A comprehensive investigation of memory impairment in attention deficit hyperactivity disorder and oppositional defiant disorder

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Background: We conducted a comprehensive and systematic assessment of memory functioning in drug-naïve boys with attention deficit hyperactivity disorder (ADHD) and oppositional defiant disorder (ODD). **Methods:** Boys performed verbal and spatial working memory (WM) component (storage and central executive) and verbal and spatial storage load tasks, and the spatial span, spatial executive WM, spatial recognition memory and verbal recognition memory tasks from the Cambridge Neuropsychological Test Automated Battery. Groups comprised: (a) ADHD only ($N = 21$); (b) ADHD+ODD ($N = 27$); (c) ODD only ($N = 21$); and (d) typically developing (TYP) boys ($N = 26$). Groups were matched for age ($M = 9.7$ years) and sex (all boys). **Results:** Confirmatory factor analyses confirmed the presence of five factors: verbal functioning, spatial functioning, WM storage, WM central executive and long-term memory (LTM). All three clinical groups demonstrated impaired memory performance. Boys with ODD and ODD+ADHD but not ADHD alone performed poorly on verbal memory tasks, whilst all three clinical groups showed impaired performance on spatial memory tasks. All three clinical groups performed poorly on the storage and central executive WM factors and the LTM factor. **Conclusions:** ADHD and ODD are characterised by impaired performance storage and central executive WM tasks and LTM tasks. This is, we believe, the first report of impaired WM and LTM performance in ODD. This study suggests that verbal memory difficulties are more closely associated with ODD than ADHD symptoms and that combined ADHD+ODD represents a true comorbidity. The data also support a small but growing number of suggestions in the literature of impaired LTM in ADHD. **Keywords:** Working memory, long-term memory, executive function, ADHD, ODD.

Introduction

Attention deficit hyperactivity disorder (ADHD) is clinically heterogeneous with high rates of comorbidity with a range of psychiatric disorders (Willcutt, Sonuga-Barke, Nigg, & Sergeant, 2008). Accumulating evidence suggests that there is also significant causal heterogeneity (Coghill, Nigg, Rothenberger, Sonuga-Barke, & Tannock, 2005) with altered functioning of dopaminergic and noradrenergic pathways across multiple neural networks including the prefrontal cortex and connecting areas. Current causal models suggest that these underlying pathophysiological factors are associated with multiple neuropsychological 'endophenotypes' including various aspects of executive functioning, an aversion to delay, temporal processing and response variability (Castellanos, Sonuga-Barke, Milham, & Tannock, 2006; Rhodes, Coghill, & Matthews, 2004, 2005; Willcutt et al., 2008).

Whilst several groups of researchers have identified working memory (WM) deficits in ADHD (e.g. Martinussen, Hayden, Hogg-Johnson, & Tannock, 2005; Rhodes et al., 2004, 2005; Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005), others have not (Barkley, Edwards, Laneri, Fletcher, & Metevia, 2001; Karatekin, 2004; Shue & Douglas, 1992). It is often assumed that deficits in WM are synonymous with deficits in executive functioning but this is not necessarily the case. Of the various models of WM that have been proposed Baddeley's (1986, 2007; Baddeley & Hitch, 1974) theoretical WM component model is the best supported. This model identifies WM storage components (the phonological loop and visuospatial sketchpad) and a 'central executive' wherein stored information is controlled and manipulated. The central executive is involved under conditions where a high level of processing is involved while the storage of information in memory is thought to involve the domain-specific storage components. A common processing mechanism is thought to underlie both verbal and spatial information (Baddeley & Logie, 1999) although some alternative models (e.g. Cowan, 1999) argue that domain general resources are responsible for processing and storage. Accumulating evidence supports the Baddeley

model (e.g. Alloway, Gathercole, & Pickering, 2006; Bayliss, Jarrold, Gunn, & Baddeley, 2003) and indeed evidence from developmental studies suggest that this theoretical structure of WM with a common processing mechanism but distinct storage systems is evident from as young as 4 years of age (Alloway et al., 2006).

Martinussen and Tannock (2006) suggest that ADHD is associated with deficits on spatial storage and executive tasks and on verbal executive, but not storage, tasks. Our previous studies identified that stimulant naive boys with ADHD were impaired on memory tasks both with and without executive components (Rhodes et al., 2004, 2005) and that basic storage may be one of the core impairments in ADHD (Coghill, Rhodes, & Matthews, 2007). Interestingly, methylphenidate improved storage but not executive aspects of WM (Coghill et al., 2007; Rhodes, Coghill, & Matthews, 2006; Rhodes et al., 2004). Unfortunately as most previous studies have used different tasks to measure storage and central executive demands, only indirect comparisons within and between studies have been possible. Several groups have proposed that impaired memory in ADHD extends beyond WM to aspects of long-term memory (LTM; Kempton et al., 1999; Krauel et al., 2007; Rhodes et al., 2005), but evidence is mixed (Skowronek, Leichtman, & Pillemer, 2008).

