Clinical Neurophysiology 129 (2018) 1410–1417

Contents lists available at ScienceDirect

Clinical Neurophysiology

journal homepage: www.elsevier.com/locate/clinph

Temporal stability of posterior EEG alpha over twelve years

Craig E. Tenke [a,b,c], Jürgen Kayser [a,b,c,*], Jorge E. Alvarenga [a], Karen S. Abraham [a], Virginia Warner [b,d], Ardesheer Talati [b,c,d], Myrna M. Weissman [b,c,d], Gerard E. Bruder [a,c]

[a] Division of Cognitive Neuroscience, New York State Psychiatric Institute, New York, NY, USA
[b] Division of Translational Epidemiology, New York State Psychiatric Institute, New York, NY, USA
[c] Department of Psychiatry, Columbia University College of Physicians & Surgeons, New York, NY, USA
[d] Mailman School of Public Health, Columbia University, New York, NY, USA

ARTICLE INFO

Article history:
Accepted 20 March 2018
Available online 16 April 2018

Keywords:
EEG
Temporal stability
Trait biomarker
Surface Laplacian
Current source density (CSD)
Principal components analysis (PCA)

HIGHLIGHTS

- Evaluates test-retest reliability of posterior EEG alpha at rest in recordings separated by 12 years.
- Reliability was excellent in adults for overall alpha and acceptable for net and overall asymmetry.
- Reliability was substantially weaker in children or adolescents (aged <18 at first test session).

ABSTRACT

Objective: We previously identified posterior EEG alpha as a potential biomarker for antidepressant treatment response. To meet the definition of a trait biomarker or endophenotype, it should be independent of the course of depression. Accordingly, this report evaluated the temporal stability of posterior EEG alpha at rest.

Methods: Resting EEG was recorded from 70 participants (29 male; 46 adults), during testing sessions separated by 12 ± 1.1 years. EEG alpha was identified, separated and quantified using reference-free methods that combine current source density (CSD) with principal components analysis (PCA). Measures of overall (eyes closed-plus-open) and net (eyes closed-minus-open) posterior alpha amplitude and asymmetry were compared across testing sessions.

Results: Overall alpha was stable for the full sample (Spearman-Brown [r_{SB}] = .834, Pearson's r = .718), and showed excellent reliability for adults (r_{SB} = .918; r = 0.848). Net alpha showed acceptable reliability for adults (r_{SB} = .750; r = .600). Hemispheric asymmetries (right-minus-left hemisphere) of posterior overall alpha showed significant correlations, but revealed acceptable reliability only for adults (r_{SB} = .728; r = .573). Findings were highly comparable between 29 male and 41 female participants.

Conclusions: Overall posterior EEG alpha amplitude is reliable over long time intervals in adults.

Significance: The temporal stability of posterior EEG alpha oscillations at rest over long time intervals is indicative of an individual trait.

1. Introduction

The hope offered by biomarkers and endophenotypes (Beauchaine, 2009) of antidepressant treatment response rests on the elimination or reduction of costly treatment delays by supplementing or replacing trial-and-error testing with individualized treatment (Trivedi et al., 2016). Resting EEG activity in the alpha and theta bands is predictive of a positive response to a range of antidepressants (for reviews, see Alhaj et al., 2011; Bruder et al., 2013). Greater alpha power, particularly at posterior scalp sites, has been reported in patients who respond to antidepressants compared to those who do not (Tenke et al., 2011; Ulrich et al., 1986), especially over right hemisphere sites (Bruder et al., 2008). Owing to these clinical interests, the temporal stability of EEG measures has been frequently studied on a time scale conforming to typical treatment protocols, with high test-retest reliability of EEG alpha and theta power reported for depressed patients (Bruder et al., 2008: 12 weeks) as well as healthy adults

* Corresponding author at: New York State Psychiatric Institute, Unit 50, 1051 Riverside Drive, New York, NY 10032, USA.
E-mail address: kayserj@nyspi.columbia.edu (J. Kayser).

https://doi.org/10.1016/j.clinph.2018.03.037
1388-2457/

(Enoch et al., 2008: 2 years; Smit et al., 2005: 1.8 years; Tomarken et al., 1992: 3 weeks; Tenke et al., 2017a: 1 week).

In marked contrast to the brain changes that underlie clinical depression or treatment outcome, an endophenotype should precede the onset of the disorder and persist independent of its course or the outcome following treatment (Beauchaine, 2009; Gottesman and Gould, 2003; Gould and Gottesman, 2006). Consequently, individual differences in EEG measures should be demonstrable and persistent not just in clinical populations, but in preclinical and healthy populations as well, with trait-like stability over substantial periods of time. High test-retest correlations have been reported for broad band spectral amplitude measures at time scales of months (Salinsky et al., 1991), and for spectral patterns over 1–3 years (Näpflin et al., 2007).

This report assessed the temporal stability of posterior EEG alpha in a sample of participants in a longitudinal study of familial risk of major depressive disorder (MDD), with EEG testing sessions separated by a span of twelve years. Following our previous recommendations (Tenke et al., 2017a), posterior EEG alpha was identified and quantified using reference-free measures that combine current source density (CSD) frequency spectra with principal components analysis (CSD-fPCA; Tenke and Kayser, 2005; Tenke et al., 2011, 2013, 2017a, 2017b). Given prior evidence that overall alpha amplitude is in part unrelated to the classical Berger (1929) effect, that is, the condition-dependent blockade of resting alpha with eyes open as opposed to closed (net alpha; Tenke et al., 2015), we evaluated both aspects of posterior alpha as core characteristics of the resting EEG paradigm.

