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Comorbidity in ADHD: A Neuropsychological Perspective

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1. Introduction

ADHD is commonly defined as a condition that affects attention, learning and behavior (American Psychiatric Association, 2001). Despite that over twenty years of scientific research has defined ADHD as a neurodevelopmental disorder, with the higher heritability among psychiatric disorders; clinical-empirical taxonomy (DSM-IV) still prevail (McGough & McCracken, 2006).

From an empirical point of view, *other* difficulties and also disorders that accompany this condition are considered “comorbid” (meaning: secondary). However several studies have found that quality of life in adolescents and adults with ADHD-child-diagnostic is not only related with attention deficits-severity, but also-mainly to comorbidity (Klassen, Miller & Fine, 2004). Yang et al. (2007) studied a sample of 1000 children and adolescents with ADHD, finding that comorbidity symptoms increases in relations to age, reaching the highest impact on behavior by the end of adolescence. Other studies are in agreement that in transition to adulthood comorbidity tends to: a) increase in intensity, and/or b) to appear for the first time (Kessler et al., 2006).

Particularly conduct and negativistic disorders are highly related with other types of comorbidity (Rhee, Willcutt, Hartman, Pennington & Defries, 2007); some studies have found that comorbidity-type could be more important than attention-deficits-severity to produce and explain clinical behaviors: in general literature indicates that when ADHD and conduct disorder are clearly present and combined, the psychological impact is far more important than the effects produced separately by each disorder (Waschbusch, 2002); this impact is not found in the same magnitude for anxiety, depression or bipolar disorder (Pliszka, 2006). Flory et al. (2003) studied 481 adults with childhood-ADHD diagnosis, finding that subjects with higher characteristics for conduct disorder presented higher levels of drug consumption; in this same perspective Donohew et al. (1999) reported a relationship between the sensation seeking behavior profile and drug consumptions in adolescents (alcohol and marihuana). Harty et al. (2009) studied 87 children with ADHD, selecting from this sample subjects with oppositional defiant disorder diagnosis, and conduct disorder, applying diverse behavioral scales to set an initial evaluation during childhood (aggression, anger, and hostility), and a second evaluation teen years later. Finding that the ADHD-

conduct disorder group presented higher levels of physical aggression in adolescence; both comorbid groups presented significant increment of anger (compared with adolescents with ADHD-only). Authors propose that *emotional dysregulation* could be an important component for ADHD during transition from childhood to adolescence.

Specific results on ADHD from the national comorbidity survey replication (Kessler et al., 2006) show a very important negative relation between years of school and symptoms prevalence: adults with ADHD profile presents fewer years of education, even adults with childhood onset without current full-symptoms presents a tendency to have mid to low education level (compared with the general population).

This report outlines that a diverse number of studies that have documented high societal costs for anxiety, mood and substance use disorders, have not included the role of comorbid ADHD. Indicating that only 10% of respondents diagnosed with ADHD reported having received treatment for adult ADHD, this percentage is significantly lower than the reported rates for anxiety, mood, or substance use disorders. Revealing that many people with adult-ADHD are in treatment for other mental or substance use disorders but not for ADHD (Kessler et al., 2006).

ADHD symptoms overlap and are frequently prevalent among patients with others psychiatric disorder (mood, anxiety, substance use, and impulse control disorders). Metanalysis have shown that DSM-IV criteria for adult-ADHD has unclear validity, provoking underdiagnosis (Simon et al. 2009). This clinical under-recognition may negatively influence treatment effectiveness and outcome success (Barkley & Brown, 2008).

McGough et al. (2009) interviewed 435 parents in 230 families with at least one affected children (present or past) with ADHD, using different rating scales based on DMSM-IV. Parents affected with ADHD (past or present affection) presented lower educational and occupational achievement, higher rates of lifetime comorbidity for depression, anxiety, disruptive behaviors, and substance use disorder. Higher risk for disruptive disorder was predicted only by male sex and ADHD; substance use disorder was mainly predicted by disruptive behavior disorders, male sex, and lower socioeconomic status.

