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Research Report

Increased neural variability in adolescents with ADHD symptomatology: Evidence from a single-trial EEG study

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ABSTRACT

Increased intrasubject variability of reaction time (RT) refers to inconsistency in an individual's speed of responding to a task. This increased variability has been suggested as a fundamental feature of attention deficit hyperactivity disorder (ADHD), however, its neural sources are still unclear. In this study, we aimed to examine whether such inconsistency at the behavioral level would be accompanied by inconsistency at the neural level; and whether different types of neural and behavioral variability would be related to ADHD symptomatology. We recorded electroencephalogram (EEG) data from 62 adolescents, who were part of a prospective longitudinal study on the development of ADHD. We examined trial-by-trial neural variability in response to visual stimuli in two cognitive tasks. Adolescents with high ADHD symptomatology exhibited an increased neural variability before the presentation of the stimulus, but when presented with a visual stimulus, this variability decreased to a level that was similar to that exhibited by participants with low ADHD symptomatology. In contrast with our prediction, neural variability was unrelated to the magnitude of behavioral variability. Our findings suggest that adolescents with higher symptoms are characterized by increased neural variability before the stimulation, which might reflect a difficulty in alertness to the forthcoming stimulus; but this increased neural variability does not seem to account for their RT variability.

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1. Introduction

Attention deficit hyperactivity disorder (ADHD[1]) is a chronic neurodevelopmental disorder, with a worldwide prevalence estimation of 5.9% of school-age children (Faraone et al., 2021; Polanczyk et al., 2014) and about 2.8% of adults (Fayyad et al., 2017; Simon et al., 2009). Individuals with ADHD display symptoms of inattention, hyperactivity, and impulsivity at levels that interfere with their day-to-day functioning (American Psychiatric Association, 2013). Anecdotally, individuals with ADHD are often described as "consistently inconsistent" in their behavior over time (Friedman et al., 2022; Kofler et al., 2013). For example, when dealing with their homework, children with ADHD may experience variability in their performance, as seen in the ability to solve a problem at one moment, but then struggle to do so on a similar item (Friedman et al., 2022). Researchers have empirically studied this behavioral inconsistency by measuring the variability of cognitive performance, specifically reaction time (RT) variability (Klein et al., 2006; Kofler et al., 2013).

RT variability refers to inconsistency in an individual's speed of responding to a task, including periodic instances of prolonged RTs (Klein et al., 2006; Kofler et al., 2013). This increased behavioral variability in ADHD has been consistently demonstrated in children, adolescents, and adults with ADHD (Gonen-Yaacovi et al., 2016; Klein et al., 2006; Kofler et al., 2013; Salunkhe et al., 2021; Tamm et al., 2012); and in a variety of cognitive tasks assessing different domains (e.g., attention, inhibitory control, and working memory), such as choice reaction time (Geurts et al., 2008; Gooch et al., 2012), stop signal task (SST; Einziger, Zilberman-Hayun, et al., 2021; Klein et al., 2006; Marx et al., 2013), go-no-go (Epstein et al., 2006; Kuntsi et al., 2005), and n-back (Saville et al., 2015). RT variability appears to be a stable individual feature that is independent of other cognitive domains (Karalunas et al., 2014; Klein et al., 2006; Vaurio et al., 2009). It was also found to correlate with the severity of ADHD symptoms but was not uniquely attributed to a specific symptom domain (i.e., inattention or hyperactivity/impulsivity) (Kofler et al., 2013; Tamm et al., 2012).

Despite such robust evidence regarding the involvement of increased behavioral variability (i.e., RT variability) in ADHD, the underlying neural processes remain unknown. A variety of theories have been suggested in the literature regarding the brain mechanisms driving behavioral variability in ADHD as reviewed in detail elsewhere (Kuntsi & Klein, 2012). For example, a general dysfunction in neuro-energetic supply may cause a deficit in lactate supply, which could compromise neurons' ability to fire rapidly and reliably (Killeen et al., 2013). A deficit in dopamine release may alter neuromodulation of the entire brain during development (Swanson et al., 2007); and attenuated dopamine neuromodulation might yield less stable cognitive performance (MacDonald et al., 2009; Salunkhe et al., 2019; Saville et al., 2014). Another theory has argued that the default mode system is not properly suppressed in individuals with ADHD (Sonuga-Barke & Castellanos, 2007), and this neural activity interferes with the neural processing of a task, therefore generating less consistent responses across trials (Di Martino et al., 2008; Helps et al., 2010; Sonuga-Barke & Castellanos, 2007).

It is therefore plausible to assume that behavioral variability might be accompanied by inconsistency at the neural level, which could be reflected in high neural variability. Neural variability refers to within-person variability of continuous moment-to-moment brain fluctuations (Faisal et al., 2008; Stein et al., 2005). These fluctuations can be measured during cognitive tasks, even in response to simple and repetitive sensory input (Dinstein et al., 2015). Evidence suggested that increased neural variability might reflect less consistent neural processes (Garrett et al., 2013; Li et al., 2001), emerging from random fluctuations in electrical activity (Hong & Rebec, 2012; Li et al., 2001), which could lead to a reduction in the stability of behavioral performance across time (Pertermann et al., 2019). Different individuals exhibit distinct magnitudes of trial-by-trial neural variability that are stable over time and across tasks, suggesting that it is a fundamental characteristic of an individual's neural function (Arazi, Gonen-Yaacovi, et al., 2017). Increased neural variability has been reported in ADHD (Gonen-Yaacovi et al., 2016; McLoughlin et al., 2014; Saville et al., 2015), schizophrenia (Yang et al., 2014) and autism (Milne, 2011; Weinger et al., 2014). Although it should be noticed that at the behavioral level, several studies have indicated that autism does not show increased variability if comorbidity for ADHD is controlled (Adamo et al., 2014; Salunkhe et al., 2021).

Electroencephalogram (EEG) and event-related potential (ERP) are especially suitable methodologies for studying neural variability; their high temporal resolution and precision are required to study fast fluctuations. Previous EEG studies have reported increased neural variability in ADHD (Gonen-Yaacovi et al., 2016; McLoughlin et al., 2014; Saville et al., 2015). For example, Saville et al. (2015) found that increased behavioral variability and increased variability in the latency of the response-locked P3b and the lateralized readiness potential (LRP) were apparent in adolescents with ADHD, compared to controls. Their results suggested that the increased behavioral variability in ADHD might arise from a deficit in response processing such as response planning and execution. McLoughlin et al. (2014) reported that adolescents with ADHD showed higher variability in frontal-midline theta oscillations; this kind of brain activity was previously suggested to be an index of cognitive control (Luu et al., 2004). Their results suggest that increased behavioral variability might arise from impaired top-down cognitive control processes (McLoughlin et al., 2014). These two findings could mean that behavioral variability in ADHD is caused by dysfunctions in high-order brain areas that fail to govern the (supposedly intact) low-order sensory and motor brain areas that perform the simple tasks commonly used to measure behavioral RT variability.

In contrast, Gonen-Yaacovi et al. (2016) focused on low-order sensory processes and found evidence for a greater overall neural variability in sensory systems of individuals with ADHD. They compared the trial-by-trial neural variability

[1] Abbreviations: ADHD = Attention-Deficit Hyperactivity Disorder; CV = Coefficient of Variation; SST = Stop Signal Task; VST = Visual Sensory Task.

between ADHD and control participants in response to visual and auditory stimuli. Increased neural variability, reflected by larger P100/N100 amplitude variability, was found in the ADHD group compared to controls in both sensory modalities. This neural variability was apparent before and after the presentation of the stimulus and even in trials in which the stimulus was omitted. Their results suggest that ongoing neural fluctuations were generally more variable among individuals with ADHD, regardless of stimulus type or appearance (Gonen-Yaacovi et al., 2016).

Although all these findings have shown evidence for increased neural variability in ADHD (Gonen-Yaacovi et al., 2016; McLoughlin et al., 2014; Saville et al., 2015), each of them implies a different neural mechanism. Specifically, both McLoughlin et al. (2014) and Saville et al. (2015) have focused on task-evoked higher-order cognitive neural responses, while Gonen-Yaacovi et al. (2016) have focused on ongoing variability and early stimulus-evoked responses. One of the major differences between these findings arises from measuring neural variability in different cortical regions and different stages of processing in each study. Collectively, these findings could suggest that the neural variability in ADHD might not necessarily be related to a specific cognitive deficit, but rather reflect a more general characteristic of the brain activity of individuals with this disorder, in line with the conceptualization of intrasubject variability as a unitary construct (Klein et al., 2006).

