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Additive Neurocognitive Deficits in Adults with ADHD and Depressive Symptoms

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Abstract

The purpose of this study was to examine the possible additive neurocognitive deficits in adults with both ADHD and serious depressive symptoms. Participants were 54 university students who completed a psycho-educational assessment. Three groups were examined: a group with comorbid ADHD and elevated depressive symptoms (ADHD + DEP; N = 18); a group with ADHD only (N=18); and a group with elevated depressive symptoms only (DEP; N=18). Group differences were examined on a battery of neurocognitive tests. The ADHD + DEP group performed significantly worse than the other groups on processing speed tasks and delayed recall of conceptual verbal information, and significantly worse than the ADHD group on shifting tasks. Depressive symptom severity was significantly correlated with processing speed, verbal memory performance, and shifting in the ADHD and ADHD + DEP groups. Results suggest that the co-occurrence of ADHD and depressive symptoms in adults is associated with additional neurocognitive impairment.

Keywords: adults; ADHD; depression; neurocognition; processing speed; memory.

Additive Neurocognitive Deficits in Adults with ADHD and Depressive Symptoms

Attention-deficit/hyperactivity disorder (ADHD) is a developmental neurobehavioral disorder characterised by a variety of persistent behavioral symptoms including inattention, hyperactivity, and impulsivity (American Psychiatric Association, 2000). ADHD is commonly known as a disorder of childhood; however, it is estimated that approximately 4-5% of adults in the United States have ADHD (e.g. Kessler et al., 2005). As a result, research is increasingly focusing on the continued effects of ADHD throughout adulthood, with recent findings suggesting that behavioral characteristics of children with ADHD may manifest differently in adults. For example, Adler, Barkley, and Newcorn (2008) proposed that the hyperactivity and behavioral impulsivity seen in children with ADHD may manifest as mental restlessness and excessive talking in adults. Adults with ADHD may also demonstrate difficulties with sustaining attention and organization (Wilens, 2007), which can often lead to employment or financial difficulties, poor academic performance, interpersonal problems, and risk-taking behaviors (Adler et al., 2008).

In addition to the behavioral dysfunctions related to ADHD, a number of neurocognitive deficits have also been linked to this diagnosis. Specific neurocognitive deficits identified in children with ADHD have included frontal lobe dysfunction characterized by executive function deficits (see Sergeant, Geurts, & Oosterlaan, 2002 for review), as well as difficulties with visuo-spatial working memory (Westerberg, Hirvikoski, Forssberg, & Klingberg, 2004). Similar neurocognitive deficits have also been observed in adults with ADHD. Boonstra, Oosterlaan, Sergeant, and Buitelaar's (2005) meta-analytic review identified deficits in executive functioning areas such as inhibition and set shifting, as well as in non-executive functioning areas such as speeded word reading and color naming in adults with ADHD. A recent study by Müller and

colleagues (2007) also noted significant executive function deficits in adult patients with ADHD, who performed a full standard deviation lower than normal controls on the Tower of London task and a divided attention task (Müller et al., 2007). Neurocognitive deficits in numerous other areas such as verbal and visual memory, and divided attention can also be maintained by adult ADHD patients even while on medication (Schoelin & Engel, 2005). In fact, memory deficits have frequently been identified as areas of difficulty in adults with ADHD and can include deficits in verbal working memory (Lacene, 2004; Marchetta et al., 2008), spatial working memory (McLean et al., 2004), and long- and short-term visual memory (Dige & Wik, 2005; Schoelin & Engel, 2005). Both Müller and colleagues (2007) and Marchetta and colleagues (2008) reported medium effect sizes when examining the poor performance of adults with ADHD on tasks of visual memory and verbal working memory, respectively. Slowed processing speed has also been noted in some adults with ADHD (Lacene, 2004), especially during attentional conflict paradigms (McLean et al., 2004). For example, adults with ADHD performed two standard deviation units slower than controls on a task requiring individuals to attend and respond to relevant targets while inhibiting their responses to distractor targets (McLean et al., 2004).

