Assessing Trial-by-Trial Electrophysiological and Behavioral Markers of Attentional Control and Sensory Precision in Psychotic and Mood Disorders

Megan A. Boudewyn 1,*, Molly A. Erickson 2, Kurt Winsler 3, Deanna M. Barch 4,*, Cameron S. Carter 5, Michael J. Frank 6, James M. Gold 7,*, Angus W. MacDonald III 8, J. Daniel Ragland 1, Steven M. Silverstein 9, Andrew P. Yonelinas 3, and Steven J. Luck 1,*

1Department of Psychology, University of California, Santa Cruz, California, USA; 2Department of Psychiatry and Behavioral Neuroscience, University of Chicago, Chicago, Illinois, USA; 3Department of Psychology, University of California, Davis, California, USA; 4Department of Psychological and Brain Sciences, Washington University in St. Louis, St. Louis, Missouri, USA; 5Department of Psychiatry and Human Behavior, University of California, Irvine, California, USA; 6Department of Cognitive, Linguistics and Psychological Sciences, Brown University, Providence, Rhode Island, USA; 7Department of Psychiatry, University of Maryland School of Medicine, Baltimore, Maryland, USA; 8Department of Psychology, University of Minnesota, Minneapolis, Minnesota, USA; 9Department of Psychiatry, University of Rochester Medical Center, Rochester, New York, USA

To whom correspondence should be addressed; Department of Psychology, University of California, Santa Cruz, Santa Cruz, CA, USA; tel: (831) 459-2002; e-mail: mboudewyn@ucsc.edu

Background and Hypothesis: The current study investigated the extent to which changes in attentional control contribute to performance on a visual perceptual discrimination task, on a trial-by-trial basis in a transdiagnostic clinical sample. Study Design: Participants with schizophrenia (SZ; N = 58), bipolar disorder (N = 42), major depression disorder (N = 51), and psychiatrically healthy controls (N = 92) completed a visual perception task in which stimuli appeared briefly. The design allowed us to estimate the lapse rate and the precision of perceptual representations of the stimuli. Electroencephalograms (EEG) were recorded to examine pre-stimulus activity in the alpha band (~8–13 Hz), overall and in relation to behavior performance on the task. Study Results: We found that the attention lapse rate was elevated in the SZ group compared with all other groups. We also observed group differences in pre-stimulus alpha activity, with control participants showing the highest levels of pre-stimulus alpha when averaging across trials. However, trial-by-trial analyses showed within-participant fluctuations in pre-stimulus alpha activity significantly predicted the likelihood of making an error, in all groups. Interestingly, our analysis demonstrated that aperiodic contributions to the EEG signal (which affect power estimates across frequency bands) serve as a significant predictor of behavior as well. Conclusions: These results confirm the elevated attention lapse rate that has been observed in SZ, validate pre-stimulus EEG markers of attentional control and their use as a predictor of behavior on a trial-by-trial basis, and suggest that aperiodic contributions to the EEG signal are an important target for further research in this area, in addition to alpha-band activity.

Key words: EEG/visual perception/sensory precision/attention lapsing/schizophrenia/depression/bipolar disorder

Introduction

Attentional control refers to the ability to maintain focus on a task, keeping task-relevant goals in mind and avoiding distraction. However, attentional control is limited. It waxes and wanes, and current task-relevant goals can become momentarily lost—a lapse of attention. Poor attentional control is a feature of several mental health disorders such as schizophrenia (SZ). Here, we evaluated the extent to which attentional control fluctuations contribute to performance on a perceptual discrimination task, in a transdiagnostic sample including healthy control participants (HC), people with SZ, people with bipolar disorder (BP), and people with major depressive disorder (MDD).

We consider attentional control as on a continuum ranging from optimal task engagement to attentional disengagement such that task performance is significantly compromised. Behaviorally, fluctuations in attentional control can be estimated using errors on easy trial types (“catch” trials). Attentional control can also be assessed using electrophysiological activity in the alpha frequency band (~8–13 Hz), thought to reflect a perceptual gating
or inhibitory function, such that increased alpha reduces perceptual sensitivity.\textsuperscript{3–5} This interpretation is supported by studies that have found greater alpha power when attention is directed inwardly or when participants overtly fail to perceive a visual target stimulus.\textsuperscript{6–8}

Several EEG studies have examined alpha and attention lapses in psychiatrically healthy populations. For example, increased alpha during the presentation of key pieces of information in a story listening task is associated with self-reported lapsing and poorer item comprehension on tests.\textsuperscript{9,10} Pre-stimulus alpha also predicts visual discrimination performance and working memory recall, with poorer performance on trials with greater alpha power.\textsuperscript{11,12} Although fewer studies have explored this issue in clinical populations, there is some evidence for a similar link between alpha and poor language comprehension in SZ\textsuperscript{13} and with increased attention lapsing during a visual working memory task in SZ.\textsuperscript{14}

