Deficits in inhibitory force control in young adults with ADHD

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Abstract

Poor inhibitory control is a well-established cognitive correlate of adults with ADHD. However, the simple reaction time (RT) task used in a majority of studies records performance errors only via the presence or absence of a single key press. This all-or-nothing response makes it impossible to capture subtle differences in underlying processes that shape performance. Subsequently, all-or-nothing tasks may underestimate the prevalence of executive function deficits in ADHD. The current study measured inhibitory control using a standard Go/No-Go RT task and a more sensitive continuous grip force task among adults with (N = 51, 22 female) and without (N = 51, 29 female) ADHD. Compared to adults without ADHD, adults with ADHD made more failed inhibits in the classic Go/No-Go paradigm and produced greater and more variable force during motor inhibition. The amount of force produced on failed inhibits was a stronger predictor of ADHD-related symptoms than the number of commissions in the standard RT task. Adults with ADHD did not differ from those without ADHD on the mean force and variability of force produced in Go trials. These findings suggest that the use of a precise and continuous motor task, such as the force task used here, provides additional information about the nature of inhibitory motor control in adults with ADHD.

Keywords

grip force; motor control; attention-deficit/hyperactivity disorder (ADHD); response inhibition; go/no-go task

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I. INTRODUCTION

Attention deficit hyperactivity disorder (ADHD) is a common childhood-onset disorder characterized by age-inappropriate, chronic, pervasive, and impairing levels of inattention and/or hyperactivity-impulsivity (American Psychiatric Association. and American Psychiatric Association. DSM-5 Task Force., 2013). ADHD persists into adulthood in up to 65% of cases (Faraone et al., 2006; Simon et al., 2009; Turgay et al., 2012), affects the ability to gain and maintain employment (Kessler et al., 2009; Kupper et al., 2012), and is associated with an increased risk for substance abuse (Wilens et al., 1995; Upadhyaya, 2008; Groenman et al., 2013), obesity (Cortese et al., 2008; Nazar et al., 2012; Albayrak et al., 2013; Nazar et al., 2014), workplace injuries (Swensen et al., 2004; Breslin and Pole, 2009; Hodgkins et al., 2011), and traffic accidents (Barkley et al., 1993; Jerome et al., 2006a, b; Barkley and Cox, 2007; Merkel et al., 2013). Though less often discussed, motor impairments are prominent among children with ADHD (Barkley, 1998) and up to 50% of pediatric ADHD patients are also comorbid for developmental coordination disorder (Kadesjo and Gillberg, 1999; Pitcher et al., 2003; Gillberg et al., 2004). Similarly, adults with ADHD have impaired visuomotor memory in gripping tasks (Neely et al., 2016), visuomotor adaptation in reaching tasks (Kurdziel et al., 2015), deficits in oculomotor control (Feifel et al., 2004; Carr et al., 2006), increased postural sway (Hove et al., 2015), and impaired timing in finger tapping tasks (Valera et al., 2010). These findings are important because motor processes have clearer neural correlates than many of the cognitive constructs associated with ADHD. Thus, the motor system provides a good avenue to examine the neurobiology of ADHD.

Inhibitory control is the process of suppressing competing responses to select the most appropriate response. The ability to suppress inappropriate behaviors in favor of appropriate alternatives is paramount to adapting behavior in changing circumstances and is thereby a critical component for controlling behavior at all levels, including movement. Although numerous studies report poor inhibitory control in ADHD (Nigg et al., 2002; Aron and Poldrack, 2005; Alderson et al., 2007; Wodka et al., 2007; Suskauer et al., 2008; Gilbert et al., 2011; Bari and Robbins, 2013), the type of task used in the majority of studies (e.g. go-no-go or stop signal reaction time, RT, task) records performance via the presence or absence of an all-or-nothing key press. Such an approach confounds cognitive, sensory, and motor processes into a single dichotomous response. As a result, we may be overlooking critical processes that provide insight into the neural mechanisms of the disorder. For example, a great deal of motor activity can be produced even when an individual does not ultimately press a key in a standard RT task. The current study overcomes this barrier by using a continuous and precise measure of motor output in a grip force variant of the classic go/no-go task. We used force output as a measure of activity in the motor system. In order to test the validity of this measure, participants completed a continuous grip force go/no-go task with both low and high force amplitude conditions as well as a standard go/no-go task that used an all-or-nothing keypress. Trials were presented rapidly, creating a prepotency to respond. In this context, the inhibition of a prepotent response requires effortful cognitive control, whereas allowing the motor response to proceed occurs in a more automatic fashion (Bargh et al., 1996; Muraven and Baumeister, 2000). We reasoned that greater activity in the

motor system would be reflected by the production of larger forces during no-go trials. We included low and high force amplitude conditions as a means to examine response planning. In particular, the amount of force produced on a no-go trial may reflect a pre-planned response and scale to the target amplitude. Therefore, deficits in response planning would be indicated by force output (on no-go trials) that does not scale to the target amplitude.