The high rates of comorbid disorders found in adults with ADHD, however, can greatly complicate the assessment of the neurocognitive deficits that putatively drive many of the behavioral issues present in this population. Mood disorders appear to be the most common comorbid disorders found in those with ADHD, with as many as 25 to 35% of persons with ADHD also meeting criteria for comorbid major depressive disorder (MDD; McGough, 2005; Shekim, et al., 1990). Indeed, adolescents with ADHD may be 4 times more likely to develop depressive disorders than their peers in the general population (Pliszka, 1998), and 16% of adult

patients diagnosed with MDD in one clinical sample also had a diagnosis of childhood onset ADHD (Alpert et al., 1996). Adult ADHD populations also tend to suffer from more depressive symptoms than the general population (Chao, 2008; Rabiner, Anastopoulos, Costello, Hoyle, & Schwartzwelder, 2008; Rucklidge & Kaplan, 1997), as well as a greater likelihood of a diagnosis of dysthymia (Murphy, Barkley, & Bush, 2002). The effects of subsyndromal or mild depression in ADHD, however, are not yet well known.

In large part, studies examining adult ADHD with comorbid disorders have tended to focus on clinical presentation and severity of symptoms (Buckley et al., 2006; Fischer et al., 2007; Sprafkin, Gadow, Weiss, Schneider, & Nolan, 2007), as well as the behavioral or social outcomes of these comorbidities (Torgersen, 2006). Few studies have specifically examined neurocognitive functioning associated with both ADHD and common comorbid conditions such as anxiety or depression. The literature that exists in this area does, however, point to increased neurocognitive deficits in the area of working memory when ADHD is present along with anxiety (Schatz & Rostain, 2006), and indicates that adolescents with ADHD and conduct disorder/oppositional defiant disorder demonstrate marked inhibitory problems in selective attention (Pritchard, Neumann, & Rucklidge, 2008).

Although there is a paucity of research examining the neurocognitive deficits present in comorbid ADHD with mood disorders, this is likely to be a relevant comorbidity because depression alone is associated with a variety of neurocognitive deficits, many of which are similar to those seen in adults with ADHD. For example, numerous studies have identified executive dysfunction as a central deficit in young adults diagnosed with MDD (Egeland et al., 2003; Fossati et al., 1999; Smith, Muir, & Blackwood, 2006). In fact, depressed young adults perform similarly to adults with ADHD on trail-making tests and tests of verbal fluency

(Mahurin et al., 2006). Deficits in effortful information processing speed, however, have been well-established in the depression literature (Gorlyn et al., 2006, see Hartlage, Alloy, Vasquez & Dykman, 1993; Tsourtos, Thompson, & Stough, 2002), but not in the adult ADHD literature. These depression-related difficulties in processing speed may be partially accounted for by problems with attention that seem to also impact capacities in both visual and verbal memory (Basso & Bornstein, 1999; Hill, Keshavan, Thase, & Sweeney, 2004), both of which are also impaired in adult ADHD (Dige & Wik, 2005; Marchetta et al., 2008). Severity of depressive symptoms has also been reported to correlate with more pronounced neurocognitive impairments (Basso & Bornstein, 1999; Hill et al., 2004), but this has not been consistent across studies (Fossati et al., 1999; Stordal et al., 2004). Several reports also suggest an additive neurocognitive deficit when depression is comorbid with other disorders such as anxiety (Basso et al., 2007; Kaplan et al., 2006; Kizilbash, Vanderploeg, & Curtis, 2002).

Despite the fact that many adults with ADHD also experience depressive symptoms, and that both ADHD and depression have additional neurocognitive deficits when experienced with other mental disorders, the combined neurocognitive impact of these disorders has not been examined in adults to date. Thus, it is not known how the individual neurocognitive deficits associated with serious depressive symptoms and ADHD may interact and whether this interaction produces an additive effect. The purpose of the current study, therefore, was to examine the neurocognitive deficits found in adults with ADHD and clinically significant depressive symptoms by comparing their performance with that of individuals with ADHD alone or depressive symptoms alone. It was hypothesized that individuals with ADHD and clinically significant depressive symptoms would show pronounced deficits in specific neurocognitive areas known to be affected by both ADHD and depression such as processing speed, short-term,

long-term and working memory, and executive functioning and that these deficits would be more pronounced than in those with ADHD or elevated depressive symptoms alone.

Methods

Participants

Analyses for the current study were based on data drawn from a database of students who had completed a psycho-educational assessment for ADHD or other learning problems at a university-based assessment centre between 2002 and 2008. For the purposes of these analyses, we examined subjects who had an ADHD diagnosis, clinically significant depressive symptoms, defined as a T-score greater than 65 on the Personality Assessment Inventory Depression clinical scale (PAI-DEP, Morey, 1991), or both. Students who were identified as having a medical condition associated with compromised neurocognitive functions (e.g., head injury or acquired brain injury) were excluded from study analyses.

