and dementia in old persons. This disease is provoked by an abnormal accumulation of amyloid-beta and tauopathy proteins in the brain. Very recently, the first disease-modifying drug has been licensed with reserve (i.e., Aducanumab). Therefore, there is a need to identify and use biomarkers probing neurophysiological underpinnings of human cognitive functions to test the clinical efficacy of that drug. In this regard, event-related electroencephalographic potentials (ERPs) and oscillations (EROs) are promising candidates. Here, an Expert Panel from the Electrophysiology Professional Interest Area of the Alzheimer's Association and Global Brain Consortium reviewed the field literature on the effects of the most used symptomatic drug against ADD (i.e., Acetylcholinesterase inhibitors) on ERPs and EROs in ADD patients with MCI and dementia at the group level. The most convincing results were found in ADD patients with dementia. In those patients, Acetylcholinesterase inhibitors partially normalized ERP P300 peak latency and amplitude in oddball paradigms using visual stimuli. In these same paradigms, those drugs partially normalized ERO phase-locking at the theta band (4-7 Hz) and spectral coherence between electrode pairs at the gamma (around 40 Hz) band. These results are of great interest and motivate multicentric, double-blind, randomized, and placebo-controlled clinical trials in ADD patients with MCI and dementia for final cross-validation.

Keywords: EEG, oscillations, event-related, Alzheimer, treatment, mild cognitive impairment

1. INTRODUCTION

Increased expectancy of life leads to the growth of the aged population and the increase of cases of dementia, defined as severe cognitive deficits associated with loss of autonomy and disabilities in the activities of daily living. Around 50 million people suffer from dementia worldwide, and its cost reaches up to a trillion US dollars annually (WHO Guidelines, 2019; Alzheimer's Association, 2021).

Alzheimer's disease dementia is a progressive neurodegenerative disease that represents the most common cause of age-related dementing illnesses (World Alzheimer Report 2020). Its pathological changes start as an abnormal accumulation of amyloid-β and tau proteins in the brain several years earlier than the first objective clinical manifestations (Insel et al., 2021). Along the continuum of the clinical manifestations of ADD over time, amnestic mild cognitive impairment (MCI) with preserved autonomy in the activities of daily living is considered as a frequent clinical milestone among the clinical predementia stages (Albert et al., 2011).

Scientific advance on AD has been revolutionized by biomarkers and transformed treatment practices. Various biomarkers for indicating neurodegeneration and pathologies related to abnormal deposited peptides, namely amyloid-beta or tau, have been suggested as objective measures to reflect underlying pathophysiology (Jack et al., 2016; Ehrenberg et al., 2020; Jack et al., 2018; Dubois et al., 2016).

The criteria mentioned above define the presence of insidiously developing dementia with core symptoms of episodic memory and/or impairment of other cognitive domains along with biomarkers with a classification so-called ATN, derived from acronyms of two deposited peptides, Amyloid-beta and Tau, and Neurodegeneration (Jack et al., 2018). Amyloid beta and tau peptides can be detected by ligand-based positron emission tomography techniques (Janelidze et al., 2017; La Joie et al., 2020) or by a lumbar puncture to detect their levels in the cerebrospinal fluid (CSF) (Blennow and Zetterberg, 2018). Neurodegeneration can be demonstrated by brain atrophy in structural magnetic resonance imaging (Barthélemy et al., 2020) or a low level of glucose uptake indicated by FDG-positron emission tomography (Leuzy et al., 2019). The usefulness of these valid biomarkers in routine clinical use is still arguable, not only because of their invasiveness and cost, but also because of their limited specificity and sensitivity rates (Isaacs and Boenink, 2020). Very recently, plasma-based fluid markers have

entered the scene, so now, amyloid-beta, p-tau, and NfL can be measured reliably in both CSF and blood (Ashton et al., 2021). Plasma-based measures of P-tau, either phospho-tau217 (Palmqvist et al., 2020) or phospho-tau181 (Janelidze et al., 2020), show particular promise reflecting levels of the pathological precipitations in the CSF or brain. They have been announced to provide diagnosis rates of 85% and 98%, respectively, and cross validated by PET, genetic status, or CSF studies (Mielke et al., 2018; Leuzy et al., 2020; Karikari et al., 2020; Palmqvist et al. 2020). Currently, the use of the latest plasma biomarkers awaits confirmation in larger trials and their incorporation into guidelines, and all of the above mentioned methods relating to pathological features of ADD cannot be applied widely in clinical settings.

Unfortunately, there are no drugs able to cure the disease nowadays. Concerning the licensed symptomatic treatments for ADD, cholinesterase inhibitors, memantine, and a combination of a cholinesterase inhibitor and memantine have produced statistically significant but clinically small delays in various domains of cognitive and functional decline in select AD patients with dementia and MCI (Dubois et al., 2015; Schmidt et al., 2015; Matsunaga et al., 2019; Ismail et al., 2020).

In June 2021, the Food and Drug Administration (FDA) approved Aduhelm (aducanumab) for the treatment of ADD as the first disease-modifying drug receiving an approval worldwide (Knopman et al., 2021). The decision taken into an accelerated approval program was mostly based on the significant effects on amyloid-beta with a controversial clinical efficacy. Therefore, there is an urgent need to identify and use biomarkers probing neurophysiological underpinnings of human cognitive functions to test the clinical efficacy of that drug at the group level. In this regard, event-related electroencephalographic potentials (ERPs) and oscillations (EROs) are promising candidates, which are available worldwide, including lower and middle-income countries (Babiloni, Barry, et al., 2020; Babiloni, Blinowska, et al., 2020; Rossini et al., 2020).

Notably, the Steering Committee of the Electrophysiology Professional Interest Area (EPIA) of The Alzheimer's Association International Society to Advance Alzheimer's Research and Treatment (ISTAART; https://www.alz.org/) appointed expert panels for reviewing the scientific literature on ERP and ERO biomarkers in patients with ADD and related disorders to test the hypothesis that those biomarkers may reflect the effects of ADD on cerebral cognitive

systems. The outcome has been recently published (Babiloni et al., 2020, 2021; Güntekin et al., 2021) and is summarized in the following paragraphs.

Event-related EEG measures during oddball tasks may be helpful cognitive neurophysiological biomarkers of intervention clinical trials performed in individuals with AD (Güntekin et al., 2021). Previous studies confirm that validated neuroimaging and multimodal fluid AD biomarkers are significantly associated with event-related EEG responses during oddball tasks (Babiloni et al., 2020, 2021). It is well known that ERPs allow for the study of EEG activity phase-locked to sensory stimuli or motor responses during cognitive tasks. Those potentials are typically computed by averaging artifact-free EEG activity recorded during sensory stimuli using the onset of the stimulus or motor response as a zerotime. The most popular ERP paradigm used in ADD patients is the so-called "oddball task" (Donchin et al., 1973; Polich and Kok, 1995; O'Connell et al., 2012) in which frequent (70-80%) and rare (30-20%) auditory or visual stimuli are delivered, and experimental subjects have to respond to the rare stimuli by pressing a button or counting the stimuli. ERPs following the target stimuli show a parietal ample positivity (P) peaking at ~300-400 ms post-stimulus (P300), the so called P300 or P3b component, reflecting focused attention, decision making, and working memory (Donchin et al., 1973, Polich and Kok, 1995; O'Connell et al., 2012). The P300 amplitude upon rare target stimuli was smaller and its latency longer in ADMCI and ADD patients compared with cognitively unimpaired (CU) subjects (Golob et al., 2009, Polich and Corey-Bloom, 2005). The P300 has been reported as sensitive to AD neuropathology (Morgan and Murphy, 2002). Regarding the role of EEG methodologies in the prediction of clinical presentation, a recent study demonstrated that a reduction of amplitudes of memory target-related ERPs at the left frontal sites heralded an incipient MCI about five years before diagnosis of MCI in cognitively normal older people (Jiang et al., 2021), or changes in ERPs appear 10 years prior to clinical presentation in dominantly inherited ADD patients (Quiroz et al., 2011). Even though the P50 component (positive peak at +50 ms after stimulus) is mainly related to sensory and arousal activities (Yurgil and Golob, 2013), it is also associated with the attentional system and short-term episodic memory functions in neurodegenerative processes through the oddball paradigm (Golob et al., 2009).

There were also alterations in patients with ADD and CU individuals regarding ERP components after linguistic semantic stimuli and repeated words. CU persons had larger N400

amplitude over parietotemporal regions in response to semantically incongruous stimuli (Kutas and Federmeier, 2011). Linking this effect to semantic memory, semantic incongruity caused reduced responses in physiological aging, and they were further reduced or abolished N400 in ADD (Olichney et al., 2006). The late positive component (LPC or P600) is elicited during memory encoding and retrieval processes of words. In CU subjects, P600 displays a significant word repetition effect as an attenuation of their amplitude, while the attenuation of the N400 or P600 after repeated words was lower in ADMCI and predictive for ADD development (Olichney et al., 2002a, Olichney et al., 2006).

The cognitive ERP/ERO components can also monitor disease progression of AD at the group level. Over time, patients with progressive ADMCI demonstrated an increase in P50 amplitude (Papaliagkas et al., 2011; Kimiskidis et al., 2012), in N200 latency (Missonier et al., 2007), and in P300 latency (Lai et al., 2010), while also showing decreases in N200 (Papaliagkas et al., 2011; Kimiskidis et al., 2012) and P600 responses (Olichney et al., 2002b) in comparison with CU individuals. Moreover, diminished N400 during semantic processing (Bobes et al., 2010), and reduced P200, and P300 (Quiroz et al., 2011) during episodic memory task were detected in presymptomatic autopsy-proven older people (Olichney et al., 2013) and carriers of AD mutation (Golob et al., 2009) about 10 years before clinical presentation. It is also suggested that ERPs may be feasible and useful prognostic markers of ADD (Papaliagkas, 2021).

The ongoing EEG activity recorded during cognitive tasks can also be analyzed linearly to explore event-related alterations in power or phase characteristics related to the ongoing oscillatory responses or the event-related synchronization/desynchronization (ERS/ERD) at delta, theta, alpha, beta, and gamma frequency bands (Pfurtscheller and Lopes da Silva, 1999). Cognitive ERPs can be decomposed to unveil the phase-locked EEG delta, theta, alpha, beta, and gamma oscillations named EROs (Başar-Eroğlu & Başar, 1991; Herrmann and Knight, 2001; Neuper & Klimesch, 2006; Lejko et al., 2020). EROs that were elicited after digital filtering or other transformation methods were repeatedly investigated in oddball tasks. Previous EEG studies from independent research teams have consistently demonstrated reduced EROs at delta and theta frequencies in ADMCI and ADD patients over CU seniors during oddball tasks (Karrasch et al., 2006; Güntekin et al., 2008, 2019; Cummins et al., 2008; Yener et al., 2008, 2012; Caravaglios et al., 2008, 2013; Başar et al., 2010; Michalopoulos et al., 2012; Deiber et al.,

2015; Tülay et al., 2020). Patients with ADMCI had also lower theta and beta EROs than individuals with stable MCI (Hedges et al., 2016, Jiang et al., 2015, Missonnier et al., 2007).

In the current article, a multidisciplinary panel of experts aimed to review the literature about the effects of medications or interventions on ERO/ERP EEG oscillations during cognitive tasks.

2. AIMS AND METHODOLOGY

The EPIA Steering Committee formed an expert panel to review the literature and provide recommendations on candidate ERP and ERO measures for characterizing the effects of pharmacological treatments on neurophysiological oscillatory mechanisms in ADD patients with MCI and dementia. The Expert Panel included expert neurologists, psychiatrists, and neurophysiologists from EPIA, Global Brain Consortium (https://globalbrainconsortium.org), and The PDWAVES Consortium (wwwpdwaves.eu). A specific question was addressed to: What is the ERP and ERO measure that most consistently reveal effects of those treatments in ADD patients? To answer, a comprehensive literature search was completed on ERPs and EROs in AD patients with MCI (ADMCI) and dementia (ADD).

The literature search was performed on PubMed and Scopus using the keywords given in the below keywords list. Titles and abstracts were searched from these databases. The last search was conducted on February 7, 2021. Duplicated studies were eliminated as a result of two different database searches.

The keywords for the ADD patients were as follows: “Event-related potential” and treatment and Alzheimer; “Event-related potential” and medication and Alzheimer; P300 and medication and Alzheimer; “Alzheimer’s Disease OR Alzheimer” AND “Event-Related Oscillation” AND “Treatment OR Drug OR Medication”; “Alzheimer’s Disease OR Alzheimer” AND “Evoked Oscillation” AND “Treatment OR Drug OR Medication”; “Alzheimer’s Disease OR Alzheimer” AND “Event-Related Desynchronization OR Event-Related Synchronization” AND “Treatment OR Drug OR Medication.”

The keywords for the MCI patients were as follows: “Event-related potential” and treatment and mild cognitive impairment; “Event-related potential” and medication and mild cognitive impairment; “Mild Cognitive Impairment OR MCI” AND “Event-Related Oscillation”

AND “Treatment OR Drug OR Medication”; “Mild Cognitive Impairment OR MCI” AND “Evoked Oscillation” AND “Treatment OR Drug OR Medication”; “Mild Cognitive Impairment OR MCI” AND “Event-Related Desynchronization OR Event-Related Synchronization” AND “Treatment OR Drug OR Medication”.

Authors (GY, DHG, and EY) independently reviewed the articles to decide related articles for inclusion. In case of indecision, the reviewers discussed and decided on the articles in doubt. After careful revision of the searched articles, only related articles were included in the study. Namely, articles that did not include the treatment-related EEG research on ADD and/or MCI were described as irrelevant articles and not included in the current review. The reference lists of the articles included according to database searches were checked. In the reference lists, if there were studies that did not appear in the database searches but met the related article criteria, they were also included in the study.