Sex differences in comorbidity have also been found: in women drug consumption is mainly related to depression and anxiety (McGough et al., 2005). Ruhl et al. (2009) studied 2064 young-women (aged 18-25) with a structured interview according to DMS-IV criteria. Finding a 1.5% lifetime prevalence for ADHD, with 14% still suffering from ADHD since Childhood. Lifetime prevalence for conduct, somatoform, or posttraumatic stress disorder was significant higher in ADHD-women. Females with past or current ADHD-diagnosis were almost twice vulnerable to suffer from depressive or phobic disorder (than women from the general population). Authors also highlights that even when ADHD-diagnostic criteria is no longer fulfilled in adulthood, women with ADHD since childhood presents and increased prevalence for psychiatric comorbidity.

2. Is comorbidity a psychological science?

Drake and Wallach's (2007) article title is a perfect frame to develop this issue, like Stefanatos and Baron (2007) they propose scientific frames for conceptualizing and studying ADHD-comorbidity, instead of "clinical-empirical" criteria.

Barkley (2009) and other scientists (i.e. Diamond, 2005) have demonstrated conceptual limitations in DSM-IV conceptualization for ADHD: according to Barkley diagnostic sensitivity to impulsivity symptoms is underrepresented in DSM-IV; pointing out that from a scientific-cognitive perspective, behavioral and cognitive control does not depend of attentional but on executive functions (these functions are mainly dependent on prefrontal cortex development -Diamond, 2001; van Leijenhorst et al., 2010-). They also highlights that others symptoms-behaviors-characteristics that could differentiate impulsivity like another possible category in ADHD are not appropriately represented in DSM-IV criteria.

Manassis et al. (2007) have found that children with anxiety disorders present an enhanced perception for negative emotions, specifically an incremented response in attention and memory processing to threatening stimuli; whereas ADHD-anxiety children didn't exhibited this profile. Authors consider that for each group anxiety may present different psychophysiological causes and characteristics.

Studies that compared children with ADHD-only vs children with ADHD-oppositional defiant disorder, reports attenuated electrophysiological responses (less P300 activation to cues) during attentional tasks (Continuous Performance Task: CPT) specifically for children with ADHD-only; indicating difficulties with anticipation and response preparation. This electrophysiological pattern is absent in ADHD-oppositional defiant disorder, indicating that comorbidity should not be viewed as a simple clinical-addition (Luman et al., 2009).

A consistent relationship between right hemisphere activation and a perceptual bias for negative emotions (fear, anxiety and sadness) has been frequently documented: more right-frontal activity is found in shy-avoidant children, and in subjects with depression and anxiety; in contrast more left-frontal activity is found in sociable and low-social-fear children (McManis, Kagan, Snidman & Woodwad, 2000). By studying children with ADHD and their parents (affected and not affected), with electrophysiological recording during an attentional task (CPT), Hale et al. (2010) found that affected subjects (children and parents) presented an increased rightward asymmetry across frontal and central regions. An interesting relationship has been found: increased ADHD-familial loading correlated with increased rightward frontal asymmetry, instead increased rightward parietal asymmetry was correlated with reduced ADHD-family loading. Overall, this psychophysiological profile could predispose subjects with ADHD to be perceptually-biased to negative-emotional processing.

Schwartz et al. (2010) measured the orbito-frontal cortex density in normal subjects (18 years old) previously characterized in infancy as low-reactive or high-reactive subjects, finding that participants with low-reactive infant temperament presents greater thickness in the left orbito-frontal cortex, while subjects with high-reactive infant temperament present greater thickness in the right orbito-frontal cortex.

In literature it's been described that children with conduct disorder present mainly motivational problems, whereas children with ADHD present mainly attention deficits; Rubia et al. (2009) studied (by functional magnetic resonance imaging) 14 children with conduct disorder, and 18 children with ADHD; using a reward continuous performance task, and a sustained attention task. Results show brain activation differences between groups: conduct disorder subjects presented underactivation in paralimbic regions whereas ADHD subjects presented underactivation in diverse cerebral areas including prefrontal

cortex. During the reward condition, conduct disorder subjects presented underactivation in right orbito-frontal cortex, while ADHD presented underactivation in posterior cingulate and precuneus; indicating neurocognitive differences among different clinical profiles.

