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Not Always Hyperactive? Elevated Apathy Scores in Adolescents and Adults With ADHD

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Abstract

Objectives: To investigate the presence of apathy symptoms in adolescents and adults with ADHD as a behavioral manifestation of underlying motivational deficits and to determine whether apathy symptoms were associated with a specific neuropsychological profile. **Method:** A total of 38 ADHD participants (28 of the combined subtype [ADHD/C] and 10 of the inattentive subtype [ADHD/I]) and 30 healthy controls (Ctrl) were assessed on two measures of apathy administered to subjects and informants. As well, ADHD participants completed a comprehensive neuropsychological battery. **Results:** ADHD participants presented elevated scores on measures of apathy relative to controls (ADHD/I > ADHD/C > Ctrl). Informant-based ratings of apathy correlated significantly with behavioral measures of inattention. Apathy measures correlated significantly with executive tests, working memory, verbal fluency, and general intellectual abilities, only in the inattentive sample. **Conclusions:** This study stresses the relevance of motivational deficits in adult ADHD as a significant clinical dimension closely linked to inattention and executive difficulties.

Keywords

ADHD, adult, motivation, inattention, executive dysfunction

Introduction

ADHD is a complex neuropsychiatric disorder spreading from childhood to adulthood. It affects an estimated 5% of children (Polanczyk, de Lima, Horta, Biederman, & Rohde, 2007) and 2.5% of adults (Simon, Czobor, Balint, Meszaros, & Bitter, 2009). *Diagnostic and Statistical Manual of Mental Disorders* (*DSM-IV*; 4th ed., APA, 1994) classification defines it as a condition characterized by inattention, hyperactivity, and impulsiveness. Although *DSM-IV* focuses mainly on these three clusters of symptoms, other problematic areas have been described as a part of the clinical picture of ADHD, including executive dysfunction, emotional instability, or motivational deficits (Davidson, 2008; Wender, Wolf, & Wasserstein, 2001).

In particular, motivation has received less consideration in the clinical characterization of ADHD, especially in comparison to externalizing or disruptive behaviors corresponding to the hyperactive or impulsive cluster. Nevertheless, a strong link between motivation and cognitive performance has been reported in nonclinical populations. Motivational incentives are associated with better performance and greater cognitive control, as evidenced by improved behavioral outcomes and enhanced neural activity in brain areas linked to attention, executive functioning, and working memory (Gilbert & Fiez, 2004; Small et al., 2005; Szatkowska, Bogorodzki, Wolak, Marchewka, & Szeszkowski, 2008; Taylor et al., 2004). Particularly, motivation seems to be critical for the regulation of attentional effort in challenging circumstances that are typically problematic for patients with ADHD. This is often the case with the inability to "staying-on-task" during prolonged cognitive activity or regaining previous levels of performance after a detrimental event such as an external distractor (Sarter, Gehring, & Kozak, 2006). Motivational influences on cognition involve not only the reinforcing consequences of external reward on performance but also the effect of "intrinsic motivation," that is, the inherent satisfaction or interest obtained through the activity being carried out (Ryan & Deci, 2000). In

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addition, the effects of reward over cognitive outcome are mediated by individual differences in motivation style, such as sensitivity to reward, reward expectancy, and tolerance to reward delay (Berridge, 2004; Taylor et al., 2004). These individual differences, as well as intrinsic drives, are very important aspects of human motivation because goal-directed behavior frequently occurs in absence of immediate external reward. In this sense, motivation is a complex process that involves the internal generation of a purpose, the activation of behavior toward it, and the sustained allocation of effort during several steps before achieving the final outcome (Berridge, 2004; Ryan & Deci, 2000).

As it happens in nonclinical subjects, motivational factors have been linked to reduced attention in children with ADHD when tasks are longer, boring, or when they lack external supervision (Berlin, Bohlin, Nyberg, & Janols; 2003; Diamond, 2005; van der Meere, Shalev, Borger, & Gross-Tsur, 1995). As Brown (2000) suggested, *DSM-IV* criteria for ADHD include deficits in activation and sustained effort as symptoms of the inattention cluster. Also, a subgroup of patients with ADHD of the predominantly inattentive subtype has been characterized by symptoms of sluggishness, low energy, drowsiness, and daydreaming (Hartman, Willcutt, Rhee, & Pennington, 2004; Lahey, Schaughency, Hynd, Carlson, & Nieves, 1987; McBurnett, Pfiffner, & Frick, 2001). Although these observations apply mainly to childhood ADHD, adolescent and adult subjects with ADHD have been also described as showing notorious impairments in activation and goal-directed behavior, such as lack of motivation and procrastination (Adler & Cohen, 2004), impaired task and goal persistence (Dowson et al., 2004), boredom proneness (Kass, Wallace, & Vodanovich, 2003), hypersomnia (Oosterloo, Lammers, Overeem, de Noord, & Kooij, 2006), as well as "lethargic inattention" and high fatigue (Egeland, 2007).

Likewise, various theoretical accounts have stressed the importance of failures in regulating motivation and troubles to initiate or sustain activity in ADHD. For example, Douglas (1999) employed the expression "lack of intrinsic motivation" for characterizing a pervasive regulatory failure of participants with ADHD to allocate adequate attention or effort to meet tasks' demands. More specific motivation-related failures include problems with "sustained effort," "defective regulation of arousal-activation levels," and impaired "adaptation to changing demands." Also Barkley (1997) recognized difficulties in the self-regulation of motivation and effort as a feature of ADHD, derived from a deficiency in frontal behavioral inhibition. From a different theoretical stance, Sergeant (2000) emphasized that energetic mechanisms of effort, arousal, and activation are implicated in core ADHD deficits alongside cognitive factors. Finally, Diamond (2005) stated that decaying motivation—together with dysexecutive cognitive functioning—constitutes the core deficit of ADHD, predominantly inattentive type. As this author argues, when tasks become boring or too long, these patients get distracted the way patients with frontal lobe injury do.

