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Depression and attention deficit disorders in young adults and their connection to right hemisphere function

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ABSTRACT

Objectives While depression and attention deficit hyperactivity disorder (ADHD) are highly studied neuropsychiatric disorders, the results are conflicting emphasizing deficits in verbal or visual domain. In this study, we wanted to investigate if the right hemisphere dysfunction model can explain the disorders. **Methods** The extensive neuropsychological examination was executed to young adults having depression (N = 10), ADHD (N = 9), both disorders (N = 4), and controls (N = 17) to identify possible deficits in visual reasoning and visual memory functions and attentional orientation. **Results** The depressed adolescents showed no deterioration in their ability and memory performance in contrast to the adolescents with ADHD or both disorders who had verbal memory problems besides performing significantly worse in verbal (but not visual) ability tasks. As for the visual memory, the adolescents having both disorders could be detected only by the most demanding, visuospatial (12 shapes) learning task, in which they made most errors and needed more trials to complete the task compared to controls. In attentional tendencies, all individuals remembered objects better on their left side in an easy task but their visual attention was directed more to their right side in more demanding visual memory task. **Conclusions** Despite the small group sizes, our results suggest that adolescents with ADHD and both disorders might be identified by their lowered verbal ability and verbal memory scores. In visual domain, identifying them by neuropsychological tests is more difficult. Thus, the right hemisphere dysfunction model is not good candidate to explain the core deficits in young adults with depression and ADHD. Rather, we suggest that these disorders might be approached with the left hemisphere (higher functioning in depression and lower functioning in ADHD) model.

Keywords:

Depression, attention deficit disorder, ADHD, visual ability, visual memory, attentional orientation

INTRODUCTION

Our modern world is complex and sophisticated in many ways. There are several challenges and demands confronting vulnerable adolescents and young adults. They need to tolerate pressures coming from inside (e.g. identity formation and consolidation), their family (e.g. becoming physically and economically independent), society (e.g. finding a place in possibly very competed education, getting a job without work experience in uncertain and temporary labour market), and their peers (e.g. how to look, having or having not a spouse and a family).These developmental phase tasks may become especially difficult to those adolescents who have different neurobiological and/or -behavioral disorders, which might deteriorate their daily function, such as decision making. The most important health care and medical objective would be to identify and diagnose adolescents with these disorders adequately and in time to diminish a risk of cumulation of other symptoms and comorbidity.

Depression and ADHD are highly studied disorders but still, there are many conflicting and varying research results, which are complicating a general and a specified view of these disorders and their core symptoms and pathologies. The more incoherent and unidentified features there are belonging to these disorders, the larger is the probability that these disorders remain unnoticed and/or misdiagnosed.

Depression

Depression is a common mental disorder affecting people at any age phase. It threatens psychological development and challenges the transition from the adolescence to young adulthood by causing mental distress to daily life and impairing study and work ability. In fact, mental disorders (depressive mood disorders 39%) are the leading cause of work disability among young adults in Finland (Mattila-Holappa, P., 2018), and across Europe (Kaltenbrunner Bernitz, Grees, Jakobsson Randers, Gerner & Bergendorff, 2013).

Depression is associated with a variety of cognitive and functional deficits, and social impairments (Thapar, Collishaw, Pine & Thapar, 2012; Trivedi & Greer, 2014). Imaging studies have revealed impaired metabolic activation in the frontal and subcortical brain structures in depressed individuals (Bench et al., 1992; Dolan, Bench, Brown, Scott & Frackowiak, 1994; Drevets et al., 1992; Goodwin, 1997; Larisch et al., 1997). That leads to dysfunction in executive skills e.g. attention, planning, organizing, prioritizing, starting tasks, staying focused on them to completion, regulating emotions, self-monitoring (Halari, et al., 2009). This is evident in situations where more complex cognitive processing in executive functioning is needed (Langenecker et al., 2005; Wright, Kay, Avery, Giordani & Alexander, 2011).

Identifying depression is difficult because the symptoms are highly variable specifically in adolescents. Separating symptoms of depression from other behavioral signs of puberty can be demanding. The etiology of depression is also multiple, and it might be in genetics, adversive life experiences, or it might be a consequence from other diseases. Depression as the primary and mental diagnosis seems to share homologous pathogenetic mechanisms with depression following neurological diseases or acquired brain damage (Benedetti, Bernasconi & Pontiggia, 2006). Mental disorders (as well as somatic illnesses) are classified on the basis of the International Classification of Diseases (ICD-10), which is used as a diagnostic tool. In ICD-10, depression diagnoses are represented by the diagnostic codes F32-F33. In diagnostics, the severity level of depression (mild, moderate, severe, or psychotic) result from an amount of different experienced symptoms and their duration.

It has been suggested, that depression might be related to right hemisphere functions (such as visual, visuospatial reasoning and visual and visuospatial memory) and especially to their dysfunction (Kalska, Punamäki, Mäkinen-Pelli & Saarinen, 1999; Kindermann & Brown, 1997). It is possible, that the deterioration in visual memory is due to negatively biased thinking (Kalska et al., 1999), or lacking the operational strategies (Kindermann & Brown, 1997) and/or deficits in working memory function leading to problems in retrieving things from long-term memory (Grafman et al., 1990).

The right hemisphere also specializes in recognizing the emotions of the faces (Buchtel, 2001), identifying and experiencing the emotions, and possibly especially negative emotions (Silberman & Weingartner, 1986). Depression, therefore, may be a result from a disorganization of the right hemisphere (Coffey, 1987; Gainotti, Caltagirone & Zoccolotti, 1993). Severely depressed individuals are more susceptible to observing and remembering emotionally negatively nuanced than emotionally positively nuanced social cues, and this characteristic is not necessarily disappearing after depressional recovery (Leppänen, 2006). This might refer to the weakened function in the limbic and the paralimbic areas besides dysfunction in prefrontal areas (Phillips, Drevets, Rauch & Lane, 2003a, 2003b), which is correlating quite well with depressed individuals' defects to adequately observe emotions from other faces. Yang et al. have observed more activation in bilateral prefrontal areas and left amygdala during face recognition task in depressed adolescents compared to controls (Yang et al., 2009; Young et al., 2010), and they suggested that the theories of depressed adults should be expanded to concern also depressed adolescents.

Historically, researchers in their physiological studies accidentally found that transcranial magnetic stimulation (TMS) affects mood (Bickford, Guidi, Fortesque & Swenson, 1987; Pascual-Leone, Catala & Pascual-Leone Pascual, 1996). After that, the selection of cortical targets in the treatment of mood disorders has been based on pathophysiological changes considered to underlie mood and depression disorders. Functional brain imaging has shown that there is a decrease in regional cerebral blood flow (rCBF) as well as in glucose and oxygen consumption in the left frontal regions of depressed individuals (Kennedy, Javanmard & Vaccarino, 1997) reflecting a hypo-metabolic state, with concomitant hyper-metabolism in the right prefrontal regions (Bench, Frackowiak & Dolan, 1995). In fact, there have since been 2 main lines of research developed for the treatment of depression with regional TMS (rTMS): low-frequency stimulation inducing neural inhibition on the right dorsolateral prefrontal cortex (dlPFC) (presumably hyperactive in depression), and high-frequency stimulation inducing neural excitation on the left dlPFC (presumably hypoactive in depression), or a combination of two (George et al., 2000; Klein et al., 1999). Nowadays, in most clinical studies currently use multiple sessions of high-frequency (10 Hz) rTMS applied to the left dlPFC (Lefaucheur et al., 2014; Trevizol & Blumberger, 2019). There seems to be interhemispheric asymmetry in the frontal activation and the rate of asymmetry is correlated with clinical scores of depression (Diego, Field & Hernandez-Reif, 2001). Noteworthy, the dlPFC is synaptically connected to the limbic system (striatum, thalamus, and anterior cingulate) involved in mood regulation (Barbas, 2000; Petrides & Pandya, 1999).

It has been proposed, that the right hemisphere dysfunction might precede depression and that the related cognitive deficits might be constant features existing already before onset of depression (Deptula, Manevitz & Yosawitz, 1991). The deteriorated cognitive function and defects in visual and visuospatial reasoning and visual and

visuospatial memory have also been reported in patients having recovered from depression (Bulbena & Berrios, 1993; Sackeim et al., 1992). Behnken et al. (2010) found that compared to healthy controls, individuals with major depressive disorder showed persistent non-verbal memory (but not visual-spatial function) impairments in the remitted phase of depression, and they had difficulties in organizing non-verbal information appropriately during learning (modulated probably by a deficient use of organizational strategies during encoding). In late-life depression, it has been noticed that dysfunction in executive abilities is permanent although the depressional symptoms lowering the daily skills have been treated (Alexopoulos et al., 2005).