The above Authors excluded EEG studies using long stimuli (several hundreds of milliseconds) or where the analysis was not finely time-locked to the event onset. In this line, resting state EEG studies were excluded. Afterward, the mentioned co-Authors produced a first draft of the manuscript circulated to all Panel members. After some rounds of revisions, the Panel reached a unanimous consensus about the findings and recommendations. The manuscript was finalized in December 2021.

The terms and methodological procedures of the reviewed studies do not derive from daily medical practice and were not used for diagnostic, prognostic, or monitoring purposes. Furthermore, the opinions and recommendations of the expert panel do not represent guidelines for the clinical applications to the monitoring of treatments for AD. Indeed, the present methodology did not follow standard procedures typically adopted by international biomedical societies for the review of the medical intervention and practice (e.g., "GRADE", https://gdt.gradepro.org/app/handbook/handbook.html).

In the review of the ERP and ERO studies, we decided to accept those using clinical diagnostic criteria for AD not excluding AD patients with moderate cerebrovascular, non-AD hippocampal impairment (TDP-43), and Lewy body co-pathology. We also used the term ADMCI to denote patients with amnestic MCI even without a diagnosis based on in-vivo biomarkers of AD. It should be also noted that ERP and ERO studies reviewed in the present

paper used heterogeneous procedures for the detection of artifacts in preliminary EEG data analysis.

To our knowledge, this is the first international initiative designed to reach consensus recommendations on the optimal ERP and ERO measures to be used in clinical trials testing treatments for AD patients. We hypothesized that those measures may be sensitive in the detection of the treatment effects at the group level and the outcome may promote the use of them as surrogate neural endpoints for monitoring the neurophysiological effects of drugs for AD on brain cognitive systems. Notably, there are many ongoing Phase 2-3 clinical trials targeting amyloid-beta in symptomatic or asymptomatic familial AD mutation carriers (Cummings, 2021). To our knowledge, none of them use ERPs or EROs in monitorization or even in the development of these pharmacological agents to deliver their earliest reflections on neuronal activity. So far, no review has investigated treatment effects on ERPs/EROs observed in MCI/AD patient groups.

In the following section, each component of ERPs and the pharmacological effects on them will be presented separately. Furthermore, ERPs, their functions, and possible generators are presented in Table 1. Treatment/intervention effects on ERPs in Alzheimer's disease patients are presented in Table 2.

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3. EVENT-RELATED POTENTIALS (ERPS) AND TREATMENT EFFECTS

3.1. Early Components of ERPs and Treatment Effects

3.1.1. Auditory P50

P50 is an early sensory component of auditory evoked potentials in passive listening and is enhanced in amplitude after infrequent over frequent stimuli in oddball tasks (Golob et al., 2009; Grimm et al., 2011; Nakagawa et al., 2014). In previous ERP studies, relative to controls, MCI patients showed larger long-latency P50 amplitudes that evoked potentials in passive

listening (Irimajiri et al., 2005). Furthermore, MCI patients positive to CSF biomarkers of AD exhibited greater P50 amplitude in an auditory oddball task when compared to MCI patients due to other diseases (Green et al., 2015). Notably, that 50 amplitude predicted the levels of amyloid-beta 42 in those ADMCI patients (Green et al., 2015). Notably this effect on auditory P50 may be not modality specific as compared to ADMCI patients receiving a treatment with AChEIs, non-treated ADMCI patients were characterized by larger P50 amplitude in an experiment with passive somatosensory stimulations (Irimajiri et al., 2007). Instead, that effect on early potentials evoked by sensory stimuli evoked may be specific for sporadic AD and MCI status as no modulation of P50 in an auditory oddball task was observed in asymptomatic persons with familial AD carriers for the disease as compared to non-carriers (Golob et al., 2009). In the same line, that effect was not observed in ADD patients (Fruehwirt et al., 2019), possibly due to the progression of the ADD-related neurodegeneration within the P50 cortical generators. The effects of ADD on early evoked potentials may be related to cholinergic tone as a single dose of a muscarinic anticholinergic drug (scopolamine) increased the P50 amplitude following passive listening of auditory stimuli in healthy adults (Pekkonen et al., 2001). This effect may be specific as a single dose of a dopamine D2-receptor antagonist (haloperidol) in healthy adults did not produce significant effects on that P50 during passive listening of auditory stimuli. Keeping in mind the above data and considerations, P50 amplitude following passive listening of auditory stimuli may be an interesting biomarker of the cholinergic system in ADMCI patients.

3.1.2. Visual N70, N150, and N160

The N70 and N150 components of ERPs are typically elicited during visual paradigms involving stimulus pattern reversal, while the N160 component is also associated with early perceptual processing (Morrison et al., 2019). There were only two studies that examined those components in ADD patients. One study used a visual short-term memory (2 n-back) task and showed no differences in N160 amplitude or latency among groups of CU, MCI with stable cognitive status at 1-year follow-up, MCI showing progressive cognitive deficits at 1-year follow-up, and ADD persons (Missonnier et al., 2007). The other study used simple visual stimulus pattern reversals and showed no differences in N70 or N150 amplitudes or latency between ADMCI patients receiving vs not receiving an AChEI treatment (Irimajiri et al., 2007).

Given this lack of significant effects, these visual ERP components seem not to be promising for pharmacological clinical studies in ADD patients.

3.1.3. P100, N100, N170, P200

Concerning P100, N100, and N170, previous studies in ADMCI and ADD patients showed mixed findings, possibly due to their sensitivity to multiple information processes and disease status (Lijffijt et al., 2009).

As compared to CU persons, MCI patients showed increased P100 and N170 amplitude for visual stimuli with familiar vs unfamiliar faces (Saavedra et al., 2012), while ADD patients presented no effect on P100 and decreased N170 amplitude following familial vs unfamiliar faces and scenes (Cheng and Pai, 2010). Other studies showed inconsistent results in ADD patients as increased P100 amplitude and latency in the recognition of emotional face expressions (Fide et al., 2019), or decreased P100 latency in a visual attentional task with a low sensitivity (50% of the group) (Fernandez et al., 2007), or unchanged visual P100 and N100 latencies during facial discrimination and auditory oddball demands (Kurita et al., 2010).

Given this poor information about this component, it seems not to be promising for pharmacological clinical studies in ADD patients.

Similar variability of the results was reported for N100. ADMCI patients showed decreased N100 amplitude during a task asking “congruous/incongruous” statements on visual stimulus pairs (Olichney et al., 2006) whilst no difference in P100 or N100 amplitude or latency between CU and MCI or ADD patients was observed in an auditory oddball task at baseline and 1-year follow-up (Lai et al., 2010) as well as in visual tasks requiring detection of stimulus motion (Yamasaki et al., 2012), semantic priming of word pairs (Grieder et al., 2013), and working memory as 2 n-back or matching-to-sample demands (Deiber et al., 2015; Li et al., 2016). The studies investigating the effects of AChEIs did not show any effect on N100. In a small group of ADD, the physostigmine treatment resulted in no alterations in N100 amplitude or latency in an auditory oddball task (Neshige et al., 1988). In the same line, a large group of ADD patients showed no effect of about 2 years of donepezil treatment on N100 amplitude or latency in an auditory oddball task (Chang et al., 2014). Furthermore, nicotine administration did

not change P100 and N100 amplitude or latency in auditory and visual oddball tasks between tacrine-treated and non-treated ADD groups (Knott et al., 2002).

Concerning other treatments, a nootropic drug possibly acting on AMPA glutamate and cholinergic receptors (piracetam) mitigated the reduction in N100 latency of auditory and visual oddball ERPs in ADD patients as compared to CU persons (Dabic-Jeftic and Mikula, 1993). Furthermore, intravenous sodium-lactate (vasodilator, electrolyte replenisher, and an energetic material for neurons) produced just a trend for enhancing P100 and N100 during a visual semantic categorization task (Kálmán et al., 2005).

3.1.4. P200

Concerning P200, previous studies in ADMCI and ADD patients showed mixed findings as well.

Visual P200 amplitude was smaller in ADMCI patients during short-term memory retrieval (matching-to-sample) of neutral stimuli (Li et al., 2016) but not during short-term 0-back, 1-back, and 2-back conditions (Zunini et al., 2016). It was also smaller during medium-term memory retrieval of faces with negative emotional expression (Schefter et al., 2013). In ADD patients, visual P200 amplitude was smaller during visual tasks requiring detection of stimulus motion (Yamasaki et al., 2012).

Visual P200 latency was delayed during short-term (2 n-back) conditions in ADMCI (Zunini et al., 2016), and in either progressive ADMCI or ADD patients (Missonnier et al., 2007). In contrast, visual P200 latency did not differ between CU and ADD patients during facial discrimination and auditory oddball demands (Kurita et al., 2010), and between CU and ADMCI patients during medium-term memory retrieval of faces with emotional expressions (Schefter et al., 2013) or during short-term memory retrieval (matching-to-sample) of neutral stimuli (Li et al., 2016).

There are several studies investigating pharmacological effects on the P200 component in ADD patients, most of them with negative results. In those patients, no effect on P200 amplitude or latency was observed in auditory oddball tasks in relation to physostigmine, an AChEI, (Neshige et al., 1988) and donepezil, an AChEI (Lai et al., 2010; Chang et al., 2014). In contrast, intravenous sodium-lactate infusion increased P200 amplitude in ADD patients during a visual semantic categorization task (Kálmán et al., 2005).

Given this lack of significant effects, visual N70, N150, N160, P100, N100, N170, and P200 components of ERP seem not to be promising for pharmacological clinical studies in ADD patients.

3.2. Mid-To Late ERPs and Treatment Effects

3.2.1. The ERPs components, N200

The N200 component of ERPs typically peak in amplitude at 180–350 ms post-stimulus during cognitive demands, especially oddball tasks. It reflects selective attention and perceptual (stimulus discrimination) processes (Patel and Azzam, 2005; Bennys et al., 2007). In the oddball paradigm, N200 can be divided into N200a (i.e., mismatch negativity, MMN) and N200b components.

N200a (MMN) and N200b are negative-going ERP components occurring within 100-300 ms post stimulus that reflect preattentive (automatic) and conscious brain responses to deviant stimuli during oddball tasks, respectively (Näätänen et al., 1978, 2005). In this paper we did not include the event-related responses elicited after passive tasks, therefore the N200b will be mentioned as N200 from now on.

Using an active auditory oddball task, the N200 latency reliably predicted the progression from ADMCI to ADD status in relation to CSF amyloid-β levels (Papaliagkas et al., 2009), while the N200 amplitude was progressively smaller at the follow-ups of about 1 and 2 years (Papaliagkas et al., 2011).

In a recent review paper, 22% of reviewed studies between ADMCI and CU groups reported a significant difference in N200 amplitude, whilst the rate was 18% between ADD and CU groups (Paitel et al., 2021). Furthermore, no study reported an abnormality in N200

amplitude in CU ApoE4 carriers (Paitel et al., 2021). More specifically, some studies reported smaller N200 amplitude in both ADMCI and ADD groups over the CU groups (Fernandez et al., 2013; Wang et al., 2013; Bagattini et al., 2017), while some others stated no difference in N200 between ADMCI and CU groups (Cespón et al., 2015a; Mudar et al., 2016) or between CU and ADD groups (Bagattini et al., 2017). In the mentioned review paper, 47% of reviewed studies reported a significant difference in N200 latency between ADMCI and CU groups, while 48% of reviewed studies reported that difference between ADD and CU groups and 75% in CU ApoE4 carriers (Paitel et al., 2021).

Regarding ERP studies investigating pharmacological effects on the N200 component in ADD patients, studies reported no effect of AChEI, physostigmine or donepezil on N200 amplitude or latency in ADD over CU persons (Neshige et al., 1988; Lai et al., 2010; Chang et al., 2014; Vaitkevičius et al., 2015) or ADD over ADMCI patients (Lai et al., 2010). Conversely, N200 latency was found to be associated with MMSE score (Fruehwirt et al., 2019), ADD severity (Fruehwirt et al., 2019), and duration of the disease (Vaitkevičius et al., 2015), reflecting that the early cognitive processing may not be modulated by cholinergic inputs. An intravenously administered lactate treatment resulted in larger mean N200 amplitudes in ADD patients (Kálmán et al., 2005).

The above mixed results might be due to the different variants of the oddball tasks used as the kind of stimuli, the inter-stimulus intervals, the task duration, the level of required attention to the stimuli and experimental conditions, and the kind of subject's responses required during ERP recordings (Morrison et al., 2019). Other sources of variabilities are due to the procedures for the enrollment of patients and diagnosis of ADD and clinical severity of the disease. In most of the studies, the diagnosis of ADD did not use in-vivo measures of abnormal levels of amyloid-beta and tau in patients' brains. In those studies, ADMCI patients showed different domains of cognitive functions affected, namely significant factors influencing N200 in ADMCI patients (Cespón et al., 2013, 2015a, b).

Overall, the available pharmacological studies suggest that N200 do not have the required reliability for the use in clinical trials.

3.2.2. P300

The P300 response to a rarely presented "oddball" stimulus embedded in the standard stimulus (Hillyard and Kutas, 1983) is a positive wave appearing around +300 ms post-stimulus and is considered to involve several cognitive functions, including working memory, perception, attention, and learning (Başar-Eroğlu and Başar, 1991; Halgren et al., 2002; Polich and Kok, 1995; Klimesch et al., 2006; Rektor et al., 2004). During the oddball paradigm, participants have to detect the deviation from the standard stimulus as they are instructed, and to decide whether it is a target or not, and finally to perform either a mental counting or a button press for the target stimulus. All of these mental tasks cause activities in many interwoven sensory, cognitive, or motor neural networks.

P300 is elicited from sources over cortical along with subcortical structures, including basal ganglia and thalamus (Kropotov and Ponomarev, 1991; Rektor et al., 2004, 2005). A more recent study supported this finding as revealing not only thalamic regions, but also two regions of basal ganglia, the subthalamic nucleus and the globus pallidus internus, were found to be involved in task-relevant information processing (Beck et al., 2018).