Although motivation appears to be relevant in trying to understand ADHD deficits both in children and adults, no research has, till date, explicitly explored the presence of symptoms linked to lack of motivation, for example, apathy-like states, in this clinical population. Only one study with children who developed ADHD following stroke (Max et al., 2003) addressed apathy as a relevant dimension associated with this disorder. They found that inattention and apathetic behavior were related to each other and that, together, they constituted a factor that was the most consistent predictor of ADHD traits in the whole sample with and without stroke. The authors concluded that "the neural substrate of low motivation or apathy may play a central role in the pathophysiology of ADHD/traits" independently of more general cognitive impairment. In contrast with the lack of studies concerning motivation deficits in ADHD, the concept of apathy has gained increasing attention in recent years as a distinctive symptomatic dimension present in most neuropsychiatric disorders (Duffy, 2000; Starkstein & Leentjens, 2008; van Reekum, Stuss, & Ostrander, 2005). In his pioneering writings, Marin (1990, 1991) defined apathy as "lack of motivation not attributable to diminished level of consciousness, cognitive impairment, or emotional distress." According to this author, apathy could be conceptualized as a symptom, as a syndrome, or as a dimension of behavior that might vary continuously from one participant to another. The core motivational aspect of apathy is evidenced by overt deficits of goal-directed behaviors and cognitions as indicated by diminished productivity, lack of effort, lack of initiative, or perseverance; dependency on others to structure activity; and so on. On the contrary, there is no agreement about including emotional deficits as an aspect of apathy (Starkstein & Leentjens, 2008). Anatomically, apathy seems to emerge as a result of changes in the systems that generate and control voluntary actions, which involve different regions of the prefrontal cortex, mainly the anterior cingulate and dorsolateral prefrontal cortex, and structures within the basal ganglia, namely, caudate nuclei, the internal portion of the globus pallidus, and the medial-dorsal thalamus (Levy & Dubois, 2006). Remarkably, the neural areas associated with apathy have been found to be strongly implicated in ADHD (Seidman, Valera, & Bush, 2004). In addition, both ADHD and apathy symptoms improve with the use of stimulant drugs such as methylphenidate (Davidson, 2008; Padala, Burke, Bhatia, & Petty, 2007; Starkstein & Leentjens, 2008).

Besides its biological determinants, environmental and learning factors could also participate in the generation,

increase, or maintenance of apathetic behavior. Cognitive-behavioral approach to ADHD in adulthood provides a useful framework to understand the way in which negative life experiences may contribute to reduced motivation in people who suffer from this condition (Ramsay & Rostain, 2008; Safren, 2006; Young & Bramham, 2007). Failure and underachievement in different domains of functioning are common occurrences in participants with ADHD as a result of major neurobiological underpinnings in attention, executive function, and inhibitory control. Consequently, repeated experiences of frustration undermine self-esteem, leading to the formation of self-defeating negative beliefs, which in turn favor the expression of negative affect and the adoption of dysfunctional compensatory strategies, which can include procrastination or avoidance as means for coping with tasks' difficulties. More specifically, negative expectations about the future, failure anticipation, and reduced self-confidence would also affect motivation for action. In a sustained pessimistic context, the reduction of goal-directed behavior may appear as an extreme strategy for avoiding frustration from the very beginning of the cognitive conception of purposeful action. Then, this "learned dismotivation" would act as a self-fulfilling prophecy mutually reinforcing negative beliefs, apathetic states, and poor outcomes.

Accordingly, the first aim of this study was to show the relevance of motivation symptoms in the clinical characterization of adult ADHD—especially, though not exclusively, of the inattentive type. One would expect that the said motivational disturbances should be behaviorally manifested through elevated scores in apathy scales. For the purpose of this study, apathy was conceptualized as a behavioral dimension expressing pervasive and sustained motivation failures ranging from total lack of intrinsic motivation to varied degrees of reduced motivation, including troubles for taking initiatives, acting without immediate reward, or completing tasks without external support. In this sense, apathy will be evident when participants fail, whether totally or partially, to initiate or sustain effortful autonomous goal-directed behavior. Consequently, we formulated in advance the following specific hypotheses:

Hypothesis 1: Adult ADHD patients as a whole will show higher scores of apathy in comparison to healthy control participants as measured by self-report and informant-based scales

Hypothesis 2: Apathy scores in ADHD will correlate with inattention symptoms scores in behavioral measures but not with hyperactive-impulsive symptoms scores.

Hypothesis 3: Apathy scores will be strongly associated with ADHD predominantly inattentive subtype, but ADHD combined subtype will show also elevated levels of apathy compared to controls.

A second aim of the present study was to determine whether apathy symptoms were associated with a specific neuropsychological profile. According to the previous literature on neuropsychological correlates of apathy (van Reekum, Stuss & Ostrander, 2005) we expected to find negative correlations between apathy scores and (a) measures of frontal functioning (executive function and working memory), and (b) measures of fluency and processing speed. In addition, taking into account the hypothetical double pathway mentioned previously—biological and environmental—which could influence the emergence of apathetic behaviors, we hypothesized that neurocognitive measures would be a useful method to distinguish the two types of influences. Therefore, the presence of neurocognitive correlates of apathy would be interpreted as the expression of constitutive intrinsic motivation failures. Instead, increased apathy scores in absence of neurocognitive correlates would be interpreted as the effect of "learned dismotivation."

Method

Participants

ADHD patients. Participants were recruited from the Clinic of Adult ADHD at the Institute of Cognitive Neurology (INECO, Buenos Aires, Argentina). All participants were required to give their informed consent during the initial interview. A total of 38 patients fulfilled *DSM-IV* criteria for ADHD. This group included 28 patients with ADHD combined type (ADHD/C) and 10 patients with predominantly inattentive type (ADHD/I). ADHD diagnosis based on the *DSM-IV* criteria was made by two experts (AL and FM) following the assessment protocol for adults suggested by Murphy and Gordon (2006). The protocol comprises:

1. Patient and informant versions of the ADHD Rating Scale for Adults (Barkley, Murphy & Bauermeister, 1998) that itemize current symptoms and retrospective childhood symptoms corresponding to *DSM-IV* characterization of ADHD (see the following sections for further description).
2. A comprehensive clinician-guided interview to the patient based on Barkley et al. (1998) that surveys past and present ADHD symptoms, adaptive functioning, social adjustment, developmental and medical history, school and work history, psychiatric history and prior treatments, and family history of ADHD or any psychiatric or medical condition.
3. An interview with relatives or significant others (usually parent or spouse), during which they

completed the informant-based versions of questionnaires and complemented background data.
4. Neuropsychological assessment involving a wide array of tests measuring attention, memory, executive functioning, language, and general intellectual abilities (detailed in the Materials and Procedures section).

Patients were included in the study only if there was an agreement in diagnosis between the two independent raters after examination of the complete assessment protocol. All patients were examined to rule out any confounding comorbid psychiatric or neurological condition that can potentially cause apathy, including but not limited to traumatic brain injury, dementia, schizophrenia, or bipolar disorder.

Healthy comparison participants. Healthy controls (CTR, $n = 30$) were recruited from a larger pool of volunteers who were screened to reject current or previous neuropsychiatric disorders. All participants gave their informed consent earlier to the inclusion in the study. The study was approved by the ethics committee at the Institute of Cognitive Neurology.

