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The study of automatic and controlled processes in ADHD:

a reread and a new proposal

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Abstract

Attention Deficit Hyperactivity Disorder (ADHD) is described as a persistent or an ongoing pattern of inattention and/or hyperactivity-impulsivity that gets in the way of daily life or typical development. Most research in ADHD shows that the core symptoms are related to deficits in executive functions. Only few works show that deficits in ADHD are also related to automatic processes. The shortsightedness of past studies is that they are in focus when looking at the closer object of executive function deficits, but they are out of focus when they fail to include a larger context beyond the focus such as the inextricable relationship between automatic cognitive processes and executive functions deficits. The aim of the present work is to summarize data on automatic and controlled processes in ADHD subjects. Another purpose is to show that the executive functions alone cannot explain the ADHD symptoms, they have be reinterpreted and integrated in the light of new evidence. The new evidence comes from both cognitive and neurophysiological research. Finally, based on new evidences, the Cumulative

and Emergent Automatic Deficit model (CEAD) is proposed, its theoretical implications of emerging patterns and key directions for future work are discussed.

Keywords: ADHD, automatic processes; controlled processes; executive functions; cortico-subcortical neural models.

Introduction

Attention Deficit Hyperactivity Disorder (ADHD) is a neurodevelopmental disorder affecting both children and adults. It is described as a persistent or an ongoing pattern of inattention and/or hyperactivity-impulsivity that gets in the way of daily life or typical development (APA, 2013). There are three presentations of ADHD: inattentive, hyperactive-impulsive and combined inattentive and hyperactive-impulsive. The question of whether deficiencies in specific underlying mechanisms are referred to controlled or automatic processes is a key point to child psychopathology theory development.

As the main theories suggest, automatic processing is fast (Neely, 1977; Posner & Sneider, 1975), effortless (Logan, 1988; Schneider & Shiffrin, 1977) autonomous (Logan, 1988; Schneider & Shiffrin, 1977; Posner & Sneider, 1975; Zbrodoff & Logan, 1986), stereotypical (McLeod, McLaughlin, & Nimmo-Smith, 1986), unavailable to conscious awareness (Carr, McCauley, Sperber & Parmalee, 1982) and fairly error-free. It can be accomplished simultaneously with other cognitive processes without interference, it is not limited by attention capacity and it can be unconscious or involuntary. Controlled processing is effortful and can deal with only a limited amount of information at once, it is slow and prone to errors, but – at the same time, flexible and useful to deal with new tasks. When tasks interfere, this is usually meant to indicate competition for limited attentional processing resources. A controlled process requires attention: automatic process does not.

In cognitive psychological literature, there can be other words used to refer to automatic and controlled processes such as reflexes and voluntary behaviour, exogenous and endogenous attention control, top-down and bottom-up processes, stimulus driven and voluntary driven, declarative and procedural memory, implicit and explicit memory, serial and parallel processing, conscious and unconscious processes, automatic inhibitory control mechanisms and intentional inhibitory control mechanisms.

Recent studies have begun to address this question with respect to the functional working memory model of ADHD (Rapport, Chung, Shore, & Isaacs 2001). In fact, children with ADHD show deficits in executive functions, including response inhibition, working memory, and conflict resolution. Most research (Swanson et al.1998; Barkley 2003; Mayes & Calhoun 2006; Shanahan et al. 2006) argues that the three main symptoms of this disorder are due to a deficit in executive functions (EFs) and, in particular, to an inhibition deficit and to planning difficulties (Bush et al., 1999; Casey et al., 1997; Doyle, 2006; Pliszka et al., 2006; Rubia, Smith, Brammer, Toone & Taylor, 2005; Vaidya et al., 2005). The inability to inhibit or defer an answer explains many of the behavioural symptoms (hyperactivity, impulsiveness, and intolerance with frustration) and cognitive symptoms (difficulty in resisting distractors). Some other researchers recognize a difficulty in ADHD in the automatic processing of basic skills (Ackerman, Anhalt, Holcomb, & Dykman, 1986; Fabio, 2001, 2009, 2015) or in the modality of stimulus presentation (Fabio & Antonietti, 2012). Furthermore, Hazell et al. (1999) showed that participants affected by ADHD show a deficit in encoding and in the automatic processes, along with a deficit in the central controlled processes. Moreover, Hurks et al. (2004) examined the performance of ADHD children on semantic category fluency (SCF) versus initial letter fluency (ILF) tasks. Results indicate that children with ADHD symptoms show a delay in the development of automatized skills in processing abstract verbal information. Anyway, the hypothesis that hyperactive children fail to develop automatic processing is less consolidated.