The specificity of neuropsychological deficits in ADHD has not been well studied (Willcutt et al., 2008) and similar deficits are found in several other conditions that commonly co-occur with ADHD, for example, autism (Verte, Geurts, Roeyers, Oosterlaan, & Sergeant, 2005), depression (Matthews, Coghill, & Rhodes, 2008) and anxiety disorders (Toren et al., 2000). Disruptive behaviour disorders (oppositional defiant disorder or ODD; conduct disorder or CD) are the most commonly diagnosed comorbid conditions with ADHD (Faraone, Biederman, Mennin, Russell, & Tsuang, 1998). Unfortunately research comparing cognitive functioning in ADHD, ODD and ADHD+ODD is limited, has focused almost exclusively on inhibitory processes and has presented inconsistent results (Oosterlaan, Logan, & Sergeant, 1998; Oosterlaan, Scheres, & Sergeant, 2005; Rubia et al., 2008; Scheres, Oosterlaan, & Sergeant, 2001). Studies employing other executive functioning tasks are few and report similar inconsistent findings (Oosterlaan et al., 2005). Unfortunately, many studies of children with ODD/CD have failed to control for the effects of ADHD (Pennington & Ozonoff, 1996). We are aware of only one previous study that has examined memory performance (WM or LTM) in children with ODD/CD. Van Goozen et al. (2004) examined WM in children with a diagnosis of ODD or CD with and without comorbid ADHD and reported no significant impairments in WM or other associated executive functions such as planning and inhibition. It is dif-

ficult to interpret these findings as it is highly unusual that the comorbid ADHD+ODD/CD group in the study did not show impairments in WM or inhibition which have been consistently shown in the literature. WM assessment in this study was limited to one measure of spatial WM (SWM). Further research is warranted to document the profile of WM in children with oppositional behaviour problems.

The aim of the present study was to systematically examine memory functioning in ADHD and ODD. Specifically, we aimed to (a) compare verbal and spatial memory functioning in ADHD and ODD and (b) examine various aspects of memory in these disorders including WM storage, WM central executive aspects of WM and LTM (recognition and free recall).

Methods

Participants

This study was approved by NHS Fife and Forth Valley ethics committee; informed consent was obtained from a parent or legal guardian and assent obtained from the participants. Ninety-five boys participated in the study: 69 drug-naïve boys within three clinical groups (ADHD: $N = 21$; ADHD+ODD: $N = 27$; and ODD: $N = 21$) and 26 typically developing (TYP) boys aged 7–13 years. Participants for the ADHD, ADHD+ODD and ODD groups were recruited from male out-patients referred to the Developmental Psychiatry Team at the Tayside Child and Adolescent Mental Health Service (CAMHS). Participants for the TYP group were recruited from local schools.

Clinical assessments

Clinical boys. Referred boys and their parents were interviewed by a child mental health clinician using the Kiddie Schedule for Affective Disorders and Schizophrenia – Present and Lifetime version (K-SADS–PL; Kaufman, Birmher, Brent, Rao, & Ryan, 1996) and their teachers using Child Attention-Deficit Hyperactivity Disorder Teacher Telephone Interview (CHATTI; Holmes et al., 2004). Diagnoses were made according to Diagnostic and Statistical Manual of Mental Disorders (DSM–IV TR; American Psychiatric Association, 2000). For ADHD symptom criteria at least six hyperactive/impulsive and/or six inattentive symptoms are required at home and/or school. Most of the boys with ADHD in the sample met criteria for ADHD combined type with the exception of three inattentive type and four hyperactive/impulsive type boys in each of the ADHD groups. Boys with ADHD and ADHD+ODD did not differ in number of ADHD symptoms. Similarly, boys with ODD and ADHD+ODD did not differ in number of ODD symptoms (see Table 1).

TYP control group. Symptom-free boys in local schools (T-score < 60 on all subscales of the parent completed Strengths and Difficulties Questionnaire (SDQ) Conners' Parent Rating Scale(CPRS)-27 and Conners' Teaching Rating Scale (CTRS)-28) were interviewed with the screening section of the K-SADS to confirm they were not suffering from any DSM–IV