Materials and Procedures

Questionnaires. To maximize the reliability of apathy measurements, two different instruments and two different raters were employed:

1. Apathy Scale (Starkstein et al., 1992). The Apathy Scale is an abbreviated version of Marin's Apathy Scale comprising 14 items that can be rated by either patients or informants. Each item allows four possible answers "not at all," "slightly", "some", or "a lot," and total score ranges from 0 to 42. Higher scores indicate more pronounced apathy. Both patient self-report (AS-SR) and informant versions of the Apathy Scale (AS-IB) were administered. Originally developed in Argentina, the Apathy Scale showed good interrater reliability ($r = .81$) and test–retest reliability ($r = .90$), as well as an adequate internal consistency ($\alpha = .76$).
2. Frontal Systems Behavior Scale (including apathy subscale; Grace & Malloy, 2001). The FrSBe is a 46-item rating scale designed to measure behaviors associated with disturbed or damaged frontal systems of the brain. The FrSBe yields a total scale score and three scores for subscales measuring apathy, disinhibition, and executive dysfunction. It consists of two rating forms: a self-rating form to be completed by the patient, and a family rating form to be completed by an informant who has regular contact with the patient, such as a spouse, parent, or significant other. For the purpose of this study, both the self-report version of the apathy subscale of the FrSBe (FrSBe-A-SR) and the informant-based version of the same subscale (FrSBe-A-IB) were included. The FrSBe showed high internal consistency ($\alpha = .92$, .78, .80, and .87 for the total, apathy, disinhibition, and executive scores of the family form, respectively, and .88, .72, .75, and .79 for the self-report form in a normative sample; Grace & Malloy, 2001) and demonstrated to be a valid instrument to differentiate various frontal syndromes. The Spanish version of the FrSBe—apathy subscale adapted for this study manifested also adequate internal consistency in the whole sample of participants included in this article ($\alpha = .86$ for the FrSBE-A-SR and $\alpha = .79$ for the FrSBE-A-IB) and a very good concurrent validity with the Apathy Scale (see Table 2).

Additional measures administered to participants comprised ratings of ADHD behavioral symptoms and depression estimates:

1. ADHD Rating Scale for Adults (Barkley & Murphy, 1998). The ADHD Rating Scale for Adults is a self-report questionnaire that contains the 18 items based on the diagnostic criteria for ADHD in the *DSM-IV*. Respondents rate the intensity of ADHD symptoms on a 4-point Likert-type scale, ranging from 0 (*never or rarely*) to 3 (*very often*). Participants completed two versions of this scale, one targeting current symptoms and the other for childhood symptoms between ages 5 to 12 years. For the two clinical groups, these same rating scales were obtained from parents or spouses, but only participants' self-report ratings of current symptoms were taken into account for this study. Thus, for each participant, the scale provides three different scores: the sum of ADHD item ratings on the total scale (ADHD-RS-T), the sum of inattention items (ADHD-RS-I), and the sum of hyperactive–impulsive items (ADHD-RS-HI). In the whole sample of participants included in the present study, the ADHD-RS showed a good internal consistency for the total scale, $\alpha = .88$; inattention subscale, $\alpha = .80$; hyperactivity-impulsivity subscale, $\alpha = .72$. In addition, the pattern of results obtained with this instrument was mostly congruent with the expected differences between subgroups (See Demographic and Clinical Findings in the Results section).

Table 1. Demographic and Clinical Findings

	ADHD/I[a]		ADHD/C[b]		Healthy Controls[c]			
	M	*SD*	*M*	*SD*	*M*	*SD*	*F*	*p*
Age	25.20	9.57	34.32	16.65	33.03	13.08	1.55	0.219
Gender *(male/female)*	8/2		18/10		13/17			
Apathy measures								
AS-SR	14.70	6.11	14.57	5.88	7.50	5.18	13.43	<.001
AS-IB	20.90	7.37	14.64	8.22	6.47	5.20	19.98	<.001
FrSBE-A-SR	34.60	11.52	33.54	8.68	23.73	6.12	12.89	<.001
FrSBE-A-IB	34.00	7.62	29.71	8.61	22.03	8.23	10.40	<.001
Additional measures								
ADHD-RS inattention	10.50	3.89	13.21	5.45	2.97	2.94	43.16	<.001
ADHD-RS hyperactivity/impulsivity	6.60	3.69	12.61	5.25	2.87	2.49	42.92	<.001
BDI II	8.60	11.57	16.18	9.38	4.63	3.32	16.26	<.001

Note: AS-SR= Apathy scale—Self report; AS-IB= Apathy scale—Informant based; FrSBE-A-SR= Frontal System Behavior Scale—Apathy subscale—Self report; FrSBE-A-IB= Frontal System Behavior Scale—Apathy subscale—Informant based; BDI II= Beck Depression Inventory II.
a. $N = 10$.
b. $N = 28$.
c. $N = 30$.

2. Beck Depression Inventory-II (Beck, Steer, & Brown, 1996). The Beck Depression Inventory–II (BDI-II) is a widely used 21-item self-report instrument developed to measure severity of depression symptoms. It has been successfully adapted to the local population of this study (Brenlla & Rodríguez, 2006) and showed satisfactory psychometric properties ($\alpha = .88$ for the clinical sample and .86 for the normative sample, $r = .86$ for test–retest reliability, adequate concurrent validity with other depression measures and good diagnostic discriminatory power between clinical and nonclinical population).

Neuropsychological battery. ADHD participants completed a general neuropsychological battery assessing (a) current *general intellectual abilities* employing the WAIS-III vocabulary subtest and the WAIS-III matrix reasoning test (Wechsler, 1997), (b) *attention* with the forward digits span task of the Wechsler Memory Scale—Revised (WMS-R; Wechsler, 1987) and the trail making test Part A (Partington & Leiter, 1949), (c) *verbal memory* through the Rey Auditory Verbal Learning Test (RAVLT; Rey, 1941) and the logical memory subtest of the WMS-R (Wechsler, 1987) and *nonverbal memory* with the Rey Complex Figure Test (Rey, 1941), (d) *verbal fluency* with the phonological fluency task (Benton, Hamsher, & Sivan, 1983) and the semantic fluency task (Lezak, 1995), (e) *executive function* using the backward version of the Digit Span Test (Wechsler, 1997), Part B of the trail making test (Partington & Leiter, 1949), the WAIS-III letter–number sequencing subtest (Wechsler, 1997), the modified version of the Wisconsin Card Sorting Test (WCST; Nelson, 1976), and the total score of the INECO Frontal Screening (IFS; Torralva, Roca, Gleichgerrcht, López, & Manes, 2009). The IFS constitutes a new brief instrument designed to assess executive functioning through the following seven areas: motor programming, conflicting instructions, inhibitory control, numerical working memory, verbal working memory, spatial working memory, conceptualization, and verbal inhibitory control. Also, the Memory Index from the WAIS-III was obtained combining digit span, arithmetic, and letter–number sequencing subtests (Wechsler, 1997). Lastly, the Processing Speed Index from the WAIS-III was obtained from the digit symbol-coding and symbol search subtests (Wechsler, 1997).

Procedures

ADHD patients were evaluated during admission interviews to the specialized clinic of adult ADHD at INECO. All participants (patients and controls) went through a standard assessment process, including neurological, neuropsychiatric, and neuropsychological examinations, supported by routine blood exams, EEG, and MRI. All participants were assessed before initiating treatment with specific drugs for ADHD. Nonetheless, three of them were already in ongoing treatment with psychopharmacological drugs at the moment of the assessment (two of them with a mood stabilizer and the other with an antidepressant). The healthy comparison participants were recruited by word of mouth and collaborated voluntarily. Questionnaires were

completed at home with the help of a research assistant, and relatives or significant others also provided apathy ratings about control participants. Control participants included in the present study were not assessed with neuropsychological tests.