From the original database, three groups were created by matching subjects to the group with ADHD and depression. An ADHD group (ADHD; N = 18) was created by selecting students who were diagnosed with ADHD based on a clinical interview, a thorough psycho-educational assessment, and clinical observations. This group did not meet criteria for other DSM-IV Axis I disorders. A depressive symptom group (DEP; N = 18) was created by selecting students who scored above a T-score of 65 on the DEP clinical scale of the PAI. They did not meet diagnostic criteria for ADHD. The PAI cut-off was used because the referral questions at this centre did not always prompt a full DSM Axis I diagnostic interview, but the PAI was used as a standard part of assessment procedures. This cut-off of 1.5 standard deviation units represents students who had clinically significant depressive symptoms (Keiski, Shore, & Hamilton, 2007). Finally, a combined ADHD and depression group (ADHD+DEP; N = 18) was

created by selecting students who were both diagnosed with ADHD and who had a PAI-DEP T-score of 65 or above. Matching criteria, in order, were sex, age, and presence of treatment with stimulant medications, followed by ADHD subtype and severity of PAI DEP scores for the ADHD and ADHD+DEP groups, respectively. The mean age of the entire sample was 22.02 ($SD = 5.3$; range 18-43). The three groups were not significantly different in age ($F(2,52) = .01$; $p > .05$). Chi-square analysis revealed no significant differences in percent male among the ADHD (55.6%), DEP+ADHD (55.6%), and DEP (55.6%) groups, $\chi^2(2) = 0.0$, $p = .10$. Chi-square analyses revealed no significant differences in ADHD subtype between the two groups diagnosed with ADHD, $\chi^2(0) = 0.0$, $p = 1.0$, with 11.1% hyperactive, 55.6% inattentive, and 33.3% mixed subtype for both the ADHD and ADHD+DEP groups. There were no statistically significant differences in the proportion of individuals within each group who were taking any psychotropic medication, $\chi^2(2) = 2.9$, $p = .22$. There were also no significant differences in self-reported current substance abuse, $\chi^2(2) = 1.1$, $p = .11$.

Measures

Personality Assessment Inventory - Depression Clinical Scale (PAI - DEP, Morey, 1991).

The PAI is a 344-item multi-scale self-report measure that requires respondents to rate sentences regarding their personality, psychopathology and day-to-day functioning on a 4-point scale (0 = false to 3 = very true). The PAI contains a total of 22 scales (4 validity, 11 clinical, 5 treatment consideration, and 2 interpersonal scales). The DEP scale consists of 24 items and measures clinical features common to depression such as apathy, pessimism, and subjective feelings of unhappiness. The DEP scale further comprises three subscales: Cognitive (DEP-C), Affective (DEP-A), and Physiological (DEP-P). It should be noted here that the cognitive subscale refers primarily to maladaptive content of thought, as opposed to self-rated neurocognitive abilities,

except for one item referring to concentration difficulties. The PAI has been found to have high internal consistency, with median internal consistency coefficients ranging from 0.81 to 0.86 (Boyle & Lennon, 1994; Morey, 1991).

Wechsler Adult Intelligence Scale – 3rd Edition (WAIS-III; Wechsler, 1997a). The WAIS-III is one of the most widely-used standardized intelligence tests. It consists of 14 subtests that address a variety of cognitive areas. The three primary IQ scores of the WAIS-III are the Verbal IQ, the Performance IQ, and the Full Scale IQ, which is a combination of VIQ and PIQ. VIQ consists of scores on 6 subtests, while PIQ consists of scores on 5 subtests. The four main index categories are: Verbal Comprehension Index (VCI; 3 subtests), a measure of general verbal abilities such as verbal fluency, word knowledge, and verbal reasoning; Perceptual Organization Index (POI; 3 subtests), a general measure of non-verbal abilities including visual-motor skills, non-verbal reasoning, and problem-solving; Working Memory Index (WMI; 3 subtests), a measure of the ability to memorize new information in short-term memory in addition to concentration and cognitive manipulation of information; and Processing Speed Index (PSI; 2 subtests), a measure of speed of integration of information as well as attention, discrimination and accurate scanning of information.