The main purpose of the present study is to show that past research can be reinterpreted and integrated in light of new evidence coming from both studies on neuropsychological and neurophysiological levels.We examine the hypothesis that deficits of EF are at least partly due to deficits in automatic processing. The underlying logic at the neuropsychological level is that if the basic processes are not well automatized, they will result in a high cognitive load and compete for limited resources used by EF or controlled processes. The same underlying logic lies at the neurophysiological level: a high number of research studies had shown that the principal neurocognitive deficit may reside in neural substrates underlying executive functions; since neural substrates as the basal ganglia are connected to the prefrontal cortex as a gating system, a poor functioning of the basal ganglia can influence all the subcortical-cortical-subcortical neural circuit. These models are in line with the hypothesis of a deficit in automatic processes in ADHD people.

In the first part of this study, we present literature on controlled processes in ADHD and the few works that refer to automatic processes in ADHD. In the second part, we show the impossibility to separate automatic and controlled factors, and present models that soften the traditional dual model. In the third part, research on automatic and controlled underlying neural structures on ADHD subjects will be presented. In the last part a new proposal is presented allowing us to better interpret cognitive problems in ADHD: the CEAD model. We discuss theoretical implications of emerging patterns, and propose key directions for future work, including potential resolutions to several methodological challenges for cognitive empowerment of ADHD subjects.

Research on automatic and controlled processes in ADHD

The possibility that specific deficits in executive function (EF) tasks are connected to ADHD has been documented in a number of different studies (Barkley, 1998; Brown, 2013; Pennington & Ozonoff, 1996; Shallice et al., 2001; Swanson et al., 1998; Willcutt, Doyle, Nigg, Faraone & Pennington, 2005), but the possibility that automatic deficits are also connected to ADHD is less consolidated. In the following paragraphs we will examine some key issues on automatic and controlled processes in ADHD.

Controlled processes in ADHD

The most prominent neuropsychological theories of attention-deficit/hyperactivity disorder suggest that its symptoms arise from a primary deficit in executive functions. Executive functions (also known as cognitive control and supervisory attentional system) is an umbrella term for the management (regulation, control) of cognitive processes (Elliot, 2003), including working memory, reasoning, task flexibility, and problem solving (Monsell, 2003) as well as planning and execution (Chan et al., 2008) and all controlled processes. As Barkley (2012) points out, unfortunately, there is no consensus at this time on the meaning of the term EF, despite it being used prolifically in journal articles, presentations, and books about ADHD, but most articles refer to EF as neurocognitive processes that maintain an appropriate problem-solving set to attain a later goal. Willcutt, Doyle, Nigg, Faraone, and Pennington (2005) examined the validity of the EF theory and conducted a meta-analysis of 83 studies that administered EF measures to groups with ADHD (total N = 3734) and without ADHD (N = 2969). Groups with ADHD exhibited significant impairment on all EF tasks. The most

consistent effects were obtained on measures showing ADHD deficits in response inhibition, vigilance, working memory, and planning (all controlled effortful processes). This weakness was not explained by group differences in intelligence, academic achievement, or symptoms of disorders (Pennington & Ozonoff, 1996; Sechi, Corcelli & Vasquez, 1998).

Some research on executive functions has been guided by the frontal metaphor, emphasizing the planning deficits consequential on frontal injury (Grodzinski & Diamond, 1992; Levin, Eisenberg & Benton, 1991). Other studies mainly refer to the disorder of the working memory system (Barkley, 1997) or to information processing (Sergeant, 1995).

