Working memory and intraindividual variability mediate fluid intelligence deficits associated
with ADHD symptoms

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Abstract

Objective: To determine if decreased fluid intelligence was associated with ADHD, and was mediated by deficits in working memory and intra-individual variability in motor responding.

Method: The present study tested 142 young adults from the general population on a range of working memory, response time, and fluid intelligence tasks, and an ADHD self-report symptoms questionnaire.

Results: Total and hyperactive ADHD symptoms correlated significantly and negatively with fluid intelligence, but this association was fully mediated by both working memory and intra-individual variability in response time. However, inattentive symptoms were not associated with fluid intelligence.

Conclusion: These results have important implications for clinicians using speeded psychometric tests as part of their assessment battery, working memory interventions for ADHD patients that focus on performance improvement without controlling for response consistency, and also demonstrate potential differences in the neuropsychological profiles of ADHD subtypes.

Keywords: ADHD; fluid intelligence; working memory; intraindividual variability.

Working memory and intra-individual variability in response time mediate fluid intelligence deficits associated with ADHD symptomology

Attention Deficit/Hyperactivity Disorder (ADHD) is a neuropsychological disorder characterised by hyperactivity, inattention, and impulsiveness (Barkley, 1997), and is observed in approximately 5.9 – 7.1% of children (Willcutt, 2012). The condition is most commonly diagnosed in early childhood, and has been found to persist into adulthood in 30 – 50% of cases (Klein & Mannuzza, 1991, Weiss & Hechtman, 1993). Individuals suffering from ADHD commonly suffer cognitive deficits, in domains including executive functioning, processing speed, and intelligence (Seidman, 2006), which can, in turn, negatively affect long-term outcomes such as self-esteem, academic performance, and income (Ingram, Hechtman, & Morgenstern, 1999). Recent research has found that an increase in severity of ADHD symptoms was associated with decreased fluid intelligence, and that working memory fully mediated this relationship, but processing speed had no effect (Brydges, Ozolnieks, & Roberts, 2017). However, ADHD-related deficits in processing speed may be dependent upon ADHD subtype (Nigg et al., 2005), as research has found that larger intra-individual variability in response time (the temporal consistency of response times made by an individual) are robust across ADHD subtypes (Kofler et al., 2013), and accounts for a significant proportion of variance of intelligence even when the mean response time (RT) is taken into account (Jensen, 1992). As such, it may be the case that Brydges et al. (2017) examined an inappropriate construct of speed of processing – perhaps variability in the consistency of responding, rather than the speed at which the average response is made, mediates ADHD-related deficits in fluid intelligence.

Previous research has found that ADHD is associated with deficits in a range of higher-order cognitive processes (Theiling & Petermann, 2016), including fluid intelligence (Dige & Wik, 2005), working memory (Schweitzer, Hanford, Medoff, 2006), and processing

speed (Boonstra, Oosterlaan, Sergeant, & Buitelaar, 2005). Fluid intelligence (gF) is the ability to solve novel problems, independently of any previously acquired knowledge (Cattell, 1963). Working memory is the ability to store, manipulate, and update incoming information or stimuli (Baddeley & Hitch, 1974). Processing speed refers to the speed at which individuals can perform cognitive tasks (Sheppard & Vernon, 2008, Vernon, 1983). A large body of research has found that these three constructs are associated with each other (e.g., Ackerman, Beier, & Boyle, 2005; Kail & Salthouse, 1994; Kyllonen & Christal, 1990; Wilhelm & Oberauer, 2006), and that working memory and processing speed are both excellent predictors of gF (e.g., Redick et al., 2012).

Given the associations between gF, working memory, and processing speed, and that ADHD appears to be associated with deficits in these three domains, Brydges et al. (2017) tested the hypothesis that ADHD-related deficits in gF are an indirect effect of impaired working memory and/or processing speed. They found that a latent variable of working memory fully mediated the association between self-reported ADHD symptom severity and the Cattell Culture Fair Intelligence Test (a commonly used measure of gF) in 142 young adults, albeit a non-diagnosed sample. However, they also found that a processing speed latent variable did not affect the relationship between ADHD symptom severity and gF. It was suggested that this was due to the different subtypes of ADHD having opposite effects on processing speed. Specifically, Nigg et al. (2005) found that in comparison to a typical control group, inattentive-type ADHD patients had significantly longer RTs on a processing speed task, whereas hyperactive-type ADHD patients had significantly shorter RTs (though it should be noted that neither subtype was significantly associated with processing speed).