Thus, there is substantial evidence that pre-stimulus alpha can serve as a marker of attention under task conditions in which attention must be directed to behaviorally relevant incoming stimuli. It is possible that increased alpha leads to reduced attentional control. Alternatively, increased alpha may be a consequence of reduced attentional control, along with behavioral markers such as increased error rates on a task. In the former case, we might expect that average alpha levels would be higher in clinical populations in which attentional control is impaired (eg, SZ).\textsuperscript{15,16} In the latter case, we would not necessarily expect this pattern, as other mechanisms may also produce lapses. Indeed, some previous work suggests attention lapse behavior in SZ may not be characterized by mind-wandering, as tends to be the case in HC.\textsuperscript{17}

Here, we examined behavioral performance and pre-stimulus alpha during a visual perceptual discrimination task that permitted estimation of the attention lapse rate and the precision of participants’ perceptual representations of the stimuli. Our primary goals were to (1) determine whether there were group differences in perceptual precision and/or attention lapse rate; (2) determine whether there were group differences in pre-stimulus alpha power; and (3) to examine the extent to which pre-stimulus alpha was predictive of behavioral performance on a trial-by-trial basis across diagnostic groups.

A secondary goal of this study was to leverage new analytic methods that provide greater specificity about the aspects of the EEG signal that vary by group or predict behavior. EEG is a mixture of oscillating, rhythmic activity at different frequencies (periodic activity) and non-oscillating activity (aperiodic activity) that extends across a broad band of frequencies, typically falling off in a 1/f manner (meaning that the power of the signal is inversely proportional to the frequency).\textsuperscript{18} Traditional approaches to quantifying alpha conflate periodic alpha-band oscillations with aperiodic activity that extends across frequency bands, making it difficult to know whether a difference between groups or conditions reflects a change in periodic or aperiodic activity. We computed alpha power using a standard Fourier-based approach, as well as using a novel approach that provided separate estimates of periodic and aperiodic activity.\textsuperscript{18}

**Materials and Methods**

**Participants**

Participants in this study were recruited from 5 different sites as part of the Cognitive Neuroscience Test Reliability and Computational applications for Schizophrenia Consortium (CNTRaCS): University of California, Davis, University of Chicago, Maryland Psychiatric Research Center, University of Minnesota, and Washington University in St. Louis. All recruitment, consent, and experimental procedures were approved by each site’s Institutional Review Board. EEG data on this task were collected from 270 participants. Twenty-six participants (HC: 2; SZ: 14; BP: 5; MDD: 5) were excluded from analyses for having unusable EEG or task data. Thus, final analyses were conducted on data from 244 participants (HC: 93; SZ: 58; BP: 42; MDD: 51). Demographics and clinical characteristics by group are summarized in table 1.

Diagnostic criteria were established using the DSM-5 by trained and calibrated raters. Raters were trained with remote webinars in which rating scales and anchor points were discussed. Raters also completed and discussed a set of 6 training videos. Following this training, raters then worked to achieve consensus in their ratings with “gold standard” ratings that were supplied by experienced clinicians at the Maryland and St. Louis sites for at least 6 interviews. Consensus was defined as no more than 2 items with a difference of more than 1 rating point from the standard. To maintain inter-rater reliability over the course of the study, the St. Louis site recorded an interview to rate every 2–4 weeks, and all raters participated in remote meetings to resolve any discrepancies in ratings of this interview.

All participants completed a Structured Clinical Interview (SCID) for DSM-5 with a trained researcher supervised by a doctoral-level clinician as described above. All participants were between the ages of 18–65, reported no history of serious head injury or neurological disorder, were free from a current substance use disorder, and did not have a pervasive developmental disability as indicated by a score of 16 or greater on the Weschler Test of Adult Reading (WTAR).

Additional criteria for participants with SZ: (1) DSM-5 diagnosis of SZ or schizoaffective disorder based on SCID interview; (2) no medication changes in the previous month or anticipated in upcoming month; (3) stable outpatient status.

Additional criteria for participants with BP: (1) DSM-5 diagnosis of BP based on SCID interview; (2) no
medication changes in the previous month or anticipated in upcoming month; (3) stable outpatient status.

Additional criteria for participants with MDD: (1) DSM-5 diagnosis of major depression based on SCID interview; (2) no medication changes in the previous month or anticipated in upcoming month; (3) stable outpatient status; (4) at least 2 episodes within last 2 years.