A recent meta-analysis (Kofler, Rapport, Bolden, Sarver, & Raiker, 2010) and some experimental studies (Martinussen, Hayden, Hogg-Johnson, & Tannock, 2005; Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005; Brocki, Randall, Bohlin, & Kerns, 2008; Rapport, et al., 2008) are also highly consistent in documenting working memory impairments in children with attention-deficit/hyperactivity disorder (ADHD) relative to typically developing children. Converging evidence indicates that children with ADHD are impaired in all three components of working memory, with the largest deficits found in the domain-general central executive (CE) system, followed by visuospatial (VS) storage/rehearsal and then phonological (PH) storage/rehearsal subsystems (Martinussen et al., 2005; Rapport, Alderson et al., 2008).

Taking stock, children with ADHD have difficulty in maintaining attention, in focusing their attention on a task, and, in particular in inhibiting visual and sound distrac¬tors; they also show a deficit in working memory and EFs and, in particular, an inhibition deficit and planning difficulties (Bush et al., 1999; Brown, 2013; Casey et al., 1997; Doyle, 2006; Fabio, Castriciano & Rondanini, 2015; Pliszka et al., 2006; Rubia, Smith, Brammer, Toone & Taylor, 2005; Vaidya et al., 2005). As mentioned before, most theorists focus on executive deficit, including response inhibition, working memory, and con¬flict resolution (Barkley, 1997, 1998; Bush et al., 1999; Casey et al., 1997; Doyle, 2006; Pennington & Ozonoff, 1996; Pliszka et al., 2006; Rubia, Smith, Brammer, Toone, & Taylor, 2005; Sechi, Corcelli & Vasques, 1998; Sergeant, 1999; Shallice, Marzocchi, Coser, Del Savio, Menter & Rumiati, 2001; Swanson, Posner, et al., 1998; Vaidya et al., 2005). The nature of such control components is based on inhibitory control. Barkley (1997) refers to them as three interconnected processes, including inhibiting prepotent responses, inhibiting ongoing responses, and interference control (Barkley, 1997). It is implicated in the control of both behavioural actions and cognitive processes (Fillmore, 2003). Roberts, Fillmore and Millich (2011) present an example, in which inhibitory

control represents an individual's ability to filter out distractors and retain task-relevant information. As part of typical cognitive development, inhibitory control is implied in other cognitive constructs and abilities, such as working memory (Ridderinkhof & van der Molen, 1997), the effective execution of goal directed behaviour, and sustaining attention in the presence of distractions (Barkley, 1997). Summing up again, all these findings have singled out impaired inhibitory control as a central trait of the disorder (Nigg, 2001; Pennington & Ozonoff, 1996). Furthermore, Hawk, Yartz, Pelham and Lock (2003) as well as Fillmore Milich, and Lorch (2009) found that children with ADHD showed an intentional, but not automatic, inhibitory deficit on a prepulse inhibition task. However, it is important to note that different theoretical accounts of ADHD emphasize different types of inhibitory dysfunction (e.g., executive inhibition, motivational inhibition; Barkley, 1997; Quay, 1997; Nigg, 2001).

Automatic processes in ADHD

The hypothesis that hyperactive children fail to develop automatic processing is less consolidated.

In this view, a first line of work (Ackerman, et al., 1986; Borcherding, et al., 1988; Ott & Lyman, 1993) suggests that ADHD children have no deficit in innate, automatic skills but they may exhibit problems in performing acquired automatic skills (Ackerman, et al., 1986). A second line of research attempted to clarify if the deficit is due to automatic or executive functions by studying the comorbidity of ADHD and Learning Disabilities. Since subjects with Learning disabilities show deficits in automatic processes (Lum, Conti-Ramsden, Page & Ullman, 2012), subgroups of ADHD with and without learning disabilities have been studied (Ackerman, Anhalt, Holcomb & Dikman, 1986; Barkley, 1997; Denkla, 1996; Fabio & Urso, 2014; Hazell, et al., 1999; Levi, Sechi & Graziani, 1991), but results are still undetermined. The co-occurrence of attention deficit-hyperactivity disorder (ADHD) and reading disability is well documented (August & Garfinkel, 1990; Dykman & Ackerman 1991; Trzesniewski, Moffitt, Caspi, Taylor, & Maughan, 2006; Willcutt & Pennington, 2000; Willcutt, Pennington, & DeFries, 2000). It reflects a strong phenotypic association between reading disability and ADHD inattention symptoms, largely attributed to shared genes (Martin et al. 2006; Willcutt & Pennington, 2000; Willcutt, Pennington, & DeFries, 2000). Willcutt et al. (2010) recently conducted a systematic meta-analysis of all published neuropsychological studies of childhood disorders to find cognitive risk factors capable of explaining comorbidity between RD, ADHD,