Intra-individual variability (IIV) in RT has been studied in clinical samples for over 90 years (Head, 1926), and has been found to be significantly increased in individuals diagnosed with ADHD relative to non-clinical groups (see Kofler et al., 2013, for a meta-

analysis of 319 studies). Seeing as increased IIV is associated with mind wandering and lapses in attention (e.g., McVay & Kane, 2012; West, Murphy, Armilio, Craik, & Stuss, 2002) – a commonly reported observational characteristic of ADHD patients – it stands to reason that there may be an association between IIV and ADHD symptom severity. There are several neurological explanations that could explain this effect. First, brain regions associated with IIV are also commonly implicated in ADHD, including much of the lateral and medial prefrontal cortices, such as the inferior frontal gyrus, superior frontal gyrus, and dorsal anterior cortex (Bellgrove, Hester, & Garavan, 2004; MacDonald, Nyberg, & Backman, 2006). Second, increased IIV in ADHD is theorised to be caused by dysfunctional dopaminergic and noradrenergic neurotransmission, which has been observed in several imaging studies of patients diagnosed with ADHD. This is supported by current pharmacotherapy approaches in treating ADHD, which involves the use of psychostimulants such as methylphenidate and dexamphetamine that target the dopaminergic and noradrenergic systems (Faraone & Buitelaar, 2010). Additionally, twin studies have found a genetic link between hyperactivity and IIV (Kuntsi, Oosterlaan, & Stevenson, 2001; Kuntsi & Stevenson, 2001), and adolescents carrying two copies of a high-risk allele for ADHD (a ten-repeat allele of the dopamine transporter gene) displayed increased IIV in an attention task (Bellgrove, Hawi, Kirley, Gill, & Robertson, 2005).

Considering that IIV is predictive of intelligence, even when average RT is taken into account (Jensen, 1992), it is possible that a) there is more than one measurable ‘speed’ construct in the brain, and if so, b) ADHD may differentially affect these multiple speed domains. The current study was a reanalysis of the data presented by Brydges et al. (2017). Specifically, the processing speed tasks, from which median RTs were extracted, were recalculated and IIV in RT measures were extracted from each of the tasks. The current study aimed to determine if associations between ADHD symptom severity and fluid intelligence

were mediated by working memory and intra-individual variability. It was hypothesised that ADHD is associated with deficits in gF, working memory, and IIV. Hence, it was predicted that the severity of ADHD symptoms combined across hyperactivity and inattentiveness sub-scores would negatively predict gF and working memory performance (i.e., lower scores), and would positively predict IIV (i.e., greater variability in RTs). It was also hypothesised that gF, working memory, and intra-individual variability are all related constructs. As such, it was predicted that these three constructs would significantly correlate in a confirmatory factor analysis (CFA), and that working memory and intra-individual variability would be predictive of gF in a structural equation model (SEM). Additionally, it was hypothesised that ADHD-related deficits in gF are only observable due to deficits in working memory and IIV. Hence, it was predicted that the ADHD-gF association would be fully mediated in an SEM by working memory and IIV. Lastly, the same analyses were conducted on hyperactivity and inattentiveness sub-scores with the same hypotheses that the sub-score-gF associations would be fully mediated by working memory and IIV.

Method

Participants

The sample examined in the current study is the same as that reported in Brydges et al. (2017). Participants were 142 adults aged 18-40 years ($M = 22.24$, $SD = 4.20$), of which 91 were female. All participants demonstrated competency in English and reported normal or corrected-to-normal hearing and vision. No other inclusion/exclusion criteria were applied. The majority of the participants were undergraduate psychology students at an Australian university, with the remainder recruited from the general population. The sample had a mean fluid IQ of 116.01 (given that the Cattell Culture-Fair Intelligence Test has a standardised mean of 100 and SD of 24, this is within normal range). Participants were offered a choice of

1.5 participation credit hours in order to partially fulfil course requirements, or entry to a draw to win a \$50 gift card.

Materials

All working memory and intraindividual variability tasks were from the Psychology Experiment Building Language (PEBL) version 0.13 (Mueller & Piper, 2014). Additionally, the ADHD symptoms questionnaire was administered through the PEBL Survey program.

