Behavioral and cognitive correlates of the aperiodic (1/f-like) exponent of the EEG power spectrum in adolescents with and without ADHD

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A B S T R A C T

Efficient information processing facilitates cognition and may be disrupted in a number of neurodevelopmental conditions. And yet, the role of inefficient information processing and its neural underpinnings remains poorly understood. In the current study, we examined the cognitive and behavioral correlates of the aperiodic exponent of the electroencephalogram (EEG) power spectrum, a putative marker of disrupted, inefficient neural communication, in a sample of adolescents with and without ADHD ($n = 184$ $n_{ADHD} = 87$; $M_{age} = 13.95$ years, $SD = 1.36$). Exponents were calculated via FOOOF (Donoghue et al., 2020a) from EEG data recorded during an 8-minute baseline episode. Reaction time speed and variability, as well as drift diffusion parameters (including the drift rate parameter, a cognitive parameter directly related to inefficient information processing) were calculated. Adolescents with ADHD had smaller aperiodic exponents (a "flattened" EEG power spectrum) relative to their typically-developing peers. After controlling for ADHD, aperiodic exponents were related to reaction time variability and the drift rate parameter, but not in the expected direction. Our findings lend support for the aperiodic exponent as a neural correlate of disrupted information processing, and provide insight into the role of cortical excitation/inhibition imbalance in the pathophysiology of ADHD.

1. Introduction

Attention deficit/hyperactivity disorder (ADHD) is a neurodevelopmental disorder that is associated with cognitive control impairments (Huang-Pollock et al., 2012; Kofler et al., 2013; Willcutt et al., 2005). Although there is heterogeneity in the ADHD population (Fair et al., 2012; Nigg, 2005), at the group level, consistent impairments have been found in attention, working memory, and response inhibition (Huang-Pollock et al., 2012; Kofler et al., 2013; Martinussen et al., 2005; Nikolas and Nigg, 2013; Willcutt et al., 2005). Mounting evidence also points to intraindividual variability—indexed by greater reaction time variability (RTV)—as a core cognitive dysfunction for some individuals with ADHD. RTV is of particular interest in ADHD because it yields one of the largest group differences of any cognitive measure (Kofler et al., 2013) and may mediate other cognitive impairment in the disorder (Karalunas and Huang-Pollock, 2013).

Although increased RTV in ADHD is well-documented, there is not yet agreement on its neural correlates or mechanisms (e.g., Karalunas et al., 2014; Killeen et al., 2013; Kofler et al., 2013; Sonuga-Barke, and Castellanos, 2007). One problem in narrowing these mechanisms is that commonly used measures of reaction time—mean (RT) and standard deviation (SDRT)—are influenced by multiple processes (e.g., speed-accuracy trade-offs). Each of these has been implicated in cognitive theories of ADHD. Perhaps for this reason, RTV correlates broadly with deficits in networks associated with attentional control (e.g., Bellgrove et al., 2004; Simmonds et al., 2007) but also with regions related to response selection and motor output (Kanai and Rees, 2011).

Recently, progress in clarifying cognitive mechanisms underlying RTV has been made using computational approaches to separately model the multiple processes affecting the decision process. Within ADHD, the emphasis has been on well-validated variants of sequential sampling models (SSMs) (Ratcliff and Rouder, 1998). SSMs assume that information about a stimulus is accumulated via an information accumulation process (*drift rate*) until a decision criterion (*boundary separation*) is met, at which point a response is initiated. Processes that are not related to the response decision (e.g., motor preparation) are also

modeled (*non-decision time*). The evidence accumulation process is described as "noisy" because random neural activity unrelated to the decision process is thought to influence the efficiency with which a person is able to accumulate decision-relevant information.

SSMs have helped clarify the nature of cognitive impairments in ADHD (Karalunas and Huang-Pollock, 2013; Karalunas et al., 2012a; Weigard et al., 2016). Children with ADHD show a reduced drift rate relative to their peers without ADHD, with inconsistent differences in other parameters (Karalunas et al., 2014; Weigard et al., 2018). This inefficient information processing is thought to reflect weaker signal-to-noise in the underlying neural circuits (Ratcliff, 2006; Ratcliff and Rouder, 1998), but has not been directly demonstrated in ADHD.

Disruptions to the excitatory (E) to inhibitory (I) balance may contribute to impairment in specific psychiatric populations (Voytek and Knight, 2015), with a speculative link that a shift away from cortical inhibition may be related to disrupted information processing. This suggestion is consistent with prior studies that have linked reduced GABAergic and increased glutamatergic activity with ADHD (Edden et al., 2012; Hammerness et al., 2012; Zimmerman et al., 2015), signaling a possible E/I imbalance. Prior studies have also shown disrupted neural communication in ADHD and related neurodevelopmental disorders (Groom et al., 2010; Dinstein et al., 2012; Milne, 2011) using single-trial electroencephalogram (EEG) and canonical measures of EEG signal variability (McLoughlin et al., 2014), lending support to neural theories of information processing deficits in ADHD.