and other complex disorders (Willcutt et al., 2000). The results of the review and a series of empirical studies, overall, suggest that the strongest candidates for a shared cognitive weakness in RD and ADHD are processing speed, response variability, and verbal working memory (Rucklidge & Tannock, 2002; Shanahan et al., 2006; Willcutt, Chhabildas, Pennington, 2001; Willcutt, Pennington, et al., 2001; Willcutt, Pennington, Olson, Chhabildas, & Hulslander, 2005). In addition, several studies unexpectedly found deficits in response inhibition in groups with RD (Purvis & Tannock, 2000; Willcutt, Chhabildas et al., 2001; Willcutt, Pennington, et al., 2001), pointing out that additional research is needed to clarify the nature of this association.

A third line of research tries to understand if ADHD without comorbidity can show deficit in automatic processing (Ackerman, et al.,1986; Fabio, 2005, 2009; Fabio, et al., 2015) or in the stimulus presentation type (Fabio & Antonietti, 2012). Hazell et al. (1999) suggested that participants affected by ADHD show deficits in the encoding and in the automaticity of processes, along with deficits in the central controlled processes. Kofler, Rapport, Bolden, Sarver, & Raiker (2010) propose a model hypothesizing a functional relationship between working memory deficits and inattentive behaviour. Their study investigated whether the inattentive behaviour in children with ADHD was functionally related to the domain-general central executive and/or subsidiary storage/rehearsal components of working memory. Objective observations of children's attentive behaviour by independent observers were conducted while children with ADHD and typically developing children completed counterbalanced tasks that differentially manipulated central executive, phonological storage/rehearsal, and visuospatial storage/rehearsal demands. Their results show that children with ADHD (a) place demands on central executive processing even with low cognitive loads, and (b) exceed storage/rehearsal capacity. Fabio et al. (2015) using the Merrill's (1992) procedure on automaticity with the dual task interference paradigm, confirm that subjects with ADHD showed deficits in auditory vigilance tests and became less careful when the interference was introduced. The results were discussed in terms of deficits in automaticity processes. Furthermore, Hurks et al. (2004) examined the performance of ADHD children on semantic category fluency (SCF) versus initial letter fluency (ILF) tasks. For each participant, word production was recorded for each 15-s time slice on each task. The authors hypothesized that children with ADHD perform significantly worse on both types of information processing (automatic and controlled) than do healthy control participants. The results were taken to

indicate that children with ADHD symptoms show a delay in the development of automating skills for processing abstract verbal information.

For some researchers (Assan & Azzam, 2012) the problem in ADHD arises even more periferically: in the sensorial integration of input stimuli. The neural systems bridging the gap between sensation and action provide the substrates for ‘intermediary’ or ‘integrative’ processing (Miller, Nielsen, Schoen, & Brett-Green, 2009). Sensory integration disorder 'SID' is a neurological disorder resulting from the brain's inability to integrate information received from the body's five basic sensory systems (vision, auditory, touch, olfaction, and taste), the sense of movement (vestibular), and/or the positional sense (proprioception). Sensory information is felt normally, but perceived abnormally affecting participation in functional daily life routines and activities (Bundy et al., 2002). Around 16 percent of the general population has symptoms of SID. In ADHD, the frequency of SID rises to 40 - 84% as reported in different studies (Ben-Sasson et al., 2009; Dunn & Bennett, 2002; Mulligan, 1996). Another study of Kim, Liu, Glizer, Tannock & Woltering (2013) investigated the neural and behavioral correlates of visual encoding during a working memory task in young adults with and without Attention-Deficit/Hyperactivity found neural differences between the groups. Specifically, as the hypothesis of the SIS does, it provides the first evidence of neural differences in the encoding stage of WM in young adults with ADHD, suggesting ineffective allocation of attentional resources involved in encoding of information in WM.