ADHD questionnaire

World Health Organization adult ADHD self-report scale (ASRS; Kessler et al., 2005): The ASRS is a self-administered questionnaire designed to measure current ADHD symptoms in adults. It consists of 18 items based on *DSM-IV* symptoms or criteria for ADHD that are measured on a 5-point scale (0 = *never* and 4 = *very often*), with higher scores indicative of more severe symptoms. Nine items each related to the hyperactivity and inattentive subtypes of ADHD. The total score (which could range from 0 – 72) was used as the indicator for ADHD symptoms, with each subtype having a range of 0 – 36. The ASRS has internal consistency in the range .63 – .72 and test-retest reliability (Pearson correlations) in the range .58 – .77 (Kessler et al., 2007), and also correlates highly with the clinician-administered ADHD Rating Scale ($r = .84$; Adler et al., 2006).

Reaction-time intraindividual variability (IIV) measures

Simple Motor Response: A single stimulus (an ‘X’) would appear on screen, and participants were required to press the X key as quickly as possible. There were four blocks of 50 trials, and to ensure participants were paying attention and reduce the temporal predictability of responding, the inter-stimulus interval varied between 250 and 2500 ms.

Two-choice Motor Response: In the two-choice motor response task, a fixation cross would appear in the centre of the computer screen for 1250 ms, before being replaced with the target stimulus (a letter) for 100 ms. The stimulus would then disappear and be replaced with three non-alphabetical characters (e.g. '&#@'), with the target stimulus appearing either to the left or right of centre, and a distractor stimulus on the other side. Participants were required to press a button (Z or /) to indicate which side of the screen the target stimulus appeared on. There were 150 trials administered in a single block.

Four-choice Motor Response: In the four-choice motor response task, a 2x2 grid was presented on screen. A cross would appear in one of the four squares, and the participants were required to press a keyboard button (F, V, J, or N) to indicate with square the cross appeared in. There were 50 trials administered in a single block.

Working memory measures

Backward Digit Span: Participants were required to remember and then recall lists of numbers of increasing length in reverse order. There were two trials of each list length, starting with three digits. The task ended once participants recorded two consecutive incorrect trials. The indicator of working memory was the number of correct trials.

Memory Span: Participants were sequentially presented with a series of pictures on a computer screen and had to recall them in order. This task had 16 trials, starting with four pictures to be recalled, and used a staircase method to determine memory span. This final value from the staircase method was the indicator used.

Corsi Block Task: Participants were presented with nine navy blue boxes on a computer screen, one of which at a time would light up. Participants were required to remember and then recall the order in which the boxes lit up in. There were two trials of each

length, starting with two boxes lighting up. The task ended once participants recorded two consecutive incorrect trials. The indicator of working memory was the number of correct trials.

Fluid intelligence measure

Cattell Culture Fair Intelligence Test (Scale 3, Form A; Cattell, 1973): The Cattell Culture Fair Intelligence Test (CCFIT) is a commonly used, non-verbal measure of gF. The task requires inductive reasoning about perceptual patterns, and consists of four timed subtests (series completion, odd-one-out, matrices, and topology), with items increasing in difficulty within each subtest. The indicator for gF was the raw score of this measure, which is the total number of correct items across all subtests. The CCFIT is an excellent measure of fluid intelligence with high reliability and convergent validity with other tests of general intelligence ($r = .70-.81$; Cattell, 1973).

Procedure

Testing took place in laboratory settings in a single session that lasted approximately 1.5 hours. The order of task administration was fixed for all participants, with the constraint that no two tasks that were supposed to tap into the same construct occurred consecutively. The order was: CCFIT; Two-choice Reaction Time; Backward Digit Span; Simple Reaction Time; Corsi; Four-Choice Reaction Time; Memory Span; ASRS.

Computation of Intraindividual Variability

For the three intra-individual variability tasks, extremely fast or slow RTs were removed as they may be a source of measurement error. A lower bound of 150 ms was set for all three tasks based on previous research (Hultsch, MacDonald, & Dixon, 2002), and upper bounds involved computing the task mean and *SD*, and removing any trials that fell more

than 3 *SDs* from the mean. Missing values were then replaced with the mean RT, as this decreases within-subject variation and represents a conservative approach to examining intra-individual variability.