Table 1 Demographic characteristics of participants

Group	ADHD	ADHD+ODD	ODD	TYP	F, p	Post hoc summary
Age	9.47 (2.01)	9.77 (1.82)	9.91 (1.94)	9.69 (1.46)	$F < 1$, NS	N/A
BPVS percentile rank	41.29 (25.8)	39.63 (23.66)	33.19 (21.97)	65.27 (21.28)	$F = 9.1$, $p < .001$	TYP > clinical groups
DSM–IV: no. of attention symptoms	6.57 (1.78)	6.50 (1.51)	2.64 (2.06)	N/A[a]	$F = 19.05$, $p < .001$	ODD < ADHD and ADHD+ODD
DSM–IV: no. of hyperactive/impulsive symptoms	6.43 (1.7)	5.43 (2.56)	2.73 (1.85)	N/A[a]	$F = 10.8$, $p < .001$	ODD < ADHD and ADHD+ODD
DSM–IV: no. of ODD symptoms	0.58 (0.99)	5.44 (1.94)	5.63 (1.51)	N/A[a]	$F = 39.27$, $p < .001$	ADHD < ADHD+ODD and ODD

Data shown are M (SD) values. ADHD, attention deficit hyperactivity disorder; ODD, oppositional defiant disorder; TYP, typically developing; BPVS, British Picture Vocabulary Scale; DSM–IV, Diagnostic and Statistical Manual of Mental Disorders.
[a]TYP boys were assessed with the initial screening section of the Kiddie Schedule for Affective Disorders and Schizophrenia only.

disorder. Eligible boys who matched with the clinical participants for age and sex participated in the study.

All boys. All boys completed the British Picture Vocabulary Scale (BPVS–II; Dunn, Dunn, Whetton, & Burley, 1997) providing an index of verbal ability. The BPVS scale has been used extensively to match clinical/patient populations (e.g. Jarrold, Baddeley, & Hewes, 1999; Rhodes, Riby, Matthews, & Coghill, 2011; Rhodes et al., 2005) and was chosen over traditional IQ tests to avoid confounds created by the inclusion of executive function skills that appear in most IQ/mental ability test batteries. Exclusion criteria were: current or previous history of post-traumatic stress disorder, major depressive disorder, bipolar disorder, pervasive developmental disorder, psychosis, seizures, neurological disorder, history of alcohol or drug abuse and major vision problems.

Materials and procedure

Tasks were designed/selected to assess verbal, spatial and pattern-based memory and also to assess the various components of memory; WM storage and WM central executive, and LTM.

WM component tasks. The verbal and spatial 'WM component tasks' were based on tasks originally designed by D'Esposito, Postle, Ballard, and Lease (1999), which have been subsequently used in several clinical studies (e.g. Cannon et al., 2005; Kim, Glahn, Nuechterlein, & Cannon, 2004). They were adapted for use in children by SR and programmed in Eprime® (Schneider, Eschmann, & Zuccolotto, 2002) and include tasks that tap (a) the phonological loop and visuospatial sketchpad (*verbal* and *SWM storage tasks*), and (b) WM central executive functioning (*verbal* and *SWM executive tasks*).

In the storage tasks, participants were presented with either a string of three letters (target) or three dots in different locations for 2.5 s which had to be maintained in memory during a 6-s delay. Participants then had to decide whether a probe was a 'match' or 'nonmatch' to the target. In the case of the verbal probe, this was presented in the same central screen location as the target. In the central executive demand tasks, participants had to alphabetise the letters or flip the dots along the vertical axis of the screen and hold this manipulated version of the stimuli in memory across the delay and decide whether the probe presented was a 'match' or 'nonmatch' of the manipulated version. Each of these 'WM component tasks' had 6 practice trials followed by 20 experimental trials. The key measure for each task was accuracy indexed by % correct.

Participants also performed 'load' versions of the storage tasks. They were presented with 3, 5, 7 or 9 letters (target) or 1, 3, 5 or 7 dots in various locations on the screen (target) for a duration of 5 s and asked to maintain the letter or dot locations in memory over a 3-s delay. A probe was then presented consisting of one letter or a dot in one location and the participant had to indicate if it was a 'match' or 'nonmatch' for the target. Each of the load tasks had 8 practice trials followed by 48 experimental trials (12 trials per memory load). Again the key measure was accuracy indexed by % correct.