Age and gender have an effect on the P300 wave in healthy adults, and its topology is affected by task difficulty or novelty effect or mental counting (Polich 1997, 2007). Age is an important factor for ERP markers. Current literature suggests aging can be tracked by means of some ERP waves (Morrison et al., 2019).

Sufficient evidence exists to suggest that the amplitude and latency of the P300 change in AD (Polich, 1989; Pokryszko-Dragan et al., 2003; Katada et al., 2004; Polich and Corey-Bloom, 2005; Ally et al., 2006; Muscoso et al., 2006; Caravaglios et al., 2008; Bonanni et al., 2010; Lai et al., 2010; Pedroso et al, 2012; Babiloni et al., 2020). Furthermore, characteristics of the P300 are also altered in individuals with MCI (Frodl et al., 2002; Golob et al., 2002; Bennys et al., 2007; van Deursen et al., 2009; Lai et al., 2010; Parra et al, 2012; Cid-Fernandez et al., 2019; Babiloni et al., 2020). Other studies suggest that features of the P300 wave might provide evidence for conversion of MCI into AD (Golob et al., 2002, 2009; Papaliagkas et al., 2008; van Deursen et al., 2009; Babiloni et al., 2020). Although prolongation of latency occurs as a function of time in physiological aging (Papaliagkas et al., 2011a), the P300 wave has been demonstrated to be sensitive to ADD neuropathology (Morgan and Murphy, 2002; Papaliagkas

et al., 2009; Fernandez et al., 2007) as either N200 or P300 latencies correlate with Ab42 levels in MCI patients, or with baseline levels in a longitudinal study (Papaliagkas et al., 2009, 2011b), or in genetically PSEN mutation carriers that lead to familial AD, altered P300 parameters have been identified 10 years prior to the disease onset (Golob et al., 2009; Quiroz et al., 2011). In the study of Jovicich et al. 2019, the sample of CSF-markers negative MCI and CSF-markers positive MCI were followed over two years with the use of auditory ERP and fMRI. It was found that reduced parietal and posterior cingulate source activities of auditory oddball ERPs. The P300 wave has been demonstrated to be sensitive to ADD neuropathology (Morgan and Murphy, 2002; Papaliagkas et al., 2009; Fernandez et al., 2007) as either N200 or P300 latencies correlate with Ab42 levels in MCI patients, or with baseline levels in a longitudinal study (Papaliagkas et al., 2009, 2011b), or in genetically PSEN mutation carriers (Golob et al., 2009; Quiroz et al., 2011). Taken together these results suggest that the P300 could contribute to the assessment of AD. The grand averaged waveforms representing the gradual decrease of P300 responses among patients with prodromal MCI, and with ADD compared to CU persons (healthy controls, HC) are presented in Figure 1.

----------Please insert Figure 1 about here----------

Figure 1. The averaged waveforms of the P300 component over the midline electrodes of frontal, central, and parietal areas demonstrated the gradual decline among the groups.

Studies on treatment effects of cholinergic drugs in ADD on the P300 wave showed decreased latency for a limited time period up to 3 to 6 months in general (Pedroso et al, 2012; Babiloni et al., 2020). Earlier AChEI reports on the effects of physostigmine noted as increased P300 amplitudes (Dierks et al., 1994) or decreased P300 latency over the short term (Neshige et al, 1988; Katada et al., 2004) in ADD. After the approval of AChEIs in routine ADD treatment, studies on the most commonly used medications donepezil displayed beneficial effects as evidenced by a reduction of P300 latency in ADD during auditory (Reeves et al., 1999; Thomas et al., 2001; Onofrj et al., 2002; Chang et al., 2014), and visual oddball paradigm (Reeves et al., 1999) and rivastigmine reduced P300 latency that was associated with better cognitive performances in mild to moderate probable ADD (Thomas et al., 2001).

Longitudinal P300 studies on the effects of donepezil revealed the latency of P300 wave is more reliable than the amplitudes (Werber et al., 2003; Parra et al, 2012; Pedroso et al, 2012; Babiloni et al., 2020). Among the five longitudinal P300 studies studying the effects of donepezil or rivastigmine (Thomas et al.,2001; Katada et al., 2003; Lai et al., 2010, Fruehwirt et al., 2014, Vaitkevičius et al., 2015), only one reported unchanged P300 latency or neuropsychological test scores between drug-naive and donepezil-treated ADD groups after monitoring for three months (Vaitkevičius et al., 2015). The effect of AChEIs on the P300 wave improvement in ADD patients was reported as the latency decrease in the first 3 to 6 months of treatment (Thomas et al.,2001; Katada et al.,2003; Lai et al., 2010) or an increase in P300 amplitude (Knott et al., 2002) The progression rates of the P300 wave latency increase in ADD patients on AChEIs were not particularly different in 6 to 12 months (Onofrj et al., 2002; Lai et al., 2010; Fruehwirt et al., 2019) from the patients not using AChEIs (Ball et al., 1989). Similarly, the clinical outcome measures also indicate symptomatic effectiveness of AChEIs as an improvement until 3 to 6 months of treatment, and then a decline thereafter (Gauthier et al., 2002; Arai et al., 2016). Memantine is a commonly used symptomatic add-on medication to cholinergic drugs in ADD. It is an NMDA receptor antagonist and functions as a glutamatergic noncompetitive NMDA receptor antagonist that modulates calcium influx. It has a selective affinity for extrasynaptic NMDAR open channels and does not interfere with normal transmission (Xia et al., 2010). It helps to restore the signal-to-noise ratio in hyper-excited neurons (Chen et al., 1992), and exerts an improving effect on cognitive and sensorimotor functions of Alzheimer's patients (Schmidt et al., 2015). In a meta-analysis for clinical trials of ADD, memantine was found effective for cognition, behavioral disturbance, and activities of daily living (Matsunaga et al., 2015). Regarding the effect of memantine on the P300, there are only two studies in the literature, possibly due to the fact that memantine is not used alone but mostly given in combination with AChEIs in the treatment regime of ADD. In the only ERP study searching the effects of memantine monotherapy led to a shortening of P300 latency about 20 ms in 42% of individuals with ADD with no significant change in P300 peak measures at the group level (Kubova et al., 2010). Another study on individuals with ADD with combination therapy of memantine and AChEI found an increase in the latency of P300 at the 12 months of treatment compared to the baseline, suggesting despite cholinergic and memantine treatment, the cognitive EEG parameters worsen in ADD on long-term (Fruehwirt et al., 2019).

Another study assessed the nicotine effect on P300 in two groups of ADD, and nicotine was administered to tacrine-treated and non-treated patients with ADD. Tacrine is the first approved AChEI mediation in ADD treatment. Prior to nicotine administration, tacrine-treated patients displayed shorter auditory P300 latencies than non-treated patients. Acutely administered nicotine did not change auditory P300, but increased the amplitudes of visual P300s in both ADD patient groups. These electrophysiological findings reflected the effects of nicotinic cholinergic processes in ADD (Knott et al., 2002).

Experimental studies indicate AChEIs drugs increase P300 amplitude in a rat model of AD (Laursen et al., 2014), and decreased amplitudes of ERP components were described in an amyloid-β infused mice model (Kim et al., 2020) or in an animal model of tau overexpression mutations (Nouriziabari et al., 2018). Furthermore, both scopolamine (anticholinergic agent) and entorhinal tau overexpression caused the learning-related changes in the P200 component (Nouriziabari et al., 2018), implying the cholinergic role in the generation of ERPs. The term folate, also known as vitamin B9, refers to a group of water-soluble compounds that play a fundamental role in a variety of physiologic processes such as regulation of gene expression, neurotransmitter synthesis, and maintenance and repair of the genome (Naderi and House, 2018). In a study assessing the effect of folate with vitamin B12 on P300 in patients with ADMCI complicated by hyperhomocysteinemia, the ADMCI group was divided into the intervention group, which was administered with folate, and the control group. After the 24th week, the intervention group had shorter P300 latency than their baseline and the control group. The findings suggest that a decrease in total homocysteine levels at the 24th week may lead to an improvement in the cognitive function of the ADMCI group revealed by shorter P300 latencies (Jiang et al., 2020) .

Other earlier reports exist in literature studying treatment effects of other agents including nicergoline and piracetam. Under nicergoline treatment, an ergot alkaloid derivative with a wide spectrum of action, including being a selective alpha-1A adrenergic receptor antagonist, enhancing cholinergic and catecholaminergic neurotransmitter function, and inhibiting platelet aggregation, ADD patients showed decreased P300 latency (Saletu et al., 1995), suggesting an improved vigilance and information processing. On the other hand, piracetam, a nootropic agent with mild antiepileptic properties, plays a role as an AChEI, while also having an effect on NMDA glutamate receptors, showed no changes in the P300 responses

in individuals with ADD (Dabic-Jeftic and Mikula, 1993). Therefore, among the ERP components, the P300 latency might be particularly useful in reflecting cognitive decline and treatment effects in ADD (Papaliagkas et al., 2008; Lai et al. 2010, Parra et al., 2012; Babiloni et al., 2020). P300 measures are promising candidates for investigating treatment effects in ADD. Yet, the paucity of studies and small size of participants in the previous P300 studies indicate the necessity of further studies.

3.2.3. The ERPs components related to Object/Facial Recognition, N170, N230, VPP, LPP

The recognition of facial expressions (Bentin et al., 1996; Başar et al., 2006, 2007; Güntekin and Başar, 2007; Puce et al.,2013; daSilva et al., 2016; Güntekin et al., 2017, 2019; Fide et al., 2019; Güntekin and Başar, 2014) or ambiguous figures (Başar-Eroğlu et al., 1996; Mathes et al., 2006; Strüber et al., 2000; İşoğlu-Alkaç et al., 2000) are among the most complex functions in the visual cognitive processes. Under physiological conditions, emotional faces elicit a greater magnitude of ERP components than neutral faces, namely N170, N230, the vertex positive potential (VPP), and the late posterior potential (LPP) in comparison to neutral faces. Figure 2 demonstrates reduced N170 responses in ADD on occipital electrode locations in response to facial expressions compared to CU persons.

To our knowledge, there has been no study of event-related potentials related to facial emotion recognition on treatment effects in ADD or ADMCI in the literature.

----------Please insert Figure 2 about here----------

Figure 2. The grand averaged ERPs of the groups indicating individuals with ADD had reduced N170 responses on occipital electrode locations in response to facial expressions (modified from Fide et al., 2019).

3.2.4. Late ERPs components related to Semantic Processing: N400, P600sem, Late positive component (LPC)

The N400 latency is similar in ADMCI, ADD, and healthy controls, but N400 amplitudes are altered in mild stage ADD patients (Olichney et al., 2006; Spironelli et al., 2013). Similarly, the attenuation of P600 for the repetition of congruous words is less in ADMCI and mild ADD

patients (Olichney et al., 2002a,b), and it has been proposed as having a predicting value for subsequent conversion from MCI to ADD, and alterations in N400 are detected even at the presymptomatic stage of disease in individuals carrying PSEN1 mutation (Bobes et al., 2009). An intervention effect on the N400 and recognition potential (RP) was sought in a study about cognitive training programs in ADD patients who were already taking AChEIs (n=11). After the training, an improvement of RP amplitude was observed; but for the N400 component, the improvement was seen only in healthy older individuals but not in ADD patients (Spironelli et al., 2013) (Table 4).

Briefly, the literature covers only one study investigating treatment effect on N400, P600, LPC components with non-promising result in ADD.

3.2.5. Memory Related Late Potentials

Among late memory-related potentials, the PNwm, which is elicited by memory tasks, distinguished stable MCI from progressive ADMCIs (Missonnier et al., 2005) and ADMCI patients from CU persons (Deiber et al., 2015). However, a study investigating the effects of combination therapy (AChEI and memantine) by late ERPs using a number-letter paradigm failed to show any difference between the medicated and the unmedicated "converted to dementia" groups (Chapman et al., 2013).

Therefore, with the current findings in the literature, later ERP components seem not to have a potential to monitor treatment effects in ADD due to a wide variety of tasks including semantic tasks or not displaying any significant change after interventions.

4. Event-Related Oscillations (EROs) and Treatment Effects

The pioneering work on oscillatory dynamics on animals was reported by Freeman (1975), Başar et al. (1975a), and Başar (1980), showing the distributed oscillatory responses in all parts of the brain. According to Başar et al. (2001), event-related potentials constitute from the superposition of oscillations in certain frequency bands by applying time-frequency (TF) analyses to ERPs activity (Başar et al., 1999, 2001; Başar-Eroğlu et al., 1992, 2001; Demiralp et al., 2001; Karakaş et al., 2000; Yordanova et al., 2002; Makeig et al., 2002; Gilmore et al., 2010).

Although the averaged ERPs are useful and commonly used methods, their further computation yields information about the brain's intrinsic activity and dynamical changes even more. As mentioned previously, the brain oscillatory activities after an "event" display almost totally inverse dynamics of those during the resting condition (Başar, 1980; Başar-Eroğlu, Başar, 1991; Babiloni et al., 2020). In case of evoked potentials elicited by an "event" or stimuli are enhanced in amplitude when preceded by low-amplitude pre-stimulus alpha or theta rhythms (Başar et al., 1984; Jasiukaitis and Hakerem, 1988; Başar-Eroğlu et al., 1992; Babiloni et al., 2008). In these studies, delta and theta EROs responsiveness in frontal lobes was interpreted as an indication of well functioning of the hippocampo–fronto–parietal system during cognitive processes. For this reason, the role of oscillatory activities in certain frequency bands will be listed and explained below. Furthermore, treatment effects on EROs in ADD patients are presented in Table 3.