Luman et al. (2009) found some neuropsychological differences among ADHD-children vs. ADHD-oppositional defiant disorder (ODD): children with ADHD-only presented difficulties in inhibition, timing estimation and reinforcement sensitivity, whereas children with ADHD-ODD presented an increased variability in timing estimation. Authors concluded that ADHD-ODD do not represent a *more severe* form of ADHD, instead a different one.

These studies indicate that neurocognitive/neuropsychological profiles are more specific and clinically illustrative for understanding comorbidity. Diverse authors have outlined that comorbidity is not only accompanying the disorder, and do not represent a *clinical ambiguous variant* for a given disorder. Instead the disorder itself and other -apparently not primarily related- clinical characteristics could depend on common cognitive, psychological and/or neuropsychological mechanisms.

Due to that ADHD-comorbidity pathophysiology is highly heterogeneous, it's been proposed that -scientific- models of comorbidity should be proposed. (Drake & Wallach, 2007; Stefanatos & Baron; 2007, Taurines et al. 2010). And although a neuropsychological approximation to understand the basic mechanisms of "psychological" disorders is continuously increasing, the neuropsychological profile for each disorder hasn't yet acquired a central role in diagnosis and treatment (Beblo, Sinnamoni & Baune, 2011: *Neuropsychology Review*). However with this approximation an optimal comprehension for this "attentional" disorder could be achieved (Diamond & Anso, 2008).

3. A neuropsychological perspective in ADHD

From a neuropsychological perspective comorbidity is considered part of the same brain and cognitive mechanisms that produces attentional and or behavioral difficulties (Barkley, 2009; Diamond, 2005): children with attentional-variant presents a different (opposite) pattern of cognitive and behavioral profile than children with the hyperactive-variant. Inattentive profile includes social isolation, behavioral passivity, and cognitive slowness; in contrast hyperactive profile includes aggressive behavior, emotional instability, cognitive impulsivity and social disruptiveness (Flores Lázaro, 2009; Maedgen & Carlson, 2000).

Attentional mechanisms do not represent a general-valid construct to explain: rejection to follow rules and to perform organized-structured activities, immediate seeking for gratification, low motivational drive, emotional lability, etc (Diamond, 2005). Instead of comorbidity *summation*, basic and common neuropsychological mechanisms are proposed in order to explain from wider perspective attentional, behavioral and psychological profiles (Barkley, 1997, 2009; Stefanatos & Baron, 2007).

The principal neuropsychological characteristics seen in ADHD could be explained by deficits in executive function, these functions depend on different brain circuits mainly located in prefrontal cortex (Barkley, 2010); for example children with combined-ADHD present more deficits in strategic memory -an executive function- than children with inattentive-ADHD type (Castel, Humphreys & Moore, 2011). Executive functions permit

complex cognitive and behavioral control, and are critical for success in school and daily life (Diamond, 2011).

4. Attentional sub-type as an ADHD-cortical form

Metanalysis of 19 developmental-functional neuroimaging studies in normal subjects has found that: fronto-parietal networks-interaction allows conscious-cognitive control of attentional processes. During brain development, functional integration between these and others networks is constructed; this phenomena also permits functional integration among several executive functions like inhibitory control, set switching and working memory (Fair et al., 2007). Inattentive predominant profile in children with ADHD includes difficulties in working memory, volume and processing speed, primary learning difficulties, and social isolation; the absence of behavioral difficulties as dominant profile, implicates neo-cortical mechanism that explains predominant cognitive consequences (Diamond, 2005).

Attentional control (focusing, stability, and alternation) and attentional processing (volume, processing speed) for cognitive academic and working environmental demands, depends mainly on cortical control -top-down regulation-; during child development, delay and inadequate integration between fronto-parietal networks leads to difficulties in attentional control and other executive functions (Fair et al., 2007). Stimulants like atomoxetine enhances prefrontal cortex functioning in ADHD-subjects -through adrenergic and dopamine receptors- (Gamo et al., 2010); with positive psychophysiological results.