The EEG is comprised of periodic (oscillations) and aperiodic (offset, exponent) signals (Donoghue et al., 2020a). The aperiodic exponent reflects power across frequencies of the power spectral density (PSD). This neural marker is characterized by the χ parameter of a $1/f^{\chi}$ function, and has been linked to the E/I balance in cortical circuits (Gao et al., 2017). A shift away from cortical inhibition is reflected as a smaller exponent (flatter PSD). EEG studies on ADHD are potentially consistent with a flattening of the power spectrum (e.g., Barry et al., 2009; Loo et al., 2013), but have been interpreted in terms of ratios between canonical frequency bands. This may be problematic: recent findings indicate that band ratio measures (e.g., theta/beta ratio) are conflated by the aperiodic signal (Donoghue et al., 2020b).

Emerging evidence suggests that ADHD may be linked to difference in aperiodic activity. Robertson et al. (2019) found that young children with ADHD who were stimulant medication-naïve had steeper PSDs relative to their typically-developing peers and children with ADHD who were prescribed stimulant medication. In a pioneering but small study ($N = 61$, $n_{ADHD} = 29$), Pertermann and colleagues (2019) found that children with ADHD had flatter PSDs relative to their peers when engaged in response inhibition. This effect disappeared after methylphenidate treatment, indicating a potential role of dopamine in normalizing the relative contributions of E/I. Thus, findings suggest that differences in the exponent may associate with cognitive impairments in ADHD, although the direction of effect remains unclear. Findings require replication in larger samples and extension to other ADHD-related cognitive deficits.

In this study, we examined relationships between the aperiodic exponent and measures of RTV among adolescents with and without ADHD. We pre-registered an analysis plan (osf.io/vwtqn) that quantified the PSD using the power spectrum slope; however, during the time the manuscript was under review a parameterization method that disentangled aperiodic from periodic activity became available (Donoghue et al., 2020a). Thus, we report our primary result using this newer method; results using the other method are reported in the Supplement. Other details of our analytic plan (e.g., hypotheses, sample selection) follow the pre-registration. We hypothesized that adolescents with ADHD would have smaller exponents relative to their peers. Recognizing cognitive heterogeneity in ADHD, we predicted that slower RT and higher RTV would be associated with a smaller exponent in adolescents with and without ADHD. Finally, we predicted that a smaller exponent would be related to slower drift rate, a cognitive indicator of

inefficient information processing, but not boundary separation or non-decision time.

2. Materials and methods

2.1. Participants

Two-hundred and thirty-seven individuals between the ages of 11–17 years were recruited as part of an ongoing longitudinal study. Participants were initially recruited between the ages of 7–11 years using a community-based strategy based on public advertising and outreach. A parent/legal guardian provided written informed consent for themselves and their child. Adolescents provided written assent. Ethics approval was obtained from the Institutional Review Board at Oregon Health & Science University.

2.1.1. Baseline diagnostic assessment

At enrollment, a parent/guardian and teacher completed a research diagnostic evaluation including standardized behavior rating scales (ADHD Rating Scale, Conners'-3, Strengths & Difficulties Questionnaire) and a semi-structured clinical interview (Kiddie Schedule for Affective Disorders and Schizophrenia; parent only). Children completed behavior ratings (Multidimensional Anxiety Scale for Children and Children's Depression Inventory) and IQ and academic achievement screening (Wechsler Intelligence Scale for Children, 4th Ed., Vocabulary, Block Design, and Information and Wechsler Individual Achievement Test, 2nd Ed. Word Reading and Math Reasoning). Using all available information, baseline diagnoses were made by a clinical diagnostic team that included a board-certified child psychiatrist with over 25 years of experience and a licensed child neuropsychologist with over 10 years of experience. Blind to one another's ratings, they formed a diagnostic opinion based on all available information. Their agreement rate was excellent (ADHD diagnosis kappa = .88). Disagreements were conferenced and consensus reached. Cases where consensus was not readily achieved were excluded from the longitudinal study. Additional information about recruitment procedure and enrollment criteria can be found elsewhere (see Karalunas et al., 2014; Alperin et al., 2017).

2.1.2. Diagnostic assessment at year of EEG recording

All children in the longitudinal study were invited to participate in an optional EEG visit at a single time point (Year 5, 6, or 8 depending on the date of their initial enrollment). Complete diagnostic assessment was repeated and was identical to baseline assessment at this visit, including parent, teacher, and child standardized behavioral ratings and semi-structured clinical interview with parent. Cases were reviewed by the same diagnostic team as described for Year 1. In addition, total symptom counts were determined by combining parent (K-SADS) and teacher (ADHD-RS) report using an "OR" algorithm (Pelham et al., 2005). Following the DSM, final diagnostic groups at the year of the EEG visit were determined as follows: Individuals with ADHD were required to have ≥ 6 hyperactive or ≥ 6 inattention symptoms, as well as parent reported impairment on the K-SADS. Individuals in the control group were required to have ≤ 3 hyperactive, ≤ 3 inattention symptoms, and ≤ 4 total symptoms with no reported impairment.