2. Methods

2.1. Participants

Participants (N = 70, 41 female, 59%) were part of a multiwave, three-generation longitudinal study of individuals at high and low risk for major depression based on family history (Weissman et al., 1997, 2005, 2016a, 2016b). In the original wave of the study, probands with moderate to severe major depressive disorder were selected from outpatient clinics for the psychopharmacologic treatment of mood disorders, and nondepressed, demographically-matched control participants were selected from an epidemiologic sample of adults with no psychiatric history from the same community. The sample was recruited from an urban setting (greater New Haven area, Connecticut, US), and consisted of Caucasian, working or middle class individuals. Clinical assessments were conducted by independent interviewers who were blind to the participant's previous clinical history (personal or family). All assessments were approved by the institutional review boards at Yale University and at Columbia University/New York State Psychiatric Institute (NYSPI). Written informed consent was obtained from all participants, or from the legal guardian for minors.

Participants were included in this study solely based on the availability of EEG data at both time points, in marked contrast to cross-sectional analyses from this sample aimed at distinguishing EEG differences reflecting familial risk status for MDD, generation, lifetime history of MDD, and interview items associated with protection against, or resilience following, MDD (Bruder et al., 2005, 2007; Tenke et al., 2013, 2017b). Collection of resting EEG was added to the study protocol at Wave 4 (approximately 20-year time point at Yale) and repeated at Wave 6 (30-year time point at Columbia/NYSPI). The mean time interval elapsed between test and retest was 12 ± 1.1 years (range 9.4–15.5 years). Most participants were children (n = 34) or grandchildren (n = 29) of the proband, with approximately equal representation of high (n = 33) and low (n = 30) familial risk status. Participants at high familial risk had significantly greater lifetime rates of MDD at Wave 6 (60.6%) than those in the low risk group (30.5%; Fisher exact test, one-sided, p = .027). The remaining seven participants were unrelated to either proband (i.e., they married-into the study); however, as family risk status is of secondary importance for the primary objective of evaluating the temporal stability of posterior EEG alpha, these individuals were included in the overall analysis to increase sample size and statistical power. Wave 4 EEG from 18 of these participants (Tenke et al., 2013; N = 52), and Wave 6 EEG from 46 participants (Tenke et al., 2017b; N = 73) were included in previous reports using comparable EEG methods.

The participants' mean ages were 26 ± 13.3 years (range = 5.2–47.6) at Wave 4 and 38 ± 13.2 years (range = 17.1–59.2) at Wave 6. To account for anticipated developmental differences in EEG, temporal stability was computed separately for the full current sample (N = 70) and for an adult subsample (≥18 years; n = 46).

2.2. EEG methods

As many aspects of EEG acquisition, processing and analysis have been detailed in prior reports (e.g., Tenke et al., 2011, 2013, 2017a, 2017b), we will present a brief overview with methodological details that are unique to this report.

2.2.1. Resting EEG paradigm

EEG at rest was measured while participants sat quietly during four 2-min periods (order of eyes-open and eyes-closed counterbalanced across participants and assessments) after being instructed to avoid blinking and eye or body movements (fixation cross used for eyes-open condition; e.g., Tenke et al., 2011).

2.2.2. Wave 4: EEG acquisition

Thirteen scalp EEG channels based on the 10/20 system (4 frontal: F7/8, F3/4; 5 central: T7/8, C3/4, Cz; 4 parietal: P7/8, P3/4), two EEG reference channels (right and left ear, digitally re-referenced to linked ears), and bipolar recordings to monitor blinks (above vs. below right eye) and horizontal eye movements (right vs. left canthi) were recorded at Yale University using an electrode cap (Electro Cap International; see Tenke et al., 2013). The EEG was acquired using a Bioamplifier system (James Long Company) at a gain of 10 K and a band pass of .01–30 Hz. EEG data were continuously acquired at 200 samples/s (NeuroScan, 2003) and segmented off-line into consecutive 1.28-s epochs every .64 s (50% overlap). Epochs contaminated by blinks, eye movements, or movement-related artifacts were excluded using a rejection criterion of ±100 μV on any channel, followed by interactive rejection of remaining artifacts (Bruder et al., 2005).

2.2.3. Wave 6: EEG acquisition

Continuous data (256 samples/s) were recorded at NYSPI using an Active2 recording system (BioSemi, 2001) with a 72-channel 10/10 system scalp montage (Jurcak et al., 2007; Pivik et al., 1993; see Tenke et al., 2017a) including the nose as a reference. Data were blink-corrected offline using a spatial, singular value decomposition (NeuroScan, 2003), and segmented into 2-s epochs (75% overlap). Epoched data were screened for electrolyte bridges (Alschuler et al., 2014; Tenke and Kayser, 2001), and affected channels interpolated via spherical splines (Perrin et al., 1989). Additional EEG data were preserved by identifying isolated EEG channels containing artifacts or noise on any given epoch (e.g. amplifier drift, residual eye activity, muscle or movement-related artifacts) using a reference-free approach (Kayser and Tenke, 2006b; Tenke et al., 2017a), which were then replaced by spherical spline interpolations (Perrin et al., 1989) from artifact-free channels (i.e., artifacts identified in less than 25% of all channels for that epoch). Bipolar vertical and horizontal EOGs were interpolated via