1.1. *Different components of neural variability*

One of the advantages of using neuroimaging and electrophysiology techniques to study neural variability is the ability to decompose it into different components, by measuring it at different time intervals (see review in Dinstein et al., 2015). Post-stimulus variability refers to the variability of the amplitude or latency of the neural response that is evaluated regarding a specific stimulus. Pre-stimulus variability is exhibited before the presentation of a stimulus and is not necessarily related to a specific task or stimulus but might reflect preparatory attention to a forthcoming stimulus. Evidence has consistently demonstrated that post-stimulus variability is smaller compared to pre-stimulus variability (e.g., Arazi, Gonen-Yaacovi, et al., 2017; Churchland et al., 2010); this phenomenon of reduction in variability after a presentation of a stimulus is referred to as variability quenching. There is evidence supporting the involvement of variability quenching in perceptual and cognitive performance (Arazi, Censor, et al., 2017; Arazi et al., 2019; Daniel & Dinstein, 2021; Dinstein et al., 2015; Garrett et al., 2013; Schurger et al., 2010; 2015; Xue et al., 2010), including attentional processes (Arazi et al., 2019), that might be relevant to ADHD. For example, variability quenching was found to be modulated by aspects of attention (i.e., alertness and spatial attention); specifically, a relevant cue that was presented to alert the subject to an upcoming stimulus generated larger variability quenching, compared to an identical cue that was presented in a control experiment without a task (Arazi et al., 2019). Arazi and colleagues have suggested that the relative level of change in variability from the pre-stimulus interval to the post-stimulus interval (which is reflected in variability quenching) was a better predictor of individual differences in perception, compared to the absolute value of variability (Arazi, Censor, et al., 2017). However, since variability quenching has not been measured in ADHD it is not clear whether it has any relation to its symptoms.

1.2. *The present study*

The current study is part of a larger longitudinal study on the development of ADHD and its associated core neurocognitive deficits. The primary aim was to corroborate Gonen-Yaacovi et al.'s (2016) findings regarding increased ongoing neural variability in ADHD, using a sample of adolescents displaying varying levels of ADHD symptomatology. By examining this relationship in a non-clinical sample, it was possible to analyze the continuous range of symptoms, rather than focusing solely on the more severe cases that are typically included in clinical samples. To achieve this, EEG was recorded during the same visual sensory task used in Gonen-Yaacovi et al.'s study, which was designed to elicit a large and robust P100 response; we also adopted their methodology to analyze the data.

Additionally, participants completed the SST (as reported in Einziger, Ben-Shachar, et al., 2021), which has been widely used to measure behavioral variability (e.g., Klein et al., 2006; Marx et al., 2013). The SST was also suitable for examining pre-stimulus neural variability, which allowed us to test our hypotheses in additional experimental settings.

We computed trial-by-trial neural variability in a sample of 17-year-old participants and examined its relation with earlier and concurrent ADHD symptoms and behavioral variability. We calculated the following types of variability: pre-stimulus variability, which appears before the presentation of the stimulus, and post-stimulus variability, which appears shortly after the presentation of the stimulus. Following Arazi and colleagues (2017, 2019), we also computed variability quenching as an additional measure, describing the reduction in variability from the pre-stimulus to the post-stimulus interval.

Such a separation of neural variability into its different types could shed light on the cognitive processes that underline its relation with ADHD. Neural variability that is observable solely in the pre-stimulus interval might reflect preparatory attentional processes. Alternatively, variability that is observable solely in the post-stimulus interval might reflect variability in the processing of the stimulus. In both cases, the elevated levels of neural variability in individuals with ADHD symptoms might be linked to a specific cognitive process. However, variability that occurs in both the pre-stimulus and post-stimulus intervals might reflect a more generalized process, indicating ongoing fluctuations in brain activity over time, irrespective of any specific cognitive process.

Moreover, given the evidence demonstrating the association between variability quenching and cognitive performance (Arazi, Censor, et al., 2017; Arazi et al., 2019), it is important to investigate its relation with ADHD symptomatology. If the strongest association is found between variability quenching and ADHD symptoms, rather than pre- and post-stimulus variability, it would suggest that the relative

change in variability is more informative than the initial absolute level of variability.

We hypothesized that (1) higher ADHD symptoms (measured at 13 and 17 years, using parent and self-reports) would be associated with higher neural variability in the pre-stimulus interval and post-stimulus interval, and with lower levels of variability quenching. (2) Behavioral variability would be positively related with the different components of neural variability; and (3) both behavioral and neural variability would uniquely and positively predict ADHD symptomatology in adolescence; however, the strength of these associations may vary depending on the different types of neural variability.

2. Materials and methods

2.1. Participants

The current sample consisted of 62 male adolescents ($M = 17.36$ years, $SD = .41$, range = 16.52–18.48) who participated in a prospective longitudinal study since birth. Families were recruited to the study from the maternity ward of a local hospital, based on their fit to several inclusion criteria. First, because of the higher prevalence of ADHD among males compared to females (American Psychiatric Association, 2000), only families with male newborns were recruited. Second, families were recruited based on fathers' ADHD symptomatology. Fathers' symptoms were initially assessed at the hospital, via a yes/no format questionnaire that included 18 ADHD items taken from the DSM-IV (Diagnostic and Statistical Manual of Mental Disorders, 4th edition; American Psychiatric Association, 2000). This assessment was used to assign the infants to either a risk group (i.e., infants whose fathers had seven or more symptoms) or a comparison group (i.e., infants whose fathers had three or fewer symptoms). At 2–6 months of age, a more extensive assessment of parental symptomatology was conducted by measuring the ADHD symptomatology of both parents, using self and spousal reports of the Conners' Adults ADHD Rating Scale (Conners et al., 1998). This continuous parental symptom score was used in our study to indicate familial risk for ADHD (Einziger, Ben-Shachar, et al., 2021; Einziger, Zilberman-Hayun, et al., 2021). Our sample included children ranging from low to high risk of developing ADHD. All infants who entered the study were born healthy, with normal birth weight ($M = 3{,}296.07$ g, $SD = 419.75$) and gestational age ($M = 39.19$ weeks, $SD = 1.59$). Families who were recruited for the longitudinal study were two-parent families of either native-born residents or immigrants who studied in the country and spoke the local language. At the beginning of the study, the mean age of parents was 29.95 years ($SD = 4.90$) for mothers and 33.65 years ($SD = 5.44$) for fathers. The mean number of years of education was 12.80 ($SD = 1.72$) for mothers and 12.32 ($SD = 1.77$) for fathers. For the current study, we tried to re-contact all the families from the study; participants who eventually took part in the current stage of the study did not differ from the rest of the original sample (Ns = 50–114, which included participants who decided to discontinue their participation or with whom we lost contact) in all study variables (including parents' education, parents' ADHD symptoms, children's birth weight, and gestational age), all $ts < |1.66|$, $ps > .09$. Informed consent was obtained from all the participants included in the study.

2.2. Measures

2.2.1. ADHD symptoms assessment

2.2.1.1. The Conners' Rating Scales-Revised (CRS-R; Conners, 1997). The CRS-R was used to assess adolescent ADHD symptoms at 17 and 13 years of age. Mothers were asked to rate specific behaviors (e.g., "difficulty doing or completing homework") exhibited by their adolescent in the past month, on a scale ranging from 0 (*the behavior rarely or never occurs*) to 3 (*the behavior occurs very often*). Cronbach's alphas for the total ADHD subscale was .88 at age 13 years and .90 at age 17 years.

2.2.1.2. The strengths and difficulties questionnaire (SDQ; Goodman, 1997). The self-report version of this questionnaire was completed by the adolescents during a home visit at 13 years. The SDQ contains 25 items (e.g., "I get very angry"), with each item being scored on a 3-point scale of 0 = not true, 1 = somewhat true, and 2 = certainly true. The items comprised five different subscales of five items each, measuring: emotional problems, conduct problems, hyperactivity-inattention problems, peer problems, and prosocial behavior. We used the hyperactivity-inattention subscale, which had a Cronbach alpha of .70.

2.2.2. Variability assessment

2.2.2.1. A visual sensory task – VST. During a lab visit at the age of 17 years, participants completed the VST (Gonen-Yaacovi et al., 2016) while EEG was continuously recorded. The stimulus of interest in this task was a circular, doughnut-shaped checkerboard with an inner radius of 6° and an outer radius of 3.7°, which participants did not respond to. At the fixation point, an unrelated infrequent brightness-detection task was presented, which was intended to divert participants' attention away from the examined sensory stimuli (i.e., the checkerboard) and to ensure that participants were attentive and engaging in the task. Participants were instructed to press a key whenever the black fixation cross changed its brightness to gray. The task included a total of 300 trials; 200 trials contained the checkerboard stimulus and in 100 trials it was absent. In each trial, the stimuli were presented for 50 msec followed by a randomized intertrial window lasting 750–1,200 msec. The unrelated brightness-detection task was conducted at the fixation cross, presented in black at the center of the screen. In 60 random trials, there was a brightness change and the fixation appeared in gray (i.e., the fixation cross remained black in 240 trials). The brightness change lasted 30 msec, and participants had 1 sec to respond. Feedback for correct and incorrect responses was given by changing the fixation cross to green or red, respectively.