Of the 237 children enrolled for the current study, 143 had ADHD at baseline and 94 were typically-developing controls. At the year of the EEG visit, 86 adolescents from the original ADHD sample continued to meet full criteria for ADHD, 19 transitioned to subthreshold (i.e., 5 symptoms of inattention or hyperactivity-impulsivity), 11 could not be readily classified as either ADHD or control based on symptom counts, and 17 transitioned from the ADHD to control group. Of the original typically-developing sample, 80 remained classified as controls, 1 now met criteria for ADHD, and 12 could no longer be classified as controls or as having ADHD. Because this is an ongoing longitudinal study, we did not have complete diagnostic information on 11 adolescents at the time of data analysis. Primary analyses for the present study were based on

diagnosis at the year of EEG recording ($n = 184$; $n_{ADHD} = 87$; males = 117).

2.2. Measures

2.2.1. EEG data collection and preprocessing

Children completed a resting baseline EEG recording as part of a longer laboratory protocol. EEG was continuously recorded during an 8-minute baseline task, which was divided into four 2-minutes blocks. Adolescents were instructed to keep their eyes open (EO) for two of the blocks, and closed (EC) for the other two blocks. Blocks alternated between EC and EO conditions (EC, EO, EC, EO).

EEG was recorded with 32 Ag-AgCl active electrodes based on the international 10–20 system. The EEG signal was amplified with Brain Products' ActiCHamp system and digitally recorded at 500 Hz using PyCorder v1.0.9. Impedance levels for each electrode was at or below 50kΩ during data collection. EEG was referenced online to the central midline electrode site (Cz), and was then down sampled to 250 Hz and re-referenced to the average of all electrodes offline. EEGLAB and ERPLAB (Delorme and Makeig, 2004; Lopez-Calderon and Luck, 2014) toolboxes in MATLAB were used to analyze the raw EEG data. We applied an infinite impulse response bandpass filter with a half-amplitude cutoff of 0.1 Hz and 50 Hz, and a 12 dB/octave roll-off to the data. An independent components analysis was used to correct eye blink artifacts for correction.

2.2.2. Aperiodic exponent

Power spectral density was calculated in 0.5 Hz increments from 1 Hz to 50 Hz using a Fourier transformation on each artifact-free epoch. The "Fitting Oscillations and One-Over-f" (FOOOF) toolbox was used to calculate the aperiodic exponent. This spectral parameterization algorithm decomposes the power spectrum into periodic and aperiodic components via an iterative process of model fitting (see Donoghue et al., 2020a for detailed description). Consistent with prior pediatric ADHD research (Robertson et al., 2019), and recommendations for a broad fitting range described on the author's website, we extracted aperiodic exponents from the 2–50 Hz frequency range of each power spectrum (*aperiodic_mode* = 'fixed', *peak_width_limits* = [1, 8], *max_n_-peaks* = 8, default settings otherwise). We used the 'fixed' setting given that we did not anticipate a "knee" in the power spectrum, a feature observable in broad frequency ranges (e.g., 77 Hz ± 14 Hz; Miller et al., 2009). This assumption was supported by visual inspection of each PSD after spectral parameterization via FOOOF.

2.2.3. Reaction time and drift-diffusion parameters

For the evaluation of reaction time speed and variability using the diffusion model, we used data from a tracking version of the dual-task "Stopping Task" (described in Logan, 1994; Logan et al., 1997; Nigg, 1999). This is a dual-task experiment in which the child completes a series of fast decision trials. The task embeds a choice reaction time task (go trials) and a stop task (stop trials). For each trial, a central fixation point appeared for 500 ms. An "X" or an "O" then appeared for 1000 ms. On 75 % of trials ("go" trials), children were asked to indicate with a key press whether an "X" or an "O" appeared in the center of the screen. On 25 % of trials ("stop" trials), an auditory tone presented after the stimulus indicated that the child should not respond. Children were given a total of 2000 ms to respond after which the next trial automatically commenced. Diffusion parameters were estimated from the go trials of the task using the full distribution of correct (pressed X when X was presented or pressed O when O was presented) and error (pressed X when O was presented or pressed O when X was presented) reaction times. Stop trials were ignored for this set of analyses. Diffusion modeling has been previously used successfully in the analysis of go-trials of a stop task in adults (Verbruggen and Logan, 2009) and in our own work in both ADHD and autism spectrum disorders (Karalunas et al., 2018; Karalunas and Huang-Pollock, 2013; Karalunas et al.,

2012a). After 32 practice trials, children completed 8 blocks of 32 experimental trials each.