Wechsler Memory Scale – 3rd Edition (WMS-III; Wechsler, 1997b). The WMS-III is designed to assess various aspects of learning and memory. It consists of eight primary index scores and four supplementary auditory process composites. In the centre in which this sample was tested, the Logical Memory subtests were administered to most individuals (ADHD, N = 8; DEP, N = 12; ADHD+DEP, N = 12). These are conceptual verbal memory tests that require the subject to listen to a story presented by the examiner and remember as much detail as possible

immediately after presentation (Logical Memory I), and after a 25 to 35 minutes delay filled by non-verbal cognitive tasks (Logical Memory II).

Trail Making Test (TMT; Reitan, 1958). The TMT is a test of visual scanning and task switching. This task includes two forms. Part A requires individuals to draw a line between 13 randomly arranged numbers on a page in sequential order from smallest to largest as quickly as possible. Part B requires individuals to draw a line between the letters A to L and numbers 1 to 13 in sequential order (1-A-2-B, etc.) as quickly as possible. Scores on each part represent the number of seconds required to complete the task. The TMT was administered to the majority of individuals in the current sample (ADHD, N = 13; DEP, N = 14; ADHD+DEP, N = 14).

Tower of London^{DX} (Culbertson & Zillmer, 2001). The TOL^{DX} is a 10-item test of higher-order executive planning abilities. This test was administered to the majority of individuals in the current sample (ADHD, N = 17; DEP, N = 17; ADHD+DEP, N = 17). Individual move three coloured balls on three wooden pegs of different lengths to form a specified pattern in a minimum number of moves. During execution of the test, the examinee must follow two types of rules. The Type I rule states that an examinee must not place or try to place more beads on a peg than it can physically support, and the Type II rule states that an examinee must not remove two beads from the pegs at the same time. The TOL^{DX} shows strong construct validity as it significantly correlates with other established problem-solving measures. The TOL^{DX} Total Correct Score indicates the number of problems solved in the minimum move count.

Woodcock-Johnson Tests of Cognitive Abilities – 3rd Edition (WJ-III COG; Woodcock, McGrew and Mather, 2001). The WJ-III COG is a series of 20 tests measuring different aspects of cognitive abilities. In this sample, all but two of the subjects in the DEP group were administered the tests that compose the Processing Speed cluster. This cluster comprises two

subtests (Visual Matching and Decision Speed) that measure an individual's cognitive efficiency in completing automatic cognitive tasks quickly and efficiently. The Visual Matching subtest is a timed measure of perceptual speed that involves identifying identical numbers in rows of six numbers, with the numbers ranging from single-digit to 3-digit numbers. The Decision Speed subtest measures the speed of processing simple concepts by quickly locating two pictures in each row that are the most similar conceptually. The Processing Speed cluster has a median reliability of .95 in adults (McGrew & Woodcock, 2001).

Procedure

Students seeking psycho-educational assessments from the centre were asked as part of the initial intake interview whether their test scores assembled during testing could be used for research purposes. The test scores of those students who provided written informed consent that was approved by the university General Research Ethics Board were entered into a general database. From this general database, a subset of students was chosen to form the groups in the current study.

Data Analysis

The relationship between depressive symptoms and neurocognitive performance in ADHD was examined with both categorical and dimensional analyses. Group differences on the neurocognitive measures were examined with univariate analysis of variance (ANOVA) tests. Post-hoc comparisons between groups were examined with the Least Significant Difference test with alpha set at .05. To examine the linearity of the relationship between depression and neurocognitive impairment, bivariate correlations were conducted with Pearson R reported.

Results

Differences between the three groups on neurocognitive tests were examined and revealed main effects for WAIS Processing Speed Index ($F(2,51) = 7.85; p = .001$), Trail Making Test B ($F(2,38) = 3.66; p = .04$), and WMS Logical Memory Delayed Recall (LM II; $F(2,29) = 4.57; p = .02$). Large effect sizes were found for each of these main effects, with eta square values of .24, .16, and .24 respectively. A trend was also identified for WJ Processing Speed Cluster with a medium effect size ($\eta^2 = .10$), but this finding was not significant ($F(2,48) = 2.75; p = .07$). The groups' scores did not differ significantly on the remainder of the psychometric tests ($F_s < 2.13; p_s > .13$).