To calculate intra-individual variability the statistical procedures proposed by Hultsch, Strauss, Hunter, & MacDonald (2008) and Bielak, Hultsch, Strauss, MacDonald, and Hunter (2010) were used to calculate the across-trial intra-individual *SD*. This method is statistically superior to other measures of IIV, such as raw RT *SD* and the intra-individual coefficient of variation (ICV), because both systematic within-subject and systematic between-subject variability are controlled for. For instance, IIV is well-known to increase with age (Bielak, Cherbuin, Bunce, & Anstey, 2014), and decrease as a result of practice effects (Hultsch, MacDonald, Hunter, Levy-Bencheton, & Strauss, 2000). As Hultsch et al. (2000) state, IIV is defined as the within-subject variability that is independent of relatively durable changes over time (e.g., practice effects). As systematic within-subject variation (e.g., practice effects) is not of interest in the current study, any systematic effects associated with trials must be removed prior to statistical analysis. Additionally, if there are group differences in mean levels of performance, it is possible that group differences in IIV are an artefact of average differences in performance. Therefore, between-subjects effects must also be statistically removed prior to any analyses. As such, potential confounds (e.g., age, practice effects) and their higher-order interactions were partialled out using split-point regression. The *SD* of the residuals extracted from this regression of the processing speed tasks for each participant was used as the measure of intra-individual variability for the three tasks.

Transformations and Outlier Analysis

The raw distributions of the ten measures all had a satisfactory level of normality, so no transformations were conducted. As CFA and SEM are very sensitive to outliers,

univariate and multivariate outlier analyses were conducted on the nine dependent variables. Specifically, a test score was considered a univariate outlier if it was greater than 3 *SDs* from the between-subjects variable mean, and was replaced with a value that was 3 *SDs* from the mean. This affected no more than 2.1% of the observations for each task. No multivariate outliers were identified when using a Cook's *D* value of > 1 (Cook & Weisberg, 1982), or had larger a Mahalanobis distance than the critical value. Little's (1988) MCAR test was non-significant [$\chi^2(79) = 100.20; p = .054$], indicating that missing data were missing completely at random. These scores were estimated using the expectation maximisation method implemented in SPSS version 24 (IBM).

Statistical Analysis

Based on guidelines summarized by Schweizer (2010), the CFA/SEM models were evaluated to be well-fitting according to the following criteria: Comparative fit index (CFI) $\geq .95$; root mean square error of approximation (RMSEA) $< .06$; and the standardized SRMR $< .08$, although values of .90 (for the CFI) and .10 (for the RMSEA and SRMR) are acceptable (Blunch, 2008). For thoroughness, the implied model chi-square statistics were reported. All models were tested in Amos 24 (Arbuckle, 2016) via maximum likelihood estimation. Significance of correlation and path coefficients was determined by conducting χ^2 difference tests when removing an individual regression parameter. If the difference in model fit was significant, it indicated that the regression path makes a significant contribution to model fit. This method is more reliable than using test statistics that are based upon comparing standard errors of parameters (Gonzalez & Griffin, 2001).

Results

Descriptive Statistics

Descriptive statistics of the eight measures are presented in Table 1, and the correlations between measures are presented in Table 2.

Associations between ADHD Symptoms and Cognitive Functioning

A six-construct CFA model was created, with correlations between the two latent variables (IIV and working memory) and four manifest variables (the three ASRS scores and CCFIT) all free to vary, presented in Figure 1. It was found that the ASRS total and hyperactivity scores significantly correlated with the CCFIT (based on the significant worsening of model fit when the paths were removed; Total $\Delta\chi^2 = 4.18$, $\Delta df = 1$, $p = .041$; Hyperactivity $\Delta\chi^2 = 5.93$, $\Delta df = 1$, $p = .015$), but the ASRS inattentiveness score did not ($\Delta\chi^2 = 1.71$, $\Delta df = 1$, $p = .19$). The working memory and IIV factors also significantly correlated with CCFIT (working memory $\Delta\chi^2 = 14.87$, $\Delta df = 1$, $p < .001$; IIV $\Delta\chi^2 = 20.70$, $\Delta df = 1$, $p < .001$), although the working memory and IIV factors did not significantly correlate with each other ($\Delta\chi^2 = 0.66$, $\Delta df = 1$, $p = .42$). After removing non-significant correlations from the CFA, the final model reported acceptable fit indices [$\chi^2(27) = 43.74$, $p = .022$, CFI = .982, RMSEA = .066, SRMR = .085].