Mean reaction time (RT) and the standard deviation of reaction time (SDRT) on correct go trials were calculated for each child. Diffusion parameters were estimated from the trial-by-trial data using the Fast-dm modeling technique and the downloadable program from the author's website (Voss and Voss, 2007). Drift rate (*v*), boundary separation (*a*), and non-decision time (*Ter*) were computed for each participant based on their performance on go trials as an index of speeded responses in the context of a forced-choice reaction time task. Anticipation reaction times (those <150 ms) were removed from the distribution because these outlier reaction times negatively impact estimation of the diffusion parameters (Vandekerckhove and Tuerlinckx, 2007). The diffusion model adequately fit the data for all adolescents, as indicated by the Kolmogorov-Smirnov statistic.

2.3. Analytic plan

Data were analyzed in R v3.5.1 (R Core Team, 2018). First, we examined whether adolescent ADHD status (ADHD/control) predicted aperiodic exponents. Second, we examined whether aperiodic exponents were associated with reaction time parameters (mean RT, SDRT, *v*, *a*, *t0*), controlling for adolescent sex and ADHD status. Third, we examined whether the interaction between ADHD and the aperiodic exponent predicted any of the reaction time parameters. Missingness was handled using the *Multivariate Imputation by Chained Equations* (MICE) package (van Buuren and Groothuis-Oudshoorn, 2011). We estimated 25 imputed datasets using 25 iterations. A linear model was fit over the imputed datasets and results were pooled and used in regression analyses.

3. Results

3.1. Sample description

EEG data were missing for 7 participants (3.8 %; $n = 5$ children where technical issues prevented recording and $n = 2$ excluded for poor data quality). Reaction time data were missing for 7 participants as well (3.8 %). Adolescents with and without missing data did not differ as a function of age, sex, or ADHD symptoms (*ps* > .23).

Descriptive information is presented in Table 1. Adolescents with and without ADHD did not differ in age ($p = .84$), but typically-developing adolescents had higher IQ scores ($p = .001$), and males are over-represented in the ADHD sample ($p = .03$), which is consistent with documented differences in prevalence between sexes (Ramtekkar et al., 2010). Parent and teacher reports on the Conners' ADHD Rating Scale were consistent with diagnostic classifications (*ps* < .001).

3.2. Preliminary analyses

Distributions and comparisons by ADHD status for each reaction time parameter are presented in Fig. 1A–E Two extreme mean RT values were excluded from analyses. Reaction time variability (SDRT) data were log transformed to account for positive skewness. All other variables had an approximately normal distribution. Aperiodic exponents for the EC and EO conditions were highly correlated ($r = .80$), and therefore averaged for primary analyses.

Aperiodic exponents did not differ based on adolescent sex, $t(175) = -1.07$, $p = .28$. Adolescent ADHD was significantly associated with greater SDRT ($t(175) = -4.35$, $p < .001$), slower drift rate ($t(175) = 5.16$, $p < .001$) and faster non-decision time ($t(175) = 2.90$, $p < .01$). Adolescent ADHD was not, however, associated with mean RT ($t(173) = 1.24$, $p = .22$) or boundary separation ($t(175) = 1.26$, $p = .21$). Overall, findings were consistent with published results in this sample at year 1 of the longitudinal study (Karalunas et al., 2012a) and with the broader literature (Karalunas et al., 2012b). Bivariate correlations among

Table 1
Descriptive information.

	ADHD (n = 87)	Controls (n = 97)		
	M (SD)	M (SD)	p	95 % CI
Age (years)	13.93 (1.45)	13.97 (1.28)	0.84	[−0.36, 0.44]
Sex (male:female)	63:24	54:43	0.03	
IQ	107.86 (16.16)	115.06 (13.67)	0.001	[2.86, 11.54]
Stimulant medication[a]	57 %	—	< 0.001	
Median income	$50,000–$74,000	$50,000–$74,000		
Caucasian/Non-Hispanic	n = 81	n = 90		
Total ADHD symptoms[b]	12.00 (3.67)	0.50 (1.33)	< 0.001	[−12.44, −10.56]
Conners' ADHD RS[c]				
Parent Int T-score	75.76 (10.75)	47.20 (9.62)	< 0.001	[−31.56, −25.57]
Teacher Int T-score	66.75 (12.52)	48.30 (7.98)	< 0.001	[−22.00, −14.91]
Parent Hyp-Imp T-score	69.55 (15.73)	48.41 (9.22)	< 0.001	[−24.89, −17.40]
Teacher Hyp-Imp T-score	64.29 (15.49)	48.00 (7.40)	< 0.001	[−20.38, −12.21]
Aperiodic exponents	1.70 (0.30)	1.79 (0.28)	0.04	[0.01, 0.18]
Mean RT (ms)	644.63 (137.45)	684.42 (165.98)	0.09	[−5.78, 85.35]
SDRT (ms)	211.97 (87.65)	167.18 (58.59)	< 0.001	[−66.69, −22.90]
Drift rate	3.15 (0.73)	3.76 (0.82)	< 0.001	[0.37, 0.84]
Boundary separation	1.55 (0.29)	1.60 (0.28)	0.21	[−0.03, 0.14]
Non-decision time	0.39 (0.11)	0.44 (0.13)	< 0.01	[0.02, 0.09]

