Working memory – not processing speed – mediates fluid intelligence deficits associated with attention deficit/hyperactivity disorder symptoms

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Attention deficit/hyperactivity disorder (ADHD) is a psychological condition characterized by inattention and hyperactivity. Cognitive deficits are commonly observed in ADHD patients, including impaired working memory, processing speed, and fluid intelligence, the three of which are theorized to be closely associated with one another. In this study, we aimed to determine if decreased fluid intelligence was associated with ADHD, and was mediated by deficits in working memory and processing speed. This study tested 142 young adults from the general population on a range of working memory, processing speed, and fluid intelligence tasks, and an ADHD self-report symptoms questionnaire. Results showed that total and hyperactive ADHD symptoms correlated significantly and negatively with fluid intelligence, but this association was fully mediated by working memory. However, inattentive symptoms were not associated with fluid intelligence. Additionally, processing speed was not associated with ADHD symptoms at all, and was not uniquely predictive of fluid intelligence. The results provide implications for working memory training programs for ADHD patients, and highlight potential differences between the neuropsychological profiles of ADHD subtypes.

Attention deficit/hyperactivity disorder (ADHD) is a psychological disorder characterized 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). Given that previous research has suggested associations between these cognitive constructs from behavioural (Demetriou et al., 2014; Redick, Unsworth, Kelly, & Engle, 2012) and neuroimaging (Duncan & Owen, 2000) perspectives, it is possible that deficits in intelligence associated with ADHD are in fact caused by deficits in other associated higher order cognitive processes (such as working

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memory and processing speed), rather than ADHD directly impairing cognitive functioning.

Fluid intelligence (gF) is the ability to solve novel problems, independently of any previously acquired knowledge (Cattell, 1963). Previous research has shown that adult ADHD patients are at increased risk of suffering deficits in gF (Goodwin, Gudjonsson, Sigurdsson, & Young, 2011). This impairment is thought to be due to structural differences in the prefrontal cortex (PFC), as the integrity and activation of the PFC (most notably its lateral surface) is heavily implicated in tasks requiring gF (Duncan, 2005; Duncan & Owen, 2000). Structural magnetic resonance imaging studies have found that adult ADHD patients have significantly smaller PFC volume than healthy controls (Seidman et al., 2006; Valera, Faraone, Murray, & Seidman, 2007). From a behavioural perspective, Dige and Wik (2005) tested 48 adult ADHD patients and 48 age-matched controls, and found that performance on Raven’s Advanced Progressive Matrices (a commonly used measure of gF) was significantly lower in the ADHD patient group, even after controlling for IQ. Hence, a compromised PFC, which is common in ADHD patients, is associated with decreased gF.

Working memory is the ability to store, manipulate, and update incoming information or stimuli (Baddeley & Hitch, 1974). There are deficits in working memory that are associated with ADHD in adults, which are thought to be caused by structural and functional neural differences (Bush, Valera, & Seidman, 2005). Working memory (and other executive functions) are commonly theorized to require the activation of a wide range of neural regions in a frontoparietal network (Niendam et al., 2012), including PFC, anterior cingulate cortex and posterior parietal cortex. These regions have been found to display decreased activation when completing working memory tasks (Bush et al., 2005; Wolf et al., 2009). These differences have also been supported behaviourally. Schweitzer, Hanford, and Medoff (2006) tested 18 typical adult controls, 17 combined-type adult ADHD patients, and 16 adult inattentive-type ADHD patients, and found that the ADHD patients had impaired performance in comparison to typical controls on several working memory tasks, suggesting that ADHD patients may suffer from working memory deficits. That is, there appears to be associations between ADHD in adults, functional and structural neuroanatomy of the frontoparietal network, and behavioural performance on working memory tasks.