2.2.2.2. The stop signal task – SST. Participants completed a computerized SST, which was based on the paradigm of Logan (1994), while EEG was continuously recorded. This task consisted of a primary simple discrimination task, also referred to as a "go task". Go stimuli were the number "2" or letter "Z"

(50% each) inside a white square. Participants were instructed to press "1" on a numbered key on an S–R (serial response) box when the go stimulus was "Z" and "4" when the go stimulus was "2". They were asked to respond as quickly and as accurately as possible. A visual stop signal (a red square outline) appeared randomly after different delays of the go stimulus in 30% of the trials. The delay was set in each trial using a staircase dynamic-tracking procedure (Logan, 1994). In stop trials, participants were instructed not to respond. The task included a practice block (40 trials) and three blocks of 80 trials each. In total, there were 168 (70%) go trials and 72 (30%) stop trials. In the current study, we only analyzed responses to go trials; however, a more detailed description of the "stop task", as well as a behavioral analysis of all task measures, including the stop signal reaction time (SSRT), can be found in Einziger, Ben-Shachar, et al. (2021).

2.2.3. *Intelligence assessment*

2.2.3.1. The Raven's standard progressive matrices. The Raven's test (Raven, 1960) was used at age 13 years to estimate general intelligence. A series of 36 diagrams, divided into three sets (C, D, E), was used from the original version. Each diagram consisted of a black and white matrix with one missing part and eight response options. In each set, if the participant made three errors in a row, the set was terminated and the next set was administered. Intelligence scores were calculated by summing the number of correct answers. It should be noted that eight participants did not complete this test. This variable was used as a control variable in all analyses; therefore, the missing value for these eight participants was imputed as the mean of the whole sample to avoid a decrease in the sample size. All analyses were also conducted with the original scores (before the imputation of missing data) and these results are mentioned in the footnotes of the relevant tables.

Digital study materials are available at https://osf.io/xzqrv/. Legal copyright restrictions prevent public archiving of Raven's test and CRS-R which can be obtained from the copyright holders in the cited references.

2.2.4. *EEG analyses*

2.2.4.1. EEG data acquisition and pre-processing. EEG data were recorded during the VST and SST from 128 scalp sites using EGI HydrocCel Geodesic Sensor Net (HCGSN) and system (Electrical Geodesics, 2003). The electrode impedance level was kept at an acceptable level for this system (i.e., below 40 kΩ) (Ferree et al., 2001). During recording, all channels were referenced to the Cz electrode, the recording frequency band was constant at .01–100 Hz, and the sampling rate was 250 Hz. Because of technical problems with two of the recorded files, the data from two participants were unusable; therefore, these participants were excluded from all EEG analyses. EEG-data preprocessing was carried out using the EEGLAB toolbox (version 14) (Delorme & Makeig, 2004), operating in the MATLAB environment (version 2017b). Continuous EEG data were first high-pass filtered offline at .5 Hz and low-pass filtered at 40 Hz. Data were re-referenced to the common average reference. We did not conduct baseline correction, to avoid altering trial-by-trial variability in the pre-stimulus interval. In the VST, data were segmented from 200 msec before the stimulus to 500 msec after the stimulus. The segmented data were visually inspected for artifacts; trials containing large artifacts and bad channels were manually removed ($M = 16.1$, $SD = 11.09$ in the VST, and $M = 7.82$, $SD = 4.93$ in the SST). Additionally, any trial in which the subject responded (i.e., pressed the key) was removed to exclude motor and response processes interference. Next, we conducted an independent component analysis using EEGLAB's runica function. Components containing artifacts that could have been clearly identified (e.g., blinks, muscle twitches) were subtracted from the data. We used an automated bad-channel and artifact detection and replacement method (EEGLAB's TBT plugin) (Ben-Shachar, 2020); trials containing 15 or more bad channels were excluded ($M = 2.88$, $SD = 5.82$ in the VST, and $M = 6.42$, $SD = 9.46$ in the SST). Then, another visual inspection was conducted in the VST in which any trials and electrodes that still had substantial artifacts were removed completely ($M = 1.11$, $SD = 2.00$). Bad channels that were removed during the pre-processing stages were interpolated based on activity from neighboring channels. A total of 60 participants had high-quality data in the VST and their data were used in the EEG analysis. The mean number of trials containing the checkerboard stimulus after preprocessing was 145.31 ($SD = 13.39$, range = 111–165); and it was not related to participants' ADHD symptoms, $r = -.13$, $p = .30$.

EEG data acquisition and pre-processing were basically the same for the SST, with a few differences. Data from successful go trials were analyzed; these trials were originally segmented from 200 msec before the stimulus to 800 msec after the stimulus (a more detailed description of the preprocessing of the SST can be found in Einziger, Ben-Shachar, et al., 2021). In the current study, we only analyzed the pre-stimulus period (i.e., baseline) of successful go trials (i.e., 200 msec before stimulus until the presentation of the stimulus), and therefore did not exclude trials with motor responses (as was done in the VST). A total of 57 participants had high-quality data in the SST and their data was used for EEG analyses; three participants were excluded from the EEG analysis because of extremely low behavioral performances (e.g., the probability of response to the go signal was ~.5) and low-quality EEG data (for full details see Einziger, Ben-Shachar, et al., 2021). The mean number of trials after preprocessing was 141.62 ($SD = 17.92$, range = 79–161).

2.2.4.2. Electrodes selection and P100 identification. Although the primary analysis of this study focused on trial-by-trial neural variability, the P100 component was also calculated as a preliminary assessment. This was conducted to identify suitable electrodes for the main analysis and to ensure that the event-related potential (ERP) waveforms and topographic maps were consistent with what would be expected during the perception of a visual stimulus. In the VST, the P100 component was identified in each participant separately. For each electrode, we calculated the ERP in trials that contained the checkerboard stimulus. We followed the procedure reported in Gonen-Yaacovi et al. (2016) and used an automatic script to identify the six electrodes in which the maximal amplitude was recorded during the P100 time window. This time window was set to 70–110 msec after the presentation of the stimulus, based on the visual inspection of the grand average ERP waveforms (see Fig. 1, panel A). All six electrodes

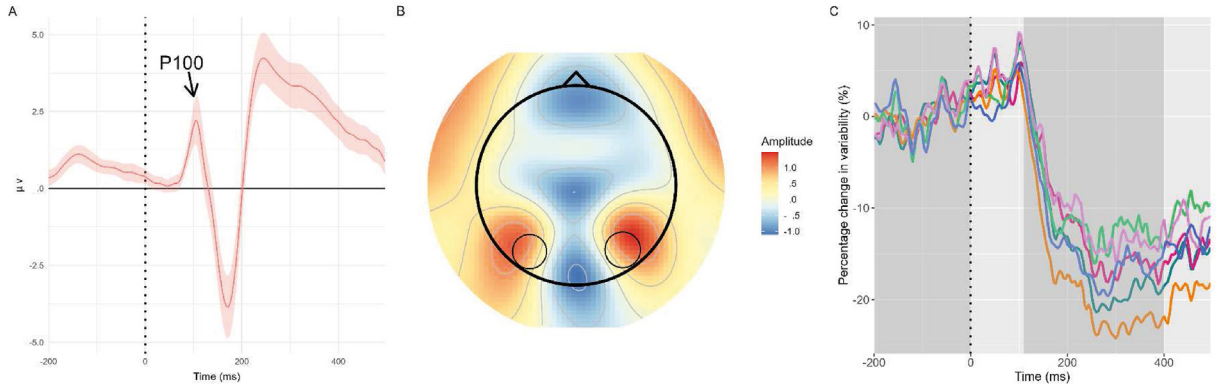

Fig. 1 – Grand average ERP waveforms from the VST, a topographic map at the P100 time window, and temporal dynamics of variability quenching across selected electrodes. *Note.* (A) Grand average ERP waveforms across subjects of trials that contained the checkerboard stimulus; the vertical dotted line indicates target-stimulus onset, and the shaded area represents 95% confidence interval. (B) A topographic map of voltages on the scalp at the P100 time window. The region of interest is marked with circles. (C) Variability quenching over time and across subjects is presented for each of the six selected electrodes (each color represents a single electrode). The shaded areas represent the pre-stimulus period and the selected time window for the variability quenching calculation; as seen at 110 msec after the presentation of the stimulus, there is a noticeable decrease in variability in all electrodes; then, there is a trend of variability increase at 400 msec. ERP = Event-related Potential; VST = Visual Sensory Task.

were in bilateral occipital areas (as marked with a circle on the topographic map in Fig. 1, panel B). Data from these electrodes were used in further analyses. The same procedure was followed in the SST; based on the grand ERP, the P100 time window was set to 100–200 msec after the presentation of the stimulus (see Fig. 2).