Task

The task was presented using PsychoPy. Task files are available for download at [https://github.com/lucklab/CNTRACS_tasks_public](https://github.com/lucklab/CNTRACS_tasks_public). As illustrated in figure 1, participants were presented with a black bar and a white bar on an invisible circle and tasked with indicating whether the white bar was presented to the left or right of the black bar. The space between the 2 bars decreased (got harder) or increased (got easier) depending on the participant’s performance. The spacing was in units of angular degrees around the circle of possible locations (separation). On 80% of trials, we used a QUEST staircase to adjust the distance between the 2 bars so that we could estimate the participant’s threshold (the distance between the 2 bars at which a participant performed at 80.35% correct). The minimum and maximum threshold values used in the

### Table 1. Demographics and Clinical Characteristics by Group

<table>
<thead>
<tr>
<th>Diagnostic Group</th>
<th>HC</th>
<th>SZ</th>
<th>BP</th>
<th>MDD</th>
<th>P Value</th>
<th>Post Hoc Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sample size (n)</td>
<td>93</td>
<td>58</td>
<td>42</td>
<td>51</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (y)</td>
<td>35.46 (SD 10.26)</td>
<td>37.28 (SD 11.6)</td>
<td>36.66 (SD 11.31)</td>
<td>31.68 (SD 7.88)</td>
<td>.03</td>
<td>SZ &gt; MDD</td>
</tr>
<tr>
<td>Gender</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>45 (48.39%)</td>
<td>17 (29.31%)</td>
<td>23 (54.76%)</td>
<td>26 (50.98%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>47 (50.53%)</td>
<td>38 (65.52%)</td>
<td>18 (42.86%)</td>
<td>21 (41.18%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nonbinary</td>
<td>1 (1.1%)</td>
<td>3 (5.17%)</td>
<td>1 (2.38%)</td>
<td>4 (7.84%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Race</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Asian</td>
<td>9 (9.68%)</td>
<td>2 (3.45%)</td>
<td>1 (2.38%)</td>
<td>3 (5.89%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Black</td>
<td>26 (27.96%)</td>
<td>26 (44.83%)</td>
<td>5 (11.9%)</td>
<td>8 (15.69%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>More than 1 race</td>
<td>6 (6.45%)</td>
<td>2 (3.45%)</td>
<td>4 (9.52%)</td>
<td>1 (1.96%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>White</td>
<td>44 (47.31%)</td>
<td>27 (46.56%)</td>
<td>30 (71.43%)</td>
<td>37 (72.55%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Other</td>
<td>6 (6.45%)</td>
<td>1 (1.72%)</td>
<td>1 (2.38%)</td>
<td>0 (0%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Unreported</td>
<td>2 (2.15%)</td>
<td>0 (0%)</td>
<td>1 (2.38%)</td>
<td>2 (3.92%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Education</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Self (y)</td>
<td>16.22 (SD 2.4)</td>
<td>13.89 (SD 2.64)</td>
<td>15.49 (SD 2.67)</td>
<td>16.26 (SD 2.22)</td>
<td>&lt;.001</td>
<td>HC, BP, MDD &gt; SZ</td>
</tr>
<tr>
<td>Parental (y)</td>
<td>14.06 (SD 3.38)</td>
<td>14.67 (SD 2.62)</td>
<td>14.72 (SD 3.13)</td>
<td>15.18 (SD 2.89)</td>
<td>.215</td>
<td></td>
</tr>
<tr>
<td>Symptom assessment</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CAINS</td>
<td>12.59 (SD 7.32)</td>
<td>9.28 (SD 5.95)</td>
<td>10.61 (SD 5.47)</td>
<td>10.61 (SD 5.47)</td>
<td>.037</td>
<td>SZ &gt; BP</td>
</tr>
<tr>
<td>BPRS—Positive</td>
<td>2.3 (SD 1.38)</td>
<td>1.32 (SD 0.48)</td>
<td>1.12 (SD 0.23)</td>
<td>1.12 (SD 0.23)</td>
<td>.001</td>
<td>SZ &gt; BP and MDD</td>
</tr>
<tr>
<td>BPRS—Disorganized</td>
<td>1.37 (SD 0.43)</td>
<td>1.25 (SD 0.33)</td>
<td>1.15 (SD 0.2)</td>
<td>1.15 (SD 0.2)</td>
<td>.004</td>
<td>SZ &gt; MDD</td>
</tr>
<tr>
<td>WTAR</td>
<td>110.7 (12.65)</td>
<td>105.14 (14.99)</td>
<td>114.08 (10.46)</td>
<td>112.71 (11.93)</td>
<td>.003</td>
<td>BP and MDD &gt; SZ</td>
</tr>
</tbody>
</table>

Data collection site key: (1) University of California, Davis; (2) University of Chicago; (3) Maryland Psychiatric Research Center; (4) University of Minnesota; (5) Washington University in St. Louis. Note: BP, bipolar disorder; HC, healthy control; MDD, major depressive disorder; SZ, schizophrenia.
staircase procedure were 0.1° and 5.1°. The other 20% of trials were “catch” trials in which the bars were so widely spaced that performance should have been perfect unless a lapse of attention occurred (6° apart for all participants). Catch trial performance was used to estimate the attention lapse rate. As participants would be expected to respond correctly (by chance) on 50% of the trials on which they made an attention lapse, for the purposes of this study the lapse rate was estimated as 2x the actual lapse rate, to better estimate the true number of attention lapses. There were 320 regular trials and 80 catch trials. Ten participants with a lapse rate greater than 50% or with the maximum threshold (an indicator of poor task comprehension) were excluded from all analyses.