Processing speed refers to the speed at which individuals can perform cognitive tasks (Sheppard & Vernon, 2008; Vernon, 1983). Previous research has shown that there are deficits in processing speed that are associated with ADHD in adults (Boonstra, Oosterlaan, Sergeant, & Buitelaar, 2005). From a neuroimaging perspective, this is thought to be due to white matter abnormalities and disrupted neuroanatomical connectivity (Konrad & Eickhoff, 2010). White matter refers to a myelinated neuron, through which neural impulses travel much faster than in non-myelinated neurons (grey matter). As such, abnormalities in white matter can impair processing speed. This is supported from a behavioural perspective, as Johnson et al. (2001) tested 56 adult ADHD patients and 38 typical control adult participants, and found that reaction times were significantly longer in the ADHD group. Additionally, Boonstra et al. (2005) conducted a meta-analysis on a range of neuropsychological constructs, and generally found medium effects (Cohen’s $d \approx .6$) of ADHD on measures of processing speed. Furthermore, Nigg et al. (2005) examined this association in more detail by examining whether processing speed was affected differentially by the hyperactive and inattentive subtypes of ADHD in 195 young adults (105 with ADHD and 90 controls), and found that hyperactivity was positively associated with processing speed (i.e., increased severity of hyperactivity...
symptoms was associated by faster performance), but inattentiveness was negatively associated (i.e., increased inattentiveness led to slower performance). These studies imply that there are processing speed deficits in ADHD patients, although processing speed may be differentially affected by different ADHD subtypes.

There is a large amount of research that examines associations between working memory and gF (Ackerman, Beier, & Boyle, 2005; Conway, Kane, & Engle, 2003; Engle, Tuholski, Laughlin, & Conway, 1999; Friedman et al., 2006; Kyllonen & Christal, 1990), processing speed and gF (Kail & Salthouse, 1994; Salthouse, 1996; Sheppard & Vernon, 2008), and working memory and processing speed (Fisk & Warr, 1996; Salthouse, 1992; Schmiedek, Oberauer, Wilhelm, Süß, & Wittmann, 2007; Wilhelm & Oberauer, 2006). Additionally, there is behavioural research that suggests that both are predictors of gF (Demetriou et al., 2014; Redick et al., 2012), and the three constructs are frequently associated with frontoparietal activation (Duncan & Owen, 2000; Niendam et al., 2012). Given these similarities and close relationships between working memory, processing speed, and gF, it could be the case that ADHD-related deficits in gF are only apparent due to impaired working memory and/or processing speed. As such, this study aimed to determine if associations between ADHD symptoms and fluid intelligence were mediated by working memory and processing speed. To the best of the authors’ knowledge, this approach to examining the neuropsychological profile of ADHD is completely novel in any population. It was hypothesized that ADHD is associated with deficits in gF, working memory, and processing speed. Hence, it was predicted that the severity of ADHD symptoms combined across hyperactivity and inattentiveness subscores would negatively predict gF and working memory performance (i.e., lower scores), and would positively predict processing speed (i.e., longer reaction times). It was also hypothesized that gF, working memory and processing speed 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 processing speed would be predictive of gF in a structural equation model (SEM). Additionally, it was hypothesized that ADHD-related deficits in gF are only observable due to deficits in working memory and processing speed. Hence, it was predicted that the ADHD-gF association would be fully mediated in an SEM by working memory and processing speed. Lastly, the same analyses were conducted on hyperactivity and inattentiveness subscores and had the same hypotheses, with one exception: It was hypothesized that hyperactivity would be positively associated with processing speed, whereas inattentiveness would be negatively associated.

**Method**

**Ethics statement**

Approval for the study was provided by the Human Research Ethics Office of the Australian University the study was conducted at. Prior to testing, participants were provided with an information sheet and were given the opportunity to ask any questions about the study, and provide written informed consent.

**Participants**

Participants were 142 adults aged 18–40 years \( (M = 22.24, SD = 4.20) \), of which 91 were female (64.08%). Four additional participants completed the testing procedure, but were
excluded on grounds of age (all were older than 40). 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 [CCFIT] has a standardized mean of 100 and SD of 24, this is within normal range). Participants were offered a choice of 1.5 participation credit hours to partially fulfil course requirements, or entry to a draw to win a $50 gift card. All participants demonstrated competency in English and reported normal or corrected-to-normal hearing and vision.

**Materials**
All working memory and processing speed 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 programme.

**ADHD Symptoms**

**World Health Organization adult ADHD self-report scale (ASRS)**
The ASRS is a self-administered questionnaire designed to measure current ADHD symptoms in adults (Kessler et al., 2005). 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 to 72) was used as the indicator for ADHD symptoms, with each subtype having a range of 0–36.

**Processing speed**

**Simple reaction time**
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, the inter-stimulus interval varied between 250 and 2,500 ms. Responses faster than 150 ms or slower than 3,000 ms were considered too fast and too slow, respectively, and were excluded from analyses. Median reaction time was used as the indicator for processing speed.