The grand ERP across participants exhibited a clear positive peak at approximately 100 msec after the stimulus, in both the VST and the SST (see Figs. 1 and 2, respectively). Consistent with Gonen-Yaacovi et al. (2016), the mean amplitude and latency of the P100 were not significantly correlated with ADHD symptoms at 17 and 13 years, with all r's < −.20, and all *p*'s > .11.

2.2.4.3. Trial-by-trial variability. In the VST, trial-by-trial neural variability was calculated at two time windows. The first window was the pre-stimulus period, from −200 msec before the presentation of the stimulus to the stimulus onset; the second window was the post-stimulus period from 110 msec to 400 msec after the presentation of the stimulus. The post-stimulus time window was selected based on the visual inspection of the timing in which variability seemed to

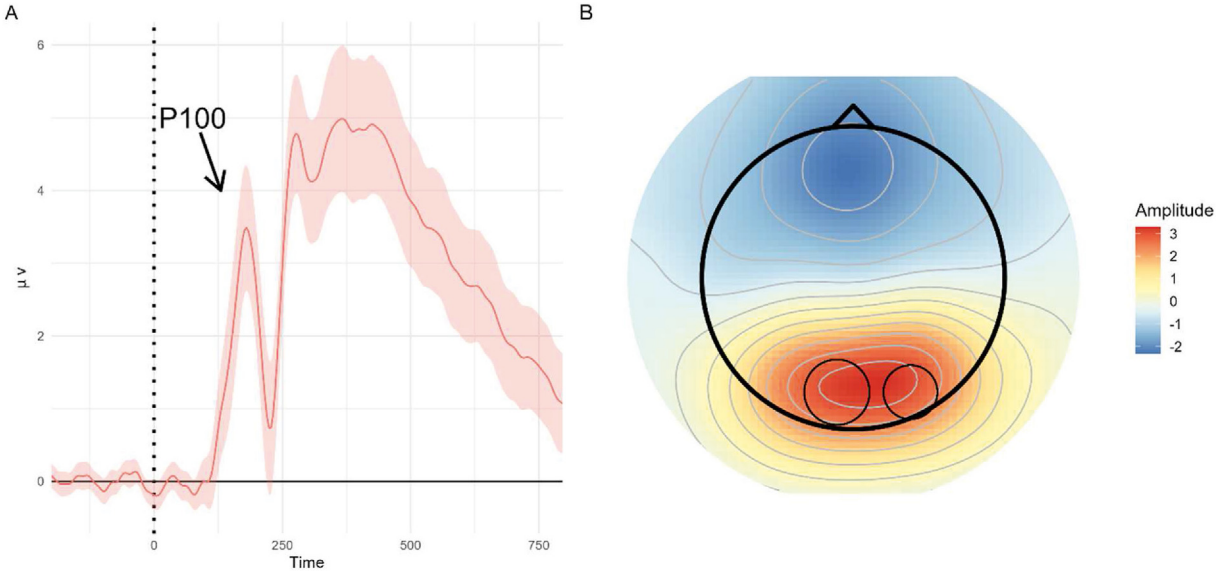

Fig. 2 – Grand average ERP waveforms from the SST and a topographic map at the P100 time window. *Note.* (A) Grand average ERP waveforms across subjects of go stimuli; the vertical dotted line indicates stimulus onset, and the shaded area represents 95% confidence interval. (B) A topographic map of voltages on the scalp at the P100 time window. The region of interest is marked with circles. ERP = Event-related Potential; SST = Stop Signal Task.

trend towards the baseline brain activity in all electrodes of interest (see Fig. 1, panel C), and following the findings of Gonen-Yaacovi et al. (2016) and Arazi et al. (2019). Trial-by-trial amplitude variability was computed for each of the six electrodes by calculating the variance across trials at each time point within the time window; then it was averaged across electrodes and time points to produce a single variability measure for each time window (i.e., pre-stimulus and post-stimulus).

Then, we calculated variability quenching based on the method reported in Arazi, Censor, et al. (2017), as follows: variability quenching $= \left(\frac{Var_{post}}{Var_{pre}} - 1\right)*100$. For each participant, continuous trial-by-trial variability in the post-stimulus time window was divided by the mean pre-stimulus variability value. From this division ratio, we subtracted 1 and then multiplied the result by 100, to transform the variability units into the percentage of change in variability units. At the end of this calculation, each participant had one value that represented their mean amount of change in variability from the pre-stimulus period to the post-stimulus period; the numeric value of variability quenching stands for the percentage of change in variability after perceiving the stimulus. Negative values indicate a decline in variability compared to the pre-stimulus period, and positive values indicate an increase in variability compared to the pre-stimulus period. Finally, each participant had three neural variability measures: pre-stimulus variability, post-stimulus variability, and variability quenching.

In the SST, the trial-by-trial amplitude variability of each participant was calculated across electrodes only for the pre-stimulus period. This was done because the SST was not originally designed to calculate neural variability across time; it has different characteristics than the VST (which was specifically designed for this purpose). For example, in the VST, participants perceived an unattended visual stimulus in the visual periphery, and trials in which there was a motor response were omitted; in the SST, participants were instructed to respond to a "go" stimulus and to withhold their response when the stop signal appeared. Because there are other cognitive processes involved in this task, we only used the pre-stimulus period, in which no stimulus was presented and no motor response was performed.

2.2.5. *Behavioral analyses*

For the behavioral analysis in both the VST and SST, we calculated the mean reaction time (RT), the standard deviation (SD) of RT, and the coefficient of variation (CV), which is calculated as the SD of RT divided by the mean RT. We removed trials with RT shorter than 100 msec. Error and post-error trials were not analyzed, as well as the first two trials in each block and the practice block.

2.3. *Data analysis plan*

The first aim was to examine the concurrent and longitudinal relation between ADHD symptoms and the different components of neural variability. This was examined with a series of regression models that were constructed hierarchically for predicting each component of variability; control variables were entered in step 1 and ADHD symptoms were entered in step 2. The second aim was to test the correlation between behavioral variability and neural variability. This was examined using partial correlation, controlling for relevant background variables. The third aim was to examine the unique contributions of both behavioral and neural variability to ADHD. This was examined with a regression model that was constructed hierarchically for the prediction of ADHD symptoms; control variables were entered in step 1, behavioral measures were entered in step 2, and neural measures were entered in step 3. Based on our unidirectional hypotheses, we used one-tailed significance tests for regression coefficients. We calculated the statistical power using G*Power version 3.1.9.7 (Faul et al., 2009). With a sample size of 62, 3–4 predictors, and an alpha of .05, we found that the statistical power for detecting medium effects was 64%–69%.

2.4. *Sample size justification, preregistration, and inclusion/exclusion*

Note that no part of the study procedures or analyses was preregistered prior to the research being conducted. We report how we determined our sample size, all data exclusions, all inclusion/exclusion criteria, whether inclusion/exclusion criteria were established prior to data analysis, all manipulations, and all measures in the study.

2.5. *Data, study materials, and analysis code availability*

Data and analysis code are available at https://osf.io/xzqrv/

3. Results

3.1. *Preliminary analyses*

Parental background variables (i.e., parents' age and years of education at birth, and aspects of socioeconomic status at the adolescent's assessment) did not correlate with study variables. Among the adolescent background variables (i.e., age at assessment, IQ scores measured at 13 years of age), IQ was positively correlated with neural variability measures ($r = .26$, $p < .05$ and $r = .24$, $p < .10$, for the pre-stimulus and post-stimulus variability in the VST, respectively, and $r = .32$, $p < .05$ for pre-stimulus variability in the SST). Also, the number of trials used for the variability calculations was correlated with pre-stimulus and post-stimulus variability in the VST ($r = -.39$, $p < .01$ and $r = -.37$, $p < .01$, respectively). Therefore, IQ and the number of trials were controlled in all analyses.