Our task was designed to be robust across modest differences in equipment and viewing conditions. The stimuli were presented at high contrast, very far above the threshold, so minor differences in displays and ambient lighting would have minimal impact on perceptibility. In addition, thresholds in tasks such as this depend on the polar angle between the stimuli around the circle of possible stimulus locations, not the absolute visual angle between the 2 items.20 Because changes in viewing distance impact the absolute visual angle and not the polar angle, performance is relatively unaffected by modest changes in viewing distance.

**EEG Recording and Preprocessing**

EEG was recorded using a Brain Products active electrode ActiCHamp system with 29 scalp electrodes and 3 electroocular (EOG) electrodes (1 below the left eye, 1 on the left outer canthus, and 1 on the right outer canthus). Our channel layout is depicted in figure 2. Channels were referenced online to electrode P9 (a proxy for left mastoid). Data were re-referenced offline to the average of P9/P10. Data were recorded with a sampling rate of 500 Hz and electrode impedances were kept below 50 kOhms.

EEG data were preprocessed offline using Matlab with EEGLab Toolbox21 and the ERPlab plugin.22 Our preprocessing approach is detailed in ref. 23. Briefly, the data were high-pass filtered using a noncausal Butterworth filter (half-amplitude cutoff of 0.05 Hz, 12 dB/octave slope) and screened for nonfunctioning channels. Nonfunctioning channels were defined as channels with unusable data during 1/3 or more of the total recording time. Independent component analysis (ICA) was used to correct for eye blinks and horizontal eye movements using the approach described in ref. 24. The nonfunctioning channels were excluded from ICA and then interpolated after ICA using a spherical spline algorithm. About 67% of all participants had zero nonfunctioning channels requiring interpolation (HC: 72.5%; SZ: 60.34%; BP: 59.5%; MDD: 74.5%); the remaining participants had 1–6 channels requiring interpolation (average: HC: 2.56; SZ: 2.78; BP: 2.24; MDD: 2.54).

After preprocessing, the continuous EEG was segmented into −1500 to 1500 epochs, time-locked to stimulus onset. Epochs were screened for any remaining artifacts, using ERPlab functions for detecting extreme values (default set to −200 to 200 µV) and moving window artifact detection. Following our standard exclusion procedure, participants with more than 50% of trials containing artifacts were excluded from analysis (N = 16). On average, 9.92% of trials were rejected by the remaining participants due to the presence of artifacts (HC: 7.6%; SZ: 12.88%; BP: 10.51%; MDD: 10.29%).

**EEG Analysis**

Single-trial estimates of total alpha power were calculated by taking the average spectral power between 8 and 13 Hz in the pre-stimulus period of each trial, calculated using a fast-Fourier transform for each electrode (Total Alpha Power). To reduce noise, the average frequency spectra for each electrode and its 3–5 neighbors (depending on whether it was an edge or middle electrode) were then calculated for each trial. Thus, for all analyses reported in the paper, each electrode represents the average of a cluster centered at that electrode.

Single-trial estimates of periodic alpha power and the aperiodic exponent of the signal were calculated using the Fitting Oscillations & One Over F (Fooof) approach
and algorithm detailed in ref. 18. This approach yielded estimates of Periodic Alpha Power, which reflects alpha-band power that surpassed the aperiodic component of the signal and represents true alpha oscillations, as well as estimates of the Aperiodic Exponent, which reflects the slope of the aperiodic (1/f) component of the signal. See supplementary materials for a detailed description. Figure 3 provides a sample power spectrum illustrating the periodic and aperiodic contributions to the signal.

**Statistical Approach**

All analyses were performed in R using the lme4 package.26

**Testing for Behavioral Differences in Task Performance Across Groups**

To test the hypothesis that the lapse rate and threshold behavioral variables would differ across patient groups, we conducted 1-way ANOVA tests for each behavioral variable. Age, gender, and data collection site were controlled by including them as predictors.

**Examining Group Differences in EEG Parameters**

To examine potential group differences in our key EEG parameters, Total Alpha Power, Periodic Alpha Power, and Aperiodic Exponent values were averaged across all trials for each participant and electrode. Separate linear mixed effect regression models were fit with each of these variables as the dependent measure. Each model contained a fixed effect for the diagnostic group (HC, SZ, BP, or MDD) and the X, Y, and Z coordinates of each electrode, following the approach used in refs. 27,28. Specifically, the X-dimension captured the anterior-to-posterior axis, the Y-dimension captured the left-to-right axis, and the Z-dimension captured the superior-to-inferior axis corresponding to electrode location (see figure 2). Random intercepts for participants and electrodes were included in each model. To obtain parameter estimates for each group, the model was refit with each group serving as the reference group in turn.