**Two-choice reaction time**
In the two-choice reaction time task, a fixation cross would appear in the centre of the computer screen for 1,250 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. Median reaction time was used as the indicator for processing speed.
Four-choice reaction time
In the four-choice reaction time task, a $2 \times 2$ 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. Median reaction time was used as the indicator for processing speed.

Working memory
Backward digit span
Participants were required to 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, which 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 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
Cattell Culture Fair Intelligence Test (Scale 3, Form A)
The CCFIT is a commonly used, non-verbal measure of gF (Cattell, 1973). 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.

Procedure
Testing took place in laboratory settings in a single session that lasted approximately 1.5 hr. 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: Cattell; Two-choice Reaction Time; Backward Digit Span; Simple Reaction Time, Corsi; Four-Choice Reaction Time; Memory Span; ASRS.
Transformations and outlier analysis
The raw distributions of the eight 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 >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, $\chi^2(45) = 48.64; p = .33$, indicating that missing data were missing completely at random. As such, these scores were estimated using the expectation maximization method in SPSS.

Statistical analysis
Amos 20 (Arbuckle, 2011) was used to estimate latent-variable models. In both CFA and SEM, several fit indices were used to evaluate the fit of each model to the data. The $\chi^2$ statistic was used, as this is a commonly used statistic in CFA and SEM (Blunch, 2008). Model fit is considered to be acceptable if the $p$ value associated with the $\chi^2$ statistic is $>0.05$. Three other fit indices recommended by Hu and Bentler (1998) were also used: Bentler’s comparative fit index (CFI), the root-mean-square error of approximation (RMSEA), and the standardized root-mean residual (SRMR). The criteria for excellent model fit based on these indices is $>.95$, $<.05$, and $<.05$ respectively. However, models are acceptable with respective values of .90, .08 and .08 (Blunch, 2008). Significance of correlation and path coefficients was determined by conducting $\chi^2$ 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 6-construct CFA model was created, with correlations between the two latent variables (processing speed and working memory) and four manifest variables (the three ASRS scores and CCFIT) all free to vary. It was found that the ASRS total and hyperactivity scores significantly correlated with the CCFIT, but the ASRS inattentiveness score did not. That is, removing these correlations one at a time (i.e., constraining it to $r = 0$) made the model fit significantly worse for the total and hyperactivity correlations ($\Delta \chi^2 = 4.31, \Delta df = 1, p = .038$ and $\Delta \chi^2 = 6.06, \Delta df = 1, p = .014$ respectively); therefore, the correlations are significant and kept in the model. Conversely, when the ASRS inattentive-CCFIT correlation was removed, the model fit was not significantly worse ($\Delta \chi^2 = 1.85, \Delta df = 1, p = .17$), so was removed from the model. Additionally, the correlations between the
three ASRS variables and the processing speed factor were all found to be non-significant (Total: $\Delta \chi^2 = 0.00$, $\Delta df = 1$, $p = 1.00$; Inattentiveness: $\Delta \chi^2 = 0.43$, $\Delta df = 1$, $p = .51$; Hyperactivity: $\Delta \chi^2 = 0.02$, $\Delta df = 1$, $p = .89$), so were removed from the model. All other correlations were significant to $p < .05$. The final model reported excellent fit indices, $\chi^2(28) = 24.62$, $p = .65$, CFI = 1.00, RMSEA = .000, SRMR = .049, and is shown in Figure 1.

**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 speed of processing (which were correlated with each other).

For the ASRS total score, initial model fit statistics were excellent, $\chi^2(16) = 15.05$, $p = .52$, CFI = 1.00, RMSEA = .000, SRMR = .045. It was hypothesized that associations between ASRS and deficits in gF are due to impaired working memory and processing speed. As such, it was predicted that the ASRS-CCFIT relationship would be fully mediated by working memory and processing speed. Removal of the ASRS-CCFIT path did not significantly worsen model fit ($\Delta \chi^2 = 0.19$, $\Delta df = 1$, $p = .66$); therefore, the relationship was no longer significant and was fully mediated.