Statistical Analysis

Comparisons between groups were made using one-way ANOVA followed by Tukey's HSD post hoc comparisons when appropriate. In addition, ANCOVA was applied when needed to control for the possible confounding effect of depression over apathy ratings. Correlations between measures were carried out by employing the Spearman correlation coefficient. Finally, when analyzing categorical variables, the Pearson chi-square test was employed.

Results

Demographic and Clinical Findings

Table 1 summarizes general demographic and clinical findings for all three groups (ADHD/C, ADHD/I and control participants). The ADHD/C sample consisted of 28 participants (10 women, 18 men), with a mean age of 34.2 ± 10.5 years. The ADHD/I sample consisted of 10 participants (2 women, 8 men), with a mean age of 25.2 ± 9.57 years. The control sample consisted of 30 participants (17 women, 13 men), with a mean age of 33.03 ± 13.08 years. No statistical differences were found between groups regarding age ($F[2, 65] = 1.552$, $p = .219$) or gender ($\chi^2 = 5.058$, $df = 2$, $p = .08$).

As expected, clinical participants showed significantly higher scores in behavioral measures of ADHD symptoms than control participants. There was an expected significant between-group difference in ADHD-RS-inattention subscale ($F[2, 65] = 43.161$, $p < .001$). Post hoc tests showed that both clinical groups (ADHD/I and ADHD/C) had significantly higher scores in inattention than control participants ($p < .001$ in both groups). Besides, there was an expected significant between-group difference in ADHD-RS-hyperactivity-impulsivity subscale ($F[2, 65] = 42.920$, $p < .001$). Post hoc tests showed that ADHD/C group had significantly higher scores in hyperactivity-impulsivity than control group and ADHD/I group ($p < .001$ in both groups). Unexpectedly, ADHD/I scored significantly higher in hyperactivity-impulsivity than control participants ($p = .035$).

Finally, there was an expected significant between-group difference in BDI-II ($F[2, 65] = 16.264$, $p < .001$). Post hoc tests revealed that ADHD/C group had significantly higher scores in depression than control group ($p < .001$) and ADHD/I group ($p = .027$). In contrast, ADHD/I group and control group did not differ significantly on BDI-II scores.

Table 2. Correlations Between Different Scores of Apathy in ADHD Participants[a]

	AS-SR	AS-IB	Frsbe-A-SR	Frsbe-A-IB
AS-SR	1			
AS-IB	0.351*	1		
Frsbe-A-SR	0.770**	0.423**	1	
Frsbe-A-IB	0.555**	0.727**	0.563**	1

Note: AS-SR= Apathy scale—Self report; AS-IB= Apathy scale—Informant based; FrSBE-A-SR= Frontal System Behavior Scale—Apathy subscale—Self report; FrSBE-A-IB= Frontal System Behavior Scale—Apathy subscale—Informant based.
a. $N = 38$.
*$p < .05$. **$p < .001$.

Consistency of Apathy Ratings Between Different Instruments and Raters

Correlations between different scores of apathy were performed to explore the consistency of measures obtained. All four ratings of apathy were significantly correlated with each other (see Table 2). Coherently, scores obtained from the same rater (patient or informant) were strongly correlated (FrSBe-A-IB–AS-IB: $r_s = .727$, $p < .001$; FrSBe-A-SR–AS-SR: $r_s = .770$, $p < .001$).

Differences in Apathy Between ADHD Participants and Control Participants (Hypothesis 1)

Scores differed significantly between the groups in the four measures of apathy applied: FrSBe-A-IB ($F[2, 65] = 10.399$, $p < .001$), FrSBe-A-SR ($F[2, 65] = 12.892$, $p < .001$), AS-IB ($F[2, 65] = 19.978$, $p < .001$), and AS-SR ($F[2, 65] = 13.430$, $p < .001$). Post hoc tests revealed that ADHD/I group had significantly higher scores in the four apathy measures than the control group (FrSBe-A-IB, $p < .001$; FrSBe-A-SR, $p < .001$; AS-IB, $p < .001$; and AS-SR, $p < .002$). Also, ADHD/C group presented higher scores than controls (FrSBe-A-IB, $p < .002$; FrSBe-A-SR, $p < .001$; AS-IB, $p < .001$; and AS-SR, $p < .001$). As expected in Hypothesis 1, both ADHD groups scored higher in apathy measures than control group.

Because ADHD/C participants had significantly higher scores in depression than control participants, we wanted to rule out the possibility that the elevated scores in apathy measures in the former group were due to the confounding effects of depression. For this purpose, an ANCOVA comparison was carried out between ADHD/C participants and control participants with total BDI-II score as a covariate. The analysis revealed that differences in apathy scores remained significant for all measures: FrSBe-A-IB

(F[1, 56] = 11.011, $p < .001$), FrSBe-A-SR (F[1, 56] = 32.813, $p < .001$), AS-IB (F[1, 56] = 14.289, $p < .005$), and AS-SR (F[1, 56] = 18.216, $p < .001$).

Correlates of Apathy in ADHD Participants (Hypothesis 2)

The two self-report ratings of apathy were significantly correlated with BDI-II ($r_s = .446$, $p = .005$ for AS-SR, and $r_s = .594$, $p < .001$ for FrSBe-A-SR). On the contrary, neither the AS-IB ($r_s = .147$, $p = .378$) nor the FrSBe-A-IB ($r_s = .307$, $p = .061$) were significantly correlated with BDI-II. Therefore, informant-based ratings seem to dissociate apathy from depression better than self-report ratings.

Regarding ADHD symptoms, FrSBe-A-IB significantly correlated with ADHD-RS-Inattention ($r_s = .322$, $p = .049$) but not with ADHD-RS-hyperactivity-impulsivity (*rho* = .151, $p = .367$), which is in congruence with Hypothesis 2. AS-IB did not show significant correlations with either ADHD-RS-I ($r_s = .276$, $p = .093$) or ADHD-RS-H/I ($r_s = –.039$ $p = .815$). The two self-report ratings of apathy presented a nonspecific pattern of significant positive correlations with both inattention and hyperactivity-impulsivity (AS-SR–ADHD-RS-I: $r_s = .348$, $p = .032$; AS-SR–ADHD-RS-H/I: $r_s = .379$, $p = .019$; FrSBe-A-SR–ADHD-RS-I: $r_s = .565$, $p < .001$; FrSBe-A-SR–ADHD-RS-H/I: $r_s = .576$, $p < .001$).