----------Please insert Table 3 about here----------

4.1. Delta frequency band (<4 Hz)

The shape of the P300 complex is formed basically by the superimposition of delta response oscillating at 2 Hz (Başar-Eroğlu, Başar, 1991; Başar-Eroğlu et al., 1991, 1993; Schürmann et al., 2001), along with prolonged theta and alpha oscillatory responses (Demiralp et al., 1999; Kolev et al., 1997), but activity changes in faster frequency bands also contribute (Karakaş et al., 2000; Sakowitz et al., 2001; Spencer and Polich, 1999). A study from 2068 participants (Bernat et al., 2007) confirmed that the major operating rhythms of the P300 were delta and theta oscillations. Not only the oddball paradigm but also others including error-related negativity, feedback negativity, N2/P3 of go-nogo tasks involved delta and theta oscillatory responses (Bernat et al., 2012; Harper et al., 2014; Schmiedt-Fehr and Başar-Eroğlu et al., 2011).

Long lasting depolarization of cortical pyramidal cells produces delta oscillations (Steriade and McCarley, 1990). Other than this, thalamocortical cells (Steriade et al., 1993), neuronal cells in nucleus accumbens (Leung and Yim, 1993), in ventral tegmental area, in the ventral pallidum (Lavin and Grace, 1996), and glial cells also yield delta rhythms (Amzica and Steriade, 2000). The delta EROs were elicited as a negative peak at +200 ms post stimulus and continued with a positive peak around +400-600 ms post stimulus. During this wave,

superimposed theta responses either enhance or dampen the signal, whilst alpha prolongation implies the achievement of a cognitive goal (Güntekin and Başar, 2016). The prestimulus delta state affects the post stimulus responses as an inverse relation between them, as suggested for the first time by Başar et al. (1984) and Başar and Stampfer (1985) as they reported when a stimulus was applied in certain interstimulus intervals, a phase reordering occurred in delta and alpha bands after the stimulus. Regarding the task's difficulty, stimulus with greater cognitive load elicited larger P300 and single-trial delta response amplitude (Mathes et al., 2012). Delta EROs (Başar and Stampfer, 1985; Stampfer and Başar, 1985) behave as a general electrophysiological marker in cognition (Güntekin and Başar, 201), and they are involved in cognitive processes related to decision making and attention processes (Knyazev, 2012). Regarding connectivity, delta synchronization is observed between frontocentral and parietal (Qassim et al., 2013) regions during attention and memory updating in a MEG (Ishii et al., 2009) and EEG study (Güntekin and Başar, 2010). In various studies, an unspecified decrease of delta ERO power decrement is encountered in ADD, ADMCI, schizophrenia, Parkinson's disease (PD), or bipolar disorder (Başar et al., 2013). In ADD, an increment in RsEEG delta band power is reported, suggesting a similarity to those in the prestimulus era (Babiloni et al., 2015, 2018a, 2018b, 2019a; Jelic et al., 2000; Caravaglios et al., 2008) and a diminished delta EROs after an event (Caravaglios et al., 2008; Yener et al., 2008, 2012), supposing an increased delta response would not be produced in such a busy network (Rahn and Başar, 1993). A delay in peak delta EROs and a gradual decrease in amplitude of delta EROs in aging of CU individuals (Emek-Savaş et al., 2016), or across the AD spectrum have been noted, (Başar et al., 2016b). Frontal delta EROs (visual and auditory) have been attenuated in ADMCI (Kurt et al., 2014; Yener et al., 2013) or ADD patients compared with CU persons (Yener et al., 2009, 2012). EROs were also sensitive to ADD progression over time (Yener and Başar, 2013). However, the delta or theta EROs power decrease cannot be considered as specific to ADD, as patients with Parkinson's disease dementia (PDD) (Güntekin et al., 2019) or PD-MCI also display lower ERP amplitudes/delta or theta EROs measurements (Yener et al., 2019; Güntekin et al., 2018, 2020; Hünerli et al., 2019). Figure 3 shows the grand averaged waveforms representing the gradual decrease of delta EROs among patients with ADMCI, and with ADD compared to CU individuals.

Regarding treatment effects on delta EROs elicited by visual or auditory oddball tasks in ADD patients, delta EROs power is reduced in both AChEI-treated and untreated denovo ADD patients in comparison to CU persons (Yener et al., 2007, 2009). Furthermore, delta ERO long

range connectivity was diminished similarly in ADD patients with or without cholinergic medication (Başar et al., 2010). Therefore, delta EROs seem to be lacking to exhibit the effect of cholinergic medication in ADD.

----------Please insert Figure 3 about here----------

Figure 3. The grand averaged delta EROs over frontal (F3,Fz,F4) electrode locations of prodromal MCI, and ADD patients in comparison with CU persons.

4.2. Theta frequency band (4-7 Hz)

During elicitation of an ERP response, oscillatory responses in theta ranges (4-7 Hz) form the early components of the P300 complex and later parts by delta response (Başar-Eroğlu et al.,1992; Kolev et al., 1997; Karakaş et al., 2000). The relation between theta oscillatory activity and working memory under physiological conditions has been implicated by many studies (Klimesch et al., 1997; Jensen and Tesche, 2002; Pavlov and Kotchoubey, 2017; Zakrzewska and Brzezicka, 2014; Borhani et al., 2021), and in ADD (Klimesch et al., 2005). Experimental studies indicate that synchrony in the theta oscillatory activity represents one of the most studied neuronal activities in the mammalian hippocampus, and it is associated with the top-down control of cognitive processes (Vertes, 2005). The hippocampal formation and the medial septum generate theta oscillations, and they act as a pacemaker for generating theta oscillatory rhythm in prefrontal cortical networks (Thierry et al., 2000). This strong connection generated by theta rhythm synchronizes medial prefrontal cortex neurons to spatially distributed cortical areas, and consequently strengthens synaptic links, and facilitates the transfer of hippocampal information to the neocortex during learning and memory (Ahnaou et al., 2014; Buzsaki, 2002; Paz et al., 2008). During active cognitive or motor tasks, enhanced EEG theta coherence has been observed between the hippocampus, prefrontal, and posterior association cortices (Womelsdorf and Fries, 2006; Seemüller et al., 2012; Schmidt et al., 2013).

The activity in theta (5–12 Hz) and beta (12–30 Hz) frequencies are elicited by stimuli that guide the selection of response (Bland and Oddie, 2001; Leventhal et al., 2012) in addition to gamma responses. Both in humans (Kaplan et al., 2014) and animals (Hyman et al., 2005; Jones and Wilson, 2005), prefrontal theta oscillations are heightened by spatial cues. In some conditions, high-frequency (gamma, beta) and low-frequency rhythms (theta, alpha, delta)

become connected through the coupling of phase-amplitude. This coordination across frequencies is a good indicator of accurate responses or learning rate (Tort et al., 2009; Canolty and Knight, 2010). Such coupling may be paramount for sensory cues to facilitate learning or memory processes (Howe et al., 2017).

In experimental studies, it has been shown that oscillations from each frequency band are considered to subserve a different function and to have a different underlying mechanism. Synchronous oscillatory rhythms in the gamma and slow theta frequencies (i.e. theta and gamma coupling) represent the main mechanism for coordinating disparate brain networks temporally during cognitive tasks, including attention and working memory (Lisman and Buzsaki, 2008; Singer, 1999; Ahnaou et al., 2014). Several studies with a focus on the generators of theta rhythm indicated specific hippocampal and other brain regions (Buzsáki and Watson, 2012). Theta oscillatory activity is considered to coordinate the information flow and establish temporal regulations to propagate across selectively distributed neuronal networks in the entorhinal cortex and the subregions of the hippocampus (Cappaert et al., 2009).

Event-related spectral perturbation evaluates the dynamic alterations in power at frequency ranges as a function of time relative to a pre-stimulus baseline (Makeig, 1993), while it also allows measuring increases and decreases in power spectrum with the use of event-related synchronization (ERS) and desynchronization (ERD). A decrease in the early induced theta ERS indicates a rapid cognitive decline among the individuals with ADMCI, and similar values of theta ERS to that of the HC group may imply a stable MCI. Even though MCI patients were successful in achieving the behavioral tests, the frontal theta (in the range of 4-6 Hz) EROsdiscriminated progressive ADMCI from both the stable MCI and the HC group and were suggested as an early electrophysiological marker of cognitive decline (Hedges et al., 2016; Jiang et al., 2015; Missonnier et al., 2007; Deiber et al., 2009). This finding can be explained on the basis of the recruitment of additional cortical networks that make the individuals achieve the behavioral tasks and maintain a high performance level, with an impaired prefrontal activity detected by electrophysiology.

Theta ERD responses are found higher in the ADMCI group than ADD group (Fraga et al., 2018), and the theta EROs power is decreased in both ADMCI and more profoundly in PDMCI groups (Yener et al., 2019), in addition to phase-locking impairment in the theta band.

ADMCI patients display reduced levels in both total delta (Tülay et al., 2020) and theta EROs power (Deiber et al., 2009; Nguyen et al., 2017; Tülay et al., 2020) at the early courses of disease, whereas reduction of evoked delta power starts in the MCI stage, but becomes apparently distinctive later in the phase of dementia of ADD (Caravaglios et al., 2010; Li et al., 2017; Tülay et al., 2020). The distinctive pattern of total and evoked the power of slow waves in the temporal evolution of ADD may be a reflection of the neurodegenerative spreading pattern that involves subcortical limbic and association cortices at the beginning and later involvement of lower level cortical areas or thalamocortical circuits. Also, theta connectivity is prominently affected in ADD (Yener and Başar 2013; Güntekin et al., 2008), and theta gamma coupling showed a gradual decrease along with the HC, ADMCI, and ADD (Goodman et al., 2018). Similar to delta band activity, theta EROs are not specifically reduced in ADD, but other cognitive impairments such as PD (Yener et al., 2019; Güntekin et al., 2018, 2019, 2020).

Regarding treatment effects, cognitive theta EROs power was similarly reduced in treated and untreated ADD (Yener et al., 2009), whilst ADD patients treated with AChEI medications had an increase in event-related frontal theta phase locking in comparison to non-treated ADD group during oddball task in a cross-sectional study (Yener et al., 2007; Figure 4). So far, there is no longitudinal or cross-sectional EROs study in the literature confirming these medication effects.

----------**Please insert Figure 4 about here**----------

Figure 4. The grand averaged waveforms represent reduced visual event-related theta phase-locking in non-treated ADD. The thick black line shows the grand averages of each group to the target stimuli elicited by a classical visual oddball paradigm. The thin gray line demonstrates the average of single sweeps from a single subject (modified from Yener et al., 2007).

4.3. Alpha frequency band (8-12 Hz)

Alpha EROs in ADD patients display a more complex picture than other slow wave EEG-EROs responses. They have been shown to relate memory-related cognitive processes (Klimesch et al., 1997, 2006, 2007; Maltseva et al., 2000; Doppelmayr et al., 2005; Sauseng et al., 2005; Wang et al., 2017). However, there are controversies on the direction of alpha responses and memory processes. Some researchers found alpha ERD responses during semantic

memory processes as a functional correlate of brain activation (Klimesch, 1997; Klimesch et al., 2007). On the other hand, some other groups demonstrated increased cognitive function and attentional processes in relation to the increased alpha responses (Jensen et al., 2002; Palva and Palva, 2007; Tuladhar et al., 2007; Scheeringa et al., 2009), or some reported post-stimulus event-related alpha synchronization in relation to "signal quality at the time point of stimulus onset" including the amplitude of or phase-angle of the pre-stimulus alpha activity (Başar, 2012; Başar and Güntekin, 2012). The variety of findings on alpha EROs responses may be based on their having multiple roles in sensory, cognitive, emotional, and motor-related processes; and the inverse relationship with pre-stimulus EEG and post-stimulus alpha power may influence the consequent behavioral performance (Ergenoğlu et al., 2004; Busch et al., 2009; Babiloni et al., 2000, 2008; Samaha and Postle, 2015).

In line with these views under physiological conditions, several ERD/ERS studies reported contradictory results spanning from post-stimulus alpha ERD decreases in ADD and ADMCI patients (Fraga et al., 2017), to finding decreased alpha ERS in ADD (Babiloni et al., 2000) and ADMCI (Karrasch et al., 2006); or decreased ERD over the anterior regions during the pre-event era, while an increased ERS over the posterior regions during post-stimulus era in ADMCI patients (Caravaglios et al., 2015). A further study on the multidomain MCI group had a more profound alpha ERS decrease than single domain MCI (Deiber et al., 2010). Also, power or phase-locking measurements of alpha EROs were diminished (Deiber et al., 2010) in progressive ADMCI compared to CU individuals (Michalopoulos et al., 2012). In several intra-hemispheric alpha EROs coherence studies indicating brain functional connectivity, ADD patients showed decreased memory-related connectivity (Başar et al., 2010; Hogan et al., 2003). On the other hand, the inter-hemispheric EEG coherence was higher in ADMCI patients when memory demand increased (Zheng et al., 2007). A compensating rsEEG hyperconnectivity in the early stages of AD has been emphasized in recent studies (Bonanni et al., 2021). Yet, other explanations can be made for these contradictory findings. Prodromal ADD patients show abnormal thalamocortical interactions, possibly due to impairment of the cortical gray matter, especially in posterior regions (Babiloni et al., 2014). This abnormality of wakefulness cortical alpha sources can be based on a progressive alteration in the interplay of thalamocortical high threshold GABA-ergic interneurons, thalamocortical relay-mode, and cortical pyramidal neurons (Hughes and Crunelli, 2007; Crunelli and Hughes, 2010). During wakefulness, under physiological conditions, glutamatergic and cholinergic signaling to this complex network

augments the generation of cortical and thalamocortical alpha rhythms, resulting in cycles of excitation and inhibition within a time frame of approximately 70-100 milliseconds (Hughes and Crunelli, 2007; Crunelli and Hughes, 2010; Jovicich et al., 2019). In order to understand these complicated responses in alpha frequency ranges, further studies are needed to explore the dynamic alpha changes during the task in ADD patients, especially taking both prestimulus era, and post-stimulus era alpha changes into consideration.