Attention deficit hyperactivity disorder (ADHD)

ADHD is the most commonly diagnosed pediatric neurobehavioral disorder in the world (Faraone, Sergeant, Gillberg & Biederman, 2003; Polanczyk, de Lima, Horta, Biederman & Rohde, 2007), and its incidence continues to be high through adolescence and into adulthood (Kessler et al., 2006; Simon, Czobor, Bálint, Mészáros & Bitter, 2009). In Diagnostic and Statistical Manual of Mental Disorders (5th ed.; DSM-5; American Psychiatric Association, 2013), ADHD have different presentations: predominantly inattentive (F90.0), predominantly hyperactive/impulsive (F90.1), or combined (F90.2) forms. The subtype and its severity are diagnosed by collecting information on the experienced (self or observer) symptoms and their duration. Clinically, ADHD might appear as two totally opposite phenotypes, and expressed behavioral symptoms. Basically, all individuals with ADHD are inattentive but others might also be hyperactive. Specifically, ADHD coming out as predominantly inattentive presentation type might be very difficult to identify because of non-apparent or difficultly observable behavioral signs. These children (with ADHD predominantly inattentive type) might be dreamy, passive, and almost hypoactive (Barkley, DuPaul & McMurray, 1990; Lahey & Carlson, 1992) and inattentiveness, unlike hyperactivity and impulsivity, appears to be pervasive with increasing age (Clarke, Barry, McCarthy & Selikowitz, 2001). In fact, it is possible that hypoactivity and hypoarousal is one further endophenotype in ADHD (Conzelmann et al., 2014; Satterfield & Dawson, 1971).

The definition of ADHD has been changing during decades. Historically, there have been descriptions of inattentive, excessive hyperactive, and impulsive children in the literature since the nineteenth century. The disorder was termed Hyperkinetic Reaction of Childhood in DSM-II (American Psychiatric Association, 1968), which as the name implies focused primarily on symptoms of excessive motor activity (Lange, Reichl, Lange, Tucha & Tucha, 2010). In the DSM-III (American Psychiatric Association, 1980) the disorder was re-conceptualized with a focus on problems with attention, impulsivity and hyperactivity, and was renamed Attention Deficit Disorder (ADD, with and without hyperactivity). The term Attention Deficit/ Hyperactivity Disorder (ADHD) was introduced in DSM-III-R (American Psychiatric Association, 1987), with the controversial elimination of ADD without hyperactivity (Epstein & Loren, 2013). Later, in the DSM-IV and the DSM-V (American Psychiatric Association, 1994, 2013, respectively) the term ADHD was retained along with the introduction of three specific subtypes (predominantly Inattentive, predominantly Hyperactive-Impulsive, and Combined), although there are subtle modifications in e.g. diagnostic criteria between the publications (Lange et al., 2010). Because of these theoretical modifications, the scientific ADHD study methodologies and constructs have also changed during

years. There are earlier studies differentiating individuals with ADD and hyperactivity from individuals with ADD but not hyperactivity. For example, Matazow and Hynd (1992a) proposed that children with ADHD and hyperactivity had more problems in tasks that measure serial, cognitive operations (guided from the anterior or frontal areas) and that children with ADHD but not hyperactivity had more problems in tasks that measure visuospatial abilities (guided from the posterior or parietal areas). Researchers (1992b) also noticed that children with ADHD and hyperactivity had similar difficulties than individuals with the right hemisphere dysfunction.

In order to the attentiveness to develop, Luria (1961) thought that the reticular activating system must first mature. Luria considered that attentiveness increases progressively with age and development, and that it can be called a verbal regulation of voluntary function. He thought that the aetiology of attentional deficit disorders is delayed development of excitatory and inhibitory regulation systems in the frontal areas. Shaw et al. (2009) found that the right-handed children with ADHD showed typical gaining of left-hemispheric cortical thickness with age in a similar posterior temporo-occipital region as the typically developing children but they showed no opposing tendency of increasing relative right-hemispheric thickness in the frontal cortex. Shaw et al. (2012) reported that in typically developing children posterior, parieto-occipital cortex matures earlier than more anterior regions. Researchers found a significant maturation delay in children and adolescents with ADHD cortical thickness (CT) and the pattern of delay was highly regional, being most marked in the anterior frontal gyri, particularly on the right. Almeida et al. (2010) showed that CT is thinner in the regions of the right superior frontal area with children, adolescents and adults having ADHD, and there is a correlation between the CT of these regions and the severity of the disorders.

The attentional and executive function processes are interconnected to the prefrontal cortex (PFC), which has been found to be dysfunctional in ADHD (Fuster, 1980; Glanzman, 2001) besides the right-sided frontostriatal system (Heilman, Voeler & Nadeau, 1991; Kasparek, Theiner & Filova, 2015; Weinberg, Harper & Brumback, 2001). In positroni emission tomography (PET) imaging, Zametkin, Nordahl, Gross and King (1990) found a lowered metabolic activity in the premotor areas and the upper parts of the prefrontal areas (Brodmann areas 8 and 9, dlPFC) in adults, who had been hyperactive since childhood. Sergeant (1995), Van der Meere (1996) and Mazaheri et al. (2010) observed that children with ADHD had slowness and/or inaccuracy in their motor reaction (guided from the frontal processes), and they also had inhibition problems in motoric function and in motor organization (Yordanova, Banaschewski, Kolev, Woerner & Rothenberger, 2001), and their early processing of the visual information was clearly ineffective compared to controls (Lenz, et al., 2010). Marzocchi et al. (2008) found that children with ADHD showed deficits on visual working memory, planning, cognitive flexibility and phonetic fluency. Magnetic imaging studies (Castellanos et al., 2000; Filipek et al., 1997; Hynd, Semrud-Clikeman, Lorys, Novey & Eliopulos, 1990) have suggested that the volume of the right prefrontal area with children having ADHD is smaller than controls. The right PFC has been found to be important in regulation of distracting factors, inappropriate behavior and incorrect emotion expression (Arnsten, 2009). The right inferior frontal cortex (IFC) has been found to have a central role in response inhibition (Aron, Fletcher, Bullmore, Sahakian & Robbins, 2003b). Sowell et al. (2003) found in their structural imaging the greatest morphological reductions in right IFC for children and adolescents with ADHD compared with matched controls. Patients with acquired lesions in right hemisphere have been found to display similar

difficulties in their attentional abilities as individuals with ADHD (Voeller & Heilman, 1988).

The PFC has also been associated to short-term memory functions. In fact, there are researchers proposing that the deficiencies in visuospatial working memory are the central neurocognitive defect in ADHD, and the most deteriorated functional area in executive functions (Rapport, Chung, Shore & Isaacs, 2001). It has been suggested that besides problems in executive skills, also motivational difficulties are necessary for generating visuospatial working memory deficiencies (Dovis, Vand der Oord, Wiers & Prins, 2012). In fact, both childhood and adult ADHD have been associated with brain abnormalities not only in fronto-cortical but also in fronto-limbic systems that mediate the control of cognition and motivation (Cubillo, Halari, Smith, Taylor & Rubia, 2012). Undoubtedly, there might be dysfunctional processes also in temporal (Sonuga-Barke, Williams, Hall & Saxton, 1996) and parietal (Mirsky, Anthony, Duncan, Ahearn & Kellam, 1991) areas. Sandson, Bachna and Morin (2000) observed that adults with ADHD (non-familial origin) made more mistakes or omissions on their left side (compared to right side) in the random letter cancellation task, which includes visuospatial features. These individuals with ADHD had also lower performance intelligent quotient (IQ) compared to other adults having ADHD (familial origin), which might refer to right hemisphere dysfunction. The theory of the PFC executing working memory (Baddeley, 1986) has been the predominant theory of the prefrontal function in nonhuman primates. At first, the working memory concept focused mostly on the short-term maintenance of information, and rather less on the manipulation or monitoring of such information or on the use of that information for decisions. Lebedev, Messinger, Kralik and Wise (2004) found in their saccadic eye movement experiment with monkeys that representation of spatial attention is dissociated from representation of spatial memory in PFC in delay-period activity. Researchers suggested that prefrontal activity during the delay-period contributes more to the process of attentional selection and selective attention than to memory storage or maintenance memory.

It has been suggested that a right hemisphere is specialized for orienting attention to novel events (Gitelman et al., 1999; Mesulam, 1998). Daffner et al. (2000) found in their electroencephalography (EEG) studies that normal controls exhibited larger event-related potentials (ERP) P3 responses at right hemisphere sites. The prefrontal region is primarily responsible for creating and maintaining an attentional set, especially when it is more difficult to direct attention to task-relevant information than to task-irrelevant information. Activation has been observed in the dlPFC of the right hemisphere regardless of the nature of information to be processed (e.g. spatial versus verbal). It has been suggested that this region is involved in control processes relevant to working memory. In that regard, the dlPFC might be conceptualized as playing a role in selective attention, choosing the relevant contents of working memory that are required to ensure correct task performance (Banich et al., 2000).

Mood and attention - two sides of the same coin?

Depression and ADHD are highly studied neuropsychiatric disorders with multiple and varying symptoms complicating the understanding of their origin and interpretation. Scientific research has been focusing in capturing different cognitive defects belonging to these disorders but the overall result is still confusing. Instead of producing more and more diverse and specified scientific knowledge, a unifying and integrating viewpoint of the disorders might be more beneficial. Health care professionals

lack useful, sensitive enough tests and indicators to recognize the disorders adequately and in time. There is also an ongoing need for more useful and effective assessment and rehabilitation methods in public and private neuropsychological clinics.

In basic psychological studies, emotion and cognition are introduced and educated as two conceptually and neurally distinct mental states or processes. Low mood (the core deficit in depression) and poor attention (the core deficit in ADHD) might then be considered dividual aspects of psychological functioning. Consistently, treatments have been mainly focusing on improving the mood in depression (e.g. antidepressants, psychotherapy) and concentration and executive skills in ADHD (e.g. neuropsychological, metacognitive individual or group rehabilitation). Nevertheless, there is growing evidence that also cognition should be regarded as a treatment target in depression (Kaser, Zaman & Sahakian, 2017; Rock, Roiser, Riedel & Blackwell, 2014) not to mention that emotion regulation should be treated in ADHD as well (Brown, 2014; Christiansen, Hirsch, Albrecht & Chavanon, 2019).