5. Hyperactive sub-type as a ADHD-fronto-striatal form

Hyperactivity, constant seek for gratification, motivational instability, cognitive impulsivity (*works with hurry*) and anxiety, has been the core symptoms in hyperactive sub-type (Rubia et al., 2009). Fronto-striatal circuits are highly replicated in literature for hyperactive children, pharmacological treatment with stimulants like Methylphenidate produces positives psychophysiological changes in cortical (cognitive) and fronto-striatal (dopamine-reward systems) functioning (Zhu et al., 2011). It's been established that inhibitory control is the core mechanism for this variant (Barkley, 2009), this executive function permit optimal cognition and behavior regulation; metanalysis on 19 studies using Stroop interference measure (inhibitory control), indicates that this process is consistently compromised in ADHD (Lansbergen, Kenemans & van Engeland, 2007).

During development dynamic interactions between brain regions changes, especially in fronto-striatal circuits, as a result hyperactivity significantly diminishes during late childhood-adolescence; instead sensation seeking (for reward-goal oriented activities) develops in a \cap -shaped along childhood-adolescence-adulthood. Motivational cues for potential reward are specifically incremented in adolescence (striate hyper-responsiveness), and can lead to riskier choices during goal-oriented behavior (Somerville & Casey 2010). Diverse studies that includes functional neuroimaging have found that only during adolescence a striate hyperactivation for reward expectation occurs (Van Leijenhorst et al., 2010), and although risk-detection capacity (in normal subjects) is adult-competent since 13 years old (Crone et al., 2005); in ADHD-adults is clinically diminished (Malloy-Diniz et al., 2007).

Emotional dysregulation (e.g. high sensation seeking despite risk consequences) in ADHD could lead to greater risk for psychopathological comorbidity (Blaskey, Harris & Nigg, 2008). Grall-Bronec et al. (2011) studied 85 adults with pathological gambling (this disorder is characterized by inadequate reward-regulation – Brevers et al., 2011-), finding that more than 25% of this sample presented history of ADHD, this subgroup presented elevated anxiety and impulsivity, more severe gambling problems, a higher frequency of psychiatric comorbidities and an elevated suicidal-risk.

6. Impulsivity as a *fronto-temporal*-ADHD type

Behavioral impulsivity, aggressiveness, conduct disorder, and emotional outburst; are the core symptoms in impulsivity variant (Harty et al., 2009). Fronto-temporal mechanisms are responsible for optimum regulation of emotional-based behavior (Gupta, Kosciak, Bechara & Tranel, 2011), adequate frontal regulation to emotional-based responses represent an important prerequisite for interpersonal and social interactions, allowing subjects to regulate verbal and behavioral interactions; these processes are fundamental for socially-adjusted behavioral and personality development during childhood (Damasio, 2005). Based on genetics studies low serotonin metabolism has been outlined as the key neurotransmitter system for behavioral-impulsivity and aggression in ADHD (Oades et al., 2008). By using magnetic resonance imaging Boes et al. (2008) studied the relationship between impulsive control and volume-size for diverse brain regions (including amygdala, anterior cingulate cortex and prefrontal cortex) in 61 normal-boys from 7 to 17 years old; finding that subjects with poorer impulsivity control presented (significantly different) less volume in the right ventro-medial –orbital- cortex.

Literature indicates that ADHD and conduct disorder combination represent a very important clinical condition (Waschbusch, 2002). Barkley and Fischer (2010) studied the influence of emotional impulsiveness over social, laboral and daily live activities , in 135 ADHD-young-adults; finding that if ADHD-symptoms are still persistent in adulthood, emotional impulsiveness severity contributes to significant impairment in occupational, educational, legal, and financial aspects. Authors highlight the importance of impulsivity in ADHD-criteria, considering it's at least as important as inattention/hyperactivity dimension for the development of adequate social, occupational and interpersonal functioning. Although highly important, impulsivity dimension is under-represented in DSM-IV criteria for ADHD (Barkley, 2009).