As seen in Figure 1, follow-up comparisons revealed that the ADHD+DEP group performed significantly worse on the WAIS Processing Speed Index than both the ADHD group ($p = .02$) and the DEP group ($p < .001$). Similarly, the ADHD+DEP group performed significantly worse than the ADHD group ($p = .01$) and the DEP group ($p = .03$) on the WMS Logical Memory Delayed. The ADHD+DEP also significantly underperformed the ADHD group ($p = .01$) on the Trail Making Test B.

Correlational analyses were performed with both ADHD groups only to determine the association between depressive symptoms and neurocognitive symptoms in these groups. As seen in Table 1, the ADHD and DEP+ADHD groups revealed significant associations of PAI Depression total for the WJ Processing Speed Cluster ($r = -.40, p = .02$), WMS Logical Memory Immediate ($r = -.50, p = .03$), WMS Logical Memory Delayed ($r = -.54, p = .01$), and the Trail Making Test B ($r = -.45, p = .02$). As well, poorer performances on these neurocognitive tests appeared to be associated with cognitive and affective symptoms of depression, but not with physiological symptoms. Overall, the three PAI-DEP subscales were similar in their relationships across neurocognitive tests. Depressive symptom severity was not significantly

associated with WAIS Working Memory Index, WAIS Processing Speed Index, or Tower of London Total Correct.

Discussion

Both ADHD and depression have associated neurocognitive performance deficits. The objectives of the current study, therefore, were to examine the neurocognitive functioning of adults with co-occurring ADHD and clinically significant depressive symptoms, and to determine what deficits are present in this population and how these deficits compare to those in adults with either ADHD or depressive symptoms alone. The results of this study suggest that the co-occurrence of ADHD and clinically significant depressive symptoms is associated with additional neurocognitive impairments in certain domains. Specifically, the co-occurring ADHD and depressive symptom group had significant deficits in processing speed and long-term memory recall for verbal conceptual material when compared to the ADHD group and the depressive symptom group alone.

The poorer performances of those with co-occurring ADHD and serious depressive symptoms on tests of processing speed may indicate increased impairment in neurocognitive efficiency, and were consistent with stated hypotheses. This impairment in the co-occurring group may demonstrate an exaggeration of the cognitive slowing that has been identified in depressed patients (Tsourtos et al., 2002). Although processing speed deficits have not been identified as a primary deficit in adult ADHD, such deficits have been noted previously and may be related to attention and concentration affecting performance on timed processing tasks (Lacene, 2004; McLean et al., 2004). As such, the comorbid group's lower processing speed may have reflected an additive cognitive impairment, combining the cognitive slowing of the depressive symptoms and the sustained attention difficulties of ADHD.

In fact, increased depressive symptom severity was negatively associated with processing speed. This association, however, was only found for the processing speed scores on the WJ processing speed tests, and not with the WAIS processing speed tests. Thus, performance on the WAIS processing speed tests does not appear to be as dependent on depressive symptom severity as performance on the WJ processing speed tasks. This discrepancy may be because the Decision Speed subtest of the WJ is a test of effortful processing, as individuals are asked to make conceptual decisions quickly rather than making simple visual discriminations (Woodcock, McGrew and Mather, 2001). This added level of effortful processing, compared to the more visual search tasks of the WAIS processing speed tests, has been consistently found to be significantly impaired in those with depression (Hammar, 2003; Gorlyn et al., 2006, see Hartlage et al., 1993). Thus, effortful processing appears to be a neurocognitive area with impairment that is more strongly related to the presence of depression, and is exaggerated when combined with the processing speed deficits of ADHD. This implies that adults with both ADHD and significant depressive symptoms may experience a specific deficit in effortful processing in addition to a general processing speed deficit compared to those with ADHD alone.