There are not many alpha EROs studies investigating treatment effects in patients with ADD. In one of them, the alpha EROs coherence was found lower in ADD groups regardless of using AChEI medication. The CU group showed higher values of EROs coherence in the "delta", "theta" and "alpha" frequency bands between the left fronto-parietal electrode pairs in comparison to both the AChEI-treated ADD and the untreated ADD groups (Başar et al., 2010), implying no effect of cholinergic medication on alpha EROs connectivity.

All these above findings imply that EROs in slow wave frequency bands, possibly except from theta, are far from showing treatment effects in ADD.

4.4. Beta frequency band (13-30 Hz)

Beta oscillations increase in WM related tasks (Engel and Fries, 2010). The increased beta responses have been reported as related to attention, emotion recognition, primary sensory processing, and movement. In CU persons, increased ERO power or phase-locking of beta responses upon presentation of target stimuli in healthy subjects imply that beta EROs oscillations could shift the system to an attention state which serves as one of the bases of cognitive functions (Wróbel et al., 2000; Güntekin and Başar 2007). Another role of beta responses was reported as their role in emotional processes, especially during the perception of negative emotional stimuli (Miskovic and Schmidt, 2010; Woodruff et al., 2011; Güntekin and Başar, 2014). These results placed beta ERO responses among one of the widely used frequency bands in the EEG based emotion recognition algorithms (Zhang et al., 2016; Mohammadi et al., 2017; Munoz et al., 2018) and in movement-related cognitive functions; (Cacace and McFarland, 2003; Mazaheri and Picton, 2005; Ishii et al., 2009), or in cognitive paradigms (Tallon-Baudry et al., 1998; Peterson and Thaut, 2002; Onton et al., 2005; Ravizza et al., 2005; Güntekin et al., 2013). In their review article, Engel and Fries (2010) discussed the beta EROs may occur as a consequence of sensory processes, such as increased beta responses were elicited over the

occipital cortex by visual stimuli (Senkowski et al., 2006) and over central and temporal locations by auditory stimulation (Haenschel et al., 2000; Sakowitz et al., 2005; Senkowski et al., 2006). Also, multisensory stimuli enhanced higher beta responses than the single sensory stimuli (Sakowitz et al., 2005; Senkowski et al., 2006). In the pathological conditions, ADMCI patients show lower beta EROs (Güntekin et al., 2014; Caravaglios et al., 2018), with a gradual decrease in progressive MCI patients in comparison to stable MCI patients (Hedges et al., 2016; Jiang et al., 2015; Missonnier et al., 2007).

Regarding treatment effects, no study reported results on beta EROs in the ADD so far.

4.5. Gamma Frequency Band (30-45 Hz)

The significance of the evoked gamma band activity, especially 40 Hz, has been emphasized in the central nervous system of a variety of animals including snails, vertebrates, and humans, as an important element in processing sensory and cognitive information in neural networks (Freeman, 1975; Başar et al., 1987; Başar-Eroğlu and Başar, 1991; Eckhorn et al., 1988; Gray and Singer, 1987; Lenz et al., 2008; Traikapi and Konstantinou, 2021). Gamma frequency band is more likely to relate to attention, or attentional selection (Fries et al., 2001; Bichot et al., 2005; Womelsdorf and Fries, 2006, 2007) as heightened connectivity at gamma frequencies (30 –100 Hz) has been elicited during states of cue detection (Howe et al., 2017). The relation of memory and gamma responses has been shown in many reports (Başar, 2013; Başar-Eroğlu et al.,1996; Herrmann et al., 2004; Jokisch and Jensen, 2007; Singer, 1999; Tallon-Baudry and Bertrand, 1999). Inhibitory GABAergic interneurons have a direct modulatory effect on gamma oscillations and (Gray andMcCormick, 1996; Herrmann and Demiralp (2005), and combinations of various transmitters play a role in even the simplest cognitive responses. In previous studies, GABA, GABA/glutamate, and dopamine have been reported as the neurotransmitters that have an effect on the gamma frequency (Whittington et al., 1995; Gray and McCormick, 1996; Muthukumaraswamy et al., 2009; Kömek et al., 2012) which may modulate glutamatergic pyramidal cell activity via inhibitory GABA network (Carlen et al., 2012; Fell et al., 2001; Fries, 2009; Sederberg et al., 2003; Tallon-Baudry et al., 2005). Gamma oscillatory activity seems to establish synchronization not only in short distance local cortical networks (Buzsaki, 2006) but also play a role in long-range connectivity (Cuesta et al., 2015, Maestú et al., 2008; Jiang et al., 2008).

The abnormalities of gamma connectivity and activity can be seen both during event-related activity in ADD and ADMCI. Counterintuitively, both the power and connectivity of the EROs gamma band seem to increase in ADD (Di Lazzaro et al., 2004; Osipova et al., 2006; van Deursen et al., 2011; Ferreri and Rossini, 2013; Başar et al., 2016a; Başar et al., 2017). The uniqueness of gamma frequency band activity in that sense could be explained by an inhibitory interneuron impairment in ADD patients with a subsequent increase of gamma activity (Verret et al., 2012; Palop and Mucke, 2016). Decreased GABAergic inhibition was demonstrated in a mice model of ADD (Busche et al., 2008) and suggested as related to increased gamma responses in ADD patients (Stam et al., 2006; Rossini et al., 2006; Osipova et al., 2006; van Deursen et al., 2008, 2011; Başar et al., 2016a, 2017).

During the cognitive tasks, ADD patients respond with a 25% larger gamma response and a delay about 100 ms later in higher frequency gamma subband (40-48 Hz) (Başar et al., 2016a) without obvious fluctuations (Başar et al., 2016a; Deiber et al., 2010). Therefore, the gamma EROs display increased power in ADD in contrast to other frequency bands. The delay in cognitive gamma responses in this patient group may be related to lagged connections between cortical, thalamic, and limbic areas as a consequence of neurodegeneration during fine tuning of fast top-down and bottom-up processes related to memory, and other related cognitive functions (Canuet et al., 2015). Furthermore, larger amplitudes in gamma EROs activity may be an index of cortex hyperexcitability that has been reported repeatedly in ADD (Stam et al., 2006; Rossini et al., 2006; Osipova et al., 2006; van Deursen et al., 2008, 2011; Başar et al., 2016a, 2017; Palop and Mucke, 2016).

Regarding treatment effects, gamma EROs merit more attention regarding the alterations in both cholinergically treated and untreated (drug-naive) ADD groups indicating a connectivity increase in both ADD subgroups. Interestingly, patients on the cholinergic treatment had further coherence increases than both drug-naive ADD patients or CU persons (Başar et al., 2017). The increase in the long range (fronto-parietal, fronto-occipital) gamma EROs connectivity in treated and untreated ADD patients were observed in response to visual sensory stimulation, whilst decreased short distance (parieto-occipital) gamma ERO connectivity was noted in treated ADD patients in comparison to drug naive ADD patients (Başar et al., 2017). This observed pattern consisting of an augmented long range connectivity and a suppressed short range connectivity fits well to those previously reported on functions of acetylcholine on brain activity (Hasselmo

and Sarter, 2011). The mechanism related to increased gamma responses after cholinergic medication may be based on the coexpression of alpha7 nicotinic receptors in GABAergic interneurons (Voytenko et al., 2015) or the change in neuronal excitation/inhibition imbalance observed in AD (Maestu et al., 2021).

In brief, gamma ERO connectivity measures seemed to be a promising tool to investigate AChEIs treatment in ADD.

5. Miscellaneous Interventional and Pharmacological Treatment Effects on ERPs/EROs in Clinical and Experimental Studies

5.1. The effect of Memantine on ERPs/EROs in Clinical Studies

Memantine is a commonly used symptomatic add-on medication to cholinergic drugs in ADD. It is an NMDA receptor antagonist that weakly binds to Mg++ and displays functions that modulate calcium influx. It has selective affinity for extrasynaptic NMDAR open channels and does not interfere with the normal transmission (Xia et al., 2010), and it helps to restore the signal-to-noise ratio in hyperexcited neurons (Chen et al., 1992). Clinical ERP/ERO EEG studies are scarce on treatment effects of memantine as it is used as an add-on therapy.

The animal studies investigating the effects of memantine are not redundant in the literature. In one of them exploring the effects of memantine by means of induced EEG elicited by electrical stimuli in anesthetized rats, demonstrated that low dose memantine increased theta and gamma band activity however, high dose memantine decreased hippocampal theta oscillations (Guadagna et al., 2012). In a study on freely moving mice by Ma et al. (2015) showed LTP-enhancing effect of memantine that was blocked by injection of scopolamine, an anticholinergic drug, indicating an interplay between cholinergic and glutamatergic antagonists favoring cognitive improvement. Memantine significantly increased gamma oscillations in freely moving animals (Hiyoshi et al., 2014; Ahnaou et al., 2014; Ma et al., 2015).

Other medications such as semagacestat, a gamma secretase inhibitor that reduces production of amyloid-β, causes decreased hippocampal theta oscillatory activity induced by electrical stimuli in anesthetized mice (Hajos et al., 2013). On the other hand, piracetam, a nootropic agent, increased hippocampal theta oscillations that are induced by electrical stimuli in

anesthetized rats (Kinney et al., 1999). The treatment effects of animal studies for EEG-ERP/EROs in AD models are presented in Table 4.

----------Please insert Table 4 about here----------

In brief, experimental studies of induced EEG investigating effects of AChEIs and memantine reported the increase in power of theta and gamma bands (Ahnaou et al., 2014). In a few clinical studies investigating effects of AChEIs on EROs EEG activity EEG in medicated ADD patients in comparison to unmedicated ADD patients can be summarized as improvement of frontal theta phase-locking and altered gamma ERO connectivity. Yet, these results await to be confirmed by other clinical research groups.

5.3. Effects of Non-Pharmacological Treatments on ERPs/EROs

General rules of oscillatory activity imply a potential for modulating brain waves by resetting the oscillatory hierarchy such as 1) amplitude periodicity of the faster waves matches with those of slower waves (Amzica and Steriade, 2000; Vanhatalo et al., 2004), 2) amplitude of gamma oscillation depends on theta oscillation phase (Buzsaki et al., 2003) and a variety of cross frequency couplings occurs between frequency bands, such as those between beta-gamma or theta-alpha bands during working memory paradigms (Siebenhühner et al., 2015), and 3) ongoing cortical activity exerts an effect on processing of a stimulus (Başar et al., 1980; Polich, 1997; Fries et al., 2001; Babiloni et al., 2006).

Hence, a new avenue for ADD treatment is open to neuromodulation techniques including, TACS, TDCS, TMS, or neurofeedback techniques (Jiang et al., 2017) or training to enhance oscillations in alpha and beta bands for higher memory performance and gamma band for depression (Escolano et al., 2014) or neurofeedback in ADD (Luijmes et al., 2016; Sürmeli et al., 2016). Transcranial direct current stimulation is a technique to modulate brain oscillations by applying a direct electrical current to the scalp. In a study on individuals with ADD and CU persons using TDCS, the result favored beneficial effects of the intervention, such as increased amplitudes of P200 and P300 and increased frontal theta EROs within 150-300 ms time window (Cespón et al., 2019).

Nonpharmacological brain stimulation techniques may help to reduce brain hyperexcitability reported in ADMCI or ADD (Adaikkan et al., 2019; for review, see Toniolo et

al., 2020). Many studies performed on ADD/ADMCI patients using these techniques aimed to reduce hyperexcitability of the brain by transcranial magnetic stimulation (Koch et al 2018; Arendash et al., 2019; Sabbagh et al., 2020), or by tDCS (Khedr et al., 2019; Ferrucci et al., 2008; by tACS (Xing et al., 2020) or by 40 Hz sensory stimulation (Cimenser et al, 2021; Ismail et al., 2018) or photobiomodulation (Chao, 2019).

Recently, there has also been a focus on various types of exercise including aerobic, strengthening, and combined involvements as another non-pharmacological intervention that is associated with cognitive improvement. Using different cognitive tasks, it was suggested that involvement in physical or aerobic exercise appears to be related to increased amplitude and/or decreased latency of P300 in young and older active CU persons compared to individuals with a sedentary lifestyle (for a review, see Huang et al., 2016). In ADMCI patients, it was reported that either aerobic dance routine demonstrated decreased P300 latency (Zhu et al., 2018), or ADMCI who participated in two different types of exercise programs displayed an increase in P300 amplitudes (Tsai et al., 2018, 2019). Another longitudinal study showed that both physical exercise programs or social-gathering intervention resulted in an improvement in P300 parameters (Pedroso et al., 2018). The findings above indicate that P300 measures are possible candidates to be used in non-pharmacological treatment studies.

6. DISCUSSION

In the current article, a multidisciplinary panel of experts reviewed the literature about the effects of medications or interventions on ERO/ERP EEG oscillations during cognitive tasks. The literature on this subject seems to be too scarce to provide definitive answers. Among the most commonly used symptomatic treatment of ADD, cholinergic drugs lead to reduction in latency of P300 and an increase in amplitudes of late ERPs components (N200, P300) temporarily up to a year. Effects of cholinergic medications on EROs can be summarized as an increase in theta phase locking and gamma connectivity, yet further confirmation is needed. Effects of memantine, another licensed symptomatic medication acting as an NMDA receptor antagonist for treatment of ADD, has not been well studied on ERPs/EROs in ADD, possibly due to its common use as an add-on medication to cholinesterase inhibitors. Animal studies confirmed that cholinesterase inhibitors cause increased amplitudes of P300 like ERPs and increased levels of induced theta and gamma oscillations.

Even though there have not been many new options of treatment in the past two decades, possibly disease modifying drugs are becoming available for ADD. Many questions still remain to be answered and cannot be covered by previous literature. The current paper studying ERP/ERO EEG studies revealed that P50, P300 measures are the most promising ERP components, whilst theta and gamma ERO responses may bear a potential for monitoring treatment effects in drug trials or intervention studies in ADD.