At age 13 years, ADHD symptoms were rated both by mothers and self-reports. Their correlation was significant, $r = .24$, $p < .05$; therefore, we created an aggregate score of ADHD symptoms at age 13 by averaging their standardized scores. We then examined whether the required assumptions for regression analyses were met. Levine's test for equality of variances was not significant for any of the regression models, and all Q–Q plots of residuals matched the linear pattern and supported the normality assumption. Descriptive statistics and intercorrelations among study variables are presented in Table 1.

Table 1 – Means, standard deviations, and correlations among study variables.

Variable	M	SD	1	2	3	4	5	6	7	8	9	10	11
VST measures													
1. Pre-stimulus variability	67.10	30.68											
2. Post-stimulus variability	55.68	19.68	.91***										
3. Variability quenching	−12.92	12.57	−.61***	−.26*									
4. Mean RT	493.86	54.61	.28*	.26*	−.17								
5. SD RT	108.39	31.39	.23	.26*	−.11	.74***							
6. CV	.22	.05	.17	.22^{+}	−.05	.46***	.93***						
SST measures													
7. Pre-stimulus variability	66.74	40.58	.74***	.65***	−.43***	.20	.25^{+}	.22					
8. Mean RT (Go trials)	785.29	332.52	.24^{+}	.20	−.10	.09	−.05	−.10	.27*				
9. SD RT	244.86	139.71	.11	.12	.04	.11	−.01	−.06	.09	.86***			
10. CV	.30	.09	−.07	−.02	.15	.12	.07	.03	−.15	.28*	.70***		
ADHD symptoms													
11. ADHD symptoms – 13y	.00	.79	.27*	.13	−.31*	.25^{+}	.11	.02	.19	−.01	.07	.11	
12. ADHD symptoms – 17y	56.52	11.01	.26*	.16	−.27*	.29*	.09	−.04	.22^{+}	.11	.08	.01	.59***

Note. *M* and *SD* are used to represent mean and standard deviation, respectively. $^{+}p < .10$, $^{*}p < .05$, $^{**}p < .01$, $^{***}p < .001$ (two-tailed test). VST = Visual Sensory Task; RT = Reaction Time; SD = Standard Deviation; CV = Coefficient of Variation; SST = Stop Signal Task; ADHD = Attention-Deficit Hyperactivity Disorder.

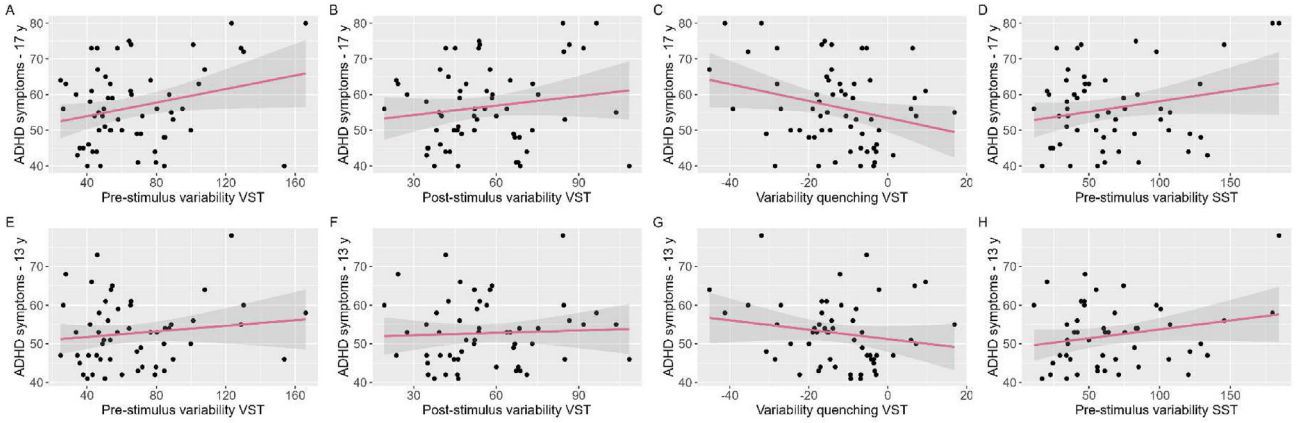

Fig. 3 – Scatter plots for the relations between neural trial-by-trial variability in the VST and SST and ADHD symptoms. ***Note.*** **Scatter plots of the correlations between pre-stimulus variability, post-stimulus variability, and variability quenching in the VST with ADHD symptoms at age 17 years (panels A–C, respectively) and 13 years (panels E–G, respectively); scatter plots of the correlations between pre-stimulus variability in the SST and ADHD symptoms at age 17 years (panel D) and 13 years (panel H). VST = Visual Sensory Task; SST = Stop Signal Task; ADHD = Attention-Deficit Hyperactivity Disorder.**

3.2. *Association of neural variability measures and ADHD symptoms*

The bivariate correlations between the different neural variability components at age 17 years and ADHD symptomatology at both 17 and 13 years are present in Table 1; the scatter plots of these correlations are present in Fig. 3. To test the relation between the different components of neural variability and ADHD symptoms, we conducted a set of regression models, constructed hierarchically, for the prediction of the different components of neural variability in both the VST and SST. The control variables of the number of trials and adolescent IQ were entered in step 1, and ADHD symptoms were entered in step 2.

3.2.1. *Predicting pre-stimulus neural variably in the VST*

As seen in Table 2, the control variables that were entered in step 1 (i.e., number of trials and IQ) made a significant contribution to the prediction. Concurrent ADHD symptoms at 17 years were entered in step 2 and significantly predicted pre-stimulus neural variability; higher levels of ADHD symptoms predicted greater neural variability in the pre-stimulus period. The entire model was significant, F (3, 56) = 9.15, $p < .001$.

As seen in Table 3, results showed a similar pattern when we tested the same model with ADHD symptoms at 13 years. The entire model was significant, F (3, 50) = 11.07, $p < .001$.

3.2.2. *Predicting post-stimulus neural variably in the VST*

As seen in Table 2, the control variables that were entered in step 1 (i.e., number of trials and IQ) made a significant contribution to the prediction. However, concurrent ADHD symptoms at 17 years were entered in step 2 and did *not* contribute to the prediction of post-stimulus neural variability. The entire model was significant, F (3, 56) = 6.55, $p < .001$.

Table 2 – Predicting neural variability in the VST from concurrent ADHD symptoms at 17 years.

Predictor	Pre-stimulus variability		Post-stimulus variability		Variability quenching	
	β	ΔR^2	β	ΔR^2	β	ΔR^2
Step 1		.29***		.25***		.06
Number of trials	−.49***		−.45***		.20+	
IQ	.38**		.35**		−.20+	
Step 2		.04+		.01		.06+
ADHD symptoms – 17 years	.19*		.09		−.24	
R^2 (Adjusted R^2)	.33 (.29)***		.26 (.22)***		.12 (.07)+	

Note. + $p < .10$, * $p < .05$, ** $p < .01$, *** $p < .001$; Results showed the same pattern (coefficients were modestly higher) when we used the original IQ score, before imputation of missing data. VST = Visual Sensory Task; ADHD = Attention-Deficit Hyperactivity Disorder.

Table 3 – Predicting neural variability in the VST from ADHD symptoms at 13 years.

Predictor	Pre-stimulus variability		Post-stimulus variability		Variability quenching	
	β	ΔR^2	β	ΔR^2	β	ΔR^2
Step 1		.36***		.30***		.09+
Number of trials	−.55***		−.51***		.26*	
IQ	.41**		.37**		−.23+	
Step 2		.04+		.00		.07*
ADHD symptoms – 13 years	.19*		.08		−.27	
R^2 (Adjusted R^2)	.40 (.36)***		.30 (.26) ***		.16 (.11)*	

Note. + $p < .10$, * $p < .05$, ** $p < .01$, *** $p < .001$; Results showed the same pattern (coefficients were modestly higher) when we used the original IQ score, before imputation of missing data. VST = Visual Sensory Task; ADHD = Attention-Deficit Hyperactivity Disorder.

As seen in Table 3, results showed a similar pattern when we tested the same model with ADHD symptoms at 13 years; ADHD symptoms did not predict post-stimulus variability. The entire model was significant, F (3, 50) = 7.34, $p < .001$.

The effect of higher neural variability in the pre-stimulus period, but not in the post-stimulus period, is demonstrated in Fig. 4. The figure presents the EEG traces from two example adolescents, one with relatively high concurrent ADHD symptoms, displaying high pre-stimulus variability, and one with low concurrent ADHD symptoms, displaying low pre-stimulus variability.