A deficit in long-term conceptual verbal memory was also identified in adults with co-occurring ADHD and depressive symptoms compared to the other two groups. Verbal working memory has been specifically identified as an area of difficulty in populations of adults with ADHD alone (Marchetta et al., 2008) and depression alone (Hill, Keshavan, Thase, & Sweeney, 2004), but problems with long-term conceptual verbal memory have not been consistently identified in either group. In many studies, however, verbal memory tests using word lists are utilized (e.g., California Verbal Learning Test, Rey Auditory Verbal Learning Test), rather than tests in which conceptual verbal material, such as a story, is recalled. It may be that encoding,

consolidating and recalling information in a conceptual context represents a more effortful task and is more affected in adults with ADHD when depression is present, as impairments in effortful processing are common in adults with depression. Indeed, Landrø, Stiles, and Sletvold (2001) used a test of conceptual long-term verbal memory in adults with major depression, and identified this as an area of significant deficit in this group. Additive impairment in processing speed may have also played a part in students' difficulties encoding more complex information into long-term memory. The fact that the information was both conceptual and involved delayed recall also appears to be associated with this additive deficit, as this same pattern was not demonstrated for the recall of smaller pieces of information on the working memory tasks. Pollack, Kahana-Vax, and Hoofien (2008) noted that adults with ADHD also show more retrieval errors, such as double recalls and intrusions, while completing verbal learning and recall tasks. Thus, further research should be completed to identify the specific encoding and retrieval processes that are affected in individuals with both ADHD and depressive symptoms to determine whether these long-term memory deficits are more influenced by inaccurate encoding, inaccurate retrieval, or both.

In line with stated hypotheses, individuals with ADHD and significant depressive symptoms showed additive deficits compared to individuals with ADHD alone in the area of executive functioning, specifically with regards to conceptual shifting and mental flexibility. Interestingly, no differences were found among the groups on another executive functioning task of higher order planning. The strong relationship between depressive symptoms and an executive functioning task related to mental flexibility indicates that difficulties in this area of executive functioning may be associated more with the presence of depressive symptoms than with the presence of ADHD alone. This needs to be investigated further, as it suggests that the

inconsistency of findings in the neuropsychological literature related to executive functioning deficits in ADHD may be influenced, in part, by the additive neurocognitive effects of comorbid conditions rather than the ADHD alone.

The additive neurocognitive deficits of those with ADHD and depressive symptoms, coupled with the fact that depressive symptom severity is positively associated with neurocognitive impairment in adults with ADHD, has important implications for clinicians. First, depressive symptoms must be thoroughly assessed in adults with ADHD, not only because of the high comorbidity rate of these two disorders, but because of the added neurocognitive impairment associated with their co-occurrence. Significant depressive symptoms in adults with ADHD might account for neurocognitive impairment in the areas of processing speed, cognitive flexibility and long-term memory and may be alleviated if the depressive symptoms are treated. If psychotherapeutic treatment is provided, however, treatment content should be modified for such clients with the help of memory aids (e.g., visual cues) to reduce the demand on processing speed and memory for large amounts of verbal information. Functional implications for adult patients with both ADHD and serious depressive symptoms may also include difficulty performing a number of processing tasks such as reading and writing. For these individuals, any tasks requiring speed of completion, such as a time-limited exam, may also be difficult to complete quickly and so accommodations should be provided to compensate for this added neurocognitive deficit. Future research should examine the social and adaptive behavioral consequences of an additive neurocognitive deficit, as these features of mental disorders have been found to account for more variance in functional outcomes than traditional diagnostic symptoms (Bowie et al, 2006; Jaeger et al, 2006).

Limitations

This study had some limitations that may limit the generalizability of the current results. The participants used in this study represented a sample of convenience. A study using a priori equal groups of subjects with ADHD and depression would help control these variables in future studies. It should be noted, however, that the groups in this study did not differ significantly in age or ADHD subtype. Because the current sample represents a clinical sample, the assessment test battery selection was guided by the presenting referral question and did not include a fixed battery for each participant. This limited the number of participants who received certain tests, and the lack of control of order effects may have limited the number of significant results, particularly with regards to working and short-term memory. A more thorough neurocognitive battery of tests may have revealed different patterns. However, differences were found between groups in the current study in spite of the relatively small sample sizes in each group, so it is possible that larger groups would reveal similar and more robust results. In addition, due to the nature of the sample, a normal comparison group was not included in our analyses. Thus, the level of neurocognitive impairment of the comorbid group compared to normal students is not known. Future research should include such a comparison group to delineate the level of impairment in the comorbid group compared to normal adults. Finally, due to the nature of the assessments in this sample, the participants were not specifically screened with diagnostic interviews and it is therefore not known who would have met the formal diagnostic criteria for MDD. As such, the current results can only be generalized to those with clinically significant self-reported depressive symptoms.