**Testing Whether Pre-stimulus EEG Parameters Predict Behavioral Performance**

To test the extent to which pre-stimulus EEG parameters were predictive of behavioral performance on a trial-by-trial basis across diagnostic groups, the data were analyzed with a logistic linear mixed effects regression model in which single-trial Total Alpha Power, Periodic Alpha Power, or Aperiodic Exponent estimates in the pre-stimulus period were used to predict whether the response on each trial was correct or incorrect (incorrect responses coded as 1; correct responses as 0). This trial-level approach provided a strong test of whether each pretrial parameter was associated with behavioral accuracy. For catch trials—only 20% of total trials—there was not enough data to allow the models to converge. Thus, while behavioral data from catch trials allowed us to estimate attention lapse rate, all EEG analyses were conducted using regular trials only.

For each model, the dependent measure was binary (correct or incorrect), and the models included fixed effects for Group (HC, SZ, BP, or MDD) and the EEG measure (Total Alpha Power, Periodic Alpha Power, or Aperiodic Exponent) and X, Y, and Z scalp distribution variables of each electrode. To control for age, gender, and data collection site, these were also included in the models. Two-way interactions between each of the fixed effects (Group and Total Alpha Power, Periodic Alpha Power, or Aperiodic Exponent) and distributional variables (X, Y, Z) were also included, as well as 3-way interactions among Group, EEG parameters, and the distributional variables.

The highest order [3-way] interactions among Group, EEG parameters, and each of the distributional variables were included in the model to account for the possibility that the effect of each EEG parameter on behavior varied in scalp distribution across groups. However, it was not our goal with this analysis to characterize the scalp distribution differences in this effect between each of the 4 groups: the primary goal of this analysis was to test the extent to which each EEG parameter predicted behavior on a trial-by-trial basis in each group. Furthermore, detailed scalp distribution effects for each group were obtained when refitting the model with each group as the reference group. Thus, we have chosen not to interpret the highest order 3-way interactions in the main text. Full model output can be found in supplementary materials.

![Fig. 3. Sample power spectrum showing separable periodic alpha power and aperiodic contributions to the signal. Plotted is the original power spectrum for 1 randomly selected participant (black solid line). Periodic alpha power for this participant is shaded and represents the full model fit minus the aperiodic component. The aperiodic exponent is derived from the slope of the dashed line, which represents the estimated aperiodic (1/f) activity.](image)
The random effects structure for the models included random intercepts for participants and by-participant random slopes for the effect of Total Alpha Power, Periodic Alpha Power, or Aperiodic Exponent. To obtain parameter estimates for each group and group comparison, the model was refit with each group as the reference group. Additional models were fit with all Group effects removed, in order to examine whether the pre-stimulus EEG parameters predicted behavior across all participants. * values for the logistic mixed effects regression model parameters were obtained with Z tests.

Results

Behavioral Differences in Overall Task Performance Across Groups

Lapse Rate  As shown in figure 4, the lapse rate was highest in SZ, smaller in BP, even smaller in MDD, and smallest in HC. The lapse rate differed significantly across groups (P = .007). Pairwise comparisons showed the following pattern: the lapse rate was significantly higher in SZ compared with HC (P = .0018) and MDD (P = .0139). HC and MDD did not significantly differ from one another (P = .78), and BP was not significantly different from any other diagnostic group (BP vs HC: P = .22; BP vs SZ: P = .152; BP vs MDD: P = .39). There were no significant effects of age, gender, or data collection site.

Threshold  As shown in figure 5, there was a significant main effect of the diagnostic group (P = .025) for thresholds. Note that lower threshold values correspond to relatively close spacing between the bars, indicating greater precision on the perceptual discrimination task. Follow-up contrasts by group showed that the threshold was significantly lower in MDD compared with SZ (P = .0006). The other groups did not differ significantly from one another (HC vs SZ: P = .26; HC vs BP: P = .94; HC vs MDD: P = .11; SZ vs BP: P = .4; BP vs MDD: P = .17).

There were also significant effects of age (P = .037) and data collection site (P = .019); there was no significant effect of gender (P = .195). Overall, thresholds increased along with age. Pairwise comparisons by site showed that the site effect was driven by one site (University of Minnesota) with significantly lower thresholds in comparison to nearly all other sites. (University of Minnesota was unable to start data collection at the same time as the other sites, due to local regulations during the COVID-19 pandemic. Thus, this site did not collect data from patients with SZ [who demonstrated the largest threshold values of all groups], and had a smaller sample size than the other sites [14/244 participants], which may account for this effect.) In addition, thresholds were slightly lower for data collected at Washington University compared with Maryland Psychiatric Research Center. These follow-ups are reported in full in supplementary materials.

EEG Parameter Differences Across Groups

This analysis focused on differences in EEG parameters across groups, collapsing across all trials. Example data are plotted in figure 6 to facilitate visualizing an increase or decrease in Periodic Alpha Power or Aperiodic Exponent Value.

Total Alpha Power  As can be seen in the top row of figure 7, total alpha power estimates were largest frontally across all groups, with the effect of the X- and Z-dimension distributional variable being significant for each group (all Ps < .001), and no effect for each group in the Y-dimension variable (all Ps > .05). There were no statistically significant group differences in total alpha power collapsed across trials. There were also no statistically significant effects of age, gender, or data collection site (all Ps > .05).

