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# Analysis of aperiodic activity in obsessive-compulsive disorder and major depression

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**Obsessive-compulsive disorder (OCD)** is believed to be linked to alterations in the cortical excitation–inhibition (E/I) ratio. Recent studies highlight aperiodic EEG activity as a neurophysiological marker for E/I ratio. We investigated the role of aperiodic activity during resting-state EEG in a children and adolescents sample of OCD patients ( $n=38$ ), compared to healthy controls (HC) ( $n=94$ ) and patients with major depression (MDD) ( $n=88$ ). To quantify aperiodic activity in resting-state EEG, we employed the FOOOF (Fitting Oscillations & One Over F) algorithm, which extracts the aperiodic exponent from EEG data. Compared to HC, both OCD and MDD patients exhibited increased E/I ratio as shown by decreased aperiodic exponents. Controlling for age by cohort-matching and for medication and stimulant intake on the test day rendered the statistical outcome non-significant but left the qualitative pattern of the outcomes intact. We conclude that both MDD and OCD seem to come with increased E/I ratios, as indicated by the aperiodic exponent, even though this effect may be moderated by age in OCD. This renders the aperiodic exponent a promising neural marker for psychopathology. However, the absence of a significant difference between OCD and MDD indicates that this marker may not be sensitive enough to differentiate between particular psychopathologies, which may be due to the fact that they often co-exist. The potential of using aperiodic exponents to examine the effectiveness of treatments in developmental neuropsychiatry should be explored. This approach should be carefully evaluated in future studies.

**Keywords** Aperiodic EEG activity, E/I ratio, MDD, OCD

**Obsessive-compulsive disorder (OCD)** is a complex condition in the realm of neuropsychiatry, exhibiting various peculiarities in clinical symptoms, cognitive functions, biochemical activity, and brain connectivity<sup>1,2</sup>. OCD ranks as the fourth most frequent neuropsychiatric disorder globally, with a lifetime prevalence rate between 2% and 3%<sup>3–5</sup>. The disorder is chiefly marked by the presence of recurring, unwanted, and distressing thoughts (obsessions) that typically provoke repetitive actions (compulsions), undertaken to mitigate the adverse emotions triggered by obsessions<sup>6</sup>.

OCD has been associated with a disrupted balance in cortical excitation and inhibition—specifically, an altered E/I ratio. Neurophysiological studies using transcranial magnetic stimulation (TMS) provide evidence that this imbalance manifests as both diminished inhibitory signaling (e.g., shorter cortical silent period) and altered excitatory activity (e.g., reduced or elevated intracortical facilitation) in individuals with OCD<sup>7,8</sup>. Such dysregulation may interfere with neural gain control mechanisms—analogous to maladaptive tuning in response to environmental inputs—potentially contributing to the intrusive and repetitive cognitive and behavioral symptoms characteristic of the disorder<sup>8</sup>.

Further support of altered E/I ratio has been explored indirectly through the lens of sensorimotor gating. This refers to the process of filtering relevant sensory signals from irrelevant or redundant motor activity. A key measure of sensorimotor gating is the pre-pulse inhibition (PPI) of the startle reflex, which occurs when a weak stimulus (the pre-pulse) is presented prior to a strong startling stimulus (the pulse) within an interval of 30 to 500 milliseconds<sup>9</sup>. When the neural pathways involved in the startle response are disrupted, sensorimotor gating is impaired, leading to an exaggerated startle response<sup>10</sup>. Research on PPI in individuals with OCD has yielded mixed results<sup>11–13</sup>, likely due to varying research methods employed<sup>10</sup>. Nevertheless, evidence indicates that unmedicated OCD patients, particularly those with a history of tic disorders, tend to exhibit diminished PPI<sup>14</sup>.

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Interestingly for our purposes, recent electrophysiological research has shown that aperiodic neural activity—defined as non-oscillatory EEG signals—may be a valuable avenue for investigating E/I ratio more directly<sup>15</sup>, although it represents only one of several approaches to assessing this balance<sup>16</sup>. Recent findings have demonstrated that aperiodic activity is systematically connected to levels of arousal<sup>17</sup>, performance on tasks<sup>18,19</sup>, and various cognitive processes<sup>20–25</sup>. Notably, the power spectrum reflects a characteristic distribution where spectral power decreases as frequency increases<sup>26–28</sup>. The dynamics of aperiodic activity can be reliably described by the aperiodic exponent, which denotes the negative 1/f slope resulting from the logarithmic transformation of the power spectrum, indicating the rate or steepness of power reduction across different frequencies<sup>26</sup>.