2. METHODS

2.1. Participants

We recruited young adults, ages 18 to 25, who identified as currently having ADHD or as having never been diagnosed with ADHD. Participants were community recruited through advertisements in State College, Pennsylvania. Exclusion criteria included: (1) previous concussions that resulted in a loss of consciousness for more than 10 minutes; (2) previous diagnosis of seizures, epilepsy, encephalitis, meningitis or an autism spectrum disorder; (3) previous diagnosis of a musculoskeletal or neurological disorder; and (4) previous diagnosis of any disorder involving psychosis.

2.1.1. Adults with ADHD—Adults with ADHD met DSM-V criteria including cross situational severity and impairment as determined by a semi-structured interview, the Conners' Adult ADHD Diagnostic Interview (CAADID; Multi-Health Systems Inc.). Adults had > 5 symptoms of inattention or hyperactivity, that were impairming in at least two settings (e.g. family and work). In total, 53 young adults met the criteria for ADHD. However, two participants did not complete the go/no-go task as instructed. These individuals were unable to keep pace with the speeded trial presentation in the motor task. The final ADHD group (N = 51, 29 females) had a mean age of 21.10 + 1.71 years. Adults taking a psychostimulant (N=22) completed the laboratory session after a 24-hour washout period. No participants were taking medications known to affect motor control at the time of testing, including antipsychotics, stimulants, or anticonvulsants (Reilly et al., 2008).

2.1.2. Adults without ADHD—Age- and sex- matched controls reported < 3 total symptoms and < 2 symptoms per ADHD dimension. Self-report of anxiety and/or depression was not exclusionary. A total of 73 young adults met the criteria for healthy control. The included participants (N=51, 22 females, 21.00 + 1.70 years) were chosen as age- and sex-matched controls for the ADHD group.

2.2. Procedures

The experimental task was completed as part of a larger battery of experimental and standardized measures that took place in one 3-hour session. After a complete description of the study, written informed consent was obtained from the participant. All procedures were approved by the Institutional Review Board at The Pennsylvania State University, and were consistent with the Declaration of Helsinki. All participants received monetary compensation for their participation in the study.

In advance of the laboratory session, all participants completed a brief medical history, the long form of the Connors Adult ADHD Rating Scales (CAARS), the Achenbach Adult Self

Report (ASR) (Achenbach, 2003), and the Edinburgh Handedness Inventory (Newcombe et al., 1971). Symptoms of ADHD were assessed with the self-report, long version (S:L) of the CAARS-S:L, which has 66 items and 8 factor-derived subscales: Inattention/Memory Problems, Hyperactivity/Restlessness, Impulsivity/Emotional Lability, Problems with Self-Concept, DSM-IV Inattentive Symptoms, DSM-IV Hyperactive/Impulsive Symptoms, DSM-IV ADHD Symptoms Total, and an ADHD Index. Symptoms of conditions that commonly co-occur with ADHD (e.g. anxiety, depression, and conduct problems) were evaluated with the ASR, a 123-item rating scale scored with a 3-point Lickert scale. The ASR has excellent psychometric properties and uses age-based normative data to identify normal, borderline clinical, and clinical ranges of behavior (Achenbach, 2003). Handedness was assessed with the Edinburgh Handedness Inventory (Muth et al., 1971). The 10-item inventory asks participants to indicate which hand they would use to complete common tasks, such as striking a match, throwing, or using scissors. Handedness is determined using a laterality quotient (LQ = (R-L)/(R+L) * 100), where a score of 100 reflected complete right-hand dominance, and a score of −100 reflected complete left-hand dominance.