CANTAB tasks. Participants also completed four memory tasks from the Cambridge Neuropsychological Test Automated Battery (CANTAB; Morris, Evendon, Sahakian, & Robbins, 1987). These tasks have been used extensively with child populations (e.g. Coghill et al., 2007; Happé, Booth, Charlton, & Hughes, 2006; Luciana & Nelson, 1998; Matthews et al., 2008; Rhodes et al., 2005). Spatial span (SSP) is a measure of SWM storage that assesses the ability to remember the spatial locations of a sequence of squares on a computer screen. Trials range from 3 to 9 item locations. The key measure on this task is the SSP score (the longest sequence that a subject is able to reproduce correctly within three attempts). The SWM task assesses the capacity to simultaneously store and manipulate spatial information in short-term memory placing significant demand on central executive functioning. Participants are required to 'search through' a spatial array of coloured boxes presented on the screen to collect 'blue tokens' hidden inside the boxes. Returning to a box where a token has already been found constitutes a 'between search error' (BSE). Experimental trials commence with a four-box search and the highest difficultly level involves eight box trials. The spatial recognition memory (SRM) and verbal recognition memory (VRM) tasks index LTM. The SRM is a test of visual SRM in a two-choice forced discrimination paradigm. In the recognition phase, the participants are presented with four sequences of five pairs of squares, one of which is in a place previously seen in the presentation which they must choose. The VRM assesses immediate and delayed memory of verbal information under free recall and forced choice recognition conditions. Participants are presented with a series of 12 words to remember and have to choose the words they remember from distractors. In the free recall condition the participant is asked to call aloud as many words as they can from the study phase. Accuracy of responding (% correct) is the key outcome measure for the VRM and SRM tasks. Delayed matching to sample (DMtS) a pattern-based memory task previously demonstrated to have a strong association with ADHD (Rhodes et al., 2004) was also included but will be reported separately. The CANTAB tasks were performed in the same order (SSP, SWM, SRM, VRM immediate, DMtS and VRM delay) by all boys.

The testing was divided into three sessions which were conducted in one of two counterbalanced orders (WM component verbal, WM component spatial, CANTAB or WM component spatial, WM component verbal, CANTAB).

Statistical analysis

Analyses were conducted using SPSS for Windows (v. 17; SPSS Inc., Chicago, IL) and AMOS (v. 6; SPSS Inc.).

A series of confirmatory factor analyses (CFAs) were estimated to determine the best fitting model for the proposed aspects of memory functioning (verbal vs. spatial; WM storage vs. WM central executive vs. LTM) in addition to confirming the model this approach has the added benefit that it reduces measurement error. The maximum likelihood method was used. Model
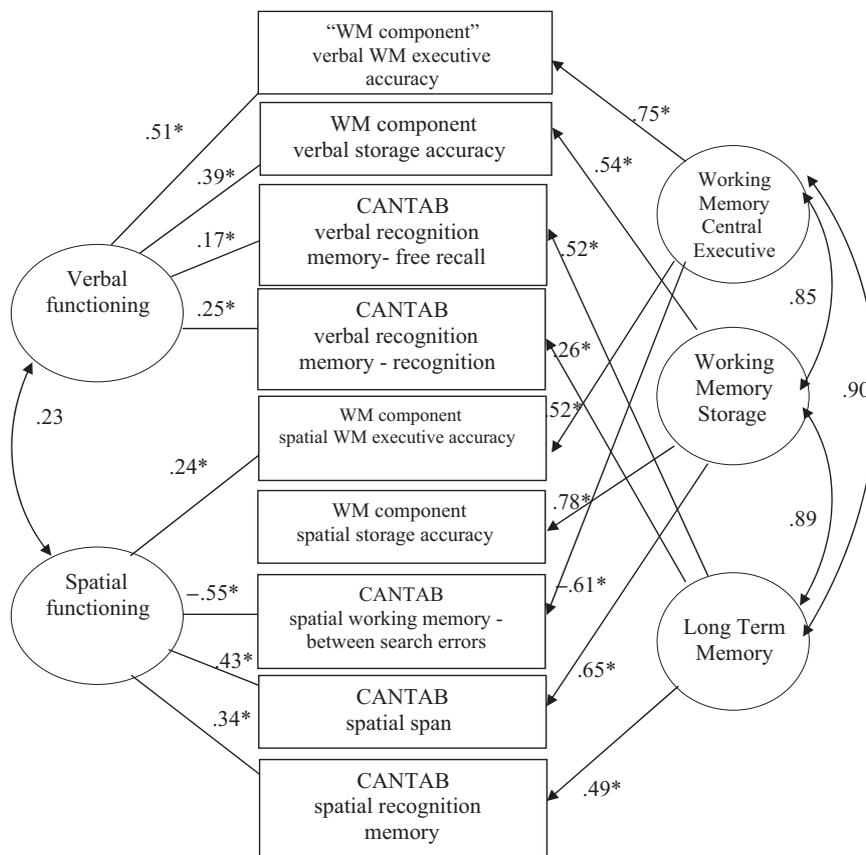

Figure 1 Confirmatory factor analysis (note all path coefficients are standardised; *p < .05)

goodness of fit was evaluated using chi-squared fit statistics, root mean square error of approximation (RMSEA) and comparative fit index (CFI). Smaller chi-square and RMSEA values and larger CFI values indicate better fit. Generally speaking, nonsignificant chi-square, RMSEA equal to or below .05, and CFI above .9 indicate good fit (Kline, 2005). The best model was determined by the best overall fit indices. Figure 1 illustrates the tasks that comprised each of the factors in the model (e.g. verbal tasks comprised all verbal storage, executive and LTM tasks).