Crucially for our goal, the aperiodic exponent can be considered an indicator of the “E/I ratio.” This ratio describes the balance between excitatory and inhibitory neural processes within the brain—where a predominance of excitatory activity suggests increased variability and dispersion of neural responses, while a predominance of inhibitory activity indicates reduced variability and greater specificity. A higher aperiodic exponent, which signals a “steeper” power spectrum, suggests a greater influence of inhibition relative to excitation, whereas a lower exponent implies the opposite<sup>29,30</sup>. Note that the relation between aperiodic exponent and E/I ratio is inverse: a low aperiodic exponent suggests a high E/I ratio, while a high aperiodic exponent reflects a low E/I ratio<sup>31</sup>.

In the current study, we were interested in examining aperiodic activity in a sample of young OCD patients. Studying young participants has the advantage of revealing how neural brain patterns mature and identifying sensitive periods that may be crucial for early intervention in neurodevelopmental disorders. Such insights can lead to early diagnosis, allowing for timely and tailored therapeutic strategies appropriate for children’s unique neurophysiological profiles<sup>32–38</sup>. Considering aperiodic activity in OCD might not only inform diagnostic and treatment practices but also contribute to advancing theoretical models of brain function and mental health, enhancing our ability to address neuropsychiatric challenges across the lifespan.

To date, only one research study has examined the 1/f slope in patients with OCD in comparison to healthy controls (HC) in an adult sample<sup>39</sup>. The study revealed no significant differences between the two groups<sup>39</sup> but, as the authors noted, the sample size may have been much too small to detect such differences<sup>39</sup>. Additionally, the authors’ failure to specify the fit range used to calculate the 1/f slope further complicates the interpretation of these null results<sup>39</sup>.

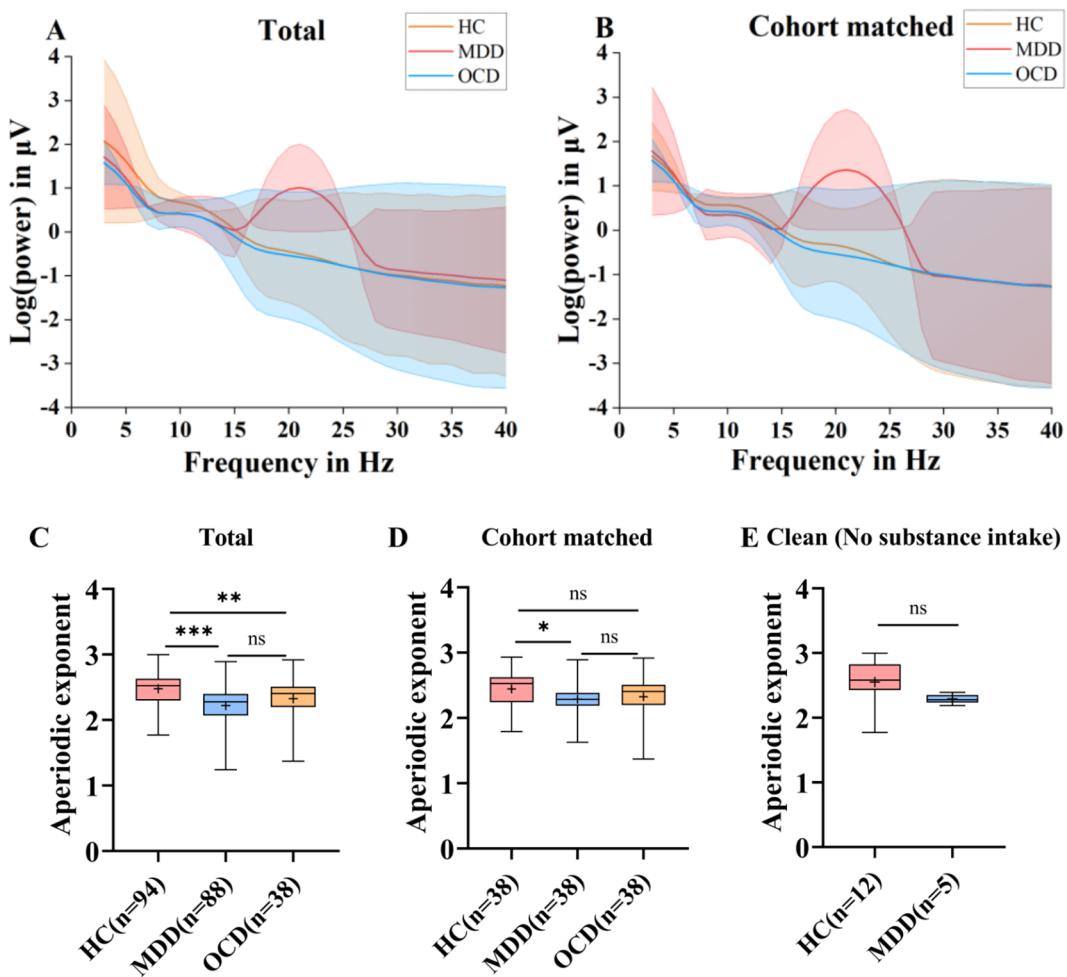
To address these limitations, we investigated the role of aperiodic activity during resting-state EEG. We utilized the spectral parameterization method known as Fitting Oscillations and One Over f (FOOOF)<sup>26</sup> to extract aperiodic exponents from EEG data, as we did in previous studies<sup>20,23,24,40</sup>. We compared a children and adolescents sample of OCD patients ( $n = 38$ ) to HC ( $n = 94$ ) and patients diagnosed with major depression (MDD) ( $n = 88$ ). Including another psychiatric group was crucial for determining to which degree possible abnormal aperiodic activity identified in OCD is specific to this disorder or whether it can also be observed in other psychiatric conditions. Analyzing the differences and similarities in aperiodic activity across various psychiatric groups could help identify specific markers of OCD, potentially leading to improved diagnostic criteria and targeted treatment approaches.