To determine whether working memory and/or processing speed are mediators, further testing was conducted. Removal of the ASRS-working memory ($\Delta \chi^2 = 9.00$, $\Delta df = 1$, $p = .003$) and working memory-CCFIT ($\Delta \chi^2 = 10.27$, $\Delta df = 1$, $p = .001$) paths both significantly worsened model fit, indicating that these paths are both significant and

### Table 1. Descriptive statistics of the attention deficit/hyperactivity disorder self-report scale (ASRS) and cognitive measures used in the analyses ($N = 142$)

<table>
<thead>
<tr>
<th>Measure</th>
<th>$M$ (SD)</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Attention deficit/hyperactivity disorder symptoms</strong></td>
<td></td>
<td></td>
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<tr>
<td>ASRS Total$^a$ (/72)</td>
<td>31.66 (11.63)</td>
<td>4–65</td>
</tr>
<tr>
<td>ASRS Inattentive$^a$ (/36)</td>
<td>16.93 (6.42)</td>
<td>3–35</td>
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<tr>
<td>ASRS Hyperactive$^a$ (/36)</td>
<td>14.51 (6.44)</td>
<td>0–32</td>
</tr>
<tr>
<td><strong>Processing speed (ms)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Simple reaction time</td>
<td>318.04 (26.22)</td>
<td>256.00–389.00</td>
</tr>
<tr>
<td>Two-choice reaction time</td>
<td>505.81 (71.31)</td>
<td>390.00–725.50</td>
</tr>
<tr>
<td>Four-choice reaction time</td>
<td>475.13 (80.18)</td>
<td>319.00–740.00</td>
</tr>
<tr>
<td><strong>Working memory</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Backward digit span$^b$ (/16)</td>
<td>6.47 (2.90)</td>
<td>1–14</td>
</tr>
<tr>
<td>Corsi$^b$ (/16)</td>
<td>9.02 (1.64)</td>
<td>5–14</td>
</tr>
<tr>
<td>Memory span$^c$ (/9)</td>
<td>4.83 (1.04)</td>
<td>3–8</td>
</tr>
<tr>
<td><strong>Fluid intelligence</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cattell Culture Fair Intelligence Test$^d$ (/46)</td>
<td>27.23 (4.48)</td>
<td>14–39</td>
</tr>
</tbody>
</table>

**Notes.** $^a$Number of points.  
$^b$Total trials correct.  
$^c$Span.  
$^d$Total items correct.
Table 2. Correlations between the attention deficit/hyperactivity disorder self-report scale (ASRS) and cognitive measures (N = 142)

<table>
<thead>
<tr>
<th></th>
<th>1</th>
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<th>3</th>
<th>4</th>
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<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. ASRS Total</td>
<td></td>
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<td></td>
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<td>1</td>
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<tr>
<td>2. ASRS Inattentive</td>
<td>.89**</td>
<td></td>
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<td>3. ASRS Hyperactive</td>
<td>.90**</td>
<td>.60**</td>
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<td>4. Simple reaction time</td>
<td>.02</td>
<td>.03</td>
<td>.01</td>
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<td>5. Two-choice reaction time</td>
<td>.04</td>
<td>.08</td>
<td>-.01</td>
<td>.43**</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>6. Four-choice reaction time</td>
<td>-.04</td>
<td>-.07</td>
<td>.00</td>
<td>.29**</td>
<td>.50**</td>
<td></td>
<td></td>
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<tr>
<td>7. Backward digit span</td>
<td>-.24**</td>
<td>-.26**</td>
<td>-.18*</td>
<td>-.11</td>
<td>-.30**</td>
<td>-.14</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>8. Corsi</td>
<td>-.11</td>
<td>-.08</td>
<td>-.13</td>
<td>-.11</td>
<td>-.31**</td>
<td>-.25**</td>
<td>.12</td>
<td></td>
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<tr>
<td>9. Memory span</td>
<td>-.05</td>
<td>-.03</td>
<td>-.05</td>
<td>-.04</td>
<td>-.16</td>
<td>-.07</td>
<td>.27**</td>
<td>.09</td>
<td></td>
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</tr>
<tr>
<td>10. Cattell Culture Fair Intelligence Test</td>
<td>-.17*</td>
<td>-.11</td>
<td>-.21*</td>
<td>-.20*</td>
<td>-.32**</td>
<td>-.29**</td>
<td>.31**</td>
<td>.20*</td>
<td>.24**</td>
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</tbody>
</table>

Note. *p < .05; **p < .01.
that working memory mediates the ASRS-CCFIT relationship. The same procedure was then conducted with the processing speed factor. However, removal of neither the ASRS-processing speed ($\Delta \chi^2 = 0.12$, $\Delta df = 1$, $p = .73$) nor that processing speed-CCFIT ($\Delta \chi^2 = 0.03$, $\Delta df = 1$, $p = .87$) paths significantly worsened model fit, indicating that these paths are both non-significant, that the ASRS is not associated with processing speed, that processing speed is not predictive of CCFIT, and that working memory fully mediates the ASRS-CCFIT relationship. The final model reported excellent fit indices, $\chi^2(19) = 15.39$, $p = .70$, CFI = 1.00, RMSEA = .000, SRMR = .046, and is presented in Figure 2.