With neuropsychological testing methods, it is possible to infer the hemispheric origin (left or right hemisphere) of emotional and cognitive dysfunction including in neuropsychiatric disorders. Accordingly, these test results are essential in planning the most effective treatment methods and selecting targeted mental and cognitive exercises. Additionally, they may help scientists to develop new and more functional tests for identification of individuals having the disorders.

AIMS OF THE STUDY

The objective was to explore right hemisphere dysfunctions in depression and ADHD. The aims were to examine the cognitive profiles of young adults with either depression or ADHD or both to find out

a) possible deficits in verbal and visual abilities,

b) possible deficits in verbal and visual memory, and

c) the tendency to direct attention (voluntary or unvoluntary) either to left or right visual side.

METHODS

Participants

There were 40 (37 women and 3 men, mean age 18.9 years, sd. 2.5) examinees in our study. Participants were recruited from colleges and vocational schools via direct informing, and via advertisements in different journals and via flyers given to parents and to health nurses in learning institutions. We composed three study groups: 1) adolescents with depression (DEP n = 10), 2) adolescents with ADHD (ADHD n = 9), and 3) adolescents having both depression and ADHD (BOTH n = 4). There were also normal controls (CTRL n = 17). Participants were matched for age, gender, and education. The study protocol was approved by the Ethical Committee of the Hospital District (Pirkanmaa and South Ostrobothnia).

Table 1.
Description of demographic and clinical data of participants

			Participants						
	All	DEP	ADHD	BOTH	CTRL	H (3)-value	***p*-value**	Groups	Adj. Sig.
Demographic variables	N = 40	N = 10	N = 9	N = 4	N = 17				
Age (years) Mean (sd.)	18.9 (2.5)	18.1 (1.2)	17.8 (1.8)	19.4 (3.2)	19.9 (3.0)	3.432	0.330		
Sex, Female N	37	10	8	3	16				
Sex, Male	3	0	1	1	1				
Diagnosis, Depression	14	10	0	4	0				
Diagnosis, ADHD	9	0	9	0	0				
With antidepressants:									
No	31	4	9	1	17				
Yes	9	6	0	3	0				
With ADHD medication:									
No	36	10	5	4	17				
Yes	4	0	4	0	0				
Physical illnesses:									
No	31	9	4	4	14				
Yes	9	1	5	0	3				
History of accidents:									
No	28	6	5	3	14				
Yes	12	4	4	1	3				
Hospital care needed (prev.)									
No	32	7	6	3	16				
Yes	8	3	3	1	1				
History of family problems:									
No	11	2	3	1	5				
Yes	29	8	6	3	12				
Family history of psychiatric disorders:									
No	24	6	4	3	11				
Yes	16	4	5	1	6				
Right-handed	39	10	8	4	17				
Education (years) Mean (sd.)	11.0 (1.8)	10.4.(1.1)	10.3 (1.5)	11.3 (2.5)	11.7 (1.9)	4.625	0.201		
Grades (upper comp. school):									
Mathematics	6.6 (1.7)	6.4 (1.9)	7.0 (1.5)	6.3 (1.9)	6.8 (1.7)	2.267	0.519		
Handwork	8.3 (0.9)	8.2 (1.0)	8.3 (1.0)	7.5 (0.6)	8.4 (0.8)	4.062	0.255		
Art	8.3 (0.9)	8.3 (1.1)	8.4 (0.9)	7.5 (1.0)	8.4 (0.7)	3.440	0.329		
The mean of all subjects	7.7 (0.7)	7.4 (0.5)	7.7 (0.8)	7.2 (0.8)	8.0 (0.7)	6.737	0.081		
The mean of reading subjects	7.3 (0.9)	6.7 (0.7)	7.3 (1.0)	6.9 (0.9)	7.7 (0.8)	5.787	0.122		
BDI-13 scores	6.9 (8.9)	18.8 (8.3)	1.9 (2.1)	12.3 (4.5)	1.2 (1.65)	27.944	**0.000**	CTRL-BOTH	**0.030**
								CTRL-DEP	**0.000**
								ADHD-DEP	**0.001**
ASRS-13 A scores	12.0 (5.2)	12.9 (3.6)	14.2 (4.6)	16.5 (3.5)	8.3 (3.5)	12.367	**0.006**	CTRL-BOTH	**0.027**
ASRS-13 B scores	19.3 (11.1)	24.7 (8.0)	25.4 (11.9)	25.0 (10.2)	11.6 (5.1)	9.843	**0.020**	CTRL-ADHD	**0.035**

Note. (sd.) = standard deviation, (prev.) = previous, because of previous accidents, (comp.) = comprehensive, upper comprehensive school. Bold indicates metrics with $p < 0.05$. (Adj. Sig.) = Significance values have been adjusted by the Bonferroni correction for multiple tests.

In the DEP group, there were 10 examinees (all female). They were all diagnosed as having Depression (F32-33, ICD-10). Six of them were using the antidepressant medication at the time of the study. One of them had mild depression, two of them had moderate depression and seven of them had severe depression defined by Finnish modification of the 13-item Beck Depression Inventory (BDI-13; Kaltiala-Heino, Rimpelä,Rantanen & Laippala, 1999), in which combined scores are meaning: 0-4 no depression, 5-7 mild depression, 8-15 moderate depression, and 16-36 severe depression. Notably, these scores indicating the severity level of depression were compatible with the severity level of examinees diagnosis.

In the ADHD group, there were 9 examinees (8 female and 1 male). They were all diagnosed as having attention deficit disorder with or without hyperactivity (F90.0,

ICD-10). Four of them were using the attention deficit disorder medication at the time of the study.

In the BOTH group, there were 4 examinees (3 female and 1 male). All 4 examinees were diagnosed as having depression (F32, ICD-10), but they did not have the diagnosis of attention deficit disorder. The likelihood of undiagnosed ADHD became apparent in interviewing the examinee and the relative, and considering their past history from childhood and adolescence. Two of these examinees were diagnosed as having also the attention deficit disorder without hyperactivity (F90.0, ICD-10) later, during a one-year follow-up. Three of them were using the antidepressant medication (but not ADHD medication) at time of the study, and two of them started also ADHD medication later, during a one-year follow-up.

In the CTRL group, there were 17 examinees (16 female and 1 male). Based on self-report, they had neither depression nor ADHD. One of them was using medication for other purposes. Description of all the groups can be found in Table 1.

Methods

An extensive neuropsychological examination (Table 2.) was carried out for all examinees (n = 40). The examination started with semi-structured, Therapeutic Assessment type (Finn, 2007) interview, which was executed to examinee and in some cases also to close relative. Subjects also fulfilled a background information blanket. The verbal and the visual reasoning functions were measured with WAIS-IV (Wechsler, 2008) for examinees 16 years and older, and with WAIS-III (Wechsler, 2005) for examinees younger than 16 years. The verbal and visual memory functions were measured with WMS-III (Wechsler, 2007), the Rey-Osterrieth complex figure test (ROCF) (Lezak, Howieson & Loring, 2004; Rey, 1941) and with the Location Learning Test Revised Edition (LLT) test (Bucks, Willison, Byrne & Kessels, 2011). Visuomotor processing and visual working memory function were measured with Cambridge Neuropsychological Test Automated Battery (CANTAB). The examinees fulfilled the Adult ADHD Self-Report Scale (ASRS-13 modified) (Kessler et al., 2005) on their attentional features and the Shortened Beck Depression Inventory (BDI-13) test (Kaltiala-Heino, Rimpelä, Rantanen & Laippala, 1999; Lukkari, Kaltiala-Heino, Rimpelä & Rantanen, 1998) on their mood.

The tendency to attend visually (overt orienting or selective, executive attention) more to left or right side was analyzed in visual learning and memory tasks. In the WMS-III Visual Reproduction I and II figures 4 and 5, the left-hand figures were summed together and right-hand figures were summed together. In the LLT task, there is a 5 x 5 grid, in which there are 10 pictures of different concrete objects (e.g. an umbrella, a knife, scissors). Two of them are located on the middle column, four on the left side columns and four on the right side columns. The visual attentional orientation was evaluated by counting the hits on the left or on the right side. In the WMS-III and the LLT, the immediate recall and the delayed recall results were analyzed separately.

Due to small sample sizes and skewed variable distributions, we used non-parametric statistics. The independent samples Kruskall-Wallis test was performed to analyze differences between the groups, and the related samples Wilcoxon signed-rank test to analyze differences in repeated measurements. Statistical analyses were corrected for multiple comparisons (Bonferroni).

Table 2.
Neuropsychological assessment methods used in the study

VERBAL reasoning and memory	VISUAL reasoning and memory	QUESTIONNAIRES
WAIS-III, WAIS-IV	WAIS-III, WAIS-IV	Adult Self-Report Scale ASRS-
Arithmetic	Digit-Symbol Coding, Coding	Beck Depression Inventory
Digit Span	Block Design	(the 13 item)
Information	Object Assembly, Visual Puzzles	
Letter-Number Sequencing	WMS-III	
WMS-III	Visual Reproduction I	
Logical Memory I	Visual Reproduction II	
Logical Memory II	Rey-Osterrieth Complex Figure (ROCF)	
Word Lists I	Location Learning Test Revised Ed. (LLT)	
Word Lists II	Cambridge Neuropsychological Test Automated Battery (CANTAB)	
	Rapid Visual Processing (RVP)	
	Spatial Recognition Memory (SRM)	
	Paired Associates Learning (PAL)	
	(clinical or high functioning mode)	

(I) = immediate recall; (II) = delayed recall.