We considered three possible outcomes with regard to OCD. First, the null hypothesis: it might be that OCD is mainly expressed in periodic EEG activity but may not affect aperiodic activity. Second, it is possible that OCD, and perhaps other psychiatric disorders (like MDD), are characterized by a significant increase of aperiodic activity, which would fit considerations that at least some psychiatric disorders come with a shift of the E/I ratio from a more inhibitory to a more excitatory mode<sup>29</sup>. Indeed, previous studies investigating MDD have identified alterations in the E/I ratio, with some reporting an increased aperiodic exponent<sup>41–44</sup>, while others have observed a decreased aperiodic exponent<sup>45,46</sup>. Third, psychiatric disorders might generally come with deviant aperiodic activity patterns, but some disorders might be characterized by a particularly low E/I ratio while others are characterized by a particularly high E/I ratio<sup>47</sup>. Accordingly, this might result in OCD (and, perhaps, MDD) coming with a lower E/I ratio than HC. Hence, given the inverse relationship between E/I ratio and the aperiodic exponent, our alternative hypotheses would correspond to these predictions regarding aperiodic exponents: (1) OCD = HC; (2) OCD < HC; and (3) OCD > HC.

## Results

The power spectral density (PSD) results are shown in Fig. 1A at the frequency from 3 Hz to 40 Hz for the three different groups. Note that the contribution of the  $\beta$  frequency band (16–26 Hz) was more pronounced for MDD (see the peak in Fig. 1A), but this did not seem to have a strong effect on the aperiodic result.