Periodic Alpha Power  As shown in the middle row of figure 7, periodic alpha power had the typical posterior scalp distribution in all groups, with the effect of the X-dimension distributional variable being significant for each group (all Ps < .001), and no effect for each group in the Y- or Z-dimension variables (all Ps > .05). HC had significantly greater periodic alpha power on average
than SZ ($\beta = -3.396 \times 10^{-2}$, SE = $1.141 \times 10^{-2}$, $P = .00321$), BP ($\beta = -2.529 \times 10^{-2}$, SE = $1.237 \times 10^{-2}$, $P = .042$), and MDD ($\beta = -2.557 \times 10^{-2}$, SE = $1.186 \times 10^{-2}$, $P = .032$).

None of the other comparisons between groups showed a significant difference in average periodic alpha power (all $P$s > .05). There were also no statistically significant effects of age or gender (all $P$s > .05). One data collection site (Washington University) had slightly higher periodic alpha power values compared with the other sites ($\beta = 3.218 \times 10^{-2}$, SE = $1.191 \times 10^{-2}$, $P = .00742$).

**Aperiodic Exponent** As shown in the bottom row of figure 7, exponent values were maximal at central electrode sites in all groups, with significant $X$- and $Z$-dimension effect for all groups (all $P$s < .001). None of the other groups showed a significant difference from each other in average exponent values (all $P$s > .05). There were also no statistically significant effects of age, gender, or data collection site (all $P$s > .05).

**Predicting Behavior With EEG Parameters**

Figure 8 shows how each of the EEG parameters predicted the likelihood of making an error on a trial-by-trial basis. Statistical results are summarized below.

**Total Alpha Power** Across all participants, Total Alpha Power in the pre-stimulus period significantly predicted a greater likelihood of making an error ($\beta = -1.662 \times 10^0$, SE = $1.920 \times 10^{-2}$, $P = .0135$). When the diagnostic group was included in the models, Total Alpha Power significantly predicted behavior only for SZ, such that increased pre-stimulus alpha predicted a greater likelihood of making an error (significant Total Alpha Power

---

**Fig. 6.** (A) Example raw data traces for a trial on which relatively high periodic alpha power was present in the pre-stimulus period (solid line). (B) Example raw data traces for a trial on which periodic alpha power in the pre-stimulus period was relatively low (dotted line). (C) Example periodic alpha power estimates corresponding to plots A and B. (D) Example slope of the aperiodic ($1/f$) activity (solid line). The dashed line shows the slope for the example aperiodic exponent value minus 0.2, while the dotted line shows the slope for the example aperiodic exponent value plus 0.2, demonstrating that larger exponent values lead to steeper slopes, while smaller exponent values lead to flatter slopes.
Fig. 7. Average pre-stimulus values for each EEG parameter (collapsing across all trials): Total Alpha Power (top), Periodic Alpha Power (middle), and Aperiodic Exponent (bottom). Topographic maps show the average value in the pre-stimulus period at each electrode, across all participants. Boxplots show the average value for each group at a representative electrode (noted on the topographic plot with a black circle). The upper and lower edges (hinge) of each box reflect the first and third quartiles. The whiskers extend from the hinge to the largest and smallest values no more than 1.5 * of the interquartile range from the hinge. Data points beyond the end of the whiskers are plotted as separate black circles.
parameter for the model in which SZ was the reference group; $\beta = 0.0860244$, SE $= 0.0384876$, $P = .0254$). There were no statistically significant effects of age, gender, or data collection site (all $Ps > .05$).

**Periodic Alpha Power** Across all participants, the only hint of a relationship between Periodic Alpha Power and behavior was a marginally significant interaction with the $Y$-dimension distribution variable ($\beta = 0.0035839$, SE $= 0.001968$, $P = .0686$), in which increased Periodic Alpha Power at relatively left-lateralized electrode sites predicted a greater likelihood of making an error. When the diagnostic group was included in the models, the parameter estimate for the effect of Periodic Alpha Power as a predictor of error responses was significant only in BP, for whom increased Periodic Alpha Power interacted with the $Y$-dimension distribution variable to predict a greater likelihood of making an error (significant Periodic Alpha Power $\times Y$ parameter for the model in which BP was the reference group; $\beta = 1.408e-02$, SE $= 4.816e-03$, $P = .00347$), with this effect being maximal at left electrode sites. There were no statistically significant effects of age, gender, or data collection site (all $Ps > .05$).