**Figure 1.** The estimated 6-construct model. Single-headed arrows have standardized factor loadings next to them. Double-headed arrows have correlations next to them. All values next to solid lines are significant to $p < .05$. The dotted lines indicate non-significant correlations. The numbers on the right are the squared multiple correlations for each indicator.
This process was then repeated for the ASRS Hyperactivity variable, and the same results were observed. ASRS Hyperactivity did not significantly predict CCFIT ($\Delta \chi^2 = 0.001$, $\Delta df = 1$, $p = .97$), and this association was fully mediated by working memory (ASRS-WM: $\Delta \chi^2 = 6.25$, $\Delta df = 1$, $p = .01$; WM-CCFIT: $\Delta \chi^2 = 12.96$, $\Delta df = 1$, $p < .001$). ASRS did not predict processing speed ($\Delta \chi^2 = 0.002$, $\Delta df = 1$, $p = .96$), and processing speed did not predict CCFIT ($\Delta \chi^2 = 0.16$, $\Delta df = 1$, $p = .69$). The final model fit statistics were excellent, $\chi^2(19) = 13.26$, $p = .83$, CFI = 1.00, RMSEA = .000, SRMR = .042.

**Discussion**

The aim of this study was to determine if associations between ADHD symptoms and fluid intelligence were mediated by working memory and processing speed. It was hypothesized that there would be ADHD-related deficits in gF, working memory, and processing speed. Previous research has shown behavioural deficits in adult ADHD patient groups in each of these three cognitive domains (Boonstra *et al.*, 2005; Dige & Wik, 2005; Schweitzer *et al.*, 2006). This hypothesis was supported with regard to gF and working memory, but not for processing speed. It is possible that a non-significant correlation between ADHD symptoms and processing speed was observed due to the different subtypes of ADHD having opposite effects on processing speed. Nigg *et al.* (2005) found that in comparison to a typical control group, inattentive-type ADHD patients had
significantly longer response times on a processing speed task, whereas hyperactive-type ADHD patients had significantly shorter response times, likely to be due to high levels of impulsiveness. In this study, however, it was found that neither subtype was significantly associated with processing speed.

It was also hypothesized that gF, working memory, and processing speed are all related to each other. As expected, a CFA found significant correlations between these three constructs; however, it was found that processing speed did not significantly predict gF in an SEM, implying that any variance common to processing speed and gF can be accounted for by working memory. Conway, Cowan, Bunting, Therriault, and Minkhoff (2002) also reported that processing speed was not a significant predictor of gF in 120 young adults, and speculated that previous research that had found links between the two constructs ‘did not consider the working memory demands of the tasks employed’ (p. 178). That is, by administering relatively complex processing speed tasks, some degree of working memory was also extracted in the analyses conducted, therefore potentially inflating the association between processing speed and gF. Like Conway et al. (2002), however, the current study employed simple processing speed measures which, while correlating with gF, do not contribute any unique predictive power to gF.

Thirdly, it was hypothesized that ADHD-related deficits in gF are only observable due to deficits in working memory and processing speed. Again, this hypothesis was partially supported. It was found that working memory fully mediated the ADHD-gF relationship, but ADHD symptoms were not significantly predictive of processing speed, and processing speed was not significantly predictive of gF. Previous research has suggested close associations between working memory, processing speed and gF from both behavioural (Ackerman et al., 2005; Conway et al., 2003; Friedman et al., 2006) and neuroimaging (Duncan & Owen, 2000) perspectives. Hence, the results of the current study imply that decreased gF is only an indirect result of ADHD. That is, ADHD is associated with impaired working memory, which in turn leads to decreased gF.