Our main hypothesis was tested by means of a planned one-way ANOVA of the aperiodic exponents as a function of group/diagnosis (see Fig. 1C). One-way ANOVAs and further independent-samples t-tests were conducted using SPSS (v26.0) for classical statistical analysis (frequentist), whereas JASP (v0.17.0) was employed for Bayesian analysis to compute the Bayes factor ( $BF_{10}$ ). The group effect was highly significant,  $F_{(2, 21)} = 16.84$ ,  $p < 0.001$ ,  $BF_{10} > 1000$  (i.e., strong Bayesian evidence for  $H_1$ ). Post-hoc t-tests showed that compared to HC ( $2.48 \pm 0.03$ ), both OCD ( $2.32 \pm 0.06$ ),  $t_{(130)} = 2.76$ , Cohen’s  $d = 0.49$ ,  $p = 0.007$ ,  $BF_{10} = 5.88$  (i.e., moderate Bayesian evidence for  $H_1$ ), and MDD patients ( $2.22 \pm 0.03$ ),  $t_{(180)} = 6.03$ , Cohen’s  $d = 0.89$ ,  $p < 0.001$ ,  $BF_{10} > 1000$  (i.e., strong Bayesian evidence for  $H_1$ ), exhibited lower exponents (increased aperiodic activity) than HC, whereas MDD and OCD groups did not differ ( $t_{(124)} = 1.63$ ,  $p = 0.11$ ). We further examined the main regions exhibiting significant differences among the groups. The results revealed widespread group differences across most brain areas, with the exception of the following electrodes: 3, 5, 8, 9, 11, 17, 18, 21, 22, 26, 29, 32, 34, 35, 36, 39, 40, 41, 42, 44, 55, 61, 114, 121, and 122. These correspond to the following positions in the International 10–10 system: AF4, FP2, Fz, FP1, AF7, FC3, F9, FT7, C3, T7, C5, CP3, T9, CPz, T10, FT10, and F8.



**Fig. 1.** (Panel A–B) Power spectral density (PSD) averaged across all electrodes and participants for the overall (A) and cohort-matched (B) analyses. Colored shaded areas denote the standard error of the mean (SEM). (Panel C–E) Aperiodic exponent results for the overall (C), cohort-matched (D), and substance-control (E) analyses. “+” denotes the mean;  $p < 0.05$ ,  $p < 0.01$ ,  $*p < 0.001$ .

Two additional explorative analyses were carried out to test the impact of two possible confounders. Both analyses used a drastically reduced sample size, which rendered the crucial ANOVA no longer significant. Nevertheless, we present the qualitative outcomes because they indicate that the basic pattern did not change.

First, we tested whether age, which was not equal in the three groups, might have moderated the group effect. To exclude this possibility, we cohort-matched participants by matching each participant of the OCD group with a participant of the other two groups in terms of age and gender (see Fig. 1B and D for PSD and exponents, respectively). This resulted in the following new groups distribution: HC ( $n = 38$ , 19 males, mean age =  $12.08 \pm 2.65$  years), MDD ( $n = 38$ , 19 males, mean age =  $12.87 \pm 1.91$  years), and OCD ( $n = 38$ , 19 males, mean age =  $11.89 \pm 3.76$  years). After this matching, only MDD patients ( $2.29 \pm 0.04$ ) showed a significant difference with HC ( $2.44 \pm 0.04$ ),  $t_{(74)} = 2.62$ , Cohen's  $d = 0.60$ ,  $p = 0.01$ ,  $BF_{10} = 4.28$  (i.e., moderate Bayesian evidence for  $H_1$ ). Similarly, a comparison between HC and MDD groups revealed significant differences across brain regions on both the left and right sides except for the outer electrodes. The non-significant electrodes were 1, 2, 3, 5, 14, 17, 24, 26, 27, 28, 29, 30, 32, 33, 34, 35, 37, 38, 39, 41, 44, 47, 93, 98, 101, 102, 106, 108, 109, 110, 112, 114, 115, 121, 122, and 123 (International 10–10 equivalents: F10, AF8, AF4, FPz, F3, AF7, F5, FC5, FC3, C1, F9, F7, FT7, CP1, FT9, C5, T9, CP5, CP4, CP6, TP8, T8, FC2, T10, FT10, F8, and F6). The difference between HC and OCD no longer reached significance ( $2.32 \pm 0.06$ ),  $(t_{(74)} = 1.64$ ,  $p = 0.11$ ). More important, however, was that the qualitative pattern of the three groups was the same as in the original analysis (see 1D).

Second, we considered that participants who reported medication or stimulant use (e.g., fluoxetine, caffeine) on the test day might have affected the outcome. However, excluding all participants who reported such use resulted in the same qualitative pattern (see Fig. 1E).