In the laboratory session, participants completed a semi-structured interview, the CAADID, which was updated to reflect the criteria of the DSM-5. Other tests conducted during the lab visit included portions of the Wechsler Adult Intelligence Scale-Fourth Edition (WAIS-IV) (Wechsler, 2008) to estimate intelligence quotient (IQ), the Purdue Pegboard Test (Buddenberg and Davis, 2000) to assess coordination, and dynamometer tests for maximum pinch grip strength.

2.2.1. Go/No-Go force task—Stimuli were presented on a 102 cm Samsung television screen with 1920 x 1080 resolution and 120 Hz refresh rate. Participants were comfortably seated in a chair (JedMed Straight Back Chair, St. Louis, MO) facing the center of the TV screen a horizontal distance of 127 cm. Their dominant arm rested at approximately 100 degrees of flexion on a height-adjustable table. As shown in Figure 1A, participants used their thumb and index finger to form a pinch grip against a custom-built force apparatus with identical button load cells on either side (Measurement Specialties, Hampton, VA). Total force produced by the thumb and index finger was measured. Voltage outputs were sent to a transducer coupler to be amplified (Coulbourn Instruments Type B V72-25B), transmitted via a 16-bit A/D converter, and digitized at 62.5 Hz. Digitized voltage signals were transformed to Newtons with a resolution of 0.0016 N. The summed force output from the load cells was presented on the television screen in real time. Voltage data acquisition, voltage-to-force transformation, and stimuli presentation were all conducted using customized programs written in LabVIEW (National Instruments, Austin, TX).

Before the force task, each participant's maximum voluntary contraction (MVC) was measured using a pinch grip dynamometer (Lafayette Hydraulic Pinch Gauge, Model J00111, Lafayette, IN). The average of three, five-second trials determined each participant's maximum voluntary contraction (MVC) in Newtons. MVC was then used to create target force amplitudes for each participant. During the experiment, the visual display consisted of two rectangular bars on a black background (Figure 1C). A stationary, white target bar represented the target force. For each run, the target was normalized to 15% (low amplitude) or 60% (high amplitude) of the participant's MVC. A second colored bar moved up with

increasing applied force and down with decreasing force. The distance moved by this bar was proportional to the amount of force produced. When no force was produced, the moveable bar was stationary. The moveable bar changed colors from trial to trial and could be red, green, aqua, orange, yellow, or blue. Participants completed 100 consecutive trials to the same target amplitude and trials were presented rapidly, creating a prepotency to respond. In this context, the inhibition of a prepotent response requires effortful cognitive control, whereas allowing the motor response to proceed occurs in a more automatic fashion (Bargh et al., 1996; Muraven and Baumeister, 2000).

Each participant completed two runs of 100 trials (25% no-go) at low and high amplitude force conditions, for a total of four runs. Each run started and ended with 12.5s of rest, during which the movable bar was red, indicating to the participant that they should not press on the load cells. Every trial onset was cued by the color of the movable bar. Go trials were cued by the colors green, aqua, orange, or yellow. No-Go trials were cued by the color blue (Figure 1B). Go and No-Go trials were randomly presented. In Go trials, participants were instructed to press as quickly and accurately as possible to match the movable bar to the amplitude prescribed by the target bar, and to release when the movable bar disappeared. In No-Go trials, participants were to refrain from pressing the load cells. Trials were 750ms in duration, followed by a 500ms inter-trial period of rest, during which only the white target bar was visible. Each participant practiced the task immediately prior to the experimental session.

2.2.2. Standard Go/No-Go task—The Go/No-Go RT task was identical to the Go/No-Go force task, with the exception that there was no white target bar and the colored bar was stationary in the middle of the screen. Visual stimuli were presented on a 23-inch monitor (Dell, Round Rock, TX) using E-Prime (Psychology Software Tools, Inc., Sharpsburg, PA). Stimuli were colored bars just as in the force task. Participants were instructed to press the space-bar on a keyboard in Go trials as quickly as possible, and to refrain from pressing the space bar in No-Go trials, cued by the color blue. No feedback concerning accuracy was provided. Participants completed four runs of 100 trials (25% no-go) in the standard RT task.