Note. RT = reaction time. SDRT = standard deviation of reaction time.
[a] Three children who no longer met ADHD criteria at the EEG visit were nonetheless prescribed stimulant medications. They were retained for primary analyses because detailed independent review by the diagnostic team describe in methods confirmed the lack of diagnosis.
[b] Total symptom counts reflect combined scores from parents (K-SADS) and teachers (ADHD-RS) report using an "OR" algorithm.
[c] Conners' ADHD rating scale (Conners, 2003). Int = Inattention, Hyp-Imp= Hyperactivity-impulsivity.

variables of interest are presented in Table 2. Aperiodic exponents were associated with boundary separation ($r = .16, p = .04$). None of the other variables were significantly correlated with aperiodic exponents ($ps > .15$).

3.3. Primary analyses

Adolescents with ADHD had smaller exponents relative to the control group ($\beta = −.16, t(175) = −2.08, p = .04$), indicative of a flattened PSD (Fig. 1F). Power spectral densities for each condition by ADHD status (in log-log and semi-log) are presented in Fig. 1G–J. Exponents did not differ by stimulant medication history, $t(172) = 0.41, p = .68$.

Regression analyses showed that, controlling for ADHD status, exponents were positively associated with SDRT ($\beta = .16, p = .03$), indicating that less intraindividual variability was related to a flattened PSD. Exponents were also associated with drift rate ($\beta = −.15, p = .03$); faster drift rate was related to a flattened PSD. These effects are plotted by diagnostic category in Fig. 2. Exponents were marginally associated with boundary separation ($\beta = .15, p = .07$). Exponents did not predict mean RT ($\beta = .05, p = .54$) or non-decision time ($\beta = .03, p = .67$). There was no significant interaction between ADHD status and aperiodic exponents on any of the reaction time or drift difusion parameters ($ps > .51$). Results remained the same when adolescent sex was included in the model.

Finally, results were similar when accounting for missingness via

MICE (van Buuren & Groothuis-Oudshoorn, 2011). Adolescents with ADHD had smaller exponents relative to the control group, $t(170.59) = −1.95, p = .05$. Controlling for ADHD status, exponents were significantly associated with SDRT ($p = .03$) and drift rate ($p = .04$). Exponents were marginally associated with boundary separation ($p = .06$), and did not predict mean RT ($p = .46$) or non-decision time ($p = .67$). Posthoc analyses showed that results were similar when the 17 adolescents in the ADHD group at Year 1 who transitioned to the control group were excluded from the primary analyses (see Supplement).

3.4. Pre-registered analyses

It is worth noting that the current analyses were inspired by a preregistered study submitted to Open Science Framework in October, 2018 (osf.io/vwtqn). Due to rapid developments in research on the aperiodic exponent (Donoghue et al., 2020a), the original method for calculating the exponent, as well as related aspects of the analysis plan, were no longer considered best practice. We therefore updated the analysis plan to what is described above. For transparency, we have included findings from analyses as described in original preregistration in the Supplement. Of note, when estimated as a linear regression coefficient estimated from 2−25 Hz (with 5−9 Hz activity removed) for each participant, the exponent was associated with adolescent age ($r = .36, p < .001$) and reaction time variability ($\beta = .15, p = .04$), but not ADHD ($\beta = −.001, p = .99$) or any of the other reaction time parameters ($ps > .08$).

4. Discussion

Efficient information processing facilitates cognition and may be disrupted in multiple neurodevelopmental disorders, including ADHD. Computational cognitive models point to inefficient information processing as a key driver of cognitive impairments in ADHD (Karalunas et al., 2014, 2012a; Weigard et al., 2018). Our results highlight the clinical utility of the aperiodic exponent as a neural correlate of ADHD and disrupted information processing in adolescence. We further recent work aimed at understanding the neural mechanism underpinning ADHD, while introducing several new questions about how to interpret the functional significance of this relationship.

ADHD is believed to be characterized by inefficient information processing that contributes to cognitive impairments for some children with the disorder (Karalunas et al., 2014; Kofler et al., 2013; Tamm et al., 2012). Emerging evidence implicates an imbalance of cortical excitation and inhibition as one neural correlate (and possible mechanism) of cognitive impairment in specific psychiatric disorders (e.g., schizophrenia; Peterson et al., 2017). Our findings contribute to this burgeoning literature, linking a noninvasive index of the E/I balance (Gao et al., 2017) to ADHD in adolescence. Consistent with our hypothesis, adolescents with ADHD had smaller exponents (flatter PSD) relative to their typically-developing peers, which may reflect abnormal E/I balance in developing cortical circuitry. Our findings are consistent with clinical and animal models of ADHD, which have shown altered GABAergic and glutamatergic activity in this population (Edden et al., 2012; Hammerness et al., 2012; Zimmerman et al., 2015), potential indicators of E/I imbalance in cortical circuitry.