## Discussion

This study aimed to compare predictions from different possible hypotheses regarding altered E/I ratio in individuals with OCD<sup>47</sup>. The aperiodic exponent is considered an indicator of the brain's E/I ratio because it

reflects the non-oscillatory, irregular component of brain activity<sup>26</sup>. Unlike periodic power, which captures structured, functional oscillations, the aperiodic exponent provides insight into the overall balance between excitatory and inhibitory processes<sup>26</sup>. A flatter slope (lower exponent) suggests increased excitation or reduced inhibition, while a steeper slope (higher exponent) indicates stronger inhibitory control and more balanced neural activity<sup>26</sup>. This makes the aperiodic exponent a valuable measure for assessing the E/I ratio across different cognitive states and clinical conditions.

In a children and adolescents sample, we assessed the E/I ratio in resting-state EEG by employing the FOOOF algorithm<sup>26</sup>, which extracts aperiodic exponents from the EEG data. Our findings reveal that both OCD and MDD patients exhibited increased E/I ratios, indicated by reduced aperiodic exponents compared to HC. Even though the statistical outcomes of our control analyses are unreliable because of drastically reduced power, our attempts to control for age and ingestion reproduced the general qualitative pattern. We therefore conclude that both OCD and MDD seem to come with increased E/I ratio, which is consistent with the second of the three scenarios we developed in the introduction.

Our results of altered E/I ratio in resting-state EEG in MDD children are in line with several studies in MDD adults showing decreased aperiodic exponents<sup>41–44</sup>. With respect to OCD, our findings provide at least preliminary evidence for the possibility that OCD may also come with increased E/I ratios—which stands in contrast to the null-effect that has been reported for adults<sup>39</sup>. However, given that the statistics of our age-matched groups are difficult to interpret, we cannot fully rule out a possible contribution of age. Hence, future studies should either study the relationship between OCD and E/I ratio as a function of age or assure a better-powered comparison of age-matched individuals. In any case, our results align with hypotheses that OCD involves glutamatergic dysregulation and cortical hyperexcitability<sup>48,49</sup>. That is, altered aperiodic activity may contribute to impairments in signal clarity within neural decision-making circuits, potentially linked to intrusive thoughts and compulsions in OCD. While a recent study focused on the predictability of theta/alpha power during deep brain stimulation, its results echo what the aperiodic exponent captures: E/I ratio<sup>50</sup>. Non-responders showed high predictability—suggestive of cortical rigidity—while responders showed reduced predictability, potentially reflecting a more balanced E/I state and greater neural adaptability. Together, both measures, aperiodic exponent and predictability of theta/alpha power, highlight how neural signal dynamics can indicate clinical state and treatment response.

It is important to note that the aperiodic exponent, which reflects the slope of the 1/f pattern in neural power spectra, is sensitive to changes in neural excitability and network dynamics<sup>26–28</sup>. During rest, it captures intrinsic, spontaneous activity, while cognitive tasks engage specific neural processes that may alter the excitatory-inhibitory balance differently than the resting state. This occurs because cognitive tasks often involve shifts in arousal, attention, and task-specific neural recruitment, which can independently modulate the aperiodic exponent, inducing changes in cortical excitation or inhibition that override baseline resting-state patterns. That is, group effects observed for resting-state data may look different for aperiodic exponents obtained during task performance. Therefore, future studies should compare the aperiodic exponent in groups with OCD and HC during cognitive tasks as well<sup>51</sup>. In this respect, it would be particularly interesting to investigate metacontrol, the ability to maintain an optimal balance between cognitive control styles that are either more persistent or more flexible<sup>52–54</sup>. Rather than viewing these styles as fixed traits, the framework proposes that individuals dynamically adjust their metacontrol states depending on situational demands. When persistence dominates, individuals maintain focus on a single goal, resist distractions, and rely on top-down processing—ideal for stable, goal-driven tasks. In contrast, flexibility supports the exploration of alternative strategies, the integration of new information, and responsiveness to change, drawing more on bottom-up processing. This dynamic adjustment is not only behaviorally relevant but is increasingly supported by neurophysiological evidence, as recent studies have demonstrated a strong and reliable link between metacontrol states and aperiodic EEG patterns<sup>20,23,24,40</sup>.

A potential limitation of this study is that baseline correction was applied to the resting-state EEG segments (0–200 ms). Although baseline correction is generally unnecessary for PSD analyses, its use may have introduced minor biases in the low-frequency components. The impact of this effect is expected to be small, but it should be considered when interpreting the results.