**Aperiodic Exponent** Across all participants, Aperiodic Exponent significantly interacted with the $X$-dimension distribution variable, such that increased Aperiodic Exponent...
Exponent values significantly predicted a greater likelihood of making an error, maximally at relatively posterior electrode sites ($\beta = -0.0107757$, SE = 0.0021169, $P = 3.57e^{-07}$). When the diagnostic group was included in the models, the parameter estimate for the effect of the Aperiodic Exponent as a predictor of error responses was significant in HC and SZ, and showed a marginal effect in MDD, such that increased Aperiodic Exponent values were associated with an increased likelihood of making an error. Specifically, Aperiodic Exponent by $X$-distribution dimension interactions reflected the posterior distribution of this effect (significant Aperiodic Exponent $\times X$ parameters for models with the following reference groups: HC: $\beta = -0.0148454$, SE = 0.0034291, $P = 1.5e^{-05}$; SZ: $\beta = -0.0105354$, SE = 0.0043711, $P = .0159$), with MDD showing a marginal effect ($\beta = -0.009094$, SE = 0.004686, $P = .0523$). There were no statistically significant effects of age, gender, or data collection site (all $Ps > .05$).

### Correlations Between EEG Parameters and Clinical Variables

We calculated the correlation between averaged levels of each EEG parameter in the pre-stimulus period measured at the electrode site where that parameter was maximal (Total Alpha Power: Fz; Periodic Alpha Power: Pz; Aperiodic Exponent: Cz) and the following behavioral and clinical scores: lapse rate, threshold, positive symptoms (Brief Psychiatric Rating Scale, BPRS), negative symptoms (Clinical Assessment Interview for Negative Symptoms, CAINS), disorganized symptoms (BPRS), depression symptoms (Patient Health Questionnaire, PHQ-9), and WTAR score, for each diagnostic group separately. We corrected for multiple comparisons within each group and EEG/behavioral measure tested (7 tests per measure: the EEG/behavioral measure and the 6 behavioral/symptom variables listed above), setting the uncorrected significance level to $P < .007$ to achieve a Bonferroni-corrected alpha of 0.05. No significant correlations were found with the EEG measures. Lapse rate was significantly correlated with WTAR score in SZ, such that higher WTAR scores were associated with lower lapse rates ($r = -0.368$; $P = .005$). Correlation tables are included in supplementary materials.

### Discussion

This study asked whether perceptual precision and attention lapse rates were increased in 3 diagnostic groups (SZ, BP, and MDD) relative to HC, whether pre-stimulus alpha-band activity differed among groups, and whether pre-stimulus alpha-band activity predicted behavioral errors in individual trials. Below, we discuss the results pertaining to each question.

**Behavioral Differences in Task Performance Across Groups**

Our behavioral approach allowed us to independently estimate the precision of perceptual representations and attention lapse rate. We found a significantly elevated lapse rate in SZ relative to HC. BP exhibited a lapse rate intermediate between HC and SZ, whereas the lapse rate in MDD was nearly identical to that in HC. In contrast, we observed only a small and nonsignificant increase in the discrimination threshold in SZ relative to HC. This is in line with previous work that found that differences between HC and SZ in the contrast-contrast effect (a visual perception effect of gain control) did not reach significance when attention lapsing was accounted for. This suggests that attention lapsing contributes, at least in part, to the increased discrimination thresholds that have been observed in SZ in visual processing tasks in which lapsing was not accounted for, similar to reported effects of attention lapsing on other types of tasks where active attention is required (eg, 17,33–35). Instead, attention lapsing may be part of the so-called “general deficit” that affects performance across a range of cognitive tasks in SZ.

**EEG Parameter Differences Across Groups**

Increased pre-stimulus alpha is associated with lapses of attention in neurotypical individuals and we asked whether this neural measure of attentional engagement differed among diagnostic groups. We first assessed Total Alpha Power, which includes periodic activity in the alpha band (true oscillations) with aperiodic activity that contributes power across the frequency spectrum. There were no significant group differences in average total alpha power. However, when we separately estimated bona fide alpha-band oscillations (periodic activity) and aperiodic 1/f activity, we found that pre-stimulus periodic alpha power was significantly larger in HC than in SZ and MDD. While our focus was on activity in the alpha frequency band, a question for future work concerns whether periodic power in other frequency bands may also be reduced in SZ and MDD.

Our results are consistent with previous studies in which traditionally calculated alpha-band activity was greater in HC than in SZ but are not what would be expected if alpha caused changes in attentional control; in that case, we would expect that generally elevated pre-trial alpha would be observed in groups that demonstrate elevated lapse rates. Additionally, when we examined whether individuals with greater periodic alpha power tended to have higher lapse rates, we did not find a significant correlation. Thus, our results do not support the hypothesis that people with major mental health disorders like SZ, BP, and MDD have more lapses of attention.
because of a general decrease in pretrial periodic alpha power. Interestingly, average aperiodic exponent values were similar across groups, although participants with MDD had greater aperiodic exponent values than participants with BP.

**Predicting Behavior With EEG Parameters**

We also asked whether pre-stimulus alpha activity predicted trial-by-trial behavioral performance in a visual perception task. When all groups were combined, greater pre-stimulus total alpha power on a given trial significantly predicted a greater likelihood of making an error. Breaking this effect down by diagnostic group showed that pre-stimulus total alpha power significantly predicted trial-by-trial behavior in the SZ group, but not in the other groups. Interestingly, estimates of pre-stimulus periodic power did not significantly predict behavior in the all-group analysis, and only predicted behavior in BP, while estimates of pre-stimulus aperiodic activity significantly predicted behavior when all groups were combined, and in the HC and SZ groups individually.