Future Research

The current study raises many important questions regarding the explicit additive cognitive effects of ADHD and depression that should be addressed in future studies.

In order to determine the effects of ADHD and more chronic depressive symptoms, the current study should be replicated in a larger comorbid sample of adults with both diagnosed ADHD and MDD and include a prospective design in which participants are matched on key demographic variables. This study should also be replicated in other samples, as it is important to determine whether findings generalize to those who are not pursuing higher education, to children and adolescents, or to those who are in the workforce.

The need for treatment studies in this area is also apparent, as the treatment needs of those suffering from neurocognitive deficits associated with comorbid ADHD and depression may be particularly difficult. Research indicates that in this comorbid group, a combination of anti-depressants and stimulants is more effective in alleviating behavioral symptoms than stimulants or anti-depressants alone (Hornig-Rohan & Amsterdam, 2002). These same medications may also help improve the neurocognitive impairments associated with this dual diagnosis, but this type of longitudinal clinical trial has not been completed to date. As well, improvements in processing speed, cognitive flexibility and verbal memory may be addressed with behavioral approaches to remediating neurocognition.

Overall, the current study has identified specific additive neurocognitive deficits present in those with co-occurring ADHD and depression. Modest additive impairments were observed in processing speed, cognitive flexibility and long-term verbal memory for conceptual material, while measures of verbal intelligence, non-verbal reasoning and problem solving, working memory, and basic visual scanning were similar between groups. The results indicate that deficits in effortful processing are more closely associated with depressive symptoms, but are nonetheless significantly impaired in the comorbid group. The fact that severity of depressive symptoms is significantly correlated with these neurocognitive impairments also implies that

those with severe MDD and ADHD may be particularly at risk for these additive deficits.

Continued research in this area is necessary to replicate these findings and investigate other neurocognitive areas that may also be affected. Treatment options such as medication and neurocognitive enhancement should also be investigated in order to determine the best treatment options for improving neurocognitive functioning in this comorbid population.

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Table 1
Pearson correlations of depressive symptoms and cognitive test scores.

Test Scores	PAI- DEP - Total	PAI-DEP - Cognitive	PAI-DEP - Affective	PAI-DEP - Physiological	WAIS - WM	WAIS - PS	WJ - PS	WMS- LMI	WMS- LMII	TMTB	TOL Total Correct
PAI-DEP - Total	-	.91**	.96**	.83**	-.29	-.28	-.40*	-.50*	-.54*	-.45*	-.25
PAI-DEP - Cognitive		-	.83**	.58**	-.24	-.21	-.35*	-.50*	-.50*	-.36	-.21
PAI-DEP - Affective			-	.75**	-.27	-.27	-.44*	-.61*	-.63*	-.44*	-.22
PAI-DEP - Physiological				-	-.28	-.29	-.29	-.21	-.32	-.45*	-.27
WAIS - WM					-	.28	.07	.14	.06	.31	-.11
WAIS - PS						-	.43*	.15	.15	.66**	.01
WJ - PS							-	.34	.21	.61**	.13
WMS-LM I								-	.85**	.47	.15
WMS-LM II									-	.31	.09
TMTB										-	.19
TOL Total Correct											-

Note. * $p < .05$; ** $p < .01$. PAI-DEP = Personality Assessment Inventory - Depression Scale; WAIS = Wechsler Adult Intelligence Scale; WJ = Woodcock-Johnson; WMS - LMI = Wechsler Memory Scale Logical Memory Immediate Recall; WMS - LMII = Wechsler Memory Scale Logical Memory Delayed Recall.

Figure 1 Legend.

ADHD = Attention deficit/hyperactivity disorder group; DEP = Depressive symptom group;

ADHD + DEP = Attention deficit/hyperactivity and depressive symptom group; WAIS VCI =

Wechsler Adult Intelligence Scale – 3rd Edition Verbal Comprehension Index; WAIS POI =

Wechsler Adult Intelligence Scale – 3rd Edition Perceptual Organization Index; WAIS WMI =

Wechsler Adult Intelligence Scale – 3rd Edition Perceptual Working Memory Index; WAIS PSI

= Wechsler Adult Intelligence Scale – 3rd Edition Processing Speed Index; WJ PS = Woodcock-

Johnson 3rd Edition Processing Speed; WMS LMI = Wechsler Memory Scales 3rd Edition

Logical Memory Immediate; WMS LM II = Wechsler Memory Scales 3rd Edition Logical

Memory Delayed Recall.