If replicated in future studies, our findings suggest that aperiodic exponents might serve as valuable neural markers in developmental neuropsychiatry, offering insights into the underlying neurophysiological mechanisms associated with various co-existing psychiatric disorders. However, the lack of a significant difference in aperiodic exponents between individuals with OCD and MDD indicates that this marker might not be sufficiently sensitive to distinguish between these two conditions, likely because of their known co-existence in many patients<sup>55,56</sup>.

To build upon this understanding, we propose that future studies should examine whether the effectiveness of OCD (and other co-occurring disorders) treatments could be assessed by quantifying E/I ratio through the measurement of changes in the aperiodic exponent over the course of therapy. By monitoring how aperiodic exponents respond to different treatment modalities—such as cognitive-behavioral therapy, pharmacotherapy, or combined strategies—we may be able to identify specific trends that correlate with symptom improvement. Furthermore, tracking these changes could aid in personalizing treatment plans, allowing clinicians to tailor interventions based on individual responses. If this method turns out to be successful, it might also be useful to identify developmental neuropsychiatric patients who respond to typical treatments, potentially leading to alternative strategies that better address their unique neurophysiological profiles.

## Methods

### Participants

The database on which this study is based was approved by the Chesapeake Institutional Review Board (<https://www.chesapeakeirb.com/>)<sup>57</sup>. The data were sourced from the Healthy Brain Network (HBN), a publicly

available dataset approved by local institutional ethics committees in the U.S. The aggregated data analyzed in this article are available via the Open Science Framework: <https://osf.io/4p632>. All participants (or their legal guardians in the case of minors) gave written informed consent. Study procedures followed institutional and ethical guidelines<sup>57</sup>. The use of this dataset was made available to Amir Jahanian Najafabadi by the Child Mind Institute. The dataset consisted of resting-state EEG data of three groups: HC ( $n = 94$ , 47 males, mean age =  $9.23 \pm 3.13$  years), patients with MDD ( $n = 88$ , 34 males, mean age =  $15.37 \pm 2.79$  years), and patients with OCD ( $n = 38$ , 18 males, mean age =  $11.80 \pm 3.77$  years). MDD and OCD patients were diagnosed clinically and included regardless of their medication status, whereas HC participants had no history of neurological or psychiatric disorders. We analyzed demographic characteristics, including age and gender (by use of independent sample t-test and chi-square test separately), to assess potential intergroup differences. The analysis indicated significant differences in age across the three groups (one-way ANOVA:  $F_{(2, 217)} = 86.97, p < 0.001$ ; post-hoc t-tests:  $t_{(180)} = 13.86, p < 0.001$ ;  $t_{(130)} = 4.20, p < 0.001$ ;  $t_{(124)} = 5.64, p < 0.001$ ). In contrast, gender distribution did not show significant differences among groups ( $\chi^2_{(2)} = 3.33, p = 0.19$ ).

### EEG recording and processing

The resting-state EEG data were recorded using a 128-channel high-density HydroCel Geodesic system (Electrical Geodesics Inc.) at a sampling rate of 500 Hz and a band pass of 0.1 to 100 Hz. For the pre-processing, all outer channels were excluded, retaining 109 channels for analysis. The data was pre-processed using the Python-based MNE package. First, the data was downsampled to 256 Hz and applied a 0.5–50 Hz band-pass filter. A notch filter at 60 Hz eliminated power line noise (following the US standard). The PyPREP library's pipeline was used to identify and remove the bad channels, followed by the interpolation of detected bad channels and a channel average-based re-referencing was conducted to improve data quality. Next, Independent Component Analysis (ICA) was used to remove common artifacts such as eye blinks and eye movements from the EEG data, where the number of components was chosen based on visual inspection and IC stability over multiple runs. To further improve data quality, we implemented an automated artifact detection system using a derivative-based thresholding approach. The method calculates the first temporal derivative of the EEG signal (i.e., the sample-to-sample amplitude difference) and flags segments where this difference exceeds 150  $\mu$ V/ms. Following detection, contaminated segments were corrected via linear interpolation using neighboring clean data points, preserving data continuity by replacing artifactual segments with interpolated estimates. Finally, the pre-processed resting-state EEG data were divided into equally sized segments of 1000 ms duration, a baseline correction applied from 0 ms to 200 ms.