Differences in Apathy Between ADHD Subtypes (Hypothesis 3)

Post hoc tests from ANOVA revealed that ADHD/I group had significantly higher apathy scores than ADHD/C in the AS-IB ($p = .043$) but not on other measures of apathy. Because the ADHD/C group presented scores of depression significantly higher than the ADHD/I group, we wanted to exclude the possibility that the differences in apathy between groups—if they existed—were being obscured by depression. Hence, an ANCOVA with BDI-II as a covariate was applied, and then the four measures of apathy showed significant differences between ADHD/I and ADHD/C—the former having the higher scores: FrSBe-A-IB (F[1, 36] = 5.782, $p = .007$), FrSBe-A-SR (F[1, 36] = 19.019, $p < .001$), AS-IB (F[1, 36] = 4.224, $p = .023$), and AS-SR (F[1, 36] = 5.939, $p = .006$).

Neuropsychological Correlates of Apathy

Correlations performed between apathy measures and neuropsychological tasks in the ADHD/I sample revealed significant relationships with the executive domain, working memory, and verbal fluency. In the executive area, letter–number sequencing was negatively correlated with the FrSBE-A-IB ($r_s = –.709$, $p = .032$), backward digits span test was negatively correlated with the FrSBE-A-SR ($r_s = –.638$, $p = .047$), and the total score of the IFS was negatively correlated with both FrSBE-A-SR ($r_s = –.637$, $p = .048$) and AS-SR ($r_s = –.669$, $p = .035$). Regarding verbal fluency, the semantic fluency task was negatively correlated with both FrSBE-A-IB ($r_s = –.699$, $p = .024$) and AS-IB ($r_s = –.659$, $p = .038$). Lastly, the Working Memory Index was negatively correlated with both FrSBE-A-IB ($r_s = –.770$, $p = .015$) and AS-SR ($r_s = –.669$, $p = .049$). No significant relationships were found between apathy and processing speed, attention, or verbal memory measures. Interestingly, significant relationships emerged between apathy and current general intellectual abilities in the ADHD/I sample: Both matrix reasoning and vocabulary subtests of the WAIS-III were negatively correlated with the FrSBE-A-IB ($r_s = –.778$, $p = .023$, and $r_s = –.857$, $p = .014$, respectively). Notably, no significant correlations were found between neuropsychological tests and apathy scores in the ADHD/C sample.

Discussion

The present investigation is the first to explore apathy in adolescents and adults with ADHD. Our study showed that both ADHD predominantly inattentive and combined subtypes presented elevated scores in apathy measures relative to healthy control participants. This main effect was highly consistent across the different instruments employed in this study, which in turn have been found to highly correlate with each other. We interpret this main result as a clear index of the presence of motivational disturbances as a part of the psychopathological features of adult ADHD. The present findings also provide empirical support for the association between apathetic behavior and ADHD traits reported in a previous study in children (Max et al., 2003) and reinforce theoretical models that place the focus on motivation, activation, and energetic factors for its explanation.

A second major finding of this study is in relation with the clear link found between inattention and symptoms of low motivation. Informant-based ratings of apathy have been found to correlate with behavioral measures of inattention and discriminated between subtypes of ADHD, showing higher scores associated with ADHD predominantly inattentive type. Also, when depression was statistically controlled, ADHD/I participants manifested significantly higher scores in apathy than ADHD/C participants in the four apathy measures applied in the study. In the neuropsychological assessment, even if attention tests included in our battery were not sensitive enough to detect associations with apathy, a pattern of correlations with apathy measures specific to the ADHD/I sample emerged in the

executive domain and working memory, as well as in verbal fluency.

These results are congruent within a framework that integrates motivation, attention, and executive function (Pessoa, 2009). Executive function coordinates and integrates a series of tasks extremely relevant for the fulfilling of motivated behavior such as response preparation and organization, implementation of activation-inhibition sequences, and balancing short and long-term goals in planning. Concomitantly, motivation has been shown to modulate executive-control and working-memory regions in the brain enhancing task performance (Locke & Braver, 2008; Pochon et al., 2002; Taylor et al., 2004). In turn, it is known that attention processes are tightly connected to the executive domain to allocate cognitive resources to the relevant tasks. The prefrontal cortex and the frontostriatal interconnections are vital for these processes, as their damage or dysfunction degrades executive control and, thus, impairing judgment, decision making and planning (Fuster, 1999; Goldman-Rakic, 1998; Manes et al, 2002; Stuss & Benson, 1986). The structures and pathways that support executive function continue to develop through early adulthood and are dependent on the development of dopaminergic and norepinephrinergic pathways. The importance of prefrontal area and striatal projections in the pathophysiology of ADHD is now well established (Arnsten, 2006; Seidman et al, 2004).

Interestingly, in our study apathy appears to be associated also with a negative intellectual performance in the group of purely inattentive patients and was not correlated with processing speed. Hence, it is possible to think that the difficulties associated with apathy would become manifest in cognitive domains that require effort, for example, initiation and planning of action, but not necessarily in the performance of tasks that are not complex or prolonged enough.

Another corollary of the link between apathy and inattention concerns the distinction between ADHD subtypes. At first glance, apathy may reflect an essential difference between ADHD/I versus ADHD/C (Diamond, 2005). This idea has partial support from this study because ADHD/I showed higher apathy scores than ADHD/C. Furthermore, the fact that neuropsychological tests correlated with apathy in the ADHD/I sample, but not in ADHD/C sample, constitutes a strong argument in favor of this idea. However, the latter group also exhibited elevated scores in apathy measures. For this reason, we cannot conclude that low motivation is exclusive of ADHD/I, at least from a phenomenological standpoint. All the same, we can hypothesize that low motivation is a very important component of the predominantly inattentive subtype and probably has more incidence in the cognitive and behavioral performance of this subgroup than in the combined subtype. Moreover, according to the interpretation of neurocognitive correlates of apathy formulated in our original hypothesis, it seems likely that low intrinsic motivation would be included in the constellation of core constitutive deficits of ADHD/I. In contrast, difficulties in motivation may appear in the ADHD/C population as an implicit, acquired consequence of sustained negative environmental reinforcement in a process that we called learned dismotivation.

Lastly, in this study, informant-based measures of apathy seemed to be more specific for isolating apathy symptoms from depression than the self-report scales. Whereas self-report forms correlated significantly with depression scores, the informant-based scales did not. An important question emerges here about whether the significant correlations between self-report measures of apathy and depression were due to the fact that both scales were completed by the same observer or to an actual difficulty for the implicated participants to differentiate between the two kinds of symptoms. If so, lack of motivation could be hard to recognize and the participants may be blind about it in a sort of "anosognosy" of their own passivity. One way or the other, the availability of an external observer would provide additional reassurance about the reliability of the distinction between apathy and depression, as revealed by the results of the present study.