SEM and Mediation Analyses

Two separate SEMs were created, with ASRS total score and ASRS Hyperactivity score as the predictor in each model (no SEM and mediation analysis was conducted with ASRS Inattentiveness as a predictor as it was not significantly correlated with CCFIT in the CFA). Each model had the relevant ASRS variable predicting CCFIT, with this association being mediated by working memory and intraindividual variability (which were not correlated with each other, given the non-significant correlation in the CFA model).

For the ASRS total score, initial model fit statistics were mixed [$\chi^2(19) = 27.80, p = .047$, CFI = .896, RMSEA = .067, SRMR = .078]. It was hypothesised that associations between ASRS and deficits in gF are due to impaired working memory and IIV. As such, it was predicted that the ASRS-CCFIT relationship would be fully mediated by working memory and processing speed. Removing the ASRS-CCFIT path from the model did not significantly worsen fit ($\Delta\chi^2 = 1.01, \Delta df = 1, p = .31$); therefore, the relationship was no longer significant and was fully mediated.

In order to determine whether working memory and/or intraindividual variability were mediators, further testing was conducted. Removal of the ASRS-working memory ($\Delta\chi^2 = 6.16, \Delta df = 1, p = .013$) and working memory-CCFIT ($\Delta\chi^2 = 16.49, \Delta df = 1, p < .001$) paths both significantly worsened model fit, indicating that these paths are both significant, and that working memory mediates the ASRS-CCFIT relationship. The same procedure was then conducted with the IIV factor. Removal of both the ASRS-intra-individual variability ($\Delta\chi^2 = 4.28, \Delta df = 1, p = .039$) and the IIV-CCFIT ($\Delta\chi^2 = 19.51, \Delta df = 1, p < .001$) paths significantly worsened model fit, indicating that these paths are both significant, that the ASRS is associated with IIV, that IIV is predictive of CCFIT, and that both working memory and IIV fully mediate the ASRS-CCFIT relationship. The final model reported acceptable fit indices [$\chi^2(18) = 28.81, p = .051$, CFI = .90, RMSEA = .065, SRMR = .080], and is presented in Figure 2. Additionally, the model yielded a $R^2 = .45$. Thus, 45% of the variance in the CCFIT was accounted for by the model that included working memory and IIV as predictors of the CCFIT.

This process was then repeated for the ASRS Hyperactivity variable, and similar results were observed. ASRS Hyperactivity did not significantly predict CCFIT ($\Delta\chi^2 = 0.04, \Delta df = 1, p = .84$), and this association was fully mediated by both working memory (WM-CCFIT: $\Delta\chi^2 = 20.90, \Delta df = 1, p < .001$; though ASRS-WM was not significant, $\Delta\chi^2 = 3.54$,

$\Delta df = 1, p = .060$) and IIV (ASRS-IIV: $\Delta\chi^2 = 4.66, \Delta df = 1, p = .031$; IIV-CCFIT: $\Delta\chi^2 = 19.56, \Delta df = 1, p < .001$). The final model fit statistics were low [$\chi^2(19) = 33.48, p = .021$, CFI = .86, RMSEA = .074, SRMR = .090].

Discussion

The aim of this study was to determine if associations between ADHD symptomology and fluid intelligence were mediated by working memory and response time IIV. It was hypothesised that there would be ADHD-related deficits in gF, working memory, and IIV. It was also hypothesised that gF, working memory, and IIV are all related to each other. Thirdly, it was hypothesised that ADHD-related deficits in gF are only observable due to deficits in working memory and IIV. On the whole, these hypotheses were generally supported.

The first hypothesis of the current study was that ADHD-related deficits would be observed in the three cognitive domains of interest: gF, working memory, and IIV. Previous research has shown behavioural deficits in ADHD patient groups in each of these three constructs (Dige & Wik, 2005; Kofler et al., 2013; Schweitzer et al., 2006), and the results of the current study were consistent with these findings. Given that neuroimaging research has found that ADHD is associated with significant structural and functional differences across the frontoparietal network (which is implicated in successful higher-order cognition; Bush, Valera, & Seidman, 2005; Niendam et al., 2012; Seidman et al., 2006), it follows that these constructs may be impacted as a result.