This is the first study, to our knowledge, to examine aperiodic activity parameterized via FOOOF in adolescents with ADHD. Our findings partially align with prior work that identified a flatter PSD in older children (mean age 9.9 years) during cognitive performance (Pertermann et al., 2019). However, Pertermann et al. (2019) relied on a method for quantifying the aperiodic signal that within the current sample yielded divergent results in comparison to FOOOF. Using FOOOF, Robertson et al. (2019) found steeper PSD in young (mean age = 5.75 years), medication-naïve children with ADHD, the opposite pattern observed here and in other studies (Pertermann et al., 2019). Aligning results across development and method is a crucial next step to reconcile the inconsistent findings reported in this emerging literature.
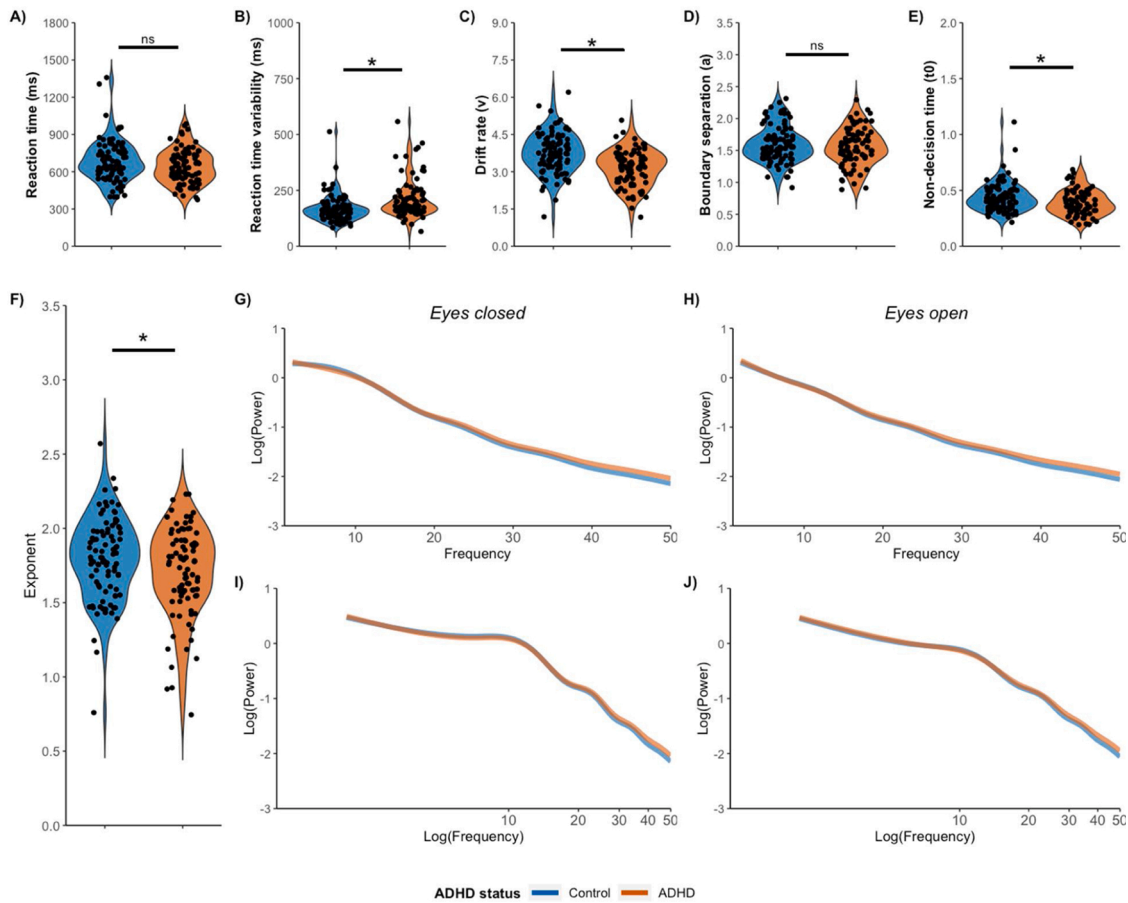
Fig. 1. Distribution and group comparison of mean reaction time (*A*), reaction time variability (*B*), drift rate (*C*), boundary separation (*D*), non-decision time (*E*), and aperiodic exponents (*F*). Power spectral densities for eyes closed (*G, I*) and eyes open (*H, J*) in semi-log (*G, H*) and log-log (*I, J*) space averaged across adolescents in the ADHD (orange) and control (blue) groups. FOOOF (Donoghue et al., 2020a) removes periodic (putative oscillations) activity that rise above the aperiodic component of the neural signal, disentangling power spectral features that are thought to have distinct physiological mechanisms. We did not expect a knee in the PSD across the examined frequency range, nor did we observe one when visually inspecting each individual PSD after spectral parameterization via FOOOF. On average, we did observe an alpha "bump" around ~10 Hz as well as a smaller beta "bump" around ~20 Hz, each of which is more prominent in the eyes closed (*I*) relative to eyes open (*J*) condition, as would be expected.