Overall, this work has theoretical and practical implications. From a conceptual stance, both previous reports and present findings remarked the usefulness of the model of frontal dysfunction in explaining the variety of ADHD disturbances (Boucugnani & Jones, 1989; Clark et al., 2007; Shue & Douglas, 1992). Prefrontal syndromes resulting from lesions or neurodegenerative diseases become apparent through a variety of symptomatic dimensions such as impulsive-disinhibited behaviors, inattention, affective instability, apathy, and dysexecutive functioning. Dysfunction of the frontostriatal circuit or structures (i.e., basal ganglia, thalamus, and frontal lobes) may cause similar abnormalities in planned, motivated behavior (Cummings, 1993). In an attenuated manner, the different subtypes of ADHD replicate the global scenario of frontal disturbances. In fact, apathy is one of the three main frontal syndromes that occurs in conjunction with dysexecutive and disinhibited syndromes (Cummings & Miller, 2006). The latter two syndromes fit well in the current conceptualization of ADHD. Importantly, however, the results of the present study strongly suggest that apathy symptoms could also be part of the complex constellation that is ADHD. From this view, it seems probable that the miscellaneous clinical panorama of ADHD subtypes could be reflecting the preponderance of one prefrontal dimension over another in one specific subtype, or even in each particular participants. As a general conceptual framework, we could assume that the diverse ADHD symptomatic dimensions are in fact the manifestations of an attenuated or "soft" prefrontal-like functional syndrome expressing along a "continuum" of impulsive-hyperactive behaviors at one extreme and

apathetic-flattened response at the other. Impulsive behaviors could be attributed to an orbtitofrontal dysfunction, and apathetic behaviors would reflect the compromise of medial frontal areas, which have been previously associated with ADHD (Seidman et al., 2004). In turn, dorsolateral involvement would be related with the executive difficulties (Cummings & Miller, 2006) and could contribute to the production of apathy (Levy & Dubois, 2006).

From a practical point of view, this study has important implications in terms of assessment and treatment of ADHD. First, apathetic behavior should be taken into account when assessing adult patients with ADHD and discriminated carefully from depression. This is an important issue because we speculate that most times, apathetic states are obscured by depression and misattributed to it. Hence, it would be necessary to incorporate specific instruments to evaluate motivation symptoms and to consider the observations of parents, spouses, or other close informants. Second, psychological treatments for ADHD should reinforce the use of motivational techniques and adapt them to the specificities of each subtype. If participants with ADHD/I express troubles related with intrinsic motivation, they should be helped to explore new activities, to expand their range of interests, to take initiatives, to generate their own goals, to structure activities by themselves, and to obtain a sense of gratification when doing things. In the case of participants with ADHD/C, current interventions based on the cognitive-behavioral model could be potentiated systematically by introducing motivational interviewing techniques and adapting them to ADHD (Young & Bramham, 2007). Motivational interviewing is a directive method for facilitating behavior change by exploring and resolving ambivalence and conflicting motivations (Miller & Rollnick, 2002). This approach could be of benefit for individuals with ADHD/C for neutralizing the discouraging effects of negative learning and for supporting self-efficacy. Furthermore, cognitive restructuring techniques (Ramsay & Rostain, 2008; Safren, Perlman, Sprich, & Otto, 2005; Young & Bramham, 2007) could be useful to address the specific dysfunctional cognitions concerning anticipation of failure and low self-confidence, both of which promote disengagement and reduction of activities as radical compensatory strategies to avoid frustration.

The main limitation of our study concerned the small sample used, especially in the case of ADHD/I group. Nevertheless, our study represents the first attempt to address the issue of clinical presentation of motivational symptoms in ADHD, and the significant differences and relationships obtained were statistically robust. Further studies with larger samples should be carried to confirm these exploratory results. Also, two additional limitations of this study should be mentioned here. First, the design lacks a predominantly hyperactive-impulsive ADHD sample. The low rate of this subtype in our clinic accounted for this absence. Hyperactive-impulsive subtype has been shown to be an early childhood presentation of ADHD very unstable over time, taking into account that most of predominantly hyperactive-impulsive children turn into the combined subtype by elementary school age (Lahey et al., 1994; Lahey, Pelham, Loney, Lee, & Willcutt 2005). Likewise, symptoms of hyperactivity tend to lessen with time in ADHD participants during adolescence (Biederman, Mick, & Faraone, 2000). Probably due to these changes in the developmental pattern of ADHD, the predominantly hyperactive-impulsive subtype eventually showed a low rate of presentation in clinically referred samples of adults similar to the samples used in our study (Michelson et al., 2003; Millstein, Wilens, Biederman, & Spencer, 1997; Murphy, Barkley, & Bush, 2002). The second criticism concerns the lack of neuropsychological data from healthy participants. This decision responded to operative restrictions, considering that the main objective of the study was to show the presence of apathy symptoms in ADHD participants and that neuropsychological data were employed only as correlates of apathy. Alongside these observations, some interesting questions fall beyond the scope of the present study, requiring an extension as well as some methodological modifications in future research. For example, are observed apathy symptoms in ADHD as profound as those seen in other neuropsychiatric syndromes? Moreover, are apathy states transitory or stable along time? Are they modifiable by therapeutic interventions? To answer these questions, we would need specific comparative studies—particularly with other syndromes involving prefrontal dysfunctions—along with longitudinal studies.

In sum, current clinical description of ADHD as outlined in *DSM-IV* appears to be too narrow to reflect the complexity of the syndrome when it evolves toward adolescence and adulthood. Different symptomatic clusters—other than inattention, hyperactivity, and impulsivity—deserve more detailed consideration. This study showed that troubles in motivation, as revealed by elevated apathy scores, constitute a relevant behavioral dimension both for patients and close relatives. This dimension seems to be associated with inattention symptoms but not restricted to the purely inattentive subtype. Furthermore, the results of this work are congruent with a model of a wide frontal-system involvement in ADHD, including medial frontal areas typically linked with apathy.

Declaration of Conflicting Interests

The authors declared that they had no conflicts of interests with respect to their authorship or the publication of this article.

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References

Adler, L., & Cohen, J. (2004). Diagnosis and evaluation of adults with attention-deficit/hyperactivity disorder. *Psychiatric Clinics of North America, 27*, 187-201.

APA. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

Arnsten, A. F. (2006) Fundamentals of attention-deficit/hyperactivity disorder: Circuits and pathways. *Journal of Clinical Psychiatry, 67*(Suppl. 8), 7-12.

Barkley, R. A. (1997). Behavioral inhibition, sustained attention, and executive functions: Constructing a unifying theory of ADHD. *Psychological Bulletin, 121*, 65-94.

Barkley R. A., & Murphy K. R. (1998). *Attention-deficit hyperactivity disorder: A clinical workbook* (2nd ed.). New York: Guilford.

Barkley, R. A., Murphy, K., & Bauermeister, J. J. (1998). *Trastorno por déficit de atención con hiperactividad: Un manual de trabajo clínico* [Attention-deficit hyperactivity disorder: A clinical workbook]. New York: Guilford.

Beck, A. T., Steer, R. A., & Brown, G. K. (1996). *Manual for the Beck Depression Inventory-II*. San Antonio, TX: Psychological Corporation.

Benton, A. L., Hamsher, K., & Sivan, A. B. (1983). *Multilingual Aphasia Examination* (3rd ed.). IA: AJA.