The results of this study highlight that complex tasks (such as those employed to measure working memory and gF) appear to become increasingly difficult as ADHD symptoms become increasingly severe, yet performance on relatively simple tasks (i.e., those employed to measure processing speed) are unimpaired. Similarly, a relatively simple construct (processing speed) is not predictive of gF, whereas a relatively complex one (working memory) is. Although processing speed has been found to not be associated with ADHD at all, and is not predictive of gF in this study, it is possible that Conway et al. (2002) alluded to a potential cause of this – ADHD symptoms, regardless of the subtype, do not cause any impairments in simple tasks/constructs, but do cause impairments in more complex tasks/constructs. Previous research has found that higher correlations between gF and processing speed are observed when more complex processing speed tasks are used, in comparison to simple processing speed tasks (Jensen, 1998). Conway et al. interpreted this as higher correlations between gF and processing speed occur when greater demands are placed on memory and attention processes in the processing speed task(s). Hence, processing speed is not associated with gF as such, rather the attentional component of processing speed tasks that is predictive of gF (Conway et al., 2002; Cowan, 1998). Within the context of the current study, this presents a potential cascade effect of ADHD symptoms on gF. Attention is commonly theorized to be associated with working memory (Awh & Jonides, 2001; Kane, Bleckley, Conway, & Engle, 2001), which in turn has been found to be associated with gF (Ackerman et al., 2005; Friedman et al., 2006), and the PFC is thought to be implicated in the application of these three constructs (Engle, Kane, & Tuholski, 1999; Kane & Engle, 2002). Therefore, in the case of ADHD, it is
possible that attention deficits cause impaired working memory, which in turn causes decreased gF, which could have implications for the efficacy of working memory training programs in ADHD populations. Although this has not been directly tested, Cornoldi, Giofrè, Calgaro, and Stupigga (2013) administered a gF intelligence test and working memory measures that required either high or low degrees of attentional control in children diagnosed with ADHD and a control group. No differences in gF or working memory requiring low levels of attentional control were observed; however, the ADHD group performed significantly worse on the working memory tasks requiring high levels of attentional control, suggesting that ‘basic’ speed is not associated with ADHD symptoms, but attentional processes and working memory are. It should be explicitly stated, though, that this is purely speculative at this point, and that – assuming the working memory training programs are effective – then increases in gF should be observed in ADHD patients as a result of the training. Previous research in this area suggests that early interventions are effective (Re, Capodieci, & Cornoldi, 2015), but attempts in adult samples have not been as successful (Stern, Malik, Pollak, Bonne, & Maeir, 2014). Future research could use latent-variable analyses in a similar vein to the current study to test this theory.

A corollary of this is the possibility that low gF could masquerade as ADHD, and there is no real relationship between gF and ADHD symptoms. That is, low gF is associated with deficits in other higher order cognitive processes that are symptomatic of ADHD, such as executive functioning (Ardila, Pineda, & Rosselli, 2000), working memory (Ackerman et al., 2005), and/or attention (Schweizer, Moosbrugger, & Goldhammer, 2005). As a result, it is possible that there is no relationship between gF and ADHD, despite the potential for highly similar neuropsychological profiles between low gF and ADHD patients. Indeed, clinicians have highlighted the difficulty in ADHD diagnosis. This may stem in part from the large conceptual overlap and behavioural similarity between low gF and ADHD symptoms.

In addition to testing this hierarchical model of ADHD symptoms and cognition, future research could examine the validity of this 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 regard to clinical groups. In particular, the current sample was mostly female and well-educated, potentially limiting the representativeness of the sample. However, Seidman (2006) suggests that gender differences in ADHD symptomology are minimal, and this was also found in analyses of the current study. Alternatively, having a wide range of symptoms scores allows for greater variation than a binomial distribution of diagnosed with ADHD versus not diagnosed with ADHD. Given that self-report symptom scores have been found to correlate highly with clinicians’ diagnoses (Adler et al., 2006), perhaps future research could use symptom severity in a group of ADHD patients, rather than whether or not an individual has been diagnosed. Additionally, examining potential differences between ADHD subtypes may be of benefit. As previously mentioned, inattentive-type patients have been found to have slower processing speed, but hyperactive-type patients display intact processing speed (Nigg et al., 2005). Research investigating differences between these two subtypes, as well as the combined-type, could potentially shed new light on the neuropsychological profiles of ADHD patients within each subtype. A third potential avenue of future research is to test this (and/or the previously hierarchical) model in a sample of children. 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, this study has shown that working memory, but not processing speed, fully mediates deficits in gF that are associated with ADHD symptoms. Although not tested in this study, this is likely due to the compromised integrity of the PFC (Duncan & Owen, 2000). It is possible that processing speed was not associated with ADHD symptoms due to different neuropsychological profiles of subtypes of ADHD. Research that uses tasks with a larger attentional component may find a cascade effect of ADHD symptoms on cognition.

References


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