Because we had three different research questions, we divided the alpha significance level 0.05 by three and considered the alpha of 0.016 or smaller as significant. Pearson's chi-square test was performed to analyze differences in the distributions of Wechler's tests between the groups. Tukey's hinges (the lower hinge is the 25th percentile, the midhinge is the 50th percentile or the median, and the upper hinge is the 75th percentile) were used as measures of positions or inclusive quartiles of the data. The analyses were completed using SPSS Statistics 26.0.

RESULTS

Verbal and visual reasoning abilities

From Tukey's Hinges percentiles, visuomotor processing (in paper and pencil task) appears slower in the ADHD group and the BOTH group than in the CTRL group but in a Kruskall-Wallis test, there were no significant differences between the groups after Bonferroni corrections. (Table 3.)

It seemed, that the scores in visual abilities were higher than the scores in verbal abilities per individual, so we analyzed (Wilcoxon signed-rank test), whether there were differences between examinees' verbal and visual reasoning level (mean standard scores, modified). For a clarification, we included the standard scores of WAIS-IV Digit Span, Arithmetic and Letter-Number Sequencing into the verbal reasoning and Digit Symbol-Coding to the visual reasoning (after WAIS-III) for the comparable results. In every group, the mean standard score level of visual reasoning abilities was a bit higher (around 1 to 1.8 standard scores) than the mean standard score level of verbal reasoning level but the differences between the medians within the groups were not statistically significant.

Although there were no differences in verbal and visual reasoning abilities (medians) between the groups, we noticed that perfor-

Table 3.
The verbal and visual abilities WAIS-III and WAIS-IV, raw scores and (standard deviations) in the groups. In visual abilities minimum, maximum and range values are also reported

	DEP group				ADHD group				BOTH group				CTRL group				H (3)-value	χ^2-value	***p-value*** Groups	Adj. Sig.
VERBAL ABILITY																				
Similarities	22.6 (3.3)				19.0 (6.6)				25.5 (4.2)				23.6 (5.4)				5.240		0.155	
Arithmetic	11.8 (4.2)				10.3 (3.0)				9.5 (3.0)				11.6 (2.2)				2.667		0.446	
Digit Span	18.0 (4.6)				15.2 (4.9)				22.3 (5.1)				21.6 (7.7)				6.755		0.080	
Information	14.2 (4.6)				11.1 (3.9)				9.3 (3.4)				11.9 (4.3)				4.124		0.248	
Letter-Number Seq.	13.8 (4.2)				12.1 (4.0)				15.3 (2.5)				14.6 (6.0)				1.556		0.670	
Verbal reas. (st.sc.)																				
Mean (mod.)	9.8 (3.2)				7.5 (3.1)				6.8 (1.9)				9.2 (2.1)							
Below average	28.0 (%)				42.2 (%)				55.0 (%)				27.1 (%)							
Average	50.0 (%)				55.6 (%)				40.0 (%)				60.0 (%)							
Above Average	22.0 (%)				2.2 (%)				5.0 (%)				12.9 (%)					15.36, df = 6	0.018	
Verbal reas. - Visual reas.	Z = 39.0,				Z = 39.0,				Z = 7.0,				Z = 113.5,							
Z = 616.5, p = **0.002**	p = 0.241				p = 0.051				p = 0.465				p = 0.019							
VISUAL ABILITY		Min.	Max.	R.		Min.	Max.	R.		Min.	Max.	R.		Min.	Max.	R.				
Picture Completion	17.6 (4.6)	10	24	14	15.1 (5.7)	6	22	16	10.8 (4.8)	5	16	11	15.3 (3.4)	10	21	11	5.616		0.132	
Digit Symbol Coding																				
Coding	70.7 (13.2)	56	101	45	55.3 (19.5)	22	77	55	56.8 (13.8)	37	69	32	76.5 (14.3)	46	94	48	9.968		0.019	ADHD-CTRL 0.032
Block Design	49.4 (12.3)	28	62	34	46.2 (13.5)	20	65	45	43.0 (9.1)	32	51	19	50.5 (10.9)	32	64	32	2.511		0.473	
Object Assembly	38.7 (7.3)	26	47	21	34.3 (10.7)	21	47	26	-				30.9 (6.9)	21	42	21	2.843		0.241	
Visual Puzzles	15.8 (5.4)	8	20	12	14.2 (4.8)	7	19	12	15.8 (3.7)	11	20	9	19.2 (4.8)	8	24	16	5.802		0.122	
Visual reas.(st. sc.)																				
Mean (mod.)	10.9 (2.8)				9.0 (3.2)				7.8 (3.1)				10.7 (2.2)							
Below average	15.0 (%)				27.8 (%)				37.6 (%)				16.2 (%)							
Average	55.0 (%)				58.3 (%)				56.2 (%)				52.9 (%)							
Above Average	30.0 (%)				13.9 (%)				6.2 (%)				30.9 (%)					9.98, df = 6	0.126	
Tukey's Hinges perc.																				
(DSC, C) *	60.0 68.5 77.0				47.0 64.0 68.0				48.5 60.5 65.0				66.0 80.0 87.0							

Note. (Seq.) = Sequencing, (Min.) = minimum, (Max.) = maximum, (R.) = range, (st. sc) = standard scores, (mod.) = modified, (reas.) = reasoning, Below average = results (standardized) in WAIS-III and WAIS-IV that have fallen below average (1, 2, 3 = Extremely Low, 4,5 = Very Low, and 6,7 = Low Average), Average = results (standardized) that have fallen on average (8-12 = Average), Above average = results (standardized) that have fallen above average (13,14 = High Average, 15, 16 = Very High, and 17, 18, 19 = Extremely High). (perc.) = percentiles, (*) = percentiles of 25, 50 and 75. (DSC) = Digit Symbol Coding, (C) = Coding. Bold indicated metrics with $p < 0.016$. (Adj. Sig.) = Significance values have been adjusted by the Bonferroni correction for multiple tests.

mance scores distributed unevenly when examining how three lowest standard scores (in Wechsler's tests) (1, 2 and 3 = extremely low, 4 and 5 = very low, and 6 and 7 = low average) and three highest standard scores (13 and 14 = high average, 15 and 16 = very high, and 17, 18 and 19 = extremely high) were divided between the groups. However, in a Pearson's chi-square test, there were no significant differences between the results.

Verbal and visual memory performance

Verbal memory performance

In the Longest Digit Span Forward (LDSF), a Kruskall-Wallis test provided evidence of a difference between at least one pair of groups. In pairwise comparisons, the ADHD group's ability of recall digits (numbers) forward was significantly lower ($p = 0.010$) than the DEP group (adjusted using the Bonferroni correction).

The Logical Memory I, a Kruskall-Wallis test provided evidence of a difference between the mean ranks of at least one pair of groups ($p = 0.016$) but in pairwise comparisons, there were no significant differences. Although, when analyzing the scores of stories A ja B1 (the Logical Memory I) and word lists A1 and B (the Word Lists I) separately, there was a significant difference between the groups scores in the immediate recall of Logical Memory story B ($p = 0.003$). The CTRL group and the DEP group succeeded much better than the ADHD group and the BOTH group. In pairwise comparisons, the BOTH group's memory recall (in a second story) was significantly worse than the CTRL group's ($p = 0.010$), adjusted using the Bonferroni correction.

In the Word Lists II, a Kruskall-Wallis test provided evidence of a difference between

Table 4.
Verbal memory performance raw scores and (standard deviations) in the groups

	DEP group	ADHD group	BOTH group	CTRL group	H (3)-value ***p*** **-value**	Groups	Adj. Sig.
VERBAL MEMORY							
Logical Memory I	48.6 (13.5)	37.9 (10.0)	30.8 (5.0)	46.3 (8.5)	H (3) = 10.310, p = **0.016**	BOTH-CTRL	0.046
Logical Memory II	31.3 (9.9)	24.4 (7.7)	18.8 (3.8)	29.8 (6.8)	H (3) = 9.045, p = 0.029		
Word Lists I	36.8 (7.3)	29.9 (7.3)	29.3 (4.9)	36.2 (4.1)	H (3) = 9.090, p = 0.028		
Word Lists II	9.1 (1.7)	7.9 (3.1)	5.5 (1.3)	9.6 (1.5)	H (3) = 10.975, p = **0.012**	BOTH-CTRL	**0.013**
Digit Span	18.0 (4.6)	15.2 (4.9)	22.3 (5.1)	21.6 (7.7)	H (3) = 6.755, p = 0.080		
Digit Span Forward	7.8 (1.5)	6.0 (2.1)	7.5 (2.4)	9.0 (2.1)	H (3) = 9.263, p = 0.026	ADHD-CTRL	0.052
						ADHD - DEP	0.032
Digit Span Backward	7.8 (1.5)	5.2 (1.3)	8.0 (1.8)	8.8 (1.8)	H (3) = 6.492, p = 0.090		
Longest DSF	5.5 (0.6)	4.4 (1.1)	5.3 (1.5)	5.7 (1.2)	H (3) = 10.179, p = 0.017	ADHD - DEP	**0.010**
Longest DSB	4.5 (1.3)	3.0 (0.7)	4.3 (1.0)	5.0 (1.2)	H (3) = 4.693, p = 0.196		
DS Sequencing	6.5 (1.9)	6.2 (2.5)	6.8 (1.5)	9.4 (1.9)	H (3) = 8.848, p = 0.031		
Longest DSS	4.8 (1.3)	4.6 (1.5)	5.0 (1.2)	6.4 (1.4)	H (3) = 6.626, p = 0.085		
Longest LNS	5.0 (0.8)	4.2 (0.8)	4.0 (0.8)	5.5 (1.3)	H (3) = 4.224, p = 0.238		
Logical Memory, story A	17.2 (6.2)	13.2 (4.1)	11.8 (1.9)	15.2 (3.0)	H (3) = 7.257, p = 0.064		
Logical Memory, story B1	13.0 (4.5)	9.6 (3.0)	6.5 (1.9)	13.8 (2.6)	H (3) = 14.004, p = **0.003**	BOTH - DEP	0.034
						BOTH - CTRL	**0.010**
Logical Memory, story B2	18.4 (4.0)	14.8 (4.5)	12.0 (4.2)	18.5 (2.9)	H (3) = 7.318, p = 0.062		
Word Lists, list A1	6.8 (2.9)	5.0 (1.4)	4.8 (1.3)	6.2 (1.3)	H (3) = 5.318, p = 0.150		
Word Lists, list B1	6.1 (1.7)	4.1 (1.6)	5.3 (1.3)	6.3 (2.2)	H (3) = 7.373, p = 0.061		
Verbal memory Mean (st.sc.) (mod.) *	10.9 (3.4)	8.0 (3.2)	5.6 (1.7)	10.7 (1.8)			
Below Average	20.0 (%)	44.4 (%)	75.0 (%)	8.7 (%)			
Average	50.0 (%)	47.2 (%)	25.0 (%)	69.2 (%)	X2 = 41.33, df = 6, p = **0.000**		
Above Average	30.0 (%)	8.4 (%)	0.0 (%)	22.1 (%)			