3.2.3. *Predicting neural variability quenching in the VST*

As seen in Table 2, the control variables that were entered in step 1 (i.e., number of trials and IQ) marginally contributed to the prediction of variability quenching. Concurrent ADHD symptoms at 17 years were entered in step 2 and showed a negative coefficient, which was opposite in direction to our hypothesis and nonsignificant.[2] Still, it had a marginally significant contribution to the prediction, F_{change} (1, 56) = 3.68, $p = .06$, and the entire model was also marginally significant, F (3, 56) = 2.51, $p = .06$.

As seen in Table 3, results showed a similar pattern when we tested the same model with ADHD symptoms at the age of 13 years,[3] F_{change} (1, 50) = 4.40, $p < .05$. The entire model was significant, F (3, 50) = 3.28, $p < .05$.

As noted, the direction of the relation between ADHD symptoms (at both 13 and 17 years) and variability quenching was not in line with our hypothesis. An illustration of the difference in variability quenching between adolescents rated with high or low symptomatology can be seen in Fig. 5 (for illustration purposes, groups were defined by the lower and upper quartile of the distribution of the symptoms at 17 years). As seen, adolescents with higher symptomatology had higher variability in the pre-stimulus period, compared to those with lower symptomatology, but there were no differences between those with high and low symptomatology in the post-stimulus period, and their variability quenching was higher. Therefore, the negative relation between variability quenching and ADHD suggests that the neural activity of individuals with high ADHD symptomatology was more variable before the presentation of the stimulus, and then decreased to a level similar to the level of variability in individuals with low symptomatology.

3.2.4. *Predicting pre-stimulus neural variably in the SST*

As seen in Table 4, among the control variables that were entered in step 1, only IQ had a unique contribution to the prediction. Concurrent ADHD symptoms at 17 years were entered in step 2 and made a significant contribution to the prediction of pre-stimulus variability; higher levels of ADHD symptoms predicted greater neural variability in the pre-stimulus period. The entire model was significant, F (3, 45) = 2.93, $p < .05$.

A similar pattern of results was found when we tested the same model with ADHD symptoms at 13 years, although the coefficient of ADHD was only marginally significant (see the right panel of Table 4). The entire model was significant, F (3, 45) = 3.98, $p < .05$.

[2] This coefficient was marginally significant in a two-tailed significance test.

[3] The coefficient of ADHD symptoms was significant in a two-tailed significance test.

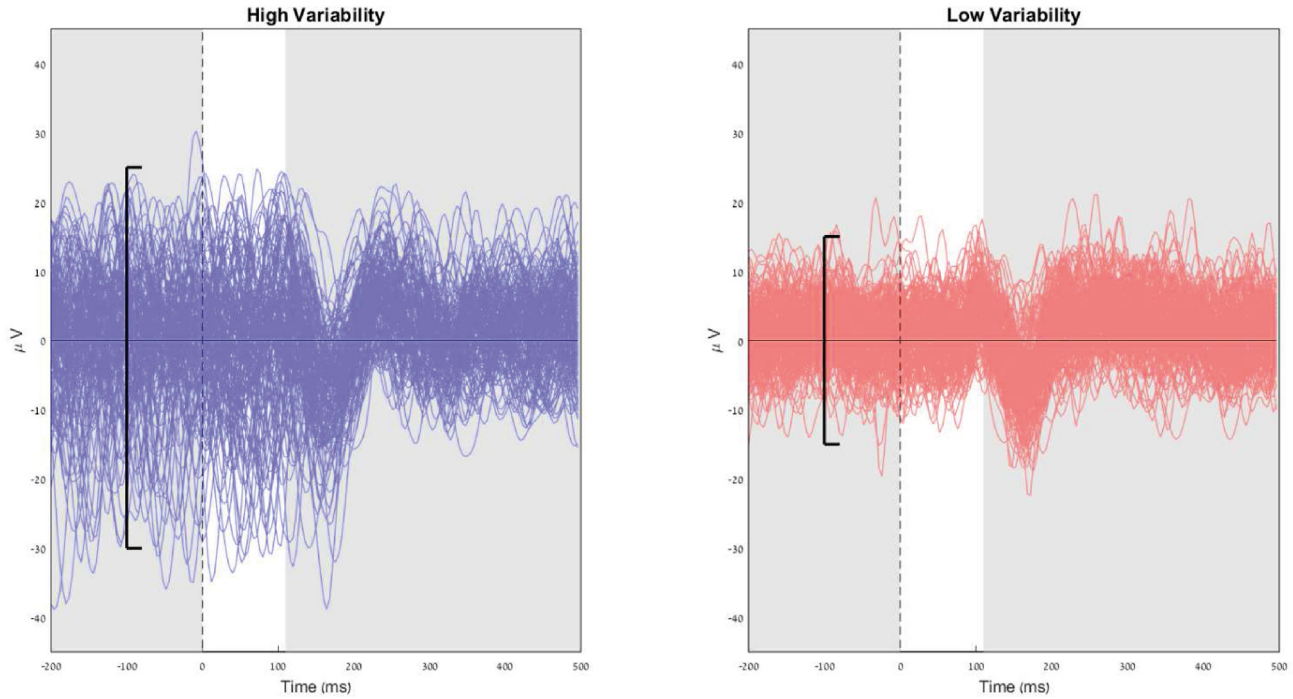

Fig. 4 – An illustration of high and low trial-by-trial neural variability. *Note.* Examples of trial-by-trial measured voltage by time, from two example participants with relatively high (left panel) or low (right panel) neural variability. In each plot, the left gray area represents the pre-stimulus time window and the right gray area represents the post-stimulus time window. The dashed line represents the stimulus onset.

Despite some differences in the overall effect size of the separate models for VST and SST, the magnitude of relationships between pre-stimulus neural variability and ADHD symptoms was rather similar in both tasks. In other words, the same pattern of results found for the VST was replicated with the SST, showing the relation between increased pre-stimulus neural variability and ADHD symptomatology in adolescents. Moreover, the partial correlation between pre-stimulus variability in both tasks, after controlling for IQ and the number of trials in both tasks, was positive and high in magnitude, $r = .71$, $p < .001$, which validated the idea that these individual differences in pre-stimulus neural variability are stable across different cognitive tasks.

3.3. *Association of behavioral measures, neural variability, and ADHD symptoms*

Partial correlations between behavioral measures (i.e., mean RT, SD RT, and CV), neural variability, and ADHD symptoms were calculated, controlling for IQ and the number of trials in the relevant task.

There were no significant correlations between behavioral variability and the different types of neural variability, in both the VST and the SST. However, mean RT for the VST was marginally correlated with pre-stimulus variability in the same task, $r = .20$, $p < .10$; and mean RT of the go stimulus for the SST was marginally correlated with the pre-stimulus variability in the same task, $r = .22$, $p < .10$.

Moreover, a significant correlation was found between mean RT for the VST and concurrent ADHD symptoms, $r = .27$, $p < .05$. The other behavioral measures (i.e., mean RT for the SST, SD RT, and CV for the VST and the SST) were not correlated with ADHD symptoms.

3.3.1. *Predicting ADHD symptoms from behavioral and neural measures*

We then examined the combined contribution of behavioral and neural measures from the VST and the SST to the prediction of ADHD symptoms at 17 years. To aggregate the effects of the different measures from both tasks into a single model, we calculated composite variables by averaging the standardized scores of equivalent variables of the VST and SST (i.e., mean RT and pre-stimulus neural variability). We then computed a regression model, constructed hierarchically, for the prediction of ADHD symptoms at age 17. Results are presented in Table 5. Overall, the control variables entered in step 1 did not contribute to the prediction of ADHD. Mean RT was entered in step 2 and had a significant contribution to the prediction of ADHD symptoms. Pre-stimulus variability was entered in step 3 (model 1) and made a significant unique contribution to the prediction, above and beyond the control variables and mean RT. The entire model was significant and explained 22% of the variance of ADHD symptoms (adjusted $R^2 = .14$), F (5, 51) = 2.77, $p < .05$.