2.3. Data analysis

2.3.1 Force data analysis—The force time series data was digitally filtered using a tenth-order low-pass Butterworth filter with a 15 Hz cut-off frequency. Figure 1C displays the time-series force data for 10 trials from an exemplar participant. A custom-written MATLAB program was used to place markers at four time points for each trial: onset of force, beginning and end of force interval, and offset of force. Each trial was then visually inspected to confirm correct placement of the time points. A second MATLAB program read in the four time points and associated force output in order to calculate the mean force amplitude for each trial, for each participant. Mean force amplitude was calculated as the average force output during the time between the second and third time-points for each trial. Standard deviation of force was calculated as the variability of mean force across Go and No-Go trials. We then obtained an average value for mean force output and standard deviation of force output for all Go and No-Go trials in the low and high amplitude force conditions, for each participant. These values were submitted to 2 (group: ADHD, CTRL)

by 2 (amplitude: low, high) mixed model analysis of variance (ANOVA). All statistical tests were evaluated at an alpha of .05. It is important to note that No-Go trials were not categorized according to behavioral outcome. In other words, mean force was calculated for all No-Go trials and trials were not labeled *post hoc* as successes or failures. In other words, mean force represents how hard individuals were pressing on the load cells and thus provides a continuous measure of motor output for both Go and No-Go trials.

2.3.2. Data analysis for standard Go/No-Go RT task—E-Prime output variables included percentage of no-go failures, i.e., "commissions", and reaction time for go successes (RT-go). Within-subject means were computed for each trial type, for each run of 100 trials. A grand mean for each participant, for each trial type, was then computed using these data. Means were submitted to three separate independent samples t-tests.

3. RESULTS

3.4.1. Participants

As reported in Table 1, independent univariate ANOVAs demonstrated that there were no differences in age or estimated IQ between the ADHD and control groups. The ADHD group reported significantly more ADHD-related symptoms in adulthood as measured by the CAADID and CAARS. As expected (Hinshaw et al., 2012; Anastopoulos et al., 2016), adults with ADHD self-reported more internalizing (e.g. anxiety or depression) and externalizing (e.g., aggression, rule-breaking behavior) difficulties on the ASR ($ps < .001$). Importantly, group means were within normal limits (*T-scores < 59*). As such, any group effects are unlikely to be attributed to the presence of a co-occurring condition instead of ADHD. Eighty-seven participants were confirmed to be right-hand dominant using the Edinburgh Handedness Inventory. The results for the left-handed (n = 7) and mixed-handed (n = 8) individuals did not differ from those who were right handed and thus they were included in the analysis. Of note, we conducted a *post hoc* power analysis using GPower (Faul et al., 2009) to confirm that the sample size (N = 102) was sufficient to detect effects and interactions. Specifically, given the lowest η_p^2 observed in our Results ($\eta_p^2 = 0.02$), the associated power is 0.953.

3.4.2. Go/No-Go force task—Mean force was evaluated by a repeated-measures ANOVA for Trial Type (Go, No-Go) and Amplitude (Low, High) with Group (Control, ADHD) as a between-subjects variable. Main effects were observed for amplitude, $F(1, 100) = 888.96$, $p < .001$, $\eta_p^2 = 0.90$, trial type, $F(1, 100) = 320.21$, $p < .001$, $\eta_p^2 = 0.06$, and group, $F(1, 100) = 6.12$, $p = .015$ $\eta_p^2 = 0.88$. Interactions were significant for group by amplitude, $F(1, 100) = 11.15$, $p = .001$, $\eta_p^2 = 0.88$, and amplitude by trial type, $F(1, 100) = 385.36$, $p < .001$, $\eta_p^2 = 0.79$. No interaction was observed for group by trial type, ($F(1, 100) = 3.40$, $p = .068$, $\eta_p^2 = .033$, or group by amplitude by trial type, $F(1, 100) = 2.31$, $p = .132$, $\eta_p^2 = 0.02$. Although the three-way interaction did not reach statistical significance, we conducted four independent samples t-tests to examine group differences for each trial type and amplitude, based on our *a priori* hypotheses. The results of the t-tests demonstrated that mean force produced in the Go trials did not differ between groups (p's > .444). In contrast, however, mean force produced in the No-Go trials was higher for adults with ADHD compared to controls for the

low (t(100) = −4.63, p < .001) and high (t(100) = −6.12, p < .001) amplitude conditions, reflecting the significant group by amplitude interaction. As shown in Figure 2B, these data are also consistent with our conclusion that adults with ADHD produce higher mean force in No-Go trials, but not Go trials, compared to controls.