A series of ANOVAs were then conducted for each of these five factors comparing the four groups (ADHD, ODD, ADHD+ODD and TYP) with post hoc comparisons where the main effect was significant. Additional repeated-measures ANOVAs were conducted comparing the four groups on the spatial and verbal short-term memory load tasks across the four load levels. An ANOVA revealed a significant difference in BPVS Percentile Rank (PR) scores between the clinical and TYP group (Table 1). Therefore, separate ANOVAs were conducted with and without BPVS as a covariate. As these yielded very similar results only the data not corrected for BPVS is reported here.

Results

Raw data from all tasks are presented in Table 2 and the correlation matrix for the memory tasks included in the CFA is presented in Table 3.

Confirmatory factor analysis

In view of the extensive correlations between the memory tasks (Table 3) three main models were tested within the CFA. All three models included separate verbal and spatial factors; in addition, the model addressing the Baddeley model of WM included three separate factors for WM storage, WM central executive and LTM. There were also two simpler models: one with two additional factors (executive and nonexecutive memory) and another with a single memory factor. The CFA confirmed that the best fit model for these data was the more complex model with verbal and spatial factors and the three memory factors (Figure 1). When fitting the

model it was necessary to remove the path between the spatial factor and the spatial storage task as inclusion of this path resulted in an inadmissible solution. The resulting model (Figure 1) proved to be an excellent fit to the data (χ^2 = 4.8, df = 15, p = .99, CFI = 1.00, RMSEA = .00]. Whilst models that separated the data into only executive and non-executive factors (χ^2 = 14.4, df = 17, p = .87) or a single memory factor (χ^2 = 27.8, df = 18, p = .89) were also supported, chi-square difference tests between these simpler models and the more complex three-factor model (WM storage tasks, WM central executive tasks and LTM) confirmed this more complex model to be significantly better than the two simpler models. All included regression weights that were significant at the p < .05 level. The standardised regression weights and correlations between factors are shown in Figure 1.

The impact of ADHD and ODD on verbal and spatial memory (Table 4)

Verbal memory. An ANOVA on the *verbal memory factor data* revealed a main effect of group [$F(3,91)$ = 6.4, p < .001] with post hoc tests revealing that the ODD (effect size Cohen's δ = .94,) and the ODD+ADHD (δ = 1.2) groups performed worse than the TYP boys but no statistically significant differences between the ADHD (δ = .72) and TYP groups or between the three clinical groups. Thirty-seven per cent of the ODD boys and 57% of the ADHD+ODD boys had a deficit in verbal memory (defined as scoring in the bottom 10% of the TYP group) compared with only 19% of the ADHD boys.

A repeated-measures ANOVA on the *verbal short-term memory load* data (Figure 2A) revealed a main effect of verbal load [$F(3,88)$ = 74.12, p < .001] with performance deteriorating as load increased, a main effect of group [$F(1,90)$ = 3.58, p < .05] but no Load × Group interaction. Post hoc comparisons revealed that the effect of the group was carried by the ADHD+ODD boys performing worse than the TYP boys. There were no differences between the other groups.

Table 2 Summary of raw task data

Measure/group	TYP	ADHD	ODD	ADHD+ODD
Verbal storage	85.19 (14.8)	76.4 (17.26)	79.76 (16.32)	74.63 (16.98)
Spatial storage	72.69 (12.75)	57.86 (15.86)	59.29 (12.68)	58.89 (19.13)
Spatial span score	5.92 (1.29)	4.95 (1.67)	4.81 (1.08)	4.35 (1.41)
Verbal executive	70.96 (14.56)	58.57 (19.82)	54.05 (17.86)	55.93 (19.12)
Spatial executive	61.54 (12.15)	49.29 (11.43)	49.52 (12.84)	45.37 (14.54)
SWM: total between search errors	35.19 (16.07)	48.5 (16.48)	46.62 (13.9)	53.96 (16.47)
Verbal load (mean %)	75.58 (12.4)	69.08 (15.18)	65.5 (15.05)	62.88 (16.06)
Spatial load (mean %)	77.81 (10.97)	62.73 (17.53)	69.9 (14.37)	64.1 (15.5)
VRM: recognition (total correct)	22.54 (2.14)	22.55 (1.57)	22.05 (2.62)	22.12 (1.86)
VRM: free recall (total correct)	6.38 (1.94)	5.4 (1.57)	5.19 (1.36)	5.08 (2.12)
Spatial recognition memory	77.5 (7.52)	68.75 (12.23)	66.19 (11.72)	64.4 (11.67)

Data shown are *M* (*SD*) values and reflect % correct unless otherwise stated. ADHD, attention deficit hyperactivity disorder; ODD, oppositional defiant disorder; TYP, typically developing; SWM, spatial working memory; VRM, verbal recognition memory.