The impossibility to separate automatic and controlled factors

In this paragraph the impossibility to separate automatic and controlled factors, and new models on the conditional automaticity and attentional sensitization model of unconscious cognition coming from the literature of normally developing subjects will be presented. Barrouillet, Corbin, Dagry & Camos (2014) point out that some old theories regarding processing and storage supported the existence of distinct mechanisms and structures (Baddeley, 1986). On the other side, some new theories (Elliot, Baird & Giesbrecht, 2016) predict that controlled or conscioussness factors aren’t a all-or-none matters. In this paragraph we analyse how recent theorizing has renewed this independence hypothesis.

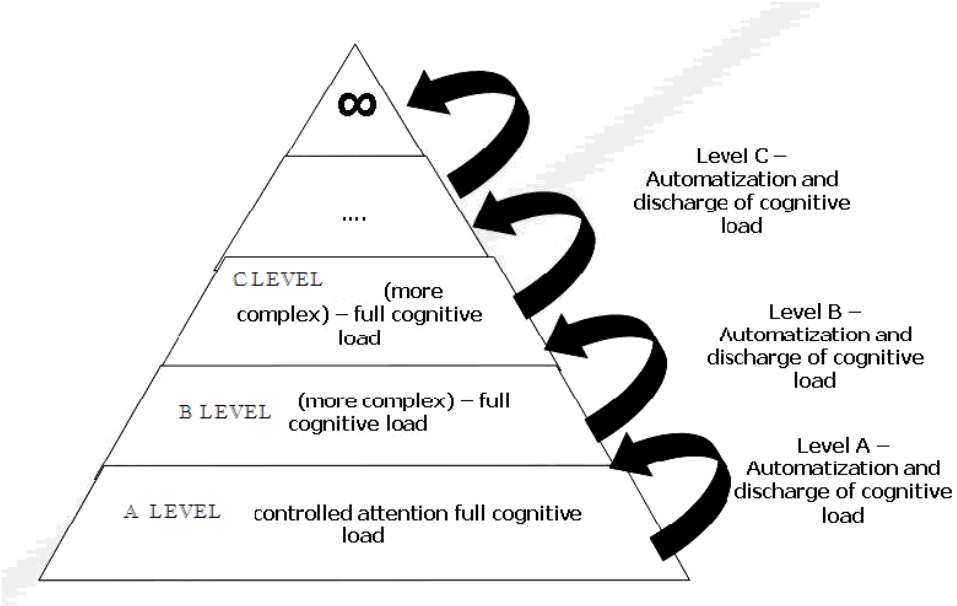
The continuum

It is not possible to separate the continuum from controlled to automatic processes. In any first step of a task, we use controlled processes of attention to learn, consequently the

performance is slow, awkward and prone to errors. The full amount of our memory load is engaged, in other words, all our cognitive resources are engaged to cope with new learning tasks. For example, we can think of a child learning to sum up two numbers, it is very difficult initially for the child: to bear in mind the first number, to memorize the second number, to recall the first and to sum up both. The child also has trouble understanding the meaning of the plus sign "to add", "to join"… but also "to enlarge", "to go on"… and so on. When the teacher asks the child to join Mary's toys with Mark's, the subject thinks long and hard, he does it slowly and finally solves the problem. During the problem solving, if someone asks something else the child can make mistakes in calculating and forget the anwser.

As training continues, performance requires less vigilance, and becomes faster, errors decrease; this transformation process can be defined as "automatization". By learning, the attentional strategies that once needed control, now become automatic. Returning to the above-mentioned child, as learning proceeds, becomes able to think of the plus sign faster and reaches the anwser easily. The subject also becomes able to reply to someone who asks something else. In other words the child automatizes learning of the plus sign. In the model below, we are positioned in the A level, when automatization appears and discharge of cognitive load on the A level takes place (fig. 1, Fabio, 2009).