In the future, EEG related methodologies may help to uncover the changes in the brain activity in response to remedies such as newly developed anti-amyloid, anti-tau, or hybrid remedies (for reviews, see Cummings, 2021; Toniolo et al., 2020; Zagórska and Jaromin, 2020). The concurrent investigation of ERPs/EROs methodologies and other well studied valid biomarkers in at-risk ADD patients may help validate the EEG methodologies to monitor the effects of treatment on brain functions. Furthermore, additional use of ERP/EROs to the RsEEG activity may offer an advantage for observing treatment effects of ADD, as gathered from limited numbers of comparative electrophysiological studies with small numbers of participants in the literature (Olichney et al., 2002; Van der Hiele et al., 2007; Deiber et al., 2009; Lopez et al., 2014; Jovicich et al. 2019).

EEG methodologies assessing treatment effects can be useful for personalized medicine. Even though a wide range of variability in the accuracy rates of ERPS/EROs limits their use on an individual basis for diagnostic purposes in the ADD, the possibility is higher when the treatment effect is considered, as each person can provide control of themselves prior to treatment. At that point, ERPs/EROs may offer advantages for monitoring intervention or treatment effects, because these electrophysiological methods provide almost an individualistic electrophysiological signature (Näpflin et al., 2008), and indicate an alteration about 10 to 30% to baseline of the same individual which is much higher rate than any other neuroimaging marker elicits (Olichney and Hillert, 2004; Başar et al., 2013).

Significant limitations of this article include (i) the restrictive criteria used for the review of literature; (ii) the inclusion of studies using various diagnostic criteria for AD in the era without current diagnostic research criteria, (iii) the included patient groups may not exclude AD patients with vascular changes in brain, TDP-43 related hippocampal impairment, and Lewy body pathology; (iv) the use of the term ADMCI to describe amnestic MCI patients, thus

considering possible prodromal ADMCI without in-vivo biomarkers of AD that were not existent in earlier studies; (v) nonhomogeneous procedures for the artifact detection in EEG analyses. Moreover, a variety of analyses and paradigms limit the use of ERPs/EROs along with lower rates of compliance of patients during the recordings. The need for participant- or user-friendly paradigms is paramount, as well as the standardized and harmonized procedures for the acquisition and analyses of ERPs/EROs.

7. CONCLUSIONS

The ERP components or event-related EEG oscillations for investigating treatment effects remains to be an unexplored field. The current diagnostic research criteria work-up for AD described by the Working Group of the National Institute of Aging and Alzheimer's Association (NIA-AA) Research Framework (Jack et al., 2018) do not include EEG measures as those for DLB (McKeith et al., 2020). As earlier studies on event-related EEG measures consistently show association with atrophy in structural MRI or disease progression in AD, they can be considered as reflecting neurodegeneration occurring in the disease course. Furthermore, keeping in mind the low rates of accessibility to the currently validated AD biomarkers, the present Expert Panel posits not only introducing the event-related EEG measures as physiological biomarker (i.e., "P" biomarker) into A-T-N Research Framework (Jack et al., 2018) but also using them to explore treatment effects in AD spectrum. These electrophysiological biomarkers may probe mechanisms of thalamocortical and subcortical neural (de)synchronization in relation to treatment effects. For the purposes of the current study on treatment effects, the biomarkers of ERP/ERO EEG markers may be represented by the mentioned P50 and P300 components, and theta and gamma ERO measures during oddball tasks.

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(2) facilitating international initiatives and clinical applications. EPIA and GBC members believe that EEG biomarkers provide an important resource for research in the field of clinical and pharmacological applications in neuropsychiatric conditions, especially in low- and middle-income countries.

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Treatment effects on event-related EEG potentials and oscillations in Alzheimer's disease

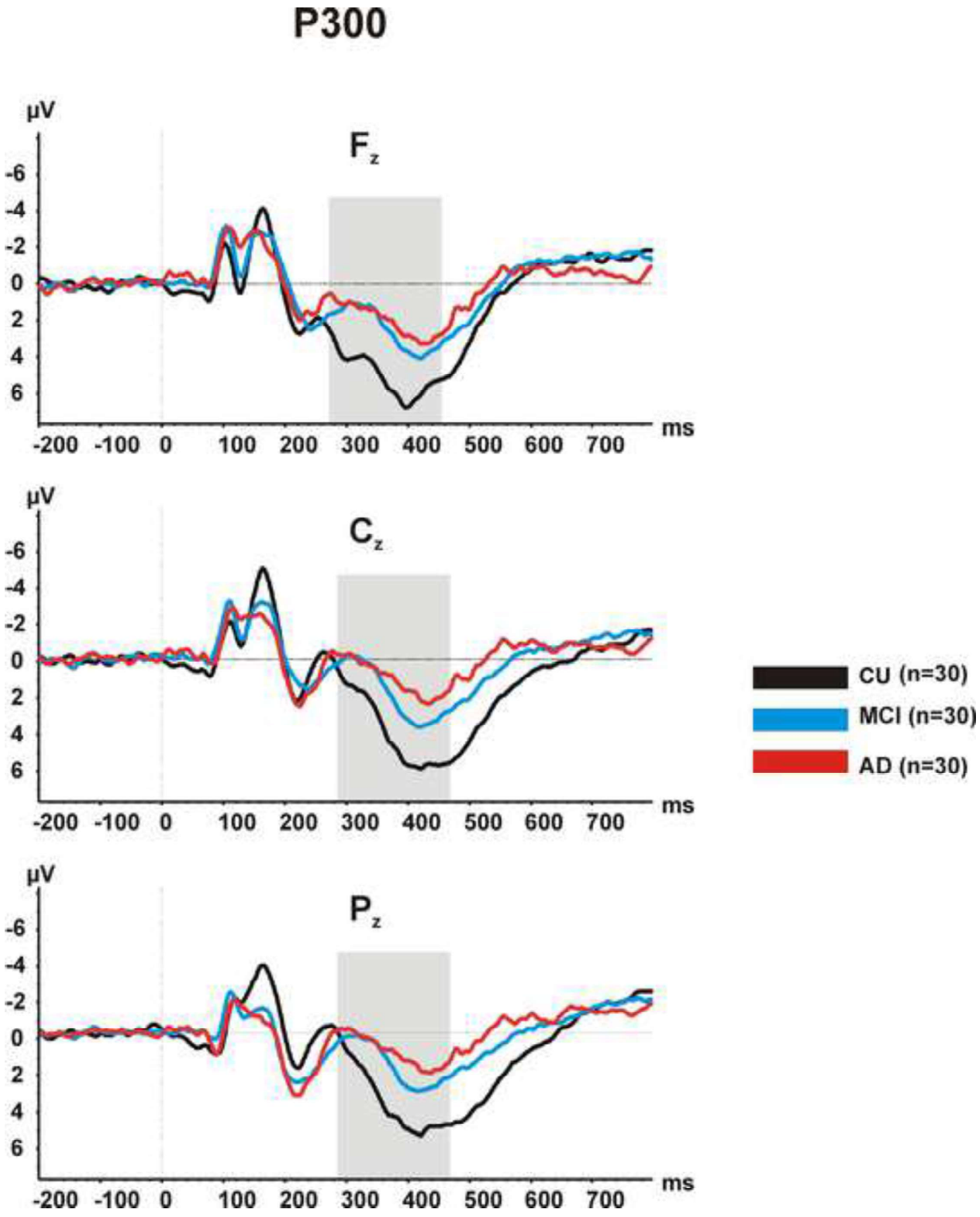

Figure 2

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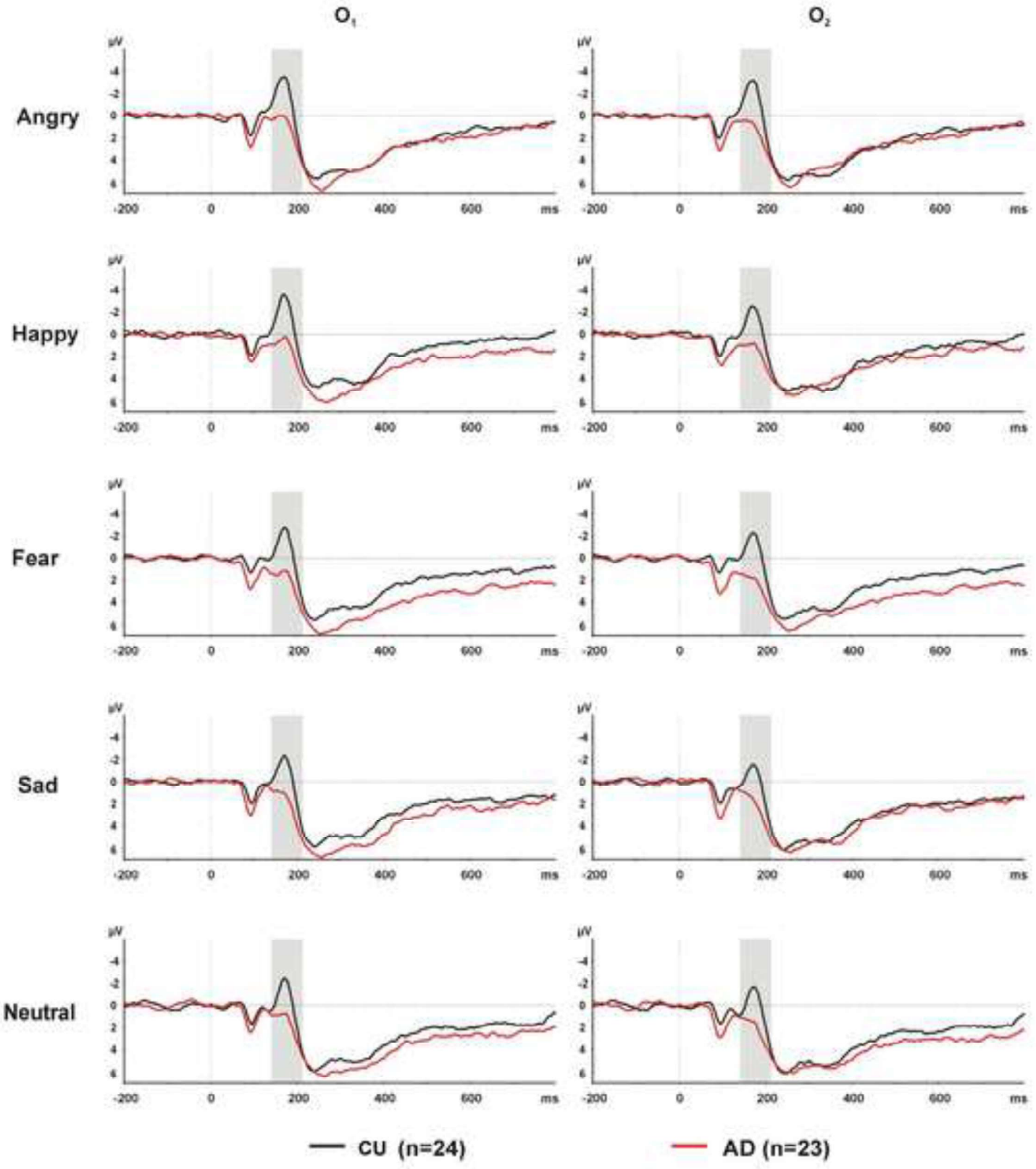
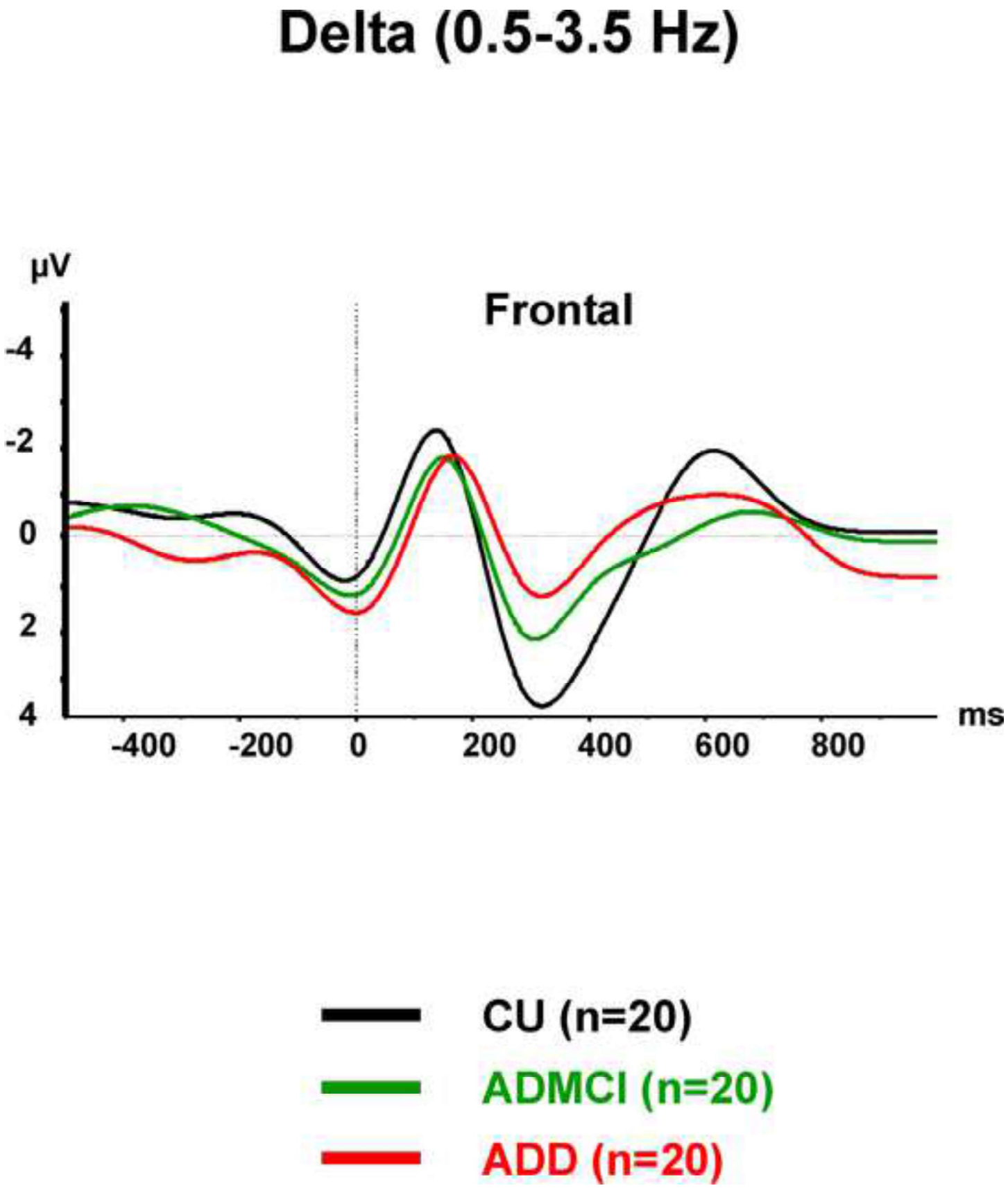
Treatment effects on event-related EEG potentials and oscillations in Alzheimer's disease