Table 3 Correlation matrix for verbal and spatial memory tasks

	CANTAB verbal recognition memory: recognition	CANTAB verbal recognition memory: free recall	CANTAB spatial recognition memory	Eprime verbal storage	Eprime spatial storage	CANTAB spatial span	Eprime verbal executive	Eprime spatial executive	CANTAB spatial working memory: BSE
CANTAB verbal recognition memory: recognition	1.00								
CANTAB verbal recognition memory: free recall	0.26*	1.00							
CANTAB spatial recognition memory	0.16	0.36**	1.00						
Eprime verbal storage	0.12	0.30**	0.19	1.00					
Eprime spatial storage	0.10	0.40**	0.38**	0.41**	1.00				
CANTAB spatial span	0.28**	0.47**	0.45**	0.38**	0.52**	1.00			
Eprime verbal executive	0.28*	0.56**	0.44**	0.57**	0.59**	0.57**	1.00		
Eprime spatial executive	0.08	0.07	0.30*	0.27*	0.42**	0.35**	0.39**	1.00	
CANTAB spatial working memory: BSE	−0.15	−0.48**	−0.51**	−0.33**	−0.48**	−0.60**	−0.50**	−0.36**	1.00

BSE, between search errors; CANTAB, Cambridge Neuropsychological Test Automated Battery.
*Pearson correlation is significant at the $p < .05$ level (two-tailed); **Pearson correlation is significant at the $p < .01$ level (two-tailed).

Table 4 Impact of ADHD and ODD on memory functioning

	TYP (N = 26) M (SD)	ADHD (no ODD; N = 21) M (SD)	ODD (no ADHD; N = 21) M (SD)	ADHD+ODD (N = 27) M (SD)	Main effect		Post hoc
Factor					F	p	
Verbal performance	0.45 (0.63)	0.01 (0.65)	−0.21 (0.76)	−0.28 (0.60)	6.4	<.001	ODD < TYP ADHD+ODD < TYP
Spatial performance	0.68 (0.58)	−0.05 (0.70)	−0.18 (0.49)	−0.46 (0.66)	16.3	<.001	ADHD < TYP ODD < TYP ADHD+ODD < TYP
Executive working memory	0.67 (0.55)	−0.05 (0.64)	−0.25 (0.70)	−0.39 (0.59)	15.1	<.001	ADHD < TYP ODD < TYP ADHD+ODD < TYP
Nonexecutive short-term memory	0.57 (0.54)	−0.13 (0.71)	−0.18 (0.65)	−0.40 (0.67)	11.2	<.001	ADHD < TYP ODD < TYP ADHD+ODD < TYP
Recognition memory	0.48 (0.54)	0.01 (0.58)	−0.24 (0.64)	−0.29 (0.61)	8.9	<.001	ADHD < TYP ODD < TYP ADHD+ODD < TYP

ADHD, attention deficit hyperactivity disorder; ODD, oppositional defiant disorder; TYP, typically developing.

Spatial memory. An ANOVA on the *spatial memory factor data* revealed a main effect of group [$F(3,91) = 16.3$, $p < .001$] with post hoc tests revealing that all three clinical groups performed worse than the TYP boys (ADHD $\delta = 1.1$; ODD $\delta = 1.6$; ODD+ADHD $\delta = 1.8$) but no statistically significant differences between the three clinical groups. Interestingly, despite the ADHD+ODD group having the largest effect size it also had the fewest individuals with a deficit (29%), compared with 63% of the ODD group and 38% of the ADHD group.

A repeated-measures ANOVA on the *spatial short-term memory load* data (Figure 2B) revealed a main effect of spatial load [$F(3,88) = 62.2$, $p < .001$] with performance deteriorating as load increased, a main effect of group [$F(1,90) = 5.42$, $p < .005$] but no Load × Group interaction. Post hoc comparisons revealed that the effect of group was carried by both the ADHD boys and the ADHD+ODD boys performing worse than the TYP boys. There were no differences between the other groups.

The impact of ADHD and ODD on the different memory components (Table 4)

An ANOVA on the WM *central executive factor* data revealed a main effect of group [$F(3,91) = 15.1$, $p < .001$]. Post hoc tests revealed that all three clinical groups performed worse than the TYP boys (ADHD $\delta = 1.1$, 43% had deficit; ODD $\delta = 1.5$, 67% had deficit, ADHD+ODD $\delta = 1.9$, 57% had deficit).