(ROCF) = Rey-Osterrieth Complex Figure, (I) = immediate recall, (II) = delayed recall, (sec.) = seconds, (min.) = minutes (*) = Verbal memory includes tests Logical Memory I & II, and Word Lists I & II. Below average = results (standardized) in WMS-III that have fallen below average (1, 2, 3 = Extremely Low, 4,5 = Very Low, and 6,7 = Low Average), Average = results (standardized) that have fallen on average (8-12 = Average), Above average = results (standardized) that have fallen above average (13,14 = High Average, 15, 16 = Very High, and 17, 18, 19 = Extremely High)Bold indicated metrics with $p < 0.016$. (Adj. Sig.) = Significance values have been adjusted by the Bonferroni correction for multiple tests.

the mean ranks (p = 0.012) and in pairwise comparisons the BOTH group's delayed recall performance was significantly worse (p = 0.013) compared to the CTRL group (adjusted using the Bonferroni correction).

We also analyzed verbal and visual memory performance levels between the groups and there was a significant difference in verbal memory (but not visual memory) between the groups (p = 0.008) but the differences were not significant in pairwise comparisons. Verbal memory results were divided unevenly between the groups (p = 0.000) and the ADHD group and the BOTH group had much more lower results (and less above average results) than the DEP group and the CTRL group. (Table 4).

Visual memory performance

In CANTAB PAL task, there were two different modes (clinical mode and high functioning mode) in use because at the beginning of the study the high functioning mode was not available. The results of those two different modes were analyzed separately. In high functioning mode results, there was a significant difference (p = 0.014) between the groups regarding a total amount of trials in 12 shapes task. The BOTH group needed significantly more trials (p = 0.011) compared to the CTRL group (adjusted using the Bonferroni correction). Although there were no differences between the duration times, from the Tukey's Hinges percentiles it can be seen that the BOTH group needed much more time to complete the task. The distribution of visual memory scores could not be tested with chi-square test because of too many low counts. It seemed though that the ADHD group and

Table 5.
Visual memory performance raw scores and (standard deviations) in the groups. Also minimum, maximum and range values are presented

	DEP group				ADHD group				BOTH group				CTRL group				H (3)-value	***p-value***
VISUAL MEMORY		Min.	Max.	R.		Min.	Max.	R.		Min.	Max.	R.		Min.	Max.	R.		
Visual Reproduction I	95.1 (8.7)	77	104	27	90.4 (10.3)	72	103	31	86.0 (17.8)	62	100	38	95.7 (7.7)	70	104	34	2.340	0.505
Visual Reproduction II	76.4 (15.8)	53	103	50	77.1 (17.2)	43	102	59	59.0 (11.1)	43	67	24	77.2 (16.0)	38	100	62	5.324	0.150
ROCF, copying time sec.	175.2 (88.1)	69	315	246	180.2 (73.9)	99	352	253	186.5 (20.5)	167	208	41	149.8 (51.6)	91	269	178	2.817	0.421
ROCF, immed. recall. time sec.	142.8 (82.3)	46	292	246	132.2 (74.6)	49	284	235	109.3(26.6)	72	135	63	151.4 (75.8)	58	321	263	1.585	0.663
ROCF, delayed recall time sec.	83.5 (41.3)	34	178	144	87.7 (33.7)	44	144	100	74.3 (8.7)	64	84	20	97.0 (34.7)	42	173	131	2.148	0.542
ROCF, copying score	33.4 (2.8)	27	36	9	33.4 (2.4)	29	36	7	33.0 (1.4)	31	34	3	34.4 (1.9)	30	36	6	2.815	0.421
ROCF, immediate recall score	17.9 (7.2)	3	29	26	21.7 (6.7)	11	30	19	15.8 (7.7)	6	24	18	19.3 (8.0)	4	30	26	2.237	0.525
ROCF, delayed recall score	18.4 (5.6)	11	28	17	21.3 (8.5)	4	30	26	15.0 (8.3)	5	25	20	19.9 (7.1)	9	32	23	2.595	0.458
LLT, learning curve, 5 trials	42.4 (5.6)	35	47	12	40.8 (8.7)	19	48	29	42.3 (3.3)	38	46	8	44.3 (5.5)	28	48	20	3.073	0.380
LLT, Total Displacement Score	11.8 (6.4)	4	19	15	16.8 (24.4)	2	81	79	17.3 (13.5)	4	35	31	9.3 (10.3)	2	40	38	2.598	0.458
LLT, Learning Index	0.9 (0.1)	0.8	1.0	0.2	0.8 (0.3)	0.1	1.0	0.9	0.8 (0.2)	0.6	1.0	0.4	0.9 (0.1)	0.5	1.0	0.5	1.240	0.743
LLT, delayed recall score	10.0 (0.0)	10	10	0	9.6 (1.0)	7	10	3	10.0 (0.0)	10	10	0	9.8 (0.6)	8	10	2	2.438	0.487
LLT, trial 1 score	5.2 (2.4)	2	8	6	5.1 (1.9)	3	8	5	5.3 (0.5)	5	6	1	6.0 (2.6)	0	9	9	1.869	0.600
RVP, Total hits	16.0 (5.1)	5	23	18	11.4 (4.7)	3	17	14	12.3 (6.8)	3	19	16	14.8 (5.0)	6	22	16	4.142	0.246
RVP, Total misses	11.0 (5.1)	4	22	18	15.6 (4.7)	10	24	14	14.8 (6.8)	8	24	16	12.2 (5.0)	5	21	16	4.195	0.241
RVP, False alarms	2.7 (3.0)	0	9	9	4.3 (4.2)	0	14	14	2.5 (0.6)	2	3	1	2.1 (1.8)	0	6	6	1.975	0.578
RVP, Total correct rejections	247.3 (9.9)	226	264	38	236.7 (12.8)	209	251	42	239.3 (13.9)	220	253	33	245.8 (10.8)	227	261	34	4.531	0.210
RVP, Probability of hit	0.59 (0.19)	0.2	0.9	0.7	0.45 (0.13)	0.3	0.6	0.3	0.45 (0.25)	0.1	0.7	0.6	0.55 (0.18)	0.2	0.8	0.6	3.771	0.287
RVP, Probability of false alarms	0.01 (0.01)	0.0	0.0	0.0	0.02 (0.02)	0.0	0.1	0.1	0.01 (0.00)	0.0	0.0	0.0	0.00 (0.00)	0.0	0.0	0.0	2.305	0.512
RVP, A'	0.89 (0.05)	0.8	1.0	0.2	0.83 (0.09)	0.6	0.9	0.3	0.85 (0.08)	0.7	0.9	0.2	0.88 (0.05)	0.8	1.0	0.2	4.558	0.207
RVP, B'	0.90 (0.13)	0.6	1.0	0.4	0.83 (0.23)	0.3	1.0	0.7	0.89 (0.08)	0.8	0.9	0.1	0.93 (0.06)	0.8	1.0	0.2	1.773	0.621
RVP, Responce latency mean	399.0 (95.7)	250.6	545.9	295.4	412.4 (116.4)	289.1	702.0	412.9	533.7 (255.5)	370.7	915.0	544.3	385.0 (59.4)	305.4	489.1	183.7	1.913	0.591
SRM, Number correct	17.2 (2.1)	13	20	7	16.4 (2.1)	12	20	8	15.5 (3.4)	12	20	8	17.9 (1.4)	15	20	5	4.970	0.174
SRM, Number incorrect	2.8 (2.1)	0	7	7	3.9 (1.7)	2	8	6	4.5 (3.4)	0	8	8	2.1 (1.4)	0	5	5	7.309	0.063
SRM, Mean correct latency sec.	2.1 (0.5)	1.2	2.8	1.6	2.1 (0.6)	1.5	3.5	2.0	2.2 (0.4)	1.9	2.8	0.9	2.6 (1.0)	1.6	6.3	4.7	5.065	0.167
SRM, Mean incorrect latency sec	2.8 (1.2)	1.1	4.7	3.6	3.4 (1.9)	1.8	7.4	5.6	3.3 (0.8)	2.7	4.2	1.5	3.8 (2.4)	1.3	8.1	6.8	1.563	0.668
SRM, Test duration min.	4.67 (0.76)	3.4	6.1	2.7	4.64 (0.53)	4.0	5.7	1.7	4.77 (0.80)	4.1	5.9	1.8	5.04 (1.07)	3.4	7.6	4.2	1.248	0.741
Visual memory Mean (st.sc.) (mod.) *	9.9 (3.6)				8.7 (3.6)				6.5 (3.5)				9.3 (2.8)					
Below Average	25.0 (%)				38.9 (%)				75.0 (%)				23.6 (%)					
Average	55.0 (%)				44.4 (%)				25.0 (%)				64.7 (%)	χ^2 could not be done due to small values				
Above Average	20.0 (%)				16.7 (%)				0.0 (%)				11.7 (%)					