Berlin, L., Bohlin, G., Nyberg, L., & Janols, L. O. (2003). Sustained performance and regulation of effort in clinical and non-clinical hyperactive children. *Child Care and Health Development, 29*, 257-267.

Berridge, K. C. (2004). Motivation concepts in behavioral neuroscience. *Physiology and Behavior, 81*, 179-209.

Biederman, J., Mick, E., & Faraone S. V. (2000). Age dependent decline of ADHD symptoms revisited: Impact of remission definition and symptom subtype. *American Journal of Psychiatry, 157*, 816-818.

Boucugnani, L. L., & Jones, R. W. (1989). Behaviors analogous to frontal lobe dysfunction in children with attention deficit hyperactivity disorder. *Archives of Clinical Neuropsychology, 4*, 161-173.

Brenlla, M. E. & Rodríguez, C. M. (2006). Adaptación argentina del Inventario de Depresión de Beck (BDI-II). In A. T. Beck, R. A. Steer, & G. K. Brown(Eds.), *BDI-II. Inventario de Depresión de Beck* (2nd ed., pp. 11-37). Buenos Aires: Paidós.

Brown, T. E. (2000). Emerging understandings of attention-deficit disorders and comorbidities. In T. E. Brown (Ed.), *Attention-deficit disorders and comorbidities in children, adolescents, and adults* (pp. 3-55). Washington, DC: American Psychiatric Press.

Clark, L., Blackwell, A. D., Aron, A. R., Turner, D. C., Dowson, J., Robbins, T. W., et al. (2007). Association between response inhibition and working memory in adult ADHD: A link to right frontal cortex pathology. *Biological Psychiatry, 61*, 1395-1401.

Cummings, J. L. (1993). Frontal-subcortical circuits and human behavior. *Archives of Neurology, 50*, 873-880.

Cummings, J. L., & Miller, B. L. (2006). Conceptual and clinical aspects of the frontal lobes. In J. L. Cummings & B. L. Miller (Eds.), *The human frontal lobes: Functions and disorders* (2nd ed., pp. 12-21). New York: Guilford.

Davidson, M. A. (2008). ADHD in adults: A review of the literature. *Journal of Attention Disorders, 11*, 628-641.

Diamond, A. (2005). Attention-deficit disorder (attention-deficit/hyperactivity disorder without hyperactivity): A neurobiologically and behaviorally distinct disorder from attention-deficit/hyperactivity disorder (with hyperactivity). *Developmental Psychopathology, 17*, 807-825.

Douglas, V. I. (1999). Cognitive control processes in attention-deficit hyperactivity disorder. In H. C. Quay & A. E. Hogen (Eds.), *Handbook of disruptive behavior disorders* (pp.105-138). New York: Kluwer Academic/Plenum Publishers.

Dowson, J. H., McLean, A., Bazanis, E., Toone, B., Young, S., Robbins, T. W., et al. (2004). The specificity of clinical characteristics in adults with attention-deficit/hyperactivity disorder: a comparison with patients with borderline personality disorder. *European Psychiatry, 19*, 72-78.

Duffy, J. (2000). Apathy in neurologic disorders. *Current Psychiatry Reports, 2*, 434-439.

Egeland, J. (2007). Differentiating attention deficit in adult ADHD and schizophrenia. *Archives of Clinical Neuropsychology, 22*, 763-771.

Fuster, J. M. (1999). Synopsis of function and dysfunction of the frontal lobe. *Acta Psychiatrica Scandinavica, 99*(Suppl. 395), 51-57.

Gilbert, A. M. & Fiez, J. A. (2004). Integrating rewards and cognition in the frontal cortex. *Cognitive, Affective, & Behavioral Neuroscience, 4*, 540-552.

Goldman-Rakic, P. S. (1998). The prefrontal landscape: Implications of functional architecture for understanding human mentation and the central executive. In A. C. Roberts, T. W. Robbins & L. Weiskrantz (Eds.), *The prefrontal cortex* (pp. 117-130). Oxford, UK: Oxford University Press.

Grace, J., & Malloy, P. F. (2001). *Frontal Systems Behavior Scale (FrSBe): Professional Manual*. Lutz, FL: Psychological Assessment Resources.

Hartman, C. A., Willcutt, E. G., Rhee, S. H., & Pennington, B. F. (2004). The relation between sluggish cognitive tempo and DSM-IV ADHD. *Journal of Abnormal Child Psychology, 32*, 491-503.

Kass, S. J., Wallace, J. C., & Vodanovich, S. J. (2003). Boredom proneness and sleep disorders as predictors of adult attention deficit scores. *Journal of Attention Disorders, 7*, 83-91.

Lahey, B. B., Applegate, B., McBurnett, K., Biederman, J., Greenhill, L., Hynd, G. W., et al. (1994). DSM-IV field trials for attention deficit hyperactivity disorder in children and adolescents. *American Journal of Psychiatry, 151*, 1673-1685.

Lahey, B. B., Pelham, W. E., Loney, J., Lee, S. S., & Willcutt, E. (2005). Instability of the DSM-IV Subtypes of ADHD from

preschool through elementary school. *Archives of General Psychiatry, 62*, 896-902.

Lahey, B. B., Schaughency, E. A., Hynd, G. W., Carlson, C. L., & Nieves, N. (1987). Attention deficit disorder with and without hyperactivity: Comparison of behavioral characteristics of clinic-referred children. *Journal of the American Academy of Child & Adolescent Psychiatry, 26*, 718-23.

Levy, R., & Dubois, B. (2006). Apathy and the functional anatomy of the prefrontal cortex-basal ganglia circuits. *Cerebral Cortex, 16*, 916-928.

Lezak, M. D. (1995). *Neuropsychological assessment* (3rd ed.). New York: Oxford University Press.

Locke, H. S. & Braver, T. S. (2008). Motivational influences on cognitive control: Behavior, brain activation, and individual differences. *Cognitive, Affective, & Behavioral Neuroscience, 8*, 99-112.

Manes, F., Sahakian, B., Clark, L., Rogers, R., Antoun, N., Aitken, M. et al. (2002). Decision-making processes following damage to the prefrontal cortex. *Brain, 125*, 624-39.

Marin, R. (1990). Differential diagnosis and classification of apathy. *American Journal of Psychiatry, 147*, 22-30.

Marin, R. (1991). Apathy: A neuropsychiatric syndrome. *Journal of Neuropsychiatry and Clinical Neuroscience, 3*, 243-254.

Max, J. E., Mathews, K., Manes, F. F., Robertson, B. A., Fox, P. T., Lancaster, J. L., et al. (2003). Attention deficit hyperactivity disorder and neurocognitive correlates after childhood stroke. *Journal of the International Neuropsychological Society, 9*, 815-29.

McBurnett, K., Pfiffner, L. J., & Frick, P. J. (2001). Symptom properties as a function of ADHD type: An argument for continued study of sluggish cognitive tempo. *Journal of Abnormal Child Psychology, 29*, 207-13.