### Parameterization of spectral data

To calculate the power spectral density (PSD) for each frequency, we used Matlab's 'pwelch' function (version R2018a), based on previous research<sup>58</sup>. The PSDs were estimated separately for each participant and electrode. To parameterize the power spectra by decomposing and extracting both aperiodic and periodic components of EEG activity<sup>26,59</sup>, we utilized the Python-based FOOOF toolbox (version 1.0.0; <https://github.com/foof-tools/fooo>). The FOOOF algorithm interprets the power spectrum by decomposing it into two components: aperiodic activity [ $L(f)$ ] and periodic oscillatory activity [ $G_n(f)$ ]. The power spectrum model can be expressed as a linear combination of these two components, where  $f$  means frequency:

$$PSD(f) = L(f) + \sum n G_n(f)$$

The PSD is obtained by integrating the aperiodic component,  $L(f)$ , with a sum of  $n$  Gaussian functions.  $L(f)$  is fitted as a function that spans the entire range of the analyzed spectrum. The precise mathematical expression for  $L(f)$  is as follows:

$$L(f) = b - x \cdot \log(f).$$

The parameter  $b$  represents the aperiodic offset, indicating the overall shift in broadband power, while  $x$  represents the aperiodic exponent, which signifies the slope of the power spectrum line when plotted on a log-log scale. The periodic/oscillatory components indicate the specific frequency regions where the power exceeds the aperiodic component. Each periodic peak is characterized by a Gaussian function and defined by three parameters. The Gaussian fitting can be described using the following model:

$$G_n(f) = \alpha_n \exp \left[ -\frac{(f - \mu_n)^2}{2\sigma_n^2} \right]$$

Each spectrum was characterized by using the parameters of amplitude ( $\alpha_n$ ), center frequency ( $\mu_n$ ), and bandwidth ( $\sigma_n$ ). Then, the spectrum was fit across the frequency range [3, 40], using the settings of peak\_width\_limits = [1, 8], max\_n\_peaks = 8, min\_peak\_height = 0.05, and aperiodic\_mode = 'fixed'. This provided an aperiodic exponent value for each participant and electrode. The spectral fits' average  $R^2$  value for HBN, MDD and OCD group were 0.99, 0.97 and 0.98, respectively, demonstrating high-quality fits.

As the aperiodic exponent, rather than the offset, was identified as sensitive to index metacontrol states<sup>25</sup>, our analysis focused solely on the exponent. Since there were no a priori hypotheses regarding the spatial distribution of aperiodic neural activity across the scalp, we employed the "global" exponent by averaging exponent values across all 109 electrodes for each participant, following the approach of previous study<sup>60</sup>, to explore the differences between groups.

## Statistical analysis

Independent-samples t-tests and one-way ANOVAs were conducted using SPSS (v26.0) and JASP (v0.17.0). SPSS was used for classical statistical analysis, while Bayesian analysis was performed using JASP to compute the Bayes factor ( $BF_{10}$ ), which represents the relative evidence for the null hypothesis ( $H_0$ ) versus the alternative hypothesis ( $H_1$ ).  $BF_{10}=1$  indicates no evidence for  $H_0$  or  $H_1$ ,  $BF_{10}>3$  indicates moderate evidence for  $H_1$ .  $BF_{10}>10$  indicates strong evidence for  $H_1$ . P-values of the multiple t-tests were corrected using the Bonferroni method. All descriptive statistics give the mean and standard error of the mean (SEM).

## Data availability

All data can be obtained from Amir Jahanian-Najafabadi ([amir.jahanian@uni-bielefeld.de]) upon reasonable request. This research was prepared using limited access to the datasets obtained from the USA BioBank provided by Child Mind Institute (CMI), Healthy Brain Network. This manuscript reflects the views of the authors and does not necessarily reflect the opinions or views of the CMI. EEG Datasets and phenotypes were credited by CMI to Dr. Amir Jahanian Najafabadi under the official data usage agreement signed by Bielefeld University.

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## Author contributions

Study design: B.H., L.C., A.J.N; data collection analysis: H.Z. A.J.N and K.B.; writing of the manuscript: H.Z., L.C., B.H. All authors reviewed the manuscript.

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## Declarations

### Competing interests

The authors declare no competing interests.

### Additional information

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