Note. (ROCF) = Rey-Osterrieth Complex Figure, (I) = immediate recall, (II) = delayed recall, (sec.) = seconds, (min.) = minutes (*) = Visual memory includes tests Visual Reproduction I & II. Below average = results (standardized) in WMS-III that have fallen below average (1, 2, 3 = Extremely Low, 4,5 = Very Low, and 6,7 = Low Average), Average = results (standardized) that have fallen on average (8-12 = Average), Above average = results (standardized) that have fallen above average (13,14 = High Average, 15, 16 = Very High, and 17, 18, 19 = Extremely High), (st. sc.) = standard scores, (mod.) = modified. Bold indicated metrics with $p < 0.016$. (Adj. Sig.) = Significance values have been adjusted by the Bonferroni correction

especially the BOTH group had more lower results compared to other groups. (Tables 5 and 6).

Visual attention orientation (left or right side)

Table 5 shows the visual orientation in the visual memory tasks. The LLT task was executed to 30 examinees (the DEP group = 5, the ADHD group = 9, the BOTH group = 4, the CTRL group = 12). Wilcoxon signed rank test (p = 0.011) provided evidence of a difference between the mean ranks concerning the orientation to left or right visual side. In LLT task I (immediate recall), 70 percent of the examinees (the DEP group 100 %, the ADHD group 66.7 %, the BOTH group 75 % and the CTRL group 58.3 %) remembered objects better on their left side. In WMS-III Visual Reproduction I (immediate recall), 65 percent of the examinees (the DEP group 60 %, the ADHD group 66.7 %, the BOTH group 50 % and the CTRL group 70.6 %) remembered features better on their right side, although the difference was not significant. In delayed recall tasks, there were no differences between the side of orientation (the mean ranks were even). (Table 7).

DISCUSSION

In this study, we were interested in finding out neurocognitive profiles of young adults having depression and/or ADHD by focus-

Table 6.

Visual memory performance PAL tests raw scores and (standard deviations) in the groups. Also minimum, maximum and range values are presented in high function mode

PAL, Paired Associates Learning	DEP				ADHD				BOTH				CTRL				H (2)-value *			
		Min.	Max.	R.		Min.	Max	R.		Min.	Max.	R.		Min.	Max.	R.	H (3)-value **	***p-value***	Groups	Adj. Sig.
Clinical mode, N	8				3				0				5							
High function mode, N	2				6				4				12							
PAL, Number of reached figures 1	8.0 (0.0)				8.0 (0.0)				0				8.0 (0.0)				0.000	1.000		
PAL, Number of reached figures 2	12.0 (0.0)				12.0 (0.0)				12.0 (0.0)				12.0 (0.0)				0.000	1.000		
PAL, Total errors adjusted 1	3.9 (3.1)				2.3 (2.5)								4.2 (3.3)				0.817	0.665		
PAL, Total errors adjusted 2	15.0 (1.4)	14	16	2	32.2 (24.3)	3	71	68	47.0 (38.5)	7	87	80	9.0 (5.1)	3	19	16	7.561	0.056		
PAL, Total errors 8 shapes 1	2.4 (2.3)				0.3 (0.6)								3.6 (3.0)				3.096	0.213		
PAL, Total errors 8 shapes 2	2.0 (1.4)	1	3	2	3.8 (4.2)	0	12	12	2.3 (3.9)	0	8	8	0.8 (1.1)	0	3	3	5.773	0.123		
PAL, Total errors 10 shapes 2	3.5 (2.1)	2	5	3	13.5 (14.2)	0	40	40	6.5 (4.8)	0	11	11	3.4 (2.5)	1	9	8	4.282	0.233		
PAL, Total errors 12 shapes 2	7.0 (1.4)	6	8	2	12.0 (7.3)	3	23	20	36.3 (30.4)	7	73	66	4.5 (3.1)	0	9	9	10.101	0.018	CTRL - BOTH	0.021
PAL, Mean errors to success 1	0.5 (0.4)				0.3 (0.3)								0.5 (0.4)				0.817	0.665		
PAL, Mean errors to success 2	3.0 (0.3)	2.8	3.2	0.4	6.4 (4.9)	0.6	14.2	13.6	10.5 (9.3)	1.4	21.8	20.4	1.8 (1.0)	0.6	3.8	3.2	7.561	0.056		
PAL, Mean trials to success 1	1.2 (0.2)				1.2 (0.2)								1.3 (0.2)				0.407	0.816		
PAL, Mean trials to success 2	1.9 (0.4)	1.6	2.2	0.6	2.5 (1.1)	1.2	3.8	2.6	3.0 (1.5)	1.4	4.5	3.1	1.7 (0.2)	1.4	2.2	0.8	4.472	0.215		
PAL, Total trials adjusted 1	9.9 (1.6)				9.3 (1.5)								10.0 (1.6)				0.407	0.816		
PAL, Total trials adjusted 2	9.5 (2.1)	8	11	3	12.7 (5.4)	6	19	13	14.0 (6.1)	7	20	13	8.3 (1.6)	7	11	4	4.382	0.223		
PAL, Total trials 8 shapes 1	2.0 (0.9)				1.3 (0.6)								2.6 (1.1)				2.930	0.231		
PAL, Total trials 8 shapes 2	2.0 (0.0)	2	2	0	2.2 (1.0)	1	4	3	1.8 (1.0)	1	3	2	1.4 (0.5)	1	2	1	4.284	0.232		
PAL, Total trials 10 shapes 2	2.0 (0.0)	2	2	0	3.7 (2.3)	1	7	6	2.8 (1.3)	1	4	3	2.4 (0.5)	2	3	1	2.617	0.455		
PAL, Total trials 12 shapes 2	2.5 (0.7)	2	3	1	3.3 (1.2)	2	5	3	6.8 (3.3)	3	10	7	2.4 (0.5)	25	36	11	10.672	**0.014**	CTRL - BOTH	**0.011**
PAL, First trial memory score 1	23.0 (2.8)				24.3 (1.5)								23.2 (2.3)				0.480	0.787		
PAL, First trial memory score 2	25.0 (2.8)	23	27	4	22.8 (8.0)	13	36	23	24.0 (9.1)	16	35	19	31.3 (3.6)	25	36	11	6.671	0.083		
PAL, Duration time min. 1	7.5 (1.2)				7.9 (0.9)								8.1 (1.0)				0.853	0.653		
PAL, Duration time min. 2	11.3 (2.1)	9.8	12.8	3	13.4 (4.0)	8.8	18.2	9.4	15.5 (5.4)	8.9	20.0	11.1	9.9 (0.8)	8.3	11.3	3.0	4.140	0.247		
Tukey's Hinges percentiles (25, 50, 75)	9.8, 11.3, 12.8				9.3, 13.3, 17.5				11.0, 16.5, 19.9				9.5, 10.0, 10.5							

Note. (Min.) = minimum, (Max.) = maximum, (R.) = range, 1 = clinical mode, 2 = high functioning mode, (*) = for clinical mode, (**) = for high functioning mode. *Bold indicated metrics with p < 0.016. (Adj. Sig.) = Significance values have been adjusted by the Bonferroni correction for multiple tests.*

Table 7.

The visual learning results (the left and the right visual side) raw scores and (standard deviations) in the groups. The percentages indicate the amount of examinees preferred orientation side in LLT and VR tasks.