Fig. 1 Fabio's model (2009).



Later the child has to learn how to multiply, and how to add and subtract. If the subject was totally (or even partially) engaged at the A level, it would be difficult to access to more complex tasks. The child can now have access to subtraction and adding (B level) thanks to the fact that the A level becomes an automatized subroutine (Fabio, 2009).

Again to solve the B level, the subject initially needs the controlled processes of attention and in consequence the performance is slow, awkward and prone to errors. The full amount of the working memory is engaged and also the cognitive resources are engaged to solve new learning.

As training proceeds, as previously seen, performance requires less vigilance, becomes faster and errors decrease, again automatization take place. In other words, with learning, the attentional strategies that once needed control become automatic. The child is now able to solve problems that need both subtraction and adding. Later more complex problem solving need to be learned incorporating 3 mathematical operations (C level). The B level that contains the A level becomes again a unique automatized subroutine thanks to a discharge of the cognitive load. So now the child can solve these more complex problems. And so on. The infinity symbol that stays at the top of the figure means that there is no limit to the possibility of having access to the increasing stages of complex thinking (Fabio, 2005). A discharge of the cognitive load of automatization happens as a continuum process. For this reason the dichotomy between “controlled” and “automatic” processing has been softened by use, so that “automaticity” may now be seen more as a matter of degree than as an all-or-none state (Elliott, Baird & Giesbrecht, 2016). As we know, all learning depends upon previous learning. Knowledge and ability develop in a hierarchical fashion; transfer from earlier learning facilitates the development of each new level. More complex forms of learning build on simpler forms of learning. When the habits, automatized skills, or cognitive structures that are prerequisite for some "new" learning have not been fully acquired, the capacity of the new learning will be impaired: learning will be retarded, inefficient, incomplete, or even impossible, depending upon the degree of inadequacy of prerequisite skills. Since learning builds on previous learning, weakness at any stage creates greater weakness in later stages, because subsequent learning depends upon transfer from prior learning.

From pure automatic process versus the conditional automaticity and versus attentional sensitization model of unconscious cognition

Kiefer (2012) point out the classical and the new theories of automatization. The core assumption of the classical view of executive control and automaticity, as we previously saw, is that executive control is exclusive to the domain of conscious cognition while unconscious, automatic processes are autonomous (Posner and Snyder, 1975; Schneider and Shiffrin, 1977) and this implies that a behavioural or neurophysiological effect has to be context-independent in order to index a "truly automatic" process (Pessoa et al., 2003). Such operational definitions of automaticity can be found in many areas of psychology and neuroscience such as object or face recognition (Pessoa et al., 2002; Wiese et al., 2008), action preparation (Bub & Masson, 2010), and emotional processing (Pessoa et al., 2002). However, it is difficult to identify processes that actually meet the classical criteria for automaticity because task demands frequently modulate behavioural and neurophysiological effects (Moors & De Houwer, 2006).

As almost all kinds of cognitive activity has to be classified as "controlled" according to classical criteria, the distinction between strategic and automatic processing becomes practically superfluous. In the view of Kiefer (2012) this renders the classical view of automaticity unsatisfactory. Furthermore, if unconscious, automatic processing were context-independent, this would result in a tremendous inflexibility of the cognitive system (Kiefer & Martens, 2010): conscious goal-directed various unconscious processes would massively influence information processing. Demands on conscious executive control would increase, because the intended action could only been ensured by inhibiting numerous interfering response tendencies induced by unconscious information processing (Botvinick, Braver, Barch, Carter, & Cohen, 2001). Refined theories of automaticity and unconscious processing allow for more flexibility and adaptability of unconscious, automatic processing (Neumann, 1990; Naccache, Blandin, & Dehaene, 2002; Moors & De Houwer, 2006; Kiefer, 2007; Kiefer & Martens, 2010). These theories posit that unconscious or automatic processing in general depends on a configuration of the cognitive system by attention and task sets. In his theory of direct parameter specification (DPS), Neumann (1990) proposes that unconscious information will only be processed and will influence the motor response to a target stimulus to the extent that it matches current intentions. Similarly, the global workspace model of consciousness by Dehaeneand and Naccache (2001) explicitly assumes that unconscious processes are susceptible to attentional amplification. Unlike classical theories, refined theories propose that executive control factors such as attention, intentions, and task sets orchestrate the unconscious processing streams toward greater optimization of task performance. Given this dependency on