3.3.2. *Additional analyses*

To further explore the contribution of variability quenching beyond our hypotheses, and to compare its predictive value with pre-stimulus variability, we constructed a separate regression model that included variability quenching as the predictor in step 3, instead of pre-stimulus variability. Steps 1 and 2 were similar in both models. Results showed that

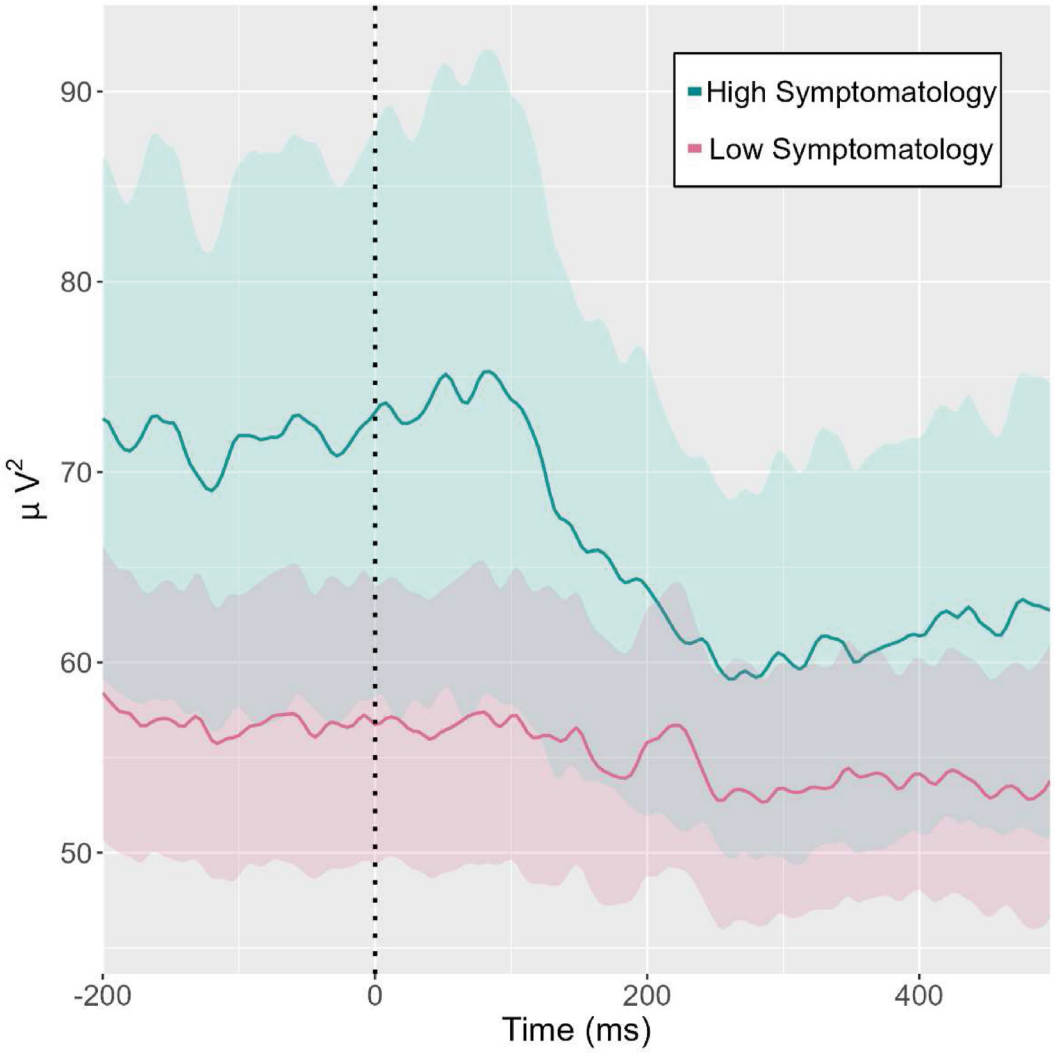

Fig. 5 – An illustration of variability quenching among adolescents with high and low ADHD symptomatology. ***Note.*** **Temporal dynamics of trial-by-trial amplitude variability over time of adolescents with high and low ADHD symptomatology (for illustration purposes, group is defined by the lower and upper quartile of the distribution of the symptoms at 17 years); the shaded area represents 95% confidence interval.**

Table 4 – Predicting pre-stimulus variability in the SST from ADHD symptoms at 17 and 13 years.

Predictor	Pre-stimulus variability (SST)		Predictor	Pre-stimulus variability (SST)	
	β	ΔR^2		β	ΔR^2
Step 1		.10+	**Step 1**		.17*
Number of trials	.00		Number of trials	−.05	
IQ	.32*		IQ	.41**	
Step 2		.06+	**Step 2**		.04
ADHD symptoms – 17 years	.25*		ADHD symptoms – 13 years	.22+	
R^2 (Adjusted R^2)	.16 (.11)*		R^2 (Adjusted R^2)	.21 (.16)*	

Note. + $p < .10$, * $p < .05$, ** $p < .01$, *** $p < .001$; Results showed the same pattern (coefficients were modestly higher) when we used the original IQ score, before the imputation of missing data. SST = Stop Signal Task; ADHD = Attention-Deficit Hyperactivity Disorder.

variability quenching significantly contributed to the prediction of ADHD symptoms above and beyond the control variables and mean RT. The overall model was significant, accounting for 22% of the variance in ADHD symptoms (adjusted $R^2 = .13$), F (5, 51) = 2.56, $p < .05$. To compare the model fit of both models, we used the Bayesian Information Criterion (BIC). The BIC measures the goodness of fit of the models, with lower values indicating a better fit. The results showed that model 2, which included variability quenching, had an equivalent fit (BIC = 156) to model 1, which included

Table 5 – Predicting ADHD symptoms in adolescence from behavioral and neural measures of both tasks.

Predictor	ADHD symptoms – 17 years	
	β	ΔR^2
Step 1		.08
Number of trials - VST	−.07	
Number of trials -SST	−.25*	
IQ	.00	
Step 2		.08+
Combined mean RT	.28*	
Step 3: Model 1		.08*
Combined pre-stimulus variability	.35*	
Step 3: Model 2	34	.07*
Variability quenching	−.30*	

Note. + $p < .10$, * $p < .05$, ** $p < .01$, *** $p < .001$; we used the combined scores of behavioral and neural measures from the VST and the SST, by averaging the standardized scores of equivalent variables. Results showed the same pattern when we entered the SD RT or SSRT at step 2, and when we used the original IQ score, before imputation of missing data, and coefficients were modestly higher. VST = Visual Sensory Task; RT = Reaction Time; SST = Stop Signal Task; ADHD = Attention-Deficit Hyperactivity Disorder.

pre-stimulus variability (BIC = 155). This suggested that variability quenching was not a better nor a worse predictor of ADHD symptoms compared to pre-stimulus variability.

4. Discussion

This study examined the relations between neural variability, behavioral variability, and ADHD. Overall, our results revealed that ADHD symptoms were related to larger neural variability before the presentation of the stimulus, but not afterward, and to higher variability quenching. Regarding the behavioral measures, mean RT, but not variability of RT, was related to both pre-stimulus neural variability and ADHD symptoms. Among the behavioral and neural measures, mean RT and pre-stimulus neural variability were predictive of ADHD symptomatology, above and beyond intelligence. These findings and their interpretations are discussed in detail.

Our main aim was to test the relations between ADHD and different types of neural variability produced in response to a visual stimulus. We found that higher symptomatology of ADHD was related to higher pre-stimulus neural variability; this was found in two different cognitive tasks (i.e., the VST and the SST), with symptoms that were measured at two separated assessments at different time points (i.e., 13 and 17 years), and using both mother and self-reports. However, we did not find evidence to support the relation between post-stimulus variability and ADHD symptoms. This is only partially consistent with Gonen-Yaacovi et al.'s (2016) findings, which found increased neural variability among individuals with ADHD in the pre-stimulus interval as well as in the post-stimulus interval, and even in trials without a stimulus. Gonen-Yaacovi et al. interpreted their findings as increased general moment-to-moment neural fluctuations, not necessarily associated with a specific cognitive process.

However, our findings of variability that appears only in the pre-stimulus interval seem to be less consistent with such interpretation, and more consistent with the idea of variability in alertness and preparatory processes being task- and stimulus-related. In other words, the increased neural variability among adolescents with high symptomatology of ADHD could reflect a mixture of ongoing neural spontaneous fluctuations as well as a difficulty in alertness to the forthcoming stimulus. In this sense, higher pre-stimulus variability in those with higher symptoms is consistent with findings showing that individuals with ADHD show a diminished amplitude of the ERP component of contingent negative variation (CNV), which reflects the anticipation of the forthcoming stimulus (Hasler et al., 2016; Kaiser et al., 2020), and alertness deficit that has been found among individuals with ADHD (Abramov et al., 2019; Johnson et al., 2008; Samyn et al., 2017).

Still, it should be noticed that the lack of the relation between post-stimulus variability and ADHD symptoms might also arise from differences in sample characteristics; the sample of Gonen-Yaacovi et al. (2016) was a clinical adult sample, while our sample was a non-clinical sample of adolescents. It could be possible that this type of variability in low-order sensory processes is a less sensitive measure for detecting individual differences in a sample of individuals with varying levels of symptomatology (compared to more prominent differences that can be found when examining a clinical sample compared to controls). Moreover, the relation between neural variability and ADHD symptoms might vary depending on the age of the participants; adults with ADHD might show general moment-to-moment variability, while adolescents might show such variability only when preparing for a stimulus. These differences should be investigated in more detail in future studies.