7. Comorbidity classification by neuropsychological criteria

In most cases in literature comorbidity is reported in relationship to prevalence (clinical-epidemiological approach), however ADHD-subtypes in adults are associated with different clinical comorbid correlates (Sprafkin et al., 2007), results coincide in describing predominant behavioral-psychological disorders in combined-ADHD, and predominant cognitive disorders at inattentive type (Murphy, Barkley & Bush, 2002). To explore comorbid profiles from a neuropsychological point of view, a sample of 61 male-children with ADHD-diagnosis (DSM-IV criteria) were selected. Participants had between 6 and 10 years-old (mean: 7.6). Inclusion criteria were: not-medicated for ADHD (or any other neurodevelopmental/psychiatric disorder), no history of psychological treatment, normal I.Q. (WISC); exclusion criteria: others non-related neurodevelopmental disorder (epilepsy,

cognitive deficit, etc). All subjects were evaluated with diverse neuropsychological tests, and evaluated by clinical-psychological criteria (see Flores Lázaro, 2009 for details). On the first analysis 20 subjects with ADHD-attentive type (ADD) (group one) were compared vs. 10 subjects with ADHD-combined (ADHD-C) type and 10 subjects with ADHD-impulsivity with or without hyperactivity-variant (ADHD-I-H) (group two: ADHD-C-I).

Based on Diamond (2005) proposal, comorbidity was divided in behavioral-psychological, and cognitive. Statistical analysis was performed by Fisher's exact test (FET). All results including tables are adapted from Flores Lázaro (2009)*

Data presented in tables 1 and 2, supports Barkley's and Diamond's basic division, illustrating that both groups are significantly different in behavioral-psychological and cognitive dimensions. These results coincide with other studies (Capdevila-Brophy, et al., 2005; Maedgen & Carlson, 2000). ADHD-C-I group is mainly characterized by behavioral-psychological comorbidity; whereas cognitive comorbidity represents the main characteristic for ADD group.

<i>Behavioral-psychological comorbidity</i>	<i>ADHD-C-I</i>	<i>ADD</i>	<i>FET</i>
Mood disorders	43 %	10%	.017
Conduct disorder an/or agresivity	39%	8%	.040
Negativistic defiant disorder	39%	0%	.032
Anxiety/anxiety disorder	35%	8%	.040
Socialization difficulties	35%	40%	.432
Avoiding to follow rules-instructions	39%	0%	.001
Infantilization	39%	0%	.002
Low tolerance to frustration	35%	17%	.101
Insecurity (low self-concept)	13%	36%	.022

Table 1. Percentage on behavioral-psychological comorbidity and differences between ADHD-C-I and ADD

<i>Cognitive comorbidity</i>	<i>ADHD-C-I</i>	<i>ADD</i>	<i>FET</i>
Visoespatial difficulties	39%	70%	.014
Mathematics learning disorder	17%	40%	.032
Reading learning disorder	26%	35%	.356
General learning disorder	4%	35%	.001
Language difficulties/dysphasia	4%	30%	.012

Table 2. Percentage on cognitive comorbidity and differences between ADHD-C-I and ADD

It's important to outlined that socialization difficulties are due to different causes in each group (evitative vs disruptive), reading learning disorder are also due to different neuropsychological causes (Roselli, Matute & Ardila, 2006). Even when general-clinical-

* Partially/totally reproduced from "Características de comorbilidad en los diferentes subtipos de trastorno por déficit de atención con hiperactividad" de J.L. Flores Lázaro, 2009, *Psicothema*, 21(4), p. 594, 595. Copyright 2009 del Colegio Oficial de Psicólogos del Principado de Asturias. Reproducido con permiso"

criteria *coincide*, psychological and neuropsychological analysis permits to scientifically differentiate specific differential-causes for the *same* criteria. Literature has progressively demonstrated that *clinical-empirical* dimensions without psychological, psychophysiological, or neuropsychological scientific bases should not serve as *professional* taxonomic criteria (McGough & McCracken, 2005; Stefanatos & Baron, 2007).