Standard deviation of force was evaluated by a repeated-measures ANOVA for Trial Type (Go, No-Go) and Amplitude (Low, High) with Group (Control, ADHD) as a between-subjects variable. Main effects were observed for amplitude, F(1, 100) = 592.59, p < .001, η_p^2 = 0.86, trial type, F(1,100) = 40.64, p < .001, η_p^2 = 0.29, and group, F(1, 100) = 8.39, p = .005, η_p^2 = 0.08. Interactions were significant for group by amplitude, F(1,100) = 7.20, p = .009, η_p^2 = 0.07, group by trial type, F(100), 17.36, p < 001, η_p^2 = 0.15, and group by trial type by amplitude, F(1, 1000) = 6.158, p = .015, η_p^2 = 0.06. We then conducted four independent samples t-tests to examine group differences for each trial type and amplitude, based on our *a priori* hypotheses. The results demonstrated that the standard deviation produced in the Go trials did not differ between groups (ps > .167). In contrast, and as shown in Figure 2C, the standard deviation for No-Go trials was higher for adults with ADHD compared to controls for the low (t(100) = −2.87, p < .005) and high (t(100) = −4.35, p < .001) amplitude conditions. This pattern mirrors the findings noted for mean force.

3.4.3. Standard Go/No-Go task—There was a main effect of group on the total number of commission errors, t(100) = −4.86, p < .001, *d* = 0.96, in which adults with ADHD had more commissions (22.6, *SD*=15.0) compared to controls (10.7, *SD*=9.0). There were no significant effects of group on mean RT for go-trials, t(100) = 1.03, p = .306 (grand mean = 348.1 ms, *SD*=45.0).

3.4.4. Clinical associations—Correlation and multiple regression analyses were conducted to examine the relationship between the CAARS S:L ADHD Index scores and three predictors: mean force output in no-go trials in the low and high amplitude conditions and number of commission errors in the no-go trials in the standard RT task. As shown in Table 2, each predictor is positively and significantly correlated with ADHD Index scores, demonstrating that those who produced higher forces and those who made more commission errors had higher ADHD Index scores. We first used hierarchical regression to determine whether the predictors contribute a significant amount of variability to the regression equation. Mean force for the low and high amplitude conditions was entered at Stage one and number of commission errors was entered at Stage two. The hierarchical multiple regression revealed that at Stage one, mean force in the low and high amplitude conditions contributed significantly to the regression model, F(2, 99) = 10.88, p < .001, and accounted for 18% of the variance in the ADHD Index score. Adding the number of commission errors to the model explained an additional 0.01% of the variation and the change in R^2 was not significant, F(1, 98) = 0.08, p = .776. Next, we reversed the order of the predictors in the model. The number of commission errors was entered at Stage one and mean force for the low and high amplitude conditions was entered at Stage two. The hierarchical multiple regression revealed that at Stage one, number of commissions contributed significantly to the regression model, F(1, 100) = 8.33, p = .005, and accounted for 7.7% of the variance in the ADHD Index score. Adding mean force output in the low and high amplitude conditions

to the model explained an additional 10.4% of the variation and the change in R^2 was significant, $F(2, 98) = 6.22$, $p = .003$. Together, these results demonstrate that the standard RT task does not predict unique variance over and above that of what is predicted by the force task, but that the force task contributes for unique variance over and above that is what is predicted by the standard RT task alone.

4. DISCUSSION

We report three novel findings. First, on Go trials of the force task, force output and variability was not different for adults with ADHD compared to adults without ADHD. Second, on No-Go trials in the force task, adults with ADHD produced greater and more variable force compared to adults without ADHD. Third, mean force output on No-Go trials was a stronger predictor of the CAARS S:L ADHD Index compared to performance in the standard RT task.

The lack of a group difference for force output and force variability on Go trials demonstrates that adults with ADHD appropriately scaled their motor output to the goals of the task in both low and high amplitude Go trials. The finding that force output and variability scaled to the target amplitude in No-Go trials, suggests that the motor system in adults with ADHD do not have difficulty calibrating response parameters to the demands of the upcoming task. That is, work in non-human primates that involves the Go/No-Go task (Wise et al., 1983; Kalaska and Crammond, 1995) and cerebellar internal models for action (Wolpert et al., 1995; Wolpert and Miall, 1996; Wolpert et al., 1998; Imamizu et al., 2000; Ito, 2005) have found that the motor system integrates sensory information with the goals of the task to estimate the parameters of the action. The cerebellum is well-positioned for such a task since the majority of its inputs are from primary motor cortex, prefrontal cortex, and posterior parietal cortex (Ramnani, 2012). Thus, it is unlikely that the fine motor deficits that are often observed in ADHD are due to difficulty creating an internal model of force amplitude. Further, the finding that force amplitude scaled to the parameters of the task, even in No-Go trials, suggests that responses were planned in advance of the visual cue. Such a movement strategy is expected in a paradigm with rapid stimulus-response requirements (Favilla et al., 1990; Ghez et al., 1991; Leocani et al., 2000).