	DEP (n = 5)		ADHD (n = 9)		BOTH (n = 4)		CTRL (n = 12)		H (3)-value	Z-value	Pdiff.	Ndiff.	NTies	***p-value***
LLTleftI	19.0 (1.0)	100.0 (%)	17.8 (2.6)	66.7 (%)	17.5 (2.4)	75.0 (%)	18.3 (2.0)	58.3 (%)	2.779					0.427
LLTrightI	16.2 (3.0)	0.0 (%)	15.9 (5.3)	22.2 (%)	17.0 (0.8)	25.0 (%)	17.7 (2.9)	25.0 (%)	1.309					0.727
Even *		0.0 (%)		11.1 (%)		0.0 (%)		16.7 (%)		293.5	21	6	3	**0.011**
		100.0 (%)		100.0 (%)		100.0 (%)		100.0 (%)						
LLTleftII	4.0 (0.0)	0.0 (%)	4.0 (0.0)	22.2 (%)	4.0 (0.0)	0.0 (%)	4.0 (0.0)	0.0 (%)	4.828					0.185
LLTrightII	4.0 (0.0)	0.0 (%)	3.7 (0.7)	0.0 (%)	4.0 (0.0)	0.0 (%)	4.0 (0.0)	0.0 (%)	0.000					1.000
Even		100.0 (%)		77.8 (%)		100.00 (%)		100.0 (%)		3.0	2	0	28	0.180
		100.0 (%)		100.0 (%)		100.0 (%)		100.0 (%)						

	DEP (n = 10)		ADHD (n = 9)		BOTH (n = 4)		CTRL (n = 17)		H (3)-value	Z-value	Pdiff.	Ndiff.	NTies	***p-value***
VRleftI	27.1 (5.5)	20.0 (%)	26.9 (4.6)	33.3 (%)	18.5 (14.5)	25.0 %	27.3 (4.3)	23.5 (%)	3.798					0.284
VRrightI	29.2 (6.1)	60.0 (%)	26.0 (7.2)	66.7 (%)	28.5 (2.4)	50.0 %	28.8 (4.3)	70.6 (%)	1.015					0.798
Even		20.0 (%)		0.0 (%)		25.0 (%)		5.9 (%)		198.5	10	26	4	0.034
		100.0 (%)		100.0 (%)		100.0 (%)		100.0 (%)						
VRleftII	23.4 (8.9)	30.0 (%)	25.0 (5.6)	66.7 (%)	10.5 (7.0)	25.0 (%)	22.0 (10.0)	41.2 (%)	2.459					0.483
VRrightII	21.4 (12.2)	60.0 (%)	19.0 (9.3)	22.2 (%)	14.5 (1.3)	50.0 (%)	18.4 (10.5)	41.2 (%)	7.397					0.060
Even		10.0 (%)		11.1 (%)		25.0 (%)		17.6 (%)		370.0	17	17	6	0.215
		100.0 (%)		100.0 (%)		100.0 (%)		100.0 (%)						

Note. (*) = no differences between orientation (the left and right) sides, (Pdiff.) = positive differences, (Ndiff.) = negative differences, (Nties) = number of ties, (I) = immediate recall, (II) = delayed recall. Bold indicated metrics with $p < 0.016$.

ing on the role of right hemisphere functioning. Our inclusion criterias were a) the age of 15 to 25 and b), and having depression or ADHD as the first diagnosis. The examinees were categorized in four groups: a) young adults having depression (n = 10), b) young adults having ADHD (with or without hyperactivity, n = 9), c) young adults having both depression and ADHD (n = 4), and d) young adults, who based on their notification did not have either depression or ADHD (n = 17). Results showed that the adolescents having ADHD or depression and ADHD had verbal memory problems besides performing significantly worse in verbal (but not visual) ability tasks. As for the visual memory, the adolescents having both disorders could be detected only by the most demanding, visuospatial (12 shapes) learning task, in which they made most errors and needed more trials to complete the task compared to controls. In attentional tendencies, there was a clear preference to attend to left side in easy task but examinees' visual attention was directed more to their right side in more demanding visual memory task.

Based on an extensive neuropsychological examination, there were no significant differences in verbal reasoning abilities between the groups. However, when analyzing the results more thoroughly, we noticed that in the ADHD group and the BOTH group, there were more extremely low, very low and low average standard scores in verbal reasoning tasks compared to the DEP group and the CTRL group. Conversely, the adolescents having ADHD and both disorders had clearly less verbal standard scores laying above average (high average, very high, or extremely high) compared to the depressed adolescents and the controls. Yet, these differences between the groups were not statistically significant, presumably due to our small sample sizes. In visual reasoning abilities, there were neither significant differences between the groups. Interestingly, we found that the scores in the visual reasoning tasks divided also unevenly - and in the same way than verbal scores - between the groups. In other words, the ADHD group and the BOTH group had more below average and less above average scores compared to the DEP group and the CTRL

group but these differences were not statistically significant.

The adolescents having ADHD or both disorders were a bit, but not significantly, slower in some tasks (e.g. Coding, ROFC copying time, PAL high functioning duration time) than the other examinees. The visuomotor processing slowness of individuals having ADHD is not a new observation. Motor difficulties have been reported in children having ADHD (Piek, Pitcher & Hay, 1999) and they have been rated as clumsy by both medical professionals and teachers on a questionnaire of motor dysfunction (Kadesjö & Gillberg, 1998), to have worse handwriting (Racine, Majnemer, Shevell & Snider, 2008), and have more frequent developmental motor delays (Yochman, Ornoy & Parush, 2006b). This kind of impaired visuomotor adaptation has also been reported in young adults having ADHD (Kurdziel, Dempsey, Zahara, Valera & Spencer, 2015). The slowness of the individuals having ADHD might not be just due to their visuoprocessing skills. It might also be that they experience paper and pencil versions of tests boring, which might be the main reason for performing poorly on these tasks. Becoming bored has been shown to lead a verbal planning impairment in adult ADHD individuals (Desjardins, Scherzer, Braun, Godbout & Poissant, 2010). Importantly, with the largest dataset to date, Hoogman et al. (2017) showed that there are subcortical brain volume differences in children and adults having ADHD. The largest effect was found specifically in amygdala, which region links ADHD to emotional regulation problems.

In Finland, Aalto-Setälä (2002), Castaneda (2010) and Gyllenberg (2012) have examined the consequences of depression to young adults' cognition with partly different and conflicting results. Hopefully, we found that depressed adolescents' cognition was not deteriorated. In fact, their performance was gifted in many cognitive areas (measured with two highest standard score categories in Wechsler's tests). For a long time, there have been assumptions of a possible link between intelligence and mood disorders. For example, Penney, Miedema and Mazmanian (2015) demonstrated that verbal intelligence in particular is a positive predictor of worry and rumination (associated in depression) as well as being predictive of severity of both processes. Karpinski, Kinase Kolb, Tetreault and Borowski (2018) found that there was a higher incidence of mood disorders in the high intelligence sample (17.3% more than the national average) compared to those with average abilities. Researchers suggested a relationship between a heightened cognitive capacity (hyper brain) and heightened psychological and subsequent physiological immune responses (hyper body). Bahrami et al. (2021) found that there is also a genetic link between intelligence and depression. They found a large number of overlapping genes shared between depression and cognitive ability, and suggested that similar genetic factors may be regulating brain pathways involved in regulating cognition and mood.

In verbal memory functions, we found that the adolescents having ADHD had the lowest capacity to recall digits (numbers) forward and backward compared to the other groups. In these digit span tasks, they could recall significantly less digits forward (Longest DSF) than the adolescents having depression. Verbal memory impairment was also found in story recalling (Logical Memory I and II) and verbal learning (Word List I) among the adolescents having ADHD and both disorders. Specifically, we found that the adolescents having depression and ADHD could recall significantly less verbal material after a cognitive load (Word List II) compared to the controls. This is notable, because they had heard and rehearsed the word list (12 words) 4 or 5 times. We also noticed that in the second story, story B1, the adolescents having

both disorders recalled significantly less verbal material compared to the controls. In visual memory functions, the performance of the groups was more stable compared to verbal memory performance. However, we found that the adolescents having both disorders had significant problems in the most challenging, software based, visuoassociative learning task (12 shapes), in which they needed more trials than the controls to succeed the task.

It seems that verbal memory is more vulnerable than visual memory in adolescents having ADHD or both disorders. In verbal memory domain, it is easier to catch these memory defects (5 to 6 digits forward are enough) but in visual domain, we need at least 12 shapes or blocks to reveal possible deterioration. In fact, the depressed adolescents showed the best verbal recall ability compared to other groups. On the contrary, the adolescents with both disorders had the weakest verbal recall in every single test. Their verbal memory impairment may be described auditive-verbal slowness or fatiguing that we could reveal by splitting verbal memory tasks into parts. This way, we could find more subtle information of the examinees' ability to attend to verbal (and visual) material. We constituted a variable, in which we summed up raw scores of verbal material heard just once before any repetitions. In this way, we found that the controls and the depressed adolescents could learn significantly more auditive-verbal material by hearing it just once compared to the adolescents having both disorders. Interestingly, we found that the adolescents having ADHD needed more repetitions of assessment questions compared to other groups.

We measured auditive-verbal short-term and working memory capacity with Wechsler's span tasks, in which a span refers to the longest sequence of correctly recalled digits or numbers. Unfortunately, it is more difficult to measure visual short-term and working memory capacity without motor component. Zhao et al. (2016) found in their spectroscopy studies that there are clear relations between the behavioral executive function scores and the resting-state functional network topological properties in the prefrontal cortex, and especially the right prefrontal cortex in the resting-state seems to be crucial to executive functions overall. Researchers related planning in the right middle frontal gyrus (MFG), working memory mainly in the right MFG and triangular interior frontal gyrus (IFG), short-term memory in the left dorsal superior frontal gyrus (SFG), and task switch in the right MFG. They examined visual short-term memory with CANTAB spatial short-term memory (SSP) task (apparently with its clinical version), in which participants were asked to repeat the order by clicking the boxes which had changed color. At the beginning nine white boxes were displayed on the screen and then some of them would change color in a specific order. The difficulty level ranged from 2 to 9 boxes and the outcome scores of span length and total errors (but not reverse version) were measured. In our opinion, this visuospatial span task forward corresponds to digit span forward DSF (and Longest DSF) tasks, which we used in our study. We found that the adolescents with ADHD had lowered capacity to recall digits than others measuring a function of short-term memory. Zhao et al. hypothesize that there seems to be a relationship between the spontaneous activity in the left frontal pole and the ability of short-term memory. If so, we propose that DSF and Longest DSF task performances are related to the left frontal pole. If so, it seems that the adolescents with ADHD have weaker regional interactions (small-world network properties) in the left frontal pole in resting-state. Then, if tasks are becoming more complex, the activation of right hemisphere is demanded. Considering also a possibility that negative emotions and/or withdrawal motivation are associ-

ated with relatively greater right frontal activity (Harmon-Jones, 2003) compared to the left frontal activity, it is understandable why the individuals having ADHD are usually performing poorly in many ordinary tasks.