Table 2
Correlation table.

	1.	2.	3.	4.	5.	6.
1. Aperiodic exponents	—					
2. Age (years)	−.11	—				
3. Mean RT	.06	−.09	—			
4. SDRT	.11	−.30 ***	.46 ***	—		
5. Drift rate	−.10	.29***	.07	−.57 ***	—	
6. Boundary separation	.16*	−.12	.56 ***	.46***	.07	—
7. Non-decision time	.06	.05	.82 ***	.18*	.22 **	.20 **

Note. RT variability data (SDRT) were natural log transformed for all analyses. RT = reaction time. SDRT = standard deviation of reaction time.

* $p < .05.$
** $p < .01.$
*** $p < .001.$

Using a SSM approach to model cognitive performance, we confirmed that adolescents with ADHD had more variable response time and slower drift rates. These results conform to prior SSM findings in ADHD and to findings in this same sample at younger ages (Karalunas et al., 2012a; Karalunas and Huang-Pollack, 2013). Researchers have

speculated that within-person, moment-to-moment fluctuations in performance, captured by the SDRT and drift rate parameters, reflect inefficient information processing (Karalunas et al., 2014; Karalunas and Huang-Pollock, 2013). Contrary to our hypotheses, we found that, when controlling for ADHD status, adolescents who had smaller exponents exhibited *less* reaction time variability, suggesting less variability in performance. This effect was mirrored in the SSM parameters, with a smaller exponent being related to *faster* drift rate, a well-validated indicator of efficient information processing that is thought to directly relate to higher signal-to-noise ratios in circuits underlying decision-making (Ratcliff et al., 2003, 2009).

These discrepant findings may speak to a U-shaped relationship between aperiodic activity and performance. It is possible that any imbalance in excitatory/inhibitory inputs may contribute to non-optimal performance, regardless of the direction of effect (Voytek and Knight, 2015). Thus, after removing the association between ADHD and the aperiodic exponent, deviations in either direction would be problematic. This would be consistent with theory suggesting a U-shaped relationship between aperiodic activity and psychiatric disorder specifically (Voytek and Knight, 2015) and brain-behavior relationships more broadly (Northoff and Tumati, 2019). Additional research with clinical populations is critically necessary to determine whether (and how) aperiodic activity relates to specific cognitive dysfunctions that contribute to psychopathology risk. Future research might also consider examining whether homogenous can be identified based on aperiodic
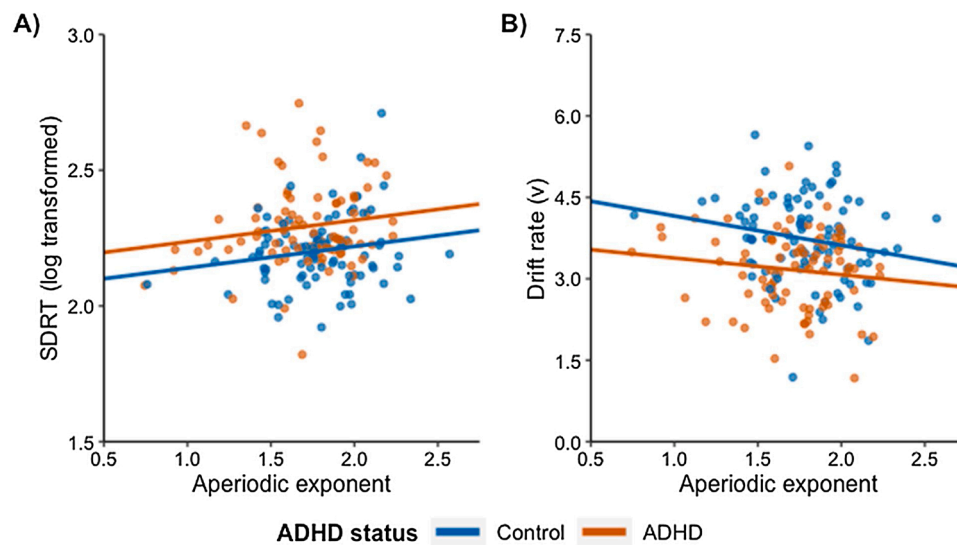
Fig. 2. Associations between aperiodic exponents and (*A*) reaction time variability and (*B*) drift rate for adolescents in the ADHD (orange) and control (blue) groups.