The lack of this relation between post-stimulus variability and ADHD is also important for the interpretation of the effect of variability quenching. The direction of the relation between variability quenching and ADHD symptoms was opposite to our hypothesis. Based on the findings of Gonen-Yaacovi et al. (2016) regarding increased variability before and after the stimulus, alongside findings that showed that larger variability quenching was related to better perceptual (Arazi, Censor, et al., 2017; Daniel & Dinstein, 2021) and attentional performance (Arazi et al., 2019), we expected to find smaller quenching among individuals with high symptomatology. However, adolescents with higher ADHD symptoms showed larger pre-stimulus variability accompanied by larger variability quenching. This outcome was due to high variability in the pre-stimulus period, rather than the post-stimulus period, resulting in a high difference between pre- and post-variability and strong quenching. Importantly, variability quenching and pre-stimulus variability had a comparable contribution to the prediction of ADHD symptoms, indicating that the higher quenching observed among those with high symptomatology was primarily driven by their higher initial level of variability.

To interpret these findings, it should first be considered that ADHD is a very heterogenic disorder, both in the level of symptoms and cognitive deficits (American Psychiatric Association, 2013; Kofler et al., 2013). Not all individuals with ADHD exhibit all cognitive deficits that are involved in this disorder (Doyle et al., 2005). There is consistent evidence to

support deficits in executive functions, at least among a subgroup of individuals with ADHD (Gau & Shang, 2010; Lin & Gau, 2019; Willcutt et al., 2005), including deficits in executive aspects of attention (Berger & Posner, 2000; Mullane et al., 2011). However, low-order sensory deficits were less examined among individuals with ADHD, and therefore, the relation between aspects of visual perception and ADHD remained relatively unclear. A meta-analysis of behavioral studies that used the continuous performance test (CPT) in those with ADHD indicated that children with ADHD showed decreased perceptual sensitivity (i.e., had difficulties distinguishing targets from nontargets, as reflected by lower d') (Huang-Pollock et al., 2012). In contrast, other studies have indicated that there were no differences in visual perception between those with ADHD and controls (e.g., as reflected in the P100 amplitude and latency; Gonen-Yaacovi et al., 2016), or that individuals with ADHD had superior performance in perceptual tasks (Sani et al., 2019). Furthermore, in a study that examined ADHD children with and without comorbid sensory processing disorder, lower visual perception performances were found among those with a comorbid sensory processing disorder (Jung et al., 2014). Therefore, it is plausible that difficulties in visual sensory processes are apparent only in a subgroup of individuals with ADHD.

In our study, and consistent with Gonen-Yaacovi et al.'s (2016) study, we did not find a significant correlation between the amplitude and the latency of the P100 and ADHD symptomatology. Therefore, it is plausible that our sample did not exhibit impairments in visual perception, however, we do not have any specific measurement to confirm this possibility. It could be that the higher level of neural variability before the stimulus, alongside the higher extent of variability quenching among those with high symptomatology, reflects an adaptive compensation mechanism that enables a "normative" visual processing, despite the initial higher levels of variability. However, this interpretation should be viewed with caution; future research is necessary to corroborate this finding and to test the potential role of variability quenching to enable such "normative" processing. Future studies should also include a direct assessment of perceptual and attentional processes, which would enable a stronger evaluation of the performance of individuals with varying levels of ADHD symptomatology.

Among the behavioral measures, mean RT was related in our study to pre-stimulus neural variability in both tasks; specifically, slower RTs were related to higher levels of pre-stimulus variability. However, such a correlation was not found for the variability of RT (i.e., SD RT and CV). It should be noticed that the lack of the correlation between neural and behavioral variability was in line with the research of Gonen-Yaacovi et al. (2016). In contrast, in the studies of both McLoughlin et al. (2014) and Saville et al. (2015), neural variability related to higher-order cognitive processes was indeed related to behavioral RT variability. Therefore, it could be that neural variability in high-order cognitive processes, but not in low-order sensory processes, might be accountable for the inconsistent behavior reflected in RT variability. Alternatively, the high heterogeneity in ADHD suggests that different individuals might have different loci of dysfunction leading to their behavioral variability. The examination of such a possibility requires an examination of different types of low-order and high-order neural variability within the same sample; we are currently investigating this within our longitudinal study.

Similarly, mean RT, but not the variability of RT, was related to ADHD symptomatology. Specifically, consistent with the literature, slower RT's were related to higher ADHD symptoms (e.g., see a meta-analysis of Huang-Pollock et al., 2012). However, behavioral variability in both the SST and the VST, measured with SD RT and CV, was not related to the level of ADHD symptoms, and this was inconsistent with the literature (Klein et al., 2006; Kofler et al., 2013; Salunkhe et al., 2021; Tamm et al., 2012). It should be noted that we previously found within the same sample that behavioral RT variability (i.e., measured with the SD RT in both the SST and the CPT) was concurrently related to ADHD symptomatology at 7 years of age. However, this relation was calculated based on a higher sample size (~100) and was modest in magnitude (Einziger, Zilberman-Hayun, et al., 2021); therefore, it could be that the current study was not adequately powered to detect such a modest effect in a smaller sample size (~60).

Although it was not the focus of our study, we found that higher pre-stimulus variability was positively related to intelligence. This finding is surprising because previous studies have shown that higher general intelligence was associated with more stable performance in simple cognitive tasks, as reflected by lower RT variability (Doebler & Scheffler, 2016; Schulz-Zhecheva et al., 2023). However, the relationship between neural variability and aspects of intelligence (as well as certain cognitive performance) has not been well established, with some studies indicating a negative relationship between the two (Arazi et al., 2019; Schurger et al., 2010; Xue et al., 2010) and others indicating a positive one (Garrett et al., 2010, 2013; McIntosh et al., 2008; Saxe et al., 2018). As these studies have utilized different approaches and methodologies, further investigation is required to establish this relation.

5. Limitations

Our study should be considered in light of several limitations. First, our sample participants were only males, which limited the generalization of the results. This was decided at the beginning of the prospective longitudinal study, based on the higher prevalence of ADHD among males (American Psychiatric Association, 2000), to increase the probability of having a sufficient number of participants displaying symptoms of ADHD. Moreover, our sample size was modest and therefore the statistical power was limited. In addition, we did not have a direct and more extensive assessment of perceptual performance; we carefully concluded that there were no differences among individuals with different levels of ADHD symptoms based on the P100 amplitude and latency. However, including another perceptual task would enable us to examine it more thoroughly. Finally, in this study we investigated the concurrent relationship between neural variability and ADHD symptomatology. To strengthen the validity of our results, we also examined this association using an earlier assessment of ADHD symptomatology. The results indicate that both self and mother's reports of symptoms at 13 years of age were predictive of pre-stimulus variability at 17 years of age. However, since we did not have a later assessment of

symptoms, we were unable to examine the ability of different types of neural variability to predict later symptomatology.

6. Conclusions

To conclude, our findings suggest that adolescents with high ADHD symptoms display increased neural variability *before* the presentation of a stimulus, which is consistent across different tasks, and could reflect a difficulty in alertness to the forthcoming stimulus. However, when presented with a visual stimulus, such increased variability decreases to a level that is similar to those with low ADHD symptoms. Our study extends previous research by directly examining brain-behavior associations in a non-clinical sample of adolescents displaying varying levels of ADHD symptoms. Future studies are required to fully understand the mechanism that underlies the larger variability quenching among those with high ADHD symptoms, and the predictive value of different types of neural variability to ADHD symptomatology over time.

Open Practices

The study in this article earned Open Data badge for transparent practices. The Data and analysis code used in this study are available at: https://osf.io/xzqrv/.

Author contribution

Tzlil Einziger: Conceptualization, methodology, validation, formal analysis, investigation, writing - original draft, project administration. **Tali Devor:** Formal analysis, visualization, software. **Mattan S. Ben-Shachar:** Software, data curation, writing - review & editing. **Ayelet Arazi:** Software, writing - review & editing. **Ilan Dinstein:** Methodology, writing - review & editing. **Christoph Klein:** Writing - review & editing. **Judith G. Auerbach:** Data curation, writing - review & editing. **Andrea Berger:** Conceptualization, methodology, supervision, resources, funding acquisition, writing - review & editing.

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Declaration of competing interest

The authors declare that they have no conflict of interest.

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