That being said, adults with ADHD did produce greater and more variable force during No-Go trials compared to adults without ADHD. One interpretation is that the excess force produced in No-Go trials is a reflection of hyperactivity in the motor system. Indeed, the idea that excess muscular tension is related to hyperactivity is not a new concept (Braud et al., 1975); however, to the best of our knowledge this has not been empirically tested. We suggest that the amount of force produced on No-Go trials provides a precise and continuous measure of inhibitory control. In particular, an individual with "good" inhibitory control produces low levels of force, whereas an individual with "poor" inhibitory control produces higher levels of force. The result is a continuum of inhibitory control performance that can be examined in relation to other behavioral, clinical, or physiological measures. Adults with ADHD produced more variable force during No-Go trials compared to adults without ADHD. Variability is inherent to biological systems and is considered to reflect noise in the system. A traditional perspective on variability in the motor system is that optimal

performance is characterized by low variability (Selen et al., 2006). However, an alternate view is that low variability leads to rigid and stereotyped movement, whereas excess variability leads to unstable movement (Stergiou et al., 2006). Both scenarios lead to less than optimal performance. In the present work, we suggest that increased force variability on No-Go trials is indicative of excess neuromuscular noise and unstable performance. Neuromuscular noise is the result of several physiological mechanisms, such as motor unit synchronization and recruitment, the level of antagonistic coactivation, and the integrity of feedback loops (for a review see (Oomen and van Dieen, 2016)). Future studies that can disentangle the physiological components of the task will be able to provide greater information about the role of neuromuscular noise in the pathology of ADHD.

More broadly speaking, adults with ADHD have impaired visuomotor memory in gripping tasks (Neely et al., 2016), visuomotor adaptation in reaching tasks (Kurdziel et al., 2015), deficits in oculomotor control (Feifel et al., 2004; Carr et al., 2006), increased postural sway (Hove et al., 2015), and impaired timing finger tapping tasks (Valera et al., 2010). The force task employed here provides a precise and continuous measure of motor output, which in future studies could be correlated with behavioral or neuroimaging data to help pinpoint the neurological cause of the impairment. Further, the force amplitude manipulation provides a means to evaluate whether adults with ADHD can appropriately plan and scale the amplitude of their response. Previous work demonstrates the importance of the basal ganglia and prefrontal cortex for force production (Vaillancourt et al., 2003; Vaillancourt et al., 2004; Vaillancourt et al., 2006; Spraker et al., 2007; Prodoehl et al., 2009; Spraker et al., 2009; Coombes et al., 2011; Neely et al., 2013a; Neely et al., 2013b). The networks for force production overlap with networks critical for inhibitory control, including the subthalamic nucleus of the basal ganglia, the supplementary motor area, and the inferior frontal cortex (Aron and Poldrack, 2005). More importantly, these brain regions are implicated in neural models of ADHD characterized by large-scale networks including anterior cingulate cortex, posterior parietal cortex, thalamus, prefrontal cortex, striatum, and cerebellum (Bush et al., 2005; Seidman et al., 2005; Makris et al., 2009; Liston et al., 2011; Cortese et al., 2012). Thus, the use of precision force tasks, which have well understood neural correlates, may provide a good framework to examine the neurobiology of ADHD.