(as well as periodic) power spectral features to improve clinical prediction (Karalunas et al., 2014).

Converging evidence indicates that the aperiodic exponent is a physiologically distinct component that coexists with periodic activity (putative oscillations) in neural signals and may underpin a range of cognitive and behavioral states (e.g., González-Villar et al., 2017; He et al., 2020; Peterson et al., 2017; Podvalny et al., 2015; Schaworonkow and Voytek, 2020; Tran et al., 2020). To this end, it has recently been argued that band ratio measures (e.g., theta/beta ratio) that are prevalent in pediatric ADHD research may be conflated by aperiodic activity (Donoghue et al., 2020b). Rather than indicating correlated change between frequency bands, as has been previously claimed, Voytek and colleagues proposed that concomitant age-related shifts in the distribution of spectral power reflects a single, unified "rotation" in the aperiodic (1/*f*-like) component of the signal (Donoghue et al., 2020a; Voytek and Knight, 2015). Our findings suggest that the historical emphasis on oscillatory dynamics in understanding cognitive impairment of neurodevelopmental disorders is too limited. Studies that simultaneously consider periodic and aperiodic changes will be critical for clarifying neural mechanisms of these impairments.

4.1. Limitations

A major unresolved limitation is that results using two different approaches for characterizing aperiodic activity do not align. The hypotheses tested here were part of a study that was preregistered in late 2018. At the time, a regression-based method for calculating the aperiodic exponent was common in the published literature (e.g., Dave et al., 2018; Voytek et al., 2015). When using that method to estimate the slope of the EEG power spectrum in this sample, we did not see an association with ADHD or SSM parameters, but did see an association with age that was not present when using FOOOF (see Supplement). Donoghue and colleagues (2020a) have since argued that the regression-based calculation method may be susceptible to periodic oscillatory activity. Instead, they emphasize the importance of parameterizing spectral features of the PSD via an iterative fitting process. We believe that the regression-based power spectral slope estimates may have been biased by individual differences in periodic activity (e.g., beta; see Fig. 1I–J). FOOOF and the regression-based method differ based on how periodic activity is removed, either by canonical frequency bands or via a person-centered approach, respectively. With this in mind, we shifted away from the preregistered analysis plan to align with evolving best practices in this area of research. Direct comparison

of these approaches is beyond the scope of this paper, but will be important for future work aimed at integrating results from this rapidly developing area of research.

Another limitation of this study was that we calculated the aperiodic exponent at rest rather than in response to stimuli. It is well-accepted that organization and functional characteristics of the brain at rest are one important piece of understanding brain-behavior relationships (Arieli et al., 1996; Cao et al., 2017; Deco et al., 2011; Fox et al., 2007; Grayson and Fair, 2017; Wang et al., 2016). Indeed, the approach of relating resting-state brain dynamics to task performance has often been applied to clinical research (Rogala et al., 2020; Szostakiwskyi et al., 2017; van Dongen-Boomsma et al., 2010v) and other studies have used this design in studying the associations of the aperiodic exponent to cognitive performance (Robertson et al., 2019). However, other studies record during cognitive tasks (Dave et al., 2018; Pertermann et al., 2019). Dave and colleagues (2018) found that exponents (estimated as EEG power spectrum slopes) were highly stable across cognitive tasks, lending support for their role as a signature of the brain's intrinsic architecture. Nevertheless, additional research is needed to determine the relationship between slopes measured at rest and during active tasks. Establishing a resting state index of disrupted information processing would afford developmental cognitive neuroscientists new opportunities to assess dynamic neural communication in populations for whom recording evoked activity can be challenging, such as infants.

Lastly, adolescents in our sample either had ADHD or had minimal (or no) symptoms. While an extreme group design provides insight into differences in a clinical disorder, it fails to capture the full range of childhood attention and hyperactivity-impulsivity. Further investigation into the functional significance of the aperiodic exponent with both normative and clinical samples across the lifespan is needed.

5. Conclusions

Efficient information processing is a critical function of the developing brain and may be disrupted in a variety of neurodevelopmental conditions, including ADHD. Nonetheless, neural mechanisms of cognitive impairment remain underspecified. EEG studies have traditionally focused on group differences in oscillatory dynamics, but recent work confirms the importance of aperiodic activity of neural power spectra as a potential confound leading to misinterpretation of oscillatory results (Donoghue et al., 2020b). Our study represents the largest investigation into the behavioral and cognitive correlates of the aperiodic exponent in children with ADHD to-date. Findings that the

aperiodic exponent, an index of cortical excitatory/inhibitory imbalance, is related to ADHD and cognitive impairments highlights the importance of this neural signature across development. Results underscore the need for additional work that considers both periodic and aperiodic activity to understand the neural mechanisms underlying cognitive dysfunctions in ADHD.

Data statement

All relevant data are within the paper and its Supporting Information files.

Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at doi:https://doi.org/10.1016/j.dcn.2021.100931.

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