The second hypothesis stated that gF, working memory, and IIV would all be related to one another. While gF was associated with both working memory and IIV, supporting previous research that have examined the relationship between the two constructs (e.g., Ackerman et al., 2005; Jensen, 1992), in our study, the correlation between working memory

and IIV was not significant. Brydges et al. (2017) found a significant association between working memory and processing speed factors when processing speed was measured with median RTs. However, Jensen (1992) suggested that ‘traditional’ processing speed (i.e., average RT) and RT variability, may be two different constructs, as these measures shared significant proportions of variance but were both significantly associated with general intelligence. It may well be the case that traditional processing speed (i.e., how fast a response is on average) is not significantly affected by ADHD or that processing speed is differentially affected by different ADHD subtypes (Nigg et al., 2005). In addition, research has shown that in complex, cognitively demanding tasks (e.g., novel rule-learning paradigms) and/or tasks that require prolonged sustained attention (e.g., the SART or ANT task), IIV (or lapses in attention) and gF are highly correlated, but only for infrequent events (e.g., rarely presented trials), with little or no relationship observed between gF and IIV for the most commonly presented trial type (Roberts, Jones, Davis, Ly, & Anderson, 2014). Future research should attempt to examine the separability of average RT and RT consistency in a clinical sample, and how each are modulated by task demands in order to better understand and examine their effect on ADHD sub-types.

The third hypothesis was that ADHD-related deficits in gF would only be observable due to deficits in working memory and IIV. That is, the ADHD-gF association would be fully mediated by working memory and IIV. This hypothesis was supported. As previously stated, previous research has suggested close associations between working memory, IIV, and gF from both behavioural (e.g., Ackerman et al., 2005; Jensen, 1992) and neuroimaging (MacDonald et al., 2006; Niendam et al., 2012) perspectives. Hence, the results of the current study imply that decreased gF could possibly be an indirect result of ADHD, although we acknowledge the study design does not imply causality. That is, ADHD is associated with

impaired working memory and increased IIV in response time, which in turn leads to decreased gF.

These results show that both working memory and IIV are important predictors of ADHD symptomology. In 1926, Henry Head stated that “an inconsistent response is one of the most striking consequences of lesions to the cerebral cortex” (p. 145). Although our sample was a nonclinical sample of university students – and the results of the study should be qualified as such – there are potential clinical implications, as simply examining the mean or median RT of a cognitive task may potentially give a misleading picture for diagnosis, assessment, and treatment of ADHD, although this needs to be tested in a clinical sample. As Brydges et al. (2017) stated, it is possible that low gF could behaviourally manifest in a similar manner to ADHD. That is, it is possible that gF is not directly affected by ADHD symptomology, but multiple predictors of gF – including working memory and IIV, based on the results of the current study – are impaired. Difficulties in clinical diagnosis of ADHD may be somewhat due to the significant conceptual overlap and behavioural similarity between low gF and ADHD symptoms.

The results of the current study are congruent with the broader neuropharmacological perspective on ADHD and clinical disorders involving the catecholamines; dopamine and noradrenaline. Both dopamine and noradrenaline are known to play crucial roles in executive attention and motor control (Braver & Cohen, 2000; Paus, 2001), and are both heavily implicated in ADHD and sustained attention (Biederman & Spencer, 1999; del Campo, Chamberlain, Sahakian, & Robbins, 2011; Levy, 1991). More importantly, medications used in the treatment of ADHD that modulate levels of dopamine and noradrenaline, such as methylphenidate, have been shown to improve IIV in RT in healthy controls (Nandam et al., 2011; Noreika, Falter, & Rubia, 2013). Reduced consistency of responding is also seen in a wide spectrum of conditions associated with these neurotransmitters, such as old age (e.g.,

Bielak & Anstey, 2015; Strauss, Biellak, Bunce, Hunter, & Hultsch, 2007), Parkinson's disease (Scatton, Javoy-Agid, Rouquier, Dubois, & Agid, 1983), Schizophrenia (Davis, Khan, Ko, & Davidson, 1991), and Depression (Dunlop & Nemeroff, 2007). In summary, although our sample was a nonclinical sample of university students, the results are consistent with the broader neuropharmacological framework of catecholamine dysfunction in ADHD.