Michelson, D., Adler, L., Spencer, T., Reimherr, F. W., West, S. A., Allen, A. J., et al. (2003). Atomoxetine in adults with ADHD: Two randomized, placebo-controlled studies. *Biological Psychiatry, 53*, 112-20.

Miller, W. R. & Rollnick, S. (2002). *Motivational interviewing: Preparing people for change* (2nd ed.). New York: Guilford.

Millstein, R. B., Wilens, T. E., Biederman, J., & Spencer, T. J. (1997). Presenting ADHD symptoms and subtypes in clinically referred adults with ADHD. *Journal of Attention Disorders, 2*, 159-166.

Murphy, K. R., Barkley, R. A., & Bush, T. (2002). Young adults with attention deficit hyperactivity disorder: Subtype differences in comorbidity, educational, and clinical history. *Journal of Nervous and Mental Disease, 190*, 147-57.

Murphy, K. R., & Gordon M. (2006). Assessment of adults with ADHD. In R. A. Barkley (Ed.), *Attention-deficit hyperactivity disorder: A handbook for diagnosis and treatment* (3rd ed., pp. 425-452). New York: Guilford.

Nelson, H. (1976). A modified card sorting response sensitive to frontal lobe defects. *Cortex, 12*, 313-324.

Oosterloo, M., Lammers, G. J., Overeem, S., de Noord, I., Kooij, J. J. S. (2006). Possible confusion between primary hypersomnia and adult attention-deficit/hyperactivity disorder. *Psychiatry Research, 143*, 293-297.

Padala, P. R., Burke, W. J., Bhatia, S. C., & Petty, F. (2007). Treatment of apathy with methylphenidate. *Journal of Neuropsychiatry and Clinical Neuroscience, 19*, 81-83.

Partington, J. E., & Leiter, R. G. (1949). Partington's pathway test. *Psychological Service Center Bulletin, 1*, 9-20.

Pessoa, L. (2009). How do emotion and motivation direct executive control? *Trends in Cognitive Science, 13*, 160-166.

Pochon, J. B., Levy, R., Fossati, P., Lehericy, S., Poline, J. B., Pillon, B., et al. (2002). The neural system that bridges reward and cognition in humans: An fMRI study. *Proceedings of the National Academy of Sciences of the United States of America, 99*, 5669-5674.

Polanczyk, G., de Lima, M., Horta, B. L., Biederman, J., & Rohde, L. A. (2007). The worldwide prevalence of ADHD: A systematic review and metaregression analysis. *American Journal of Psychiatry, 164*, 942-948.

Ramsay, J. R., & Rostain, A. L. (2008). *Cognitive behavioral therapy for adult ADHD: An integrative psychosocial and medical approach*. New York: Routledge.

Rey, A. (1941). L'examen psychologique dans les cas d'encephalopathie traumatique. *Archives de Psychologie, 28*, 286-340.

Ryan, R. M. & Deci, E. L. (2000). Self-determination theory and the facilitation of intrinsic motivation, social development, and well-being. *American Psychologist, 55*, 68-78.

Safren, S. A. (2006). Cognitive-behavioral approaches to ADHD treatment in adulthood. *Journal of Clinical Psychiatry, 67* (Suppl.8), 46-50.

Safren, S. A., Perlman, C. A., Sprich, S., & Otto, M. W. (2005). *Mastering your adult ADHD: A cognitive-behavioral treatment program, therapist guide*. New York: Guilford.

Sarter, M., Gehring, W. J., Kozak, R. (2006). More attention must be paid: The neurobiology of attentional effort. *Brain Research Reviews, 51*, 145-160.

Seidman, L. J., Valera, E. M., & Bush, G. (2004). Brain function and structure in adults with attention-deficit/hyperactivity disorder. *Psychiatric Clinics of North America, 27*, 323-347.

Sergeant, J. (2000). The cognitive-energetic model: An empirical approach to attention-deficit hyperactivity disorder. *Neuroscience and Biobehavioral Reviews, 24*, 7-12.

Shue, K. L., & Douglas, V. I. (1992). Attention deficit hyperactivity disorder and the frontal lobe syndrome. *Brain and Cognition, 20*, 104-124.

Simon, V., Czobor, P., Balint, S., Meszaros, A., & Bitter, I. (2009). Prevalence and correlates of adult attention-deficit hyperactivity disorder: Meta-analysis. *British Journal of Psychiatry, 194*, 204-211.

Small, D. M., Gitelman, D., Simmons, K., Bloise, S. M., Parrish, T., & Mesulam, M. M. (2005). Monetary incentives enhance processing in brain regions mediating top-down control of attention. *Cerebral Cortex, 15*, 1855-1865.

Starkstein, S. E., & Leentjens, A. F. G. (2008). The nosological position of apathy in clinical practice. *Journal of Neurology, Neurosurgery and Psychiatry, 79*, 1088-1092.

Starkstein, S. E., Mayberg, H. S., Preziosi, T. J., Andrezejewski, P., Leiguarda, R., & Robinson, R. G. (1992). Reliability, validity, and clinical correlates of apathy in Parkinson's disease. *Journal of Neuropsychiatry and Clinical Neuroscience, 4*, 134-139.

Stuss, D. T., & Benson, D. F. (1986). *The frontal lobes*. New York: Raven Press.

Szatkowska, I., Bogorodzki, P., Wolak, T., Marchewka, A., & Szeszkowski, W. (2008). The effect of motivation on working memory: An fMRI and SEM study. *Neurobiology of Learning and Memory, 90*, 475-478.

Taylor S. F., Welsh R. C., Wager T. D., Phan K. L., Fitzgerald K. D., & Gehring W. J. (2004). A functional neuroimaging study of motivation and executive function. *NeuroImage, 21*, 1045-1054.

Torralva, T., Roca, M., Gleichgerrcht, E., López, P., & Manes, F. (2009). INECO Frontal Screening: A brief, sensitive, and specific tool to assess executive functions in dementia. *Journal of the International Neuropsychological Society, 15*, 777-786.

van der Meere, J., Shalev, R., Borger, N., & Gross-Tsur, V. (1995). Sustained attention, activation and MPH in ADHD: A research note. *Journal of Child Psychology and Psychiatry, 36*, 697-703.

van Reekum, R., Stuss, D. T., & Ostrander, L. (2005). Apathy: Why care? *Journal of Neuropsychiatry and Clinical Neuroscience, 17*, 7-19.

Wechsler, D. (1987). *Wechsler Memory Scale-Revised*. New York: Psychological Corporation.

Wechsler, D. (1997). *Wechsler Adult Intelligence Scale-Third Edition (WAIS-III)*. San Antonio, TX: Psychological Corporation.

Wender, P. H., Wolf, L. E., & Wasserstein, J. (2001). Adults with ADHD. An overview. *Annals of the New York Academy of Science, 931*, 1-16.

Young, S., & Bramham, J. (2007). *ADHD in adults: A psychological guide to practice*. Chichester, UK: Wiley.

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