8. Hyperactive, inattentive and impulsive variants analysis

During transition from childhood to adolescence, emotional regulation represents a central component for ADHD-mental health (Barkley & Fisher, 2010), due that impulsivity variant is underrecognized in DSM-IV criteria (Barkley, 2009), and that emotional dysregulation is a core characteristic for impulsivity subtype (Harty et al., 2009); ADHD-Impulsivity variant still represents a very important clinical subgroup for scientific study.

Current knowledge on cognitive neuroscience and neuropsychology permits to validate the pertinence to investigate differences among hyperactive vs. impulsive subjects. Therefore a second analysis was performed with a three-group division: 20 children with inattentive variant (ADD), 20 children with combined variant (ADHD-C), and 21 children with impulsivity variant -with or without hyperactivity- (ADHD-I). Results are presented in tables 3 and 4, data shows a clear cognitive to behavioral-psychological comorbidity transition: inattentive-hyperactive-impulsive. As expected by neuropsychological criteria, impulsivity variant presents the highest significant-frequent behavioral-psychological comorbidity; Inattentive variant continues to present the highest significant-frequent cognitive comorbidity. Interestingly Hyperactive variant lies in the middle range on this group-division. Results illustrate Barkley's point of view that impulsivity-variant represents a sufficiently differentiated clinical-group.

Impulsivity-variant suggests fronto-temporal mechanisms as the main causes for comorbidity, differently to fronto-striatal mechanism on hyperactive-variant. However specifics neuropsychological, psychophysiological, structural and functional neuroimaging studies should differentiate fronto-parietal, fronto-striatal and fronto-temporal mechanisms for each cases.

<i>Behavioral-psychological comorbidity</i>	<i>ADHD-I</i>	<i>FET</i>	<i>ADHD-C</i>	<i>FET</i>	<i>ADD</i>
Mood disorders	47%	.202	31%	.118	10%
Conduct disorder an/or agressivity	42%	.191	26%	.204	10%
Negativistic defiant disorder	31%	.002	0%		0%
Anxiety/anxiety disorder	37%	.415	31%	.118	10%
Socialization difficulties	45%	.191	26%	.250	40%
Avoiding to follow rules-instructions	52%	.038	25%	.024	0%
Infantilization	26%	.329	42%	.002	0%
Low tolerance to frustration	31%	.585	35%	.137	15%
Insecurity (low self-concept)	5%	.284	15%	.137	36%

Table 3. Percentage on behavioral-psychological comorbidity and differences between groups

<i>Cognitive comorbidity</i>	<i>ADHD-I</i>	<i>FET</i>	<i>ADHD-C</i>	<i>FET</i>	<i>ADD</i>
Visoespatial difficulties	70%	.100	42%	.222	26%
Mathematics learning disorder	40%	.250	25%	.081	5%
Reading learning disorder	34%	.500	40%	.065	15%
General learning disorder	34%	.022	5%	.488	0%
Language difficulties/dysphasia	30%	.118	10%	.232	0%

Table 4. Percentage of cognitive comorbidity and differences between groups

9. Comorbid profiles according to ADHD-variants

Comorbid characteristics among different ADHD-subtypes analyzed are specific, behavioral-psychological comorbidity is listed from more to less frequent:

- Inattentive profile: socialization (evitative -type), and low self-concept.
- Hyperactive profile: infantilization, low-tolerance to frustration, anxiety/anxiety disorder, mood disorders, conduct disorder/aggressiveness, socialization, and avoiding to follow rules and instructions,
- Impulsivity variant: following rules, limits and instructions. Mood disorders, socialization (disruptive-type), conduct disorder/aggressiveness, anxiety/anxiety disorder, negativistic defiant disorder, low-tolerance to frustration, and infantilization.

Cognitive comorbidity is listed from more to less frequent:

- Inattentive: visoespatial, mathematics, reading, and general learning difficulties, language/dysphasia.
- Hyperactive: visoespatial, reading, and mathematics learning difficulties.
- Impulsive: visoespatial, reading learning disorder.