An ANOVA on the *WM storage factor* data revealed a main effect of group [$F(3,91) = 11.2$, $p < .001$]. Post hoc tests revealed that all three clinical groups performed worse than the TYP boys (ADHD $\delta = 1.2$, 62%
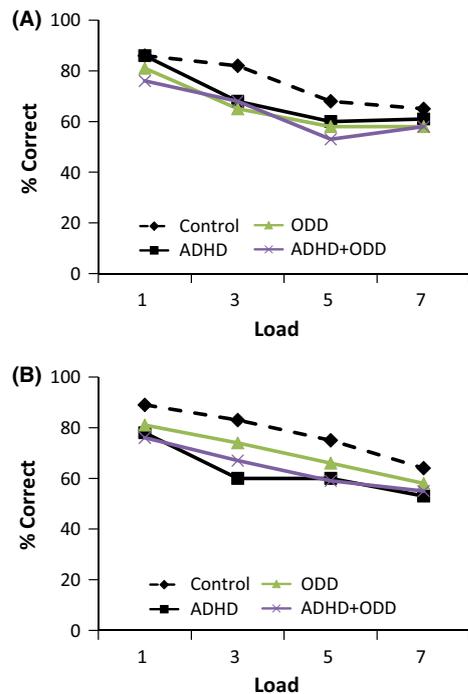
Figure 2 (A) The effects of attention deficit hyperactivity disorder (ADHD), oppositional defiant disorder (ODD) and ADHD+ODD on verbal load; (B) the effects of ADHD, ODD and ADHD+ODD on spatial load

had deficit; ODD δ = 1.3, 63% had deficit, ADHD+ODD δ = 1.6, 43% had deficit).

An ANOVA on the *LTM factor* data revealed a main effect of group [$F(3,91)$ = 8.9, $p < .001$]. Post hoc tests revealed that all three clinical groups performed worse than the TYP boys (ADHD δ = 0.9, 14% had deficit; ODD δ = 1.3, 37% had deficit, ADHD+ODD δ = 1.4, 38% had deficit).

Discussion

The present study found significantly impaired performance across a broad range of aspects of memory functioning in boys with ADHD and/or ODD. Importantly, as all participants were naive with respect to ADHD medications, these effects are not a consequence of previous exposure to or withdrawal from ADHD medication. The findings support previous findings that ADHD is characterised by substantial visual memory deficits and that these include impairments in storage and central executive WM and LTM. We believe this to be the first report of WM components in ODD which also appears to be associated with substantial deficits in storage and central executive WM and LTM extending across both verbal and spatial domains. Notwithstanding the similarities between the ADHD and ODD groups there are also important differences with respect to verbal memory. Verbal memory was intact in ADHD even under the high load conditions supporting an increasing consensus that, compared with spatial memory functioning, verbal memory is

relatively intact in ADHD (Martinussen & Tannock, 2006; Martinussen et al., 2005; Willcutt et al., 2005). In contrast boys with ODD had significantly impaired verbal memory performance, even at the lowest levels of load.

Taken together, these findings support the notion that ADHD and ODD are indeed different conditions and that ADHD+ODD represents a hybrid of the two. Both ADHD and ODD were associated with significant and large impairments in spatial memory functioning (ADHD δ = 1.1; ODD δ = 1.6) and although the boys with ODD did not show deficits on the spatial load task their performance on the overall spatial memory factor was worse than that of the ADHD boys and was associated with a very large effect size (1.6). These findings have potential clinical significance for understanding associated functional impairments that may be related to these disorders. Deficits in SWM are often associated with difficulties in arithmetic (Gathercole & Pickering, 2000) and executive SWM deficits are associated with problems in learning science (Jarvis & Gathercole, 2003). The verbal deficits associated with ODD are likely to result in significant functional impairments both at home and in the classroom where the retention of verbal information plays an important role in both learning and responding to verbal instructions. Studies have established that deficits in verbal WM are associated with language acquisition difficulties (e.g. Swanson & Howell, 2001). We are not aware of previous reports of verbal deficits in ODD samples, although there is considerable evidence from the CD literature that verbal deficits are a characteristic of this particular disruptive disorder (Narhi, Lehto-Salo, Ahonen, & Marttunen, 2010; Teichner & Golden, 2000). Further research is warranted to examine the verbal functioning profile of children with ODD. The current study contradicts the findings of Van Goozen et al. (2004) who reported no significant SWM impairment in children with ADHD+ODD/CD and ODD/CD. It is unusual that Van Goozen et al. reported no SWM impairment in the ADHD+ODD/CD sample as this impairment is widely reported in the literature. The medication status and severity of ADHD symptoms of the children in the study is unclear making the findings difficult to interpret. A number of methodological differences between the Van Goozen et al. study and the current study therefore may be responsible for the discrepancy in findings. These findings also raise the possibility that discrepancies in earlier studies of verbal WM in ADHD may have arisen because of the presence of comorbid ODD that was not controlled for in the analyses.