The results of the total alpha power analysis were broadly consistent with our hypothesis, in that pre-stimulus alpha power predicted response accuracy on the visual perceptual discrimination task at the trial level. We interpret this effect as evidence that pre-stimulus total alpha power served as a proxy measure for attentional readiness going into a given trial. Our goal in including measures of pre-stimulus periodic alpha power and aperiodic activity was to provide additional information about aspects of the total alpha signal that might be driving its link to behavior. If the link between alpha and behavior was driven solely by “true” alpha oscillations (periodic activity), we would not have expected that aperiodic contributions to the signal would have emerged as a significant predictor of behavior.

The aperiodic component of the EEG signal reflects nonrhythmic contributions, also known as the 1/f slope. Aperiodic EEG activity and its origins are less well studied than periodic activity (neural oscillations), although the exponent of the aperiodic fit (flatness or steepness of the slope) has been linked to age-related changes in cognitive functioning. Recent computational work has suggested that aperiodic components of the signal may arise from synaptic excitatory-inhibitory balance, although this is a developing research area. Here, relatively large aperiodic exponent values (indicative of relatively steep 1/f slopes) in the pre-stimulus period were associated with a greater likelihood of making an error, particularly at posterior electrode sites and most strongly for HC and SZ. However, as noted above, group differences in average estimates of the aperiodic exponent were minimal (BP showed smaller average aperiodic exponent values than MDD). This demonstrates that it is trial-to-trial variability within an individual ie predictive of behavior, rather than general changes in aperiodic activity. To the best of our knowledge, this is the first study in which this has been observed and suggests that further characterization of aperiodic contributions to the EEG signal across diagnostic groups is an important target for future research.

We note that the present analyses focused on how EEG parameters in the pre-stimulus period predicted performance on the regular trials (for which the threshold was adjusted so that participants responded with 80.35% accuracy). Catch trials provided a useful estimate of attention lapse rate (because inaccurate responding on these relatively easy trials would be best explained by a lapse in attention), but the relatively small number of catch trials and an even smaller number of catch trial errors limited our ability to assess the relation between pre-stimulus EEG parameters and performance on these trials. Although attention lapses contribute to inaccurate responding on regular trials as well, we presume that performance on regular trials represents a mixture of attentional engagement and the precision of perceptual representations of the stimuli. We conceptualize pre-stimulus alpha activity as a continuous metric of attentional readiness, rather than as an all-or-none indicator of an attention lapse (as described in refs. 47,48). Our results indicate that pre-stimulus alpha activity just prior to a visual stimulus (when the participant does not yet know the trial type) predicts the likelihood of responding correctly to that stimulus and that this effect may be driven in large part by aperiodic contributions to the signal rather than by periodic alpha oscillations.

**Summary and Conclusions**

In summary, the behavioral results of this study demonstrated elevated attention lapse rates in SZ, who had greater lapse rates compared with HC, BP, and MDD. The EEG results showed differences across diagnostic groups in averaged levels of pre-stimulus periodic alpha power, such that HC had greater average periodic alpha power compared with SZ and MDD. However, the trial-by-trial analysis showed that it was not the participant-level average that significantly predicted the likelihood of making an error, but within-participant fluctuations in pre-stimulus EEG indices, in all groups.

Specifically, the EEG results showed that separating traditionally calculated pre-stimulus alpha power into periodic and aperiodic components allowed greater specificity in identifying possible biomarkers linked to poor behavioral performance and attention lapsing. Specifically, periodic alpha power during the pre-stimulus period emerged as a weaker predictor of behavior than might have been expected given previous work, whereas aperiodic contributions to the signal emerged as a relatively strong predictor of behavioral performance across diagnostic groups. In combination with this pattern, the finding that pre-stimulus periodic alpha oscillations were largest in HC (who had the lowest lapse rate) and smallest in SZ
(who had the highest lapse rate) provides strong evidence against a simple account in which elevated lapse rates in major mental health disorders like SZ, BP, and MDD can be mapped directly on to elevated alpha activity.

These results suggest several questions for future research. First, we plan to follow up this work by examining attentional control in other domains, such as in working memory or language tasks. Second, further exploration of periodic alpha and aperiodic contributions to EEG measures of attentional control is needed, particularly given the somewhat surprising finding that aperiodic activity had the strongest connection to behavior in this study. Finally, additional research on the causes of aperiodic activity is needed to better understand the origins of this signal and its functional significance.

Supplementary Material

Supplementary material is available at https://academic.oup.com/schizophreniabulletin/.

Acknowledgments

The authors have declared that there are no conflicts of interest in relation to the subject of this study.

Funding

The current study was supported by National Institute of Mental Health grant R01 MH084821.

References