5. CONCLUSION

These current work represents a novel examination of inhibitory control in young adults. We demonstrated that the force variant of the Go/No-Go task is a precise and continuous measure of inhibitory control. As such, the resulting continuum of performance can be examined in relation to other behavioral, clinical, or physiological measures to provide greater information about the pathophysiology of inhibitory motor control. The results of the current study demonstrate that adults with ADHD have inhibitory control deficits in both the standard RT and force variant of the Go/No-Go task. Importantly, however, performance on the force task was a better predictor of ADHD symptomatology than the standard RT task. This suggests that a precisely controlled motor task may provide a more nuanced approach to examine motor control as it relates to ADHD-related symptoms. Our future work aims to identify patterns of performance in motor and cognitive tasks in a community population of young adults with ADHD who may be more impaired than the college-attending population

studied in this work. We focus on young adults because late adolescence and young adulthood is a period when individuals establish their career trajectory, have rapidly changing social patterns and relationships, and establish their independence. Further, it is well-established that young adults with ADHD have adverse outcomes. These outcomes may be related to problems in the motor system. Thus, it is critical we understand the neural basis of ADHD to inform optimal therapeutics that can be used to improve functional outcomes in young adults with ADHD.

Acknowledgments

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HIGHLIGHTS

- Adults with and without ADHD completed a force-variant of the Go/No-Go task.

- Adults with ADHD produced more force than adults without ADHD on failed inhibits.

- The amount of force was a better predictor of ADHD compared to the standard task.

- Precise and continuous motor tasks can be used to study behavioral deficits in ADHD.
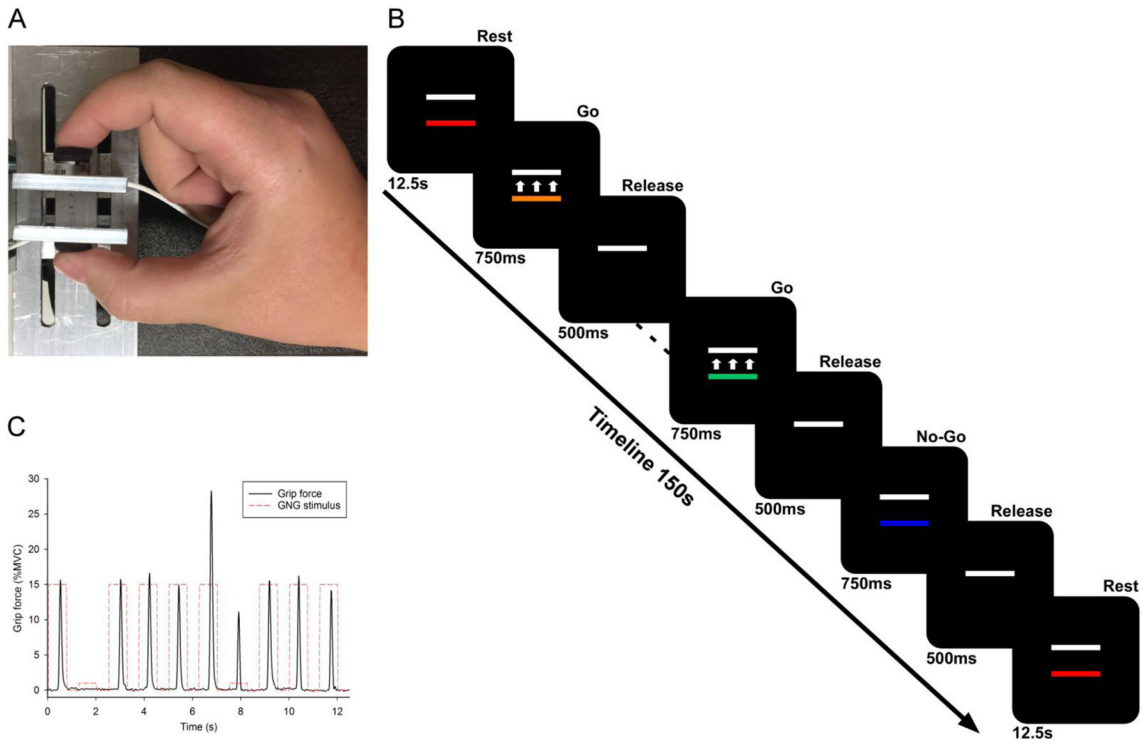
Figure 1.
(A) Participants used their thumb and index finger to grip against a custom-built force apparatus with identical button load cells. (B) The visual display consisted of two rectangular bars on a black background. A stationary, white target bar represented the target force amplitude. For each run, the target was normalized to 15% (low amplitude) or 60% (high amplitude) of the participant's maximum voluntary contraction. A second colored bar moved up with increasing applied force and down with decreasing force. (C) The experimental time-series force data for 10 trials from an exemplar participant.
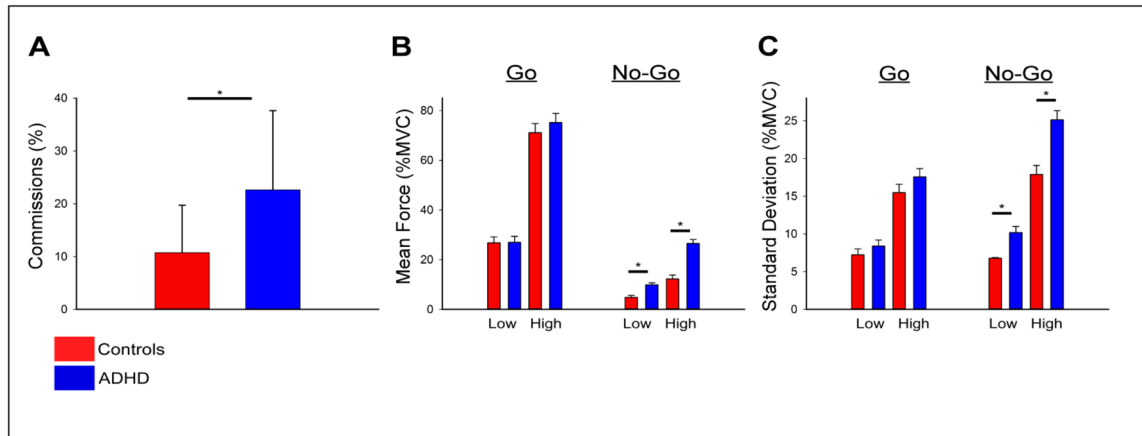
Figure 2.
(A) Adults with ADHD made more commission errors in No-Go trials in the standard RT Go/No-Go task. **(B) Adults with ADHD produced more force in No-Go, but not Go, trials compared to healthy controls, for both the low and high amplitude conditions. (C) Adults with ADHD produced more variable force in No-Go, but not Go, trials compared to healthy controls, for both the low and high amplitude conditions.**