The results of the current study indicate several potentially fruitful avenues for future research. First, as Brydges et al. (2017) stated, future research could examine the validity of the results, and this mediation model in a group of clinically diagnosed ADHD patients. One limitation of the current study was the use of the general population, which restricts the validity of the findings with regards to clinical groups, especially given that the current sample was mostly female and well-educated (see Siedman, 2006, and Theiling & Petermann, 2016, however, who suggest that gender differences in ADHD symptomology are minimal). Alternatively, having a wide range of symptoms scores allows for greater variation than a binomial distribution of diagnosed with ADHD vs. not diagnosed with ADHD. Given that self-report symptom scores have been found to correlate highly with clinicians' diagnoses (Adler et al., 2006; Silverstein et al., 2017), future research should consider using symptom severity in a group of ADHD patients, rather than simply examining whether or not an individual has been diagnosed. Additionally, examining this mediation model in a sample of children may be of benefit. Given that the prevalence of ADHD is as high as 7.1% in children (Willcutt, 2012), examining a potential hierarchical model with a view towards developing attention-training programs could be a fruitful area of research.

In conclusion, the current study has shown that both working memory and IIV fully mediate deficits in gF that are associated with ADHD symptoms, albeit in a nonclinical sample. The results of this study highlight the need for clinicians and researchers to examine the consistency of behaviour in psychometric assessment of speeded-tasks, especially when

treating or investigating complex psychiatric disorders associated with abnormal functioning of the brain's dopaminergic and noradrenergic systems. Although conducted with healthy adults from the normal population, the results of the current study suggest that ADHD symptomology and its relationship to gF are likely to be due to normal variation between individuals in their ability to store information in working memory and respond consistently.

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Table 1

Descriptive statistics of the ASRS and cognitive measures used in the analyses (N = 142)

Measure	<i>M</i> (<i>SD</i>)	Range
ADHD symptoms		
ASRS Total ^a (/72)	31.70 (11.39)	4 – 65
ASRS Inattentive ^a (/36)	16.96 (6.29)	3 – 35
ASRS Hyperactive ^a (/36)	14.51 (6.31)	0 – 32
Intraindividual variability		
Simple Motor Response ^b	61.94 (20.79)	32.61 – 153.18
Two-choice Motor Response ^b	120.56 (46.90)	40.40 – 271.55
Four-choice Motor Response ^b	122.61 (83.47)	27.41 – 450.03
Working memory		
Backward Digit Span ^c (/16)	6.51 (2.71)	1 – 14
Corsi ^c (/16)	9.02 (1.64)	5 – 14
Memory Span ^d (/9)	4.83 (1.03)	3 – 8
Fluid intelligence		
CCFIT ^e (/46)	27.23 (4.48)	14 – 39
Median Reaction Time		
Simple Motor Response ^b	318.04 (26.22)	256.0 – 389.0
Two-choice Motor Response ^b	505.81 (71.31)	390.0 – 725.5
Four-choice Motor Response ^b	475.13 (80.18)	319.0 – 740.0

Note. ^a Number of points. ^b msec. ^c Total trials correct. ^d Span. ^e Total items correct.

Table 2

Correlations between the ASRS and cognitive measures used in the analyses (N = 142)

	1	2	3	4	5	6	7	8	9	10
1. ASRS Total	-									
2. ASRS Inattentive	.89**	-								
3. ASRS Hyperactive	.90**	.60**	-							
4. Simple Motor Response	.16	.13	.19*	-						
5. Two-choice Motor Response	.13	.18*	.05	.41**	-					
6. Four-choice Motor Response	.12	.07	.15	.23**	.22*	-				
7. Backward Digit Span	-.23**	-.27**	-.15	.08	-.16	-.17*	-			
8. Corsi	-.11	-.08	-.13	-.22**	-.26**	-.15	.12	-		
9. Memory Span	-.05	-.03	-.05	-.08	-.07	-.03	.26**	.09	-	
10. CCFIT	-.17*	-.11	-.20*	-.34**	-.28**	-.23**	.31**	.20*	.24**	-

Note. * $p < .05$. ** $p < .01$.

Figure 1. The estimated six-construct model. Single-headed arrows have standardised factor loadings next to them. Double-headed arrows have correlations next to them. Emboldened lines and values are significant to $p < .05$. The thin lines indicate non-significant correlations. The numbers on the right are the squared multiple correlations for each indicator.

Figure 2. The final mediation model. The thin line from ASRS Total to CCFIT indicates a non-significant regression weight. All other coefficients are significant to $p < .05$.