† Scores converted from scaled scores ($M = 10, SD = 3$) to standard scores ($M = 100, SD = 15$).

* = ADHD+DEP < ADHD.

** = ADHD+DEP < ADHD and ADHD+DEP < DEP.

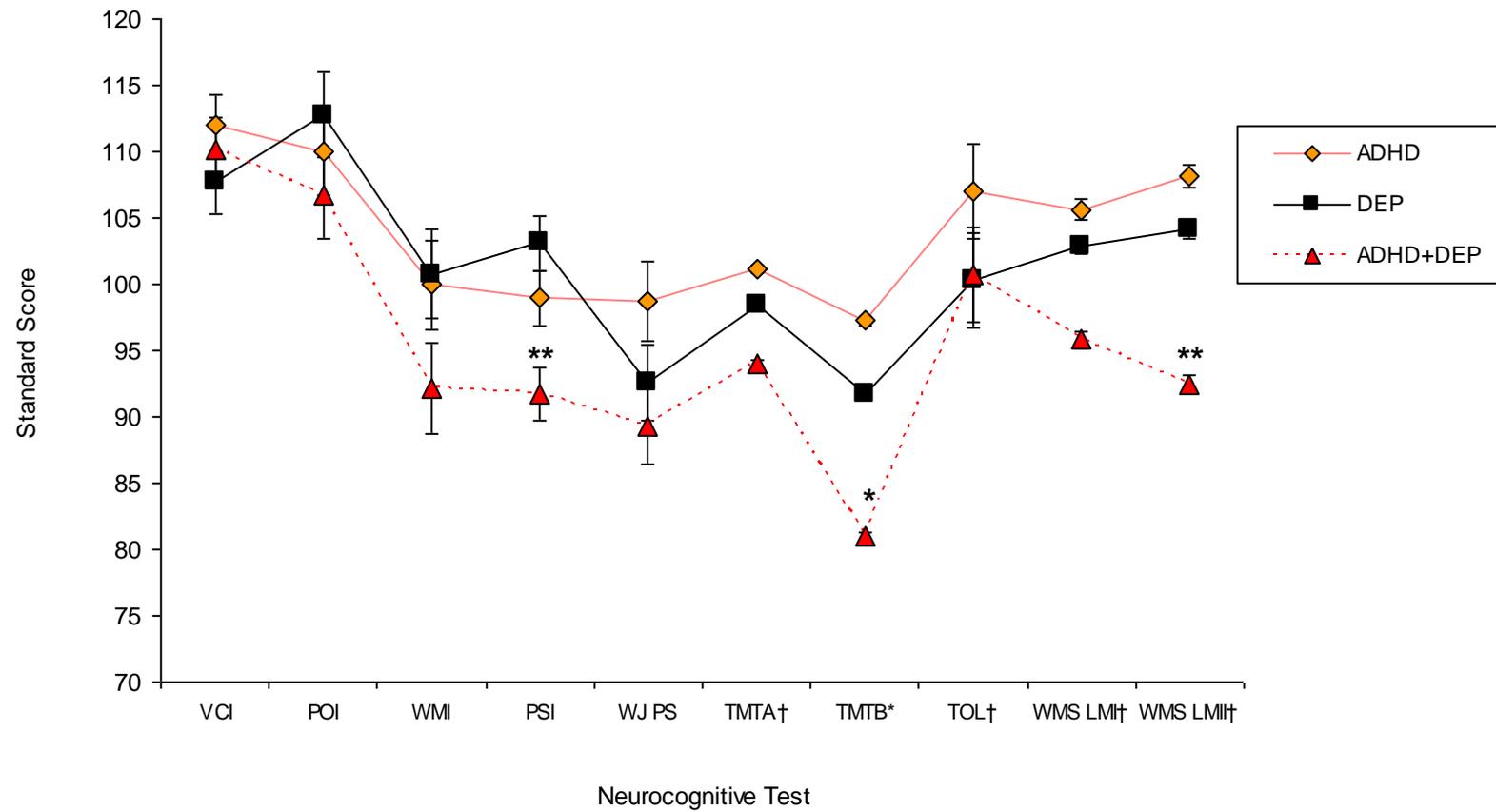


Figure 1. Neurocognitive test performance by diagnostic group.