There are researchers thinking that it is rather auditive-verbal working memory than visuospatial working memory, which deteriorates more broadly in adolescents having ADHD (Boonstra, Oosterlaan, Sergeant & Buitelaar, 2005; Hervey, Epstein & Curry, 2004; Gropper & Tannock, 2009). In our study, it really seems to be so. Besides having weaker auditive-attentional capacity, the poor performance in digit span and the Longest Digit Span Forward task might reflect a rapid motivational degradation. This kind of task might be interpreted as boring and dull, too simple and not challenging enough among the adolescents having ADHD. On the other hand, we assume that they are already familiar with their disability to attend efficiently to auditive material, and when giving them a verbal instruction to listen carefully and memorize numbers (or stories and words), they might feel a rapid, negative frustration or frightening leading first to the motivation loss and then information loss and collapsing their auditive attentional and verbal memory performance.

It has been suggested that a larger left hippocampal volume is associated with improved verbal memory performances (Engvig et al., 2012), and a larger right hippocampal volume is associated with improved visual memory performances, e.g. expertise of driving taxes in metropolis (Maguire, Nannery & Spiers, 2006) but there are also studies showing that the participation and hippocampal volume of both hemispheres are important in visuospatial memory processes (Shavitt, Johnson & Batistuzzo, 2020). We noticed that depressed adolescents' verbal and visual memory functions were not deteriorated. In fact, they could recall verbal and visual material in some tests even better than other groups (also controls). In fact, we already noticed the depressed adolescents' superiority in verbal memory in our pilot study (Yrttimaa & Jehkonen, 2012). Halvorsen, Waterloo, Sundet, Eisemann and Wang (2011) also found that young adults having mild or moderate unipolar depression were not significantly affected by verbal memory impairments over the long-term course. Noteworthy, all depressed adolescents in our study were having a treatment (medical and/or psychosocial), which is extremely important because of findings that if untreated, depression might result lower hippocampal and anterior cingulate activation and weaker memory encoding in mid-life (Bremner, Vythilingam, Vermetten, Vaccarino & Charney, 2004). Interestingly, depression does not affect memory functions in young adults, even though the depressed adolescents are experiencing considerably attentional deficits (ASRS-A) with other symptoms. Their scores in ASRS part A were very similar to adolescents with ADHD and adolescents having both disorders.

Finally, we were interested in our examinees' visual orientation preference. It has been suggested, that children and adults with ADHD have poorer ability to attend to the stimuli on the left visual field than on the right visual field (for review, see Chan, et al., 2009). Corbetta, Miezin, Shulman and Petersen (1993) found evidence in their PET studies with normal healthy participants that visuospatial attention to the left visual field is mostly controlled by one region in right parietal cortex, while attention to the right visual field is controlled by both (left and right) parietal cortices. This leftward bias has been found in visual and tactile modalities, but instead, in the auditory modality a rightward bias is observed (Sosa, Teder-Sälejärvi & McCourt, 2010). In fact, it has been suggested that there might be right-hemisphere dominance also in processing socially important cues, like faces (Etcoff, 1984). Ricciardelli, Ro and

Driver (2002) found that there is a left visual field advantage for perception of gaze direction, which gives us valuable, social information of other individuals' attentional orientation.

In this study, we found that in an easy visuospatial learning task (ten concrete pictures, many repetitions), the examinees (the DEP group 100 %, the ADHD group 66.7 %, the BOTH group 75.0 %, and the CTRL group 58.3 %) attended more to the left visual side than to the right visual side. The LLT task might be considered very easy learning task to the adolescents of this age phase. There are 5 trials (4 repeats) of 10 visual objects in the grid, and an examinee sees them for 15 seconds per trial. Even if she or he remembers all ten pictures correctly, one more repetition and trial are given. The examinee will also be told to remember the correct visuospatial order of the pictures for delayed recall. Despite the fact, that there were no significant differences between an attentionally preferred visual side (left/right) between the groups, we found that the controls (16.7 %) learned significantly more often the same amount of visual features on the left and the right side compared to others (the DEP group 0.0 %, the ADHD group 11.1 %, the BOTH group 0.0 %). Notably, the adolescents having ADHD made much more errors in delayed recall (22.2. %) than controls (8.3 %). Interestingly, all these mistakes that the adolescents with ADHD made, were on their right visual side (22.2 %), whereas only 5.9 percent of the controls made mistakes on their right side.

On the contrary, in a more complex visual memory task, the examinees in all groups were orienting more to right visual side in immediate recall. Interestingly, in delayed recall, the adolescents with depression or both disorders memorized figures better on their right visual side (like in immediate recall) but the adolescents with ADHD memorized figures better on their left visual side (unlike in immediate recall). There were no differences in the controls' visual attentional preferences. Hence, we found the leftward attentional bias in easy visual task among our examinees but when the task demanded more complex and faster visual memorizing, the right visual side is winning the attentional shift. For some reason, the adolescents having ADHD lost the most part of their right-side learned and immediately memorized figures after a cognitive load and they memorized more left-side figures than in immediate recall. This might again reflect some kind of slowness; the adolescents having ADHD are not capable of having their best capacity in their use in rapid and new situations. They seemed to forget visual material they had already learned by watching the figures 10 seconds and drawing them from the model card. This might imply that they do not benefit or learn from their visuomotor pencil work compared to others. One possibility is, that the visual engrams (the right-side figures) are not long-lasting because of dysfunctions of their left frontal pole.

Despite small sample sizes, we could emphasize many important facets in this study. We had a small group (n = 4) of the young adults having depression but it came quickly apparent that they probably have ADHD (without hyperactivity) as unrecognized, undiagnosed and untreated as the primary diagnosis. They reported of experiences of being disregarded and underestimated when trying to tell their symptoms to teachers or other professionals in health care system. For some reason, they had not had access to neuropsychiatric assessment procedures although they were assessed psychologically in their childhood because of learning deficits.

Specifically, we found that the adolescents with ADHD or both disorders had more below average scores (and less above average scores) in their verbal and visual ability and memory functions compared to other groups. One concern arises: what might happen if a young adult with this kind of low

performance level (or with some deteriorated standard scores) is directed to a special health care to have a neuropsychiatric or neuropsychological assessment? In clinics, poor cognitive performance usually evokes serious questions of its etiology, and professionals may not identify this kind of weak cognitive performance as a feature of ADHD making wrong diagnoses instead. The other concern is that young adult performing poorly in the assessment situation might receive discouraging feedback from the examiner, even though she or he would benefit just from the opposite approach - positive and encouraging feedback.

Alarmingly, if ADHD (especially inattention without hyperactivity) is ignored and/or left untreated, it may lead to depression. In this study, we could demonstrate that young adults having depression and the most probably untreated ADHD do not perform cognitively or emotionally well compared to their same aged peers without these disorders. For that reason, we scientists and clinicians in neuropsychological field have to continue regenerating new and better screening tests and methods for regular use to comprehensive schools and even to nursery schools. We all have a professional responsibility to produce new knowledge but primarily, to educate specialists in health care by giving them adequate information of depression and attention deficit disorders in simple and clear manner. In this way, we can diminish the likelihood that disorders and problems accumulate in our children, adolescents and young adults. The questionnaires we use need not necessarily to be very extensive. In our study, the ASRS part A, the six-question screener revealed differences between the BOTH group and the CTRL group but only the ASRS part B, the 12-question screener could reveal differences between the ADHD group and the CTRL group, which is contradictory to former finding, in which only the part A is sufficient to reveal ADHD in adults (Kessler et al., 2005).

We point out that it is reasonable and necessary to utilize neuropsychological testing methods in multiple and inventive way in scientific (but also in clinical) assessment situations. In this way, it is possible to have more adequate information of our examinees. Because of our small sample sizes, our results are not generalizable to apply all adolescents having depression, attention deficit disorder or both of disorders. However, it was not our aim in the first place. If we start to focus on an individual in our neuropsychological clinic on the grounds of the mean values, we might certainly lose valuable information unique to our examinee. However, having more examinees in our study would have probably given us more powerful results. Intriguingly, in one-year follow-up (which is not our focus in this article), we found that all four adolescents having depression and ADHD could improve significantly and with many standard scores in Visual Puzzles task, which might be a sign of their slowness confronting new types of visual tasks. They could perform in the task much better when it became familiar to them. This might also reflect some kind of test anxiety emerging in the visuoconstructive task in the first assessment. In any case, this reveals a vulnerability of our neuropsychological assessment methods, which we should take into account among every examinee.

In conclusion, our results suggest that adolescents with ADHD and both disorders might be identified by their lowered verbal ability and verbal memory scores. In visual domain, identifying them by neuropsychological tests is more difficult. We also found that depression is not deteriorating cognitive performance of young adolescents measured with our existing neuropsychological methods. On the contrary, depressed adolescents might have verbal giftedness in their reasoning and memory abilities.Thus, the right hemisphere dysfunction model is not good candidate to explain the core deficits in young adults with depression and ADHD. Rather, we suggest

that these disorders might be approached with the left hemisphere (higher functioning in depression and lower functioning in ADHD) model.

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