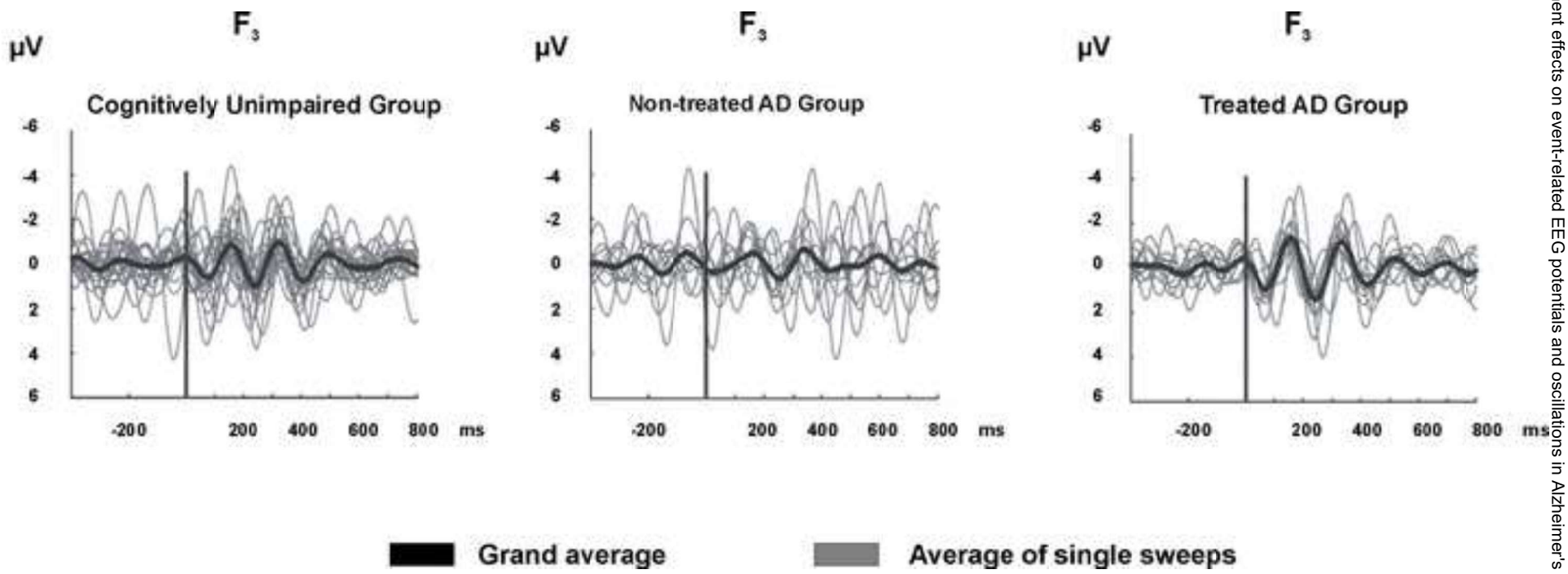


Table 1. ERP waves, their functions, and possible generators

EARLY (Sensory components)
• P50 is early stimulus-evoked potential and is associated with the prefrontal inhibition in the primary/secondary auditory cortex (Katada et al., 2004) • N100 (N1) is involved with primary perceptual processing of incoming information and early attentional allocation to visual stimuli (Lijffijt et al., 2009). • P100 (P1) is a positive wave elicited by different types of visual stimuli only and considered to be related to early visual processing (Heinze and Mangun, 1994). • P200 (P2) As an N100 component, P200 is associated with early attentional allocation to visual stimuli and is involved with primary perceptual processing of incoming information (Omoto et al., 2010). • N170 appears as a negative peak over parieto-occipital regions and is related to bottom-up perceptual processing of faces in the area of occipito-temporal cortex (Feuerriegel et al., 2015).
MID or LATE (Cognitive components)
• N200 (N2) is considered to reflect selective attention and conscious discrimination, so it is associated with information processing, but not necessarily with memory performance (Howe et al., 2014). Possible generators of the N200 include the reticular formation, frontal cortex, centro-parietal cortex thalamus and lemniscus, inferior colliculus, hippocampus, frontocentral cortical areas (Vaitkevičius et al., 2015). • VPP (vertex positive potential) is a positive peak over fronto-central regions within a similar N170 time frame. It is related to bottom-up perceptual processing of faces in the area of the occipito-temporal cortex; it was also reported to reflect an integration of top-down and bottom-up visual processing (Lu et al., 2017). • P300 is related to discrimination between target and standard stimuli that engages focused attention and decision making, or working memory demanded by task (Başar et al., 2001; Posner and Petersen, 1990; Pardo et al., 1991; Başar-Eroğlu et al., 1992; Posner, 1992; Huang et al., 2015). In brief, reverberating circuits between frontal-parietal and temporal cortical regions and possibly their connections with limbic structures take a role in the generation of the P300 potential (Knight, 1990; Soltani and Knight, 2000; Polich, 2003; Rektor et al., 2004). • N400 refers to a negative component in the average ERP that reaches its peak

amplitude approximately 400 ms after stimulus onset and is associated with linguistic and semantic processing (Olichney et al., 2006, 2008, 2011). N400 generators are bilaterally anterior fusiform and parahippocampal gyri (Olichney and Hillert, 2004).

- P600 (LPC) is a positive deflection with a centro-parietal peak at approximately 600 ms. In language studies, the P600/LPC, also known as the 'Syntactic Positive Shift', has been linked to a wide range of disagreements in syntactic rules (Kuperberg, 2007). P600 generators are median temporal lobe and paralimbic cortical regions (Katada et al., 2004; Kimiskidis and Papaliagkas, 2012).

Table 2. Treatment/intervention effects on ERPs in Patients with MCI and ADD

	Participants	Treatment	Task	ERP component	Results
Jiang et al. (2020)	- 92 MCI patients with hyperhomocysteinema- intervention group (N=46) - Control group (N=46) - Before treatment - After the 4th, 12th, 24th week of treatment	Folate of 5 mg/day and VitB 12 of 500 μg × 3/day of	Auditory Oddball Paradigm	- P300	No difference for P300 latency and amplitude in the control group. P300 latency at 24th weeks was shorter than before treatment in the intervention group.
Cespón et al. (2019)	- 14 healthy elderly subjects - 12 ADD patients	Anodal and cathodal tDCS (intensity: 1.5 mA, duration: 13 min) Sham tDCS (10 sec at the beginning and the end of the stimulation period)	n-back task	- P200 - P300	After the anodal tDCS application, the amplitude of P200 and P300 components increased in the healthy elderly. After the cathodal tDCS application, the amplitude of P200 increased in ADD patients.
Fruehwirt et al. (2019)	- 31 possible ADD patients - 32 probable ADD patients - Baseline (BL) assessment - After 6 months (FU1) assessment - After 12 months	39 patients with anti-dementia drug treatment (acetylcholinesterase inhibitors, N-methyl D-aspartate (NMDA) receptor	Auditory Oddball paradigm	- P300 - N200 - P50	P300 latency increased in probable AD patients at Follow Up-3 assessment compared to baseline. Both of P300 and

	(FU2) assessment - After 18 months (FU3) assessment	antagonists), Anti-dementia drug treatment: constant versus variable anti-dementia medication			N200 latencies related to MMSE score and ADDseverity but not P50 latency. No difference for P300 latency between the constant anti-dementia medication in comparison to the variable anti-dementia medication.
Vaitkevičius et al. (2015)	- 22 treatment-naïve ADD patients (ADD-N group) - 22 treated ADD patients (ADD-T group) - 50 healthy controls (Control Group, CG)	The stable donepezil dose of 10 mg/day for at least 3 months	Auditory Oddball task	- P300 - N200	The latencies of P300 at the Cz site; ADD-N = ADD-T>CG No difference for the amplitudes of P300 at the Cz site. -The latencies of N200 at the Cz site; ADD-T>ADD-N -P300-N200 interpeak intervals at the Cz site; ADD-N>ADD-T; ADD-N>CG -Response times; ADD-N>ADD-T ADD-N>CG
Khedr et al. (2014)	34 ADD patients - Anodal tDCS group - Cathodal tDCS group - Sham tDCS group	tDCS application: the left dorsolateral prefrontal cortex for 25	Auditory discrimination task	- P300	After both anodal tDCS and cathodal tDCS application, P300 latency reduced in ADD

	- Baseline - The end of the 10th tDCS session	min at 2 mA, daily for 10 days All of the ADD patients received memantine tablets 10 mg/day for at least 3months			patients. No difference between anodal tDCS and cathodal tDCS application for P300 latency. No difference between before and after any tDCS application for P300 amplitude.
Chang et al. (2014)	- 100 ADD patients - 20 healthy controls	The donepezil treatment (5mg/day) for 22 to 23 weeks	Auditory Oddball paradigm	- N100 - P200 - N200 - P300	ADD patients had prolonged latency of P300 and N200 compared to healthy controls at baseline assessment. No difference between groups for amplitudes of N100, P200, N200, and P300 in comparison to the healthy controls. After donepezil treatment, ADD patients had reduced P300 latency at the Pz site. No difference between pre and post-treatment for both latency and

					amplitude of N100, P200, and N200 and amplitude of P300.
Chapman et al. (2013)	30 MCI subjects - 15 subjects converted to ADD (Convert->ADD) (8 patients with treated subgroup (Meds-Convert) (7 patients with untreated subgroup (NoMeds- Convert) - 15 remained cognitively stable (Stable) (8 patients with treated subgroup (Meds-Stable) (7 patients with untreated subgroup (NoMeds- Stable)	The cholinesterase inhibitors and memantine	Number-Letter paradigm	ERP components: -C1: Slow Wave -C2: CNV -C3: C415 "P3" -C4: C250 "Memory Storage" -C5:C140 -C6: C540 -C7: C325 -C8:C185	ERP classification score was able to distinguish stable MCI from progressive MCI. No difference between those taking medications and those who were not. Convert->ADD group had larger positive classification scores, whereas Stable group had negative classification scores.
Spironelli et al. (2013)	- 11 mild/moderate ADD (treated with cholinesterase inhibitors) - 11 Healthy Control	5-week cognitive training (40 h)	The lexical decision task	-RP (Recognition Potential) -N400	For RP amplitude; After training, ADD patients had increased negativity in posterior left locations for HF words. For N400 component; In HC, NW

					stimulus elicited greater negativity than both LF and HF word stimuli, but not in ADD patients. HC had greater negativity in anterior right locations than ADD patients for NW stimulus, whereas ADD patients had greater negativity in anterior right locations for both LF and HF word stimuli.
Kubová et al. (2010)	- 17 patients with mild-to-moderate Alzheimer's disease (ADD) - Baseline - After 3 months from memantine treatment - After 6 months from memantine treatment	Memantine of 20 mg/d (10 mg twice a day)	Visual oddball paradigm	- P300	Improvement of event-related potentials (shortening of P300 peak latency by at least 20 milliseconds) occurred in 42% of patients with similar results between groups.
Lai et al. (2010)	- 20 patients with probable Alzheimer's disease (ADD) - 18 patients with mild cognitive impairment (MCI) - 14 age-matched normal controls	All ADD patients on treatment with Donepezil MCI patients had no treatment	Auditory Oddball paradigm	- N100 - P200 - N200 - P300	No difference between baseline and follow-up assessments for N100, P200, and N200. In baseline

		Longitudinal study - Baseline - 1 year follow-up			assessment, P300 latency ADD> MCI > HC No difference among groups and for P300 amplitude in baseline and follow-up assessments. In 1-year follow-up assessment, ADD patients had more prolongation of P300 latency, with no amplitude changes at Fz, Cz, and Pz sites compared to baseline assessment, whereas MCI patients had prolongation only at Cz and Pz sites.
Irimajiri et l., (2007)	- 15 HC - 8 treated MCI patients - 7 untreated MCI patients	The cholinesterase inhibitors (ChEIs: donepezil, rivastigmine, galantamine)	Visual paradigm checkerboard stimulation)	Somatosensory Evoked potentials - N20 - P50 Visual Evoked-potentials - N70 - P100 - N150	No changes at visual early components. N20 and P50 amplitudes Untreated MCI>treated MCI
Kalmann et al.	13 patients with ADD	The intravenous	The complex	- N100 (150-	After the lactate treatment,