Table 1

Participant characteristics

Variables	Group		Significant Group Differences
	Control	**ADHD**	
Sample size	51	51	
Females	22	29	
Right-handed	43	44	
Mixed-handed	4	4	
Left-handed	4	3	
Age, yrs	21.0 (1.7)	21.1 (1.8)	ADHD = CTRL, p = .779
FSIQ	106.3 (11.1)	105.3 (11.5)	ADHD = CTRL, p = .630
CAADID			
Number of inattention symptoms endorsed in adulthood	0.02 (0.14)	6.45 (1.95)	ADHD > CTRL, (F1, 100) = 557.20 p < .001
Number of hyperactive/impulsive symptoms endorsed in adulthood	0.14 (0.40)	4.90 (2.33)	ADHD > CTRL, (F1, 100) = 232.47 p < .001
Total number of symptoms endorsed in adulthood	0.16 (0.42)	11.35 (3.16)	ADHD > CTRL, (F1, 100) = 664.47 p < .001
CAARS-S:L			
DSM-IV ADHD Symptoms Total T-score	43.1 (10.0)	66.4 (15.9)	ADHD > CTRL, (F1, 100) = 78.64, p < .001
ADHD Index T-score	40.2 (7.0)	57.0 (11.1)	ADHD > CTRL, (F1, 100) = 83.36, p < .001
ASR			
Internalizing Composite T-score	44.1 (10.8)	56.3 (13.0)	ADHD > CTRL, (F1, 100) = 26.38, p < .001
Aggressive Behavior T-score	51.5 (3.6)	56.9 (7.6)	ADHD > CTRL, (F1, 100) = 20.87, p < .001
Rule-breaking Behavior T-score	52.0 (3.5)	58.9 (8.7)	ADHD > CTRL, (F1, 100) = 27.98, p < .001

Note: Values are means and standard deviations (in parentheses).

Table 2

Summary statistics, correlations, and multiple regression results.

Variables	Mean	Standard deviation	Correlation with CAARS ADHD Index	Multiple regression weights	
				b	β
CAARS ADHD Index score	48.58	12.55			
Mean force high amplitude	19.38%	13.74%	.393*	35.87	.393
Mean force low amplitude	7.35%	6.10%	.385*		
Errors of commission	16.68	13.68	.277*		

Notes: Pearson correlations with p <.001 are noted with an asterisk (*).