We were surprised by the lack of interaction between condition and load. The load conditions were included as our previous work (Rhodes et al., 2004) had suggested that for certain tasks from the CANTAB battery (e.g. SWM, DMtS and stockings of Cambridge) children with ADHD performed as well

as healthy controls on more simple task levels but not at the more demanding levels. We had therefore expected a Difficulty × Group interaction for these samples. It is possible that the tasks were not challenging enough to provoke a deterioration in performance.

Our findings also emphasise that the impairment in memory function in ADHD and ODD is not restricted to executive functioning. Both clinical groups were significantly impaired in aspects of WM storage, WM central executive functioning and in LTM. All effect sizes are large indeed apart from LTM in ADHD where the effect size is 0.9. All other effect sizes for both disorders are very large and >1.0. Overall, the effect sizes for ODD are greater than those for ADHD. This was unexpected but emphasises that ODD is unlikely to be a simple response to poor parental controls and is likely to be associated with structural and functional brain changes within the child. Future investigations of multipathway causal models for ADHD and ODD should take this into account and include a broad range of memory tasks. In particular, future studies should further investigate LTM functioning in ADHD as the entire memory literature on this disorder is currently almost exclusively focused on WM processes. The current study focused on examining WM functioning in ADHD in relation to the popular Baddeley's (2007) model. It has recently been argued that retrieval from LTM occurs in most standard measures of WM (Unsworth & Engle, 2007). Therefore, performance on the tasks used in the current study (both storage and central executive WM tasks) could reflect information that was currently maintained in and unloaded from WM and information that was not maintained, but was nevertheless retrieved from LTM. Indeed, Gibson, Condoloi, Flies, Dobrzenski, and Unsworth (2010) have recently applied this 'dual-component' model of WM to ADHD, and have shown that the primary deficit associated with ADHD is the retrieval component rather than the maintenance component. The current study methodology is not able to address this and further studies are required.

Taken together, these data suggest that ADHD and ODD have an additive effect on memory functioning and that the combination of ADHD+ODD represents true comorbidity rather than a separate clinical entity. Those with ADHD+ODD will also be more consistently, and more severely, impaired across the various aspects of WM than boys in either of the 'pure' groups. It is, however, interesting that whilst the effect sizes for the ADHD+ODD group were consistently higher than those for the other two groups the proportion of individuals with a deficit was often greatest for the pure ODD group. This implies that for the comorbid group the large effect size is driven by a relatively small group with extremely poor performance. As ODD is the most common comorbid condition to occur with ADHD, these findings strongly emphasise the need for future research in this area that examine whether or not other neuropsychological deficits thought to be central to the causal pathways for ADHD are also associated with ODD and whether accounting for these associations clarifies which types of deficit may be shared between different disorders and which are unique and can discriminate between them. With such knowledge it may be possible to target existing treatments more effectively and develop new treatments for particular patient groups.

A separate but equally important finding relates to analysis of storage and central executive aspects of WM. Studies that have separated these WM component factors in ADHD samples have produced mixed results with some suggesting no impairment on storage tasks (e.g. Karatekin, 2004) and others reporting impairment on both storage and central executive tasks (Rhodes et al., 2004, 2005). The current findings suggest that both ADHD and ODD are associated with impaired functioning across storage and central executive WM. There is already interest in the development of cognitive training in ADHD (e.g. Holmes et al., 2010; Klingberg et al., 2005). The current memory training packages focus on the executive aspects of WM. These findings suggest that programmes should focus on improving relatively basic memory retention processes and aspects of LTM in addition to executive strategic memory. These insights can also be used to support the development of specific accommodations and teaching styles for these boys, for example, the use of written notes and reminders to help the child remember classroom material. The wide range of impaired memory performance observed in the ODD sample suggests that treatments known to be effective in ADHD may be effective in alleviating some of the problems associated with ODD.

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Key points

- ADHD is characterised by WM deficits that are more often observed on spatial than verbal tasks. The limited research on LTM in ADHD has produced inconsistent findings but suggests impaired LTM in ADHD. ODD is characterised by inhibition deficits suggesting the possibility that children with the disorder may also be impaired on other aspects of executive function such as WM.
- Drug-naïve boys with ADHD are not impaired in verbal memory functioning. In contrast, boys with ODD or ADHD+ODD are impaired in verbal memory functioning.
- Boys with ADHD, ODD and ADHD+ODD are impaired in overall spatial memory functioning.
- Boys with ODD have similar impairments in both the storage and executive components of WM to boys with ADHD.
- Boys with both ADHD and ODD show impaired LTM functioning on tasks of recognition memory and free recall.
- Where an individual has both ADHD and ODD these impairments are additive. This suggests that ADHD+ODD represents a true comorbidity.
- Both ADHD and ODD are characterised by extensive WM and LTM impairments.
- Memory training programmes that currently focus on executive aspects of WM should focus on both storage and executive WM difficulties and LTM functioning.
- These memory training programmes may also be beneficial for boys with ODD.

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