(2005)	-ADD patients treated with Normal Saline -ADD patients treated with intravenous sodium-lactate	sodium-lactate (SL): 0.5 M SL (5 ml/kg) infused over 20 min	natural color images, comprising either an animal or a non-animal item	250 ms) - P200 (250-400 ms) - N200 (400-600 ms)	the mean amplitudes of N100, P200, and N200 became more negative for stimuli comprising the non-animal images, whereas the mean amplitude of P200 became more positive for stimuli comprising the animal images.
Werber et al. (2003)	32 patients with dementia - 14 patients with ADD - 10 patients with Parkinson's dementia (PDD) - 8 patients with Vascular dementia (VD) -Baseline - After 26 weeks	Cholinesterase inhibitors - Tacrine n=19 (maximally tolerated dose) - Donepezil n=5 (10 mg/day) - Rivastigmine n=8 (12 mg/day)	Auditory Oddball paradigm	-P300	-Decreased P300 latency in dementia patients after the treatment. - No difference for P300 amplitude after the treatment. - No difference between subgroups of dementia for latency or amplitude of P300.
Katada et al. (2003)	- 13 probable ADD patients	Donepezil (DPZ) 3 mg/day for the first week orally and 5 mg/day thereafter over 6 months	Auditory Oddball paradigm	-P300 Baseline (before DPZ treatment) and after 1, 3, and 6 months of the DPZ treatment	After 1 month of the DPZ treatment, decreased P300 latency versus baseline measurement of ADD patients. After 6 months of the DPZ treatment, delayed P300 latency in ADD patients versus 1-

					month follow-up.
Knott et al. (2002)	13 patients with ADD dementia - 6 patients (already taking THA) - 7 patients (non treated group) All patients were given nicotine acutely	THA treatment (cholinesterase inhibitor tacrine) The nicotine polacrilex (Nicorette; 2 mg)	The auditory continuous performance task The visual continuous performance task	- P300 - N100	Pre-nicotine administration ERPs; The THA-treated group had faster P300 latencies compared to the untreated group, with no difference for both amplitude and latency of N100. Post-nicotine administration ERPs; No difference for both amplitude and latency of auditory P300 The nicotine subgroup had larger visual P300 amplitude with no difference for auditory P300 or N100 of any modality.
Onofrj et al. (2002)	30 patients with "mild" ADD 30 patients with "moderate-severe"	Donepezil (DPZ) (10 mg/day) for 6 months		- P300	After Vitamin E treatment, P300 latency increased in both mild and

	AD 40 healthy controls - Group I Donepezil with "mild" ADD (N=15) - Group I Vitamin E with "mild" ADD (N=15) - Group II Donepezil with "moderate-severe" ADD (N=15) - Group II Vitamin E with "moderate-severe" AD (N=15)	Vitamin E (2000 IU/day) for 6 months - Baseline - 6-months follow-up			moderate-to-severe ADD groups. After Donepezil treatment, P300 latency reduced in both mild and moderate-to-severe ADD groups.
Thomas et al. (2001)	- 60 patients with mild to moderately severe probable ADD - 20 patients on Donepezil - 20 patients on Vitamine E - 20 patients on Rivastigmine - 60 control subjects	Throughout 26 weeks; Donepezil (DPZ) 10 mg/d Vitamin E (vit E) (2,000 IU) Rivastigmine (Riv) (12 mg/d)	Auditory oddball paradigm	- P300	The vitamin E-treated patients had increased P300 latency correlated with worsening neuropsychological test scores. Both DPZ-treated and Riv-treated patients had reduced P300 latency. Shortening of the P300 latency was related to higher Wechsler Adult Intelligence Scale scores and lower ADD Assessment Scale-cognitive subscale (ADAS-cog) scores.

Reeves et al. (1999)	- 12 mild-to-moderate ADD patients - Baseline - After 1-month treatment	Donepezil treatment for 1 month (5 mg daily)	Auditory and visual oddball paradigm	- P300	After the 1-month DPZ treatment, both auditory and visual P300 latency reduced in ADD patients. No difference between baseline assessment and after 1-month treatment for auditory and visual P300 amplitude.
Oishi et al. (1998)	- 10 ADD patients	The traditional Chinese medicine (astragalus root, Prunella vulgaris, pueraria root, Lycii fructus, cnidium rhizome, rhubarb, alisma rhizome, peach kernel, ginseng, oyster shell) for 3 months	Auditory oddball paradigm	- P300	P300 latency reduced after the treatment.
Saletu et al. (1995)	- 56 senile dementia of the Alzheimer type ▪ 28 patients (treated with nicergolineADD/NIC) ▪ 28 patients (placebo-AD/PLAC) - 56 multi-	The nicergoline (Sermion) 2 x 30 mg per day for 8 weeks	Auditory oddball paradigm	- P300	After the treatment, P300 latency shortened in both ADD/NIC and MID/NIC groups. After the treatment, P300 latency lengthened in both

	infarct dementia (MID) ▪ 28 patients (treated with nicergoline-MID/NIC) ▪ 28 patients (placebo-MID/PLAC)				ADD/PLAC and MID/PLAC groups.
Dierks et al. (1994)	- 6 healthy young male subjects (physostigmine and biperiden) - 10 healthy elderly subjects (pyritinol)	Physostigmine and biperiden: Physostigmine (0.25mg) for the first day and biperiden (2mg) for the second day Pyritinol (600mg)	Auditory oddball paradigm	- P300	P300 amplitude increased 1 h after application of physostigmine, whereas no change for P300 latency. P300 amplitude decreased 1 h after application of biperiden, whereas P300 latency increased. P300 amplitude increased after application of pyritinol, whereas no change for P300 latency.
Dabic-Jeftic and Mikula (1993)	- 7 patients with Alzheimer's dementia (ADD) - 15 patients with multi-infarct dementia (MID) - Healthy controls	The piracetam treatment for three months	Auditory and visual paradigm	- N100 - P300	ADD and MID patients had longer P300 latencies compared to healthy controls. ADD patients had shorter latency of N100.

					ADD and MID patients had lower P300 amplitudes compared to healthy controls. Treatment with piracetam reduced early components without any changes in P300.
Neshige et al. (1988)	- 13 patients with Alzheimer's disease (ADD) - 14 patients with multi-infarct dementia (MID) - 9 normal controls -Among them 5 patients with ADD and 5 patients with MID received physostigmine	Physostigmine oral	Auditory oddball paradigm	- N100 - P200 - N200 - P300	ADD and MID groups had prolonged N200 and P300 latencies compared to normal controls. Both demented groups had greater P300 latency than the predicted mean value for their age. No difference among groups for N100, P200, N200, and P300 amplitudes. After the treatment, P300 latency decreased in six among 10 patients who received physostigmine whereas prolonged in one MID patient.

Table 3. Treatment effects on EROs in patients with ADD

	Participants	**Task**	**Method /Frequency**	**Results**
Yener et al. (2007)	22 ADD; - 11 unmedicated, - 11 medicated (AChEIs) 20 HC	Visual oddball paradigm	Phase-locking of theta	Unmedicated ADD < HC (at F3) Unmedicated ADD < Medicated ADD (at F3) Medicated ADD and controls were not significantly different
Güntekin et al. (2008)	21 mild probable ADD; - 10 unmedicated, - 11 medicated (AChEIs) 19 HC	Visual oddball paradigm	Delta, theta, and alpha ERO coherences	- Evoked delta, theta, alpha coherences: HC > Unmedicated ADD (over left fronto-parietal electrode pair) -EROs delta coherence: HC > Medicated ADD=Unmedicated ADD (over right fronto-parietal electrode pair) No medication effect on delta. - EROs theta coherence: HC > Medicated ADD=Unmedicated ADD (over left fronto-parietal electrode pair) No medication effect on theta. -EROs alpha coherence: HC= Medicated ADD > Unmedicated ADD (over left fronto-parietal pair)
Yener et al. (2008)	22 mild probable ADD; - 10 unmedicated - 11 medicated (AChEIs) 20 HC	Visual oddball paradigm	Maximum peak-to-peak amplitudes of delta oscillations	-Delta oscillatory responses were reduced in ADD regardless of cholinergic treatment (over left and mid-central electrodes): HC > ADD (for the C_z and C_3 locations) -No medication effect on delta.

Yener et al. (2009)	22 ADD; - 11 unmedicated - 11 medicated (AChEIs) 19 HC	Simple Light stimulation	Digital filtering of sensory-EROs of theta	-No differences in delta, alpha, gamma. -Theta response: Unmedicated ADD > Medicated ADD=HC (over bi-parietal and right occipital electrodes)
Başar et al. (2010)	38 mild probable ADD; - 19 unmedicated - 19 medicated (AChEIs) 19 HC	Simple Light stimulation Visual oddball paradigm	Sensory EROs coherence of delta, theta, and alpha Cognitive EROs Coherence of delta, theta, and alpha	- No differences in Sensory EROs coherence Cognitive EROs delta, theta, and alpha coherences: HC > Medicated and Unmedicated ADD - No changes in Cognitive EROs beta and gamma coherences - Cognitive EROs alpha coherence: HC> Unmedicated ADD (over left fronto-parietal pair)
Yener et al. (2012)	34 mild ADD; - 17 de-novo - 17 medicated (AChEIs) 17 HC	Auditory oddball paradigm Simple Auditory Stimulation	Maximum peak-to-peak amplitudes of delta EROs	- Cognitive EROs HC > either medicated or unmedicated -No group differences during simple sensory Auditory task.
Başar et al. (2017)	39 probable mild ADD; - 21 unmedicated - 18 medicated (AChEIs) 21 HC	Simple Light stimulation Visual oddball paradigm	Sensory-EROs and Cognitive EROs gamma coherences	Both treated and untreated ADD > HC in three sub-gamma bands. Fronto parietal EROs gamma coherences: -Medicated ADD > Unmedicated ADD during both sensory stimulation and oddball paradigm, -Medicated ADD < Unmedicated ADD (over occipital-parietal electrodes) during oddball paradigm.

Table 4. The treatment effects of animal Studies for ERP/EROs in AD

	Participants	Treatment	Task	ERP component	Results
Kim et al. (2020)	- 12 Aβ-infused mice model (Aβ group) - 7 normal mice model (vehicle group)	Aβ-infusion	Auditory oddball paradigm	-P100 (10-25 ms) -N100 (25-45 ms) -P200 (45-200 ms)	The decreased difference of ERP responses between standard and deviant tones in the Aβ-infused mice group The reduced difference of N1 component between standard and deviant tones in the parietal region in the Aβ-infused group
Nouriziabari et al. (2018)	4 group of 12 rats -Saline-treated, GFP expressing (GFP-saline) -Saline-treated, tau-expressing (Tau-saline) -Scopolamine treated, GFP expressing (GFP-scopolamine) -Scopolamine treated, tau-expressing (Tau-scopolamine)	The low-dose scopolamine hydrobromide treatment subcutaneously	Trace eyeblink conditioning paradigm	For auditory ERPs, - P100 (15–25 ms) - P200 (40–80 ms) - P300 (140–400 ms) - N100 (25–40 ms) - N200 (80–150 ms) For visual ERPs, P100 (15–70 ms) P200 (80–150 ms) P300 (180–290 ms) N100 (50–100 ms) N200 (120–200 ms)	With learning, the amplitude of ERP components in the frontal and temporal regions came to differentiate the CS+ from CS- Scopolamine caused the learning-related changes in the temporal P2 component and other learning-unrelated components in three locations. Entorhinal tau overexpression primary affected the amplitude of temporal visual ERPs and learning-unrelated frontal and temporal auditory ERP components.
Laursen et	21 Sprague	Donepezil	Auditory	- P300-like	Reduced amplitude of

al. (2014)	Dawley rats -10 animals infused 1.25 µg IgG-192-SAP -11 animals sham-lesioned with sterile PBS	hydrochloride 1 mg/kg subcutaneously	oddball paradigm	component	P3-like ERPs in SAP-lesioned rats compare to sham-lesioned rats. After the treatment with 1 mg/kg donepezil, increased P3 amplitude in SAP-treated rats.
Guadagna et al . (2015)	Anesthetized mice	Memantine	Induced EEG activity by electrical stimulation and spontaneous EEG		Dose dependent alteration in induced theta activity in hippocampus (increase in low dose and decreased in high dose), whilst no change in spontaneous theta rhythms. Increase in spontaneous and induced-gamma power.
Hajos et al. (2013)	Anesthetized mice	Semagacestat, a gamma secretase inhibitor reducing amyloid-β	Induced EEG activity by electrical stimulation and spontaneous EEG		Decreased induced-theta activity in hippocampus
Kinney et al. (1999)	Anesthetized rats	Piracetam, a nootropic agent	Induced EEG activity by electrical stimulation		Increased induced-theta activity in hippocampus
Kinney et al. (1999)	Anesthetized rats	Apamin,a potassium channel blocker	Induced EEG activity by electrical stimulation		Increased induced-theta activity in hippocampus

(For ERP/EROs related reviews please see, Paitel et al., 2021; Rossini et al., 2020; Tarawneh et al., 2020; Horvath et al., 2018; Morrison et al., 2018; Palop and Mucke, 2016; Seer et al., 2016; Feuerriegel et al., 2015; Hedges et al., 2016; Huang et al., 2015; Nimmrich et al., 2015; Güntekin and Başar, 2014; Howe et al., 2014; Howe, 2014; Tsolaki et al., 2014; Başar, 2012;

Başar and Güntekin, 2008, 2012, 2013; Morrison et al., 2013; Yener and Başar, 2010, 2013; Farwell et al., 2012; Kimiskidis and Papaliagkas, 2012; Rêgo et al., 2012; Yamasaki et al., 2012; Drago et al., 2011; Lizio et al., 2011; Vecchio and Määttä, 2011; Dauwels, et al, 2010; Jackson and Snyder, 2008; Sauseng and Klimesch, 2008; Uhlhaas and Singer, 2008; Rossini et al. 2007; Prichep et al., 2005; Hermann and Demiralp, 2005; Polich and Corey Bloom, 2005; Jeong, 2004; Katada et al, 2004; Olichney and Hillert, 2004; Başar-Eroğlu et al., 2001; Klimesch., 1999; Başar-Eroğlu and Demiralp, 1991; and for hypotheses and rules for EROs, the following articles have been recommended; Hebb et al 1949; Başar-Eroğlu et al., 1991, 1992, 2001; Schürmann et al., 1997; Sakowitz et al 2001.