<i>Behavioral-psychological comorbidity</i>	<i>FET</i>
Mood disorders	.012
Conduct disorder an/or agressivity	.025
Negativistic defiant disorder	.003
Anxiety/anxiety disorder	.048
Socialization difficulties	.607
Avoiding to follow rules-instructions	.000
Infantilization	.014
Low tolerance to frustration	.181
Insecurity (low self-concept)	.015

Table 5. Differences on behavioral-psychological comorbidity between *extreme groups*: ADD vs ADHD-I-H

10. Extreme groups division

As shown in tables 3 and 4, ADD and ADHD-I groups represent *extreme comorbidity*: behavioral-psychological vs cognitive. In order to analyze statistical differences among these groups FET has been applied. Results are presented in table 5 and 6, data indicate

clearer/most frequent significant differences both comorbidity-types; inclusion of children with Hyperactive-variant (a transitional group) tends to diminish statistical differences (see tables 3 and 4).

<i>Cognitive comorbidity</i>	<i>FET</i>
Visoespatial difficulties	.005
Mathematics learning disorder	.006
Reading learning disorder	.100
General learning disorder	.003
Language difficulties/dysphasia	.007

Table 6. Differences on cognitive comorbidity between *extreme groups*: ADD vs ADHD-I

11. Conclusions

Neuropsychology permit to establish a conceptual bridge between clinical and basics sciences (psychology and neurosciences). Applied neuropsychological research posses high environmental validity in clinical settings (Beblo et al., 2011). Several researchers have criticized the reluctance of psychiatry-practice to move toward scientifically oriented pathophysiology -neurosciences- more common in actual days to general medicine and psychology (McGough & McCracken, 2005); from a neuropsychological point of view ADHD is mainly a disexecutive disorder in which several cognitive, behavioral and psychological consequences are present, in different form, at diverse ontogenetic moments. In ADHD, attention is *only one* of these consequences; and for an important number of cases is not the most important. Diverse clinicians and researches in this field began to underline the need for systematic neuropsychological evaluation in order to objectify attention disorders (Chambry et al., 2011), interpretations on these results should be made within valid scientific models (Drake & Wallach, 2007; Stefanatos & Baron, 2007). Professionals should understand basic neuropsychological and psychological mechanism causing ADHD: attention is *ONLY* one of the cognitive processes involved, due to inadequate development in executive function control; in most cases comorbidity is *ALSO* explained by the same psychophysiological and/or neuropsychological mechanisms; based on this comprehension intervention programs will be more specific, and above all: more scientifically oriented.

Drake and Wallach's fundamental question for psychology may be better answered in this form: *ADHD-comorbidity is -mainly- a neuropsychological science.*

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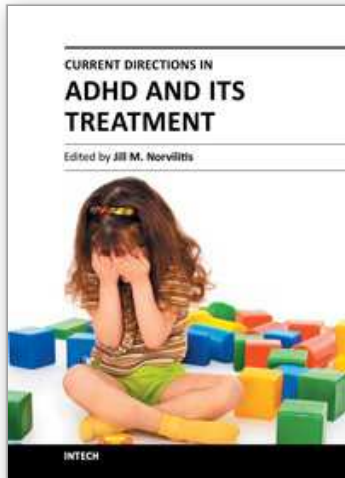
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The treatment of Attention Deficit Hyperactivity Disorder is a matter of ongoing research and debate, with considerable data supporting both psychopharmacological and behavioral approaches. Researchers continue to search for new interventions to be used in conjunction with or in place of the more traditional approaches. These interventions run the gamut from social skills training to cognitive behavioral interventions to meditation to neuropsychologically-based techniques. The goal of this volume is to explore the state-of-the-art in considerations in the treatment of ADHD around the world. This broad survey covers issues related to comorbidity that affect the treatment choices that are made, the effects of psychopharmacology, and non-medication treatments, with a special section devoted to the controversial new treatment, neurofeedback. There is something in this volume for everyone interested in the treatment of ADHD, from students examining the topic for the first time to researchers and practitioners looking for inspiration for new research questions or potential interventions.

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