the precise configuration of the cognitive system, the term “conditional automaticity” has been coined (Bargh, 1989; Logan, 1989).

Furthermore, Kiefer (2012), in contrast to classical theories, proposes attentional sensitization model of unconscious information processing that hypotized that unconscious processing is susceptible to executive control and is only elicited if the cognitive system is configured accordingly. He discusses that the assumption of attentional sensitization of unconscious information processing can accommodate conflicting findings regarding the automaticity of processes in many areas of cognition and emotion. In his model it is assumed that unconscious processing depends on the attentional amplification of task-congruent processing pathways as a function of task sets. Kiefer (2012) explains how executive control affect unconscious information processing. He introduces refined theories of automaticity with a particular focus on the attentional sensitization model of unconscious cognition that is specifically developed to account for various attentional influences on different types of unconscious information processing. In his view executive control influences unconscious cognition in the domains of visuo-motor and semantic processing: subliminal priming depends on attentional resources, is susceptible to stimulus expectations and is influenced by action intentions and task sets. This suggests that even unconscious processing is flexible and context-dependent as a function of higher-level executive control settings

Summing up the results of the critical relationship between automatic and controlled processes, it is shown that (1) Unconscious stimuli influence executive control settings in a bidirectional way. Several experiments showed that sub-liminal stimuli can modulate shifts of spatial (Ansorge et al., 2011; Scharlau and Ansorge, 2003) and modality-specific attention (Mattler, 2003, 2005) as well as task-specific control operations (Mattler, 2005, 2006) and task sets (Reuss et al., 2011; Wokke et al., 2011). (2) Furthermore, the relation between executive control and unconscious processing is bidirectional because top-down factors such as attentional resources, stimulus expectations, action intensions, or task sets, typically considered to involve executive control mechanisms (Norman and Shallice, 1986), modulate unconscious stimulus. Hence, these two lines of research suggest that executive control mechanisms interact with unconscious information processing in several ways and are thus not exclusive to the domain of conscious cognition (Kiefer et al., 2011). The same results come from the research on the neurological basis of automatic and controlled processes. Methods such as positron emission tomography (PET) and functional magnetic resonance imaging (fMRI) allow the

volumetric variations in regional cerebral blood flow that are related to cognitive activity to be indexed with millimetre-level spatial resolution.

In the next paragraph, research on automatic and controlled underlying neural structures will be presented, confirming the hypothesis that is impossible to separate the controlled and automatic processes, both at psychological and neural level.

Research on automatic and controlled processes underlying neural structures in ADHD

Classic views of controlled and automatic processing posit that different types of modes of elaboration are stored in different regions of the brain and undergo quite different processes.

This paragraph examines the current status of research on the neurological basis of automatic and controlled processes in ADHD. As Saling and Phillips (2007) point out, functional anatomical evidence supports the position that automatic processes are qualitatively different from controlled processes. As the authors point out, if automaticity involved faster processing, one would expect to see more processing as reflected by increased global cerebral activation with the acquisition of automaticity. Instead, there is a decrease in global activation or a shift in activation, particularly from cortical regions to subcortical areas. Thus, automatic processing is performed differently from controlled processing, apparently employing different, superior algorithms, which in some cases are explicit and in other cases are yet to be documented.

In line with Sailing and Philips (2007), also Satpute and Lieberman (2006) point out that reflexive systems correspond to automatic processes and include the amygdala, basal ganglia, ventromedial prefrontal cortex, dorsal anterior cingulate cortex, and lateral temporal cortex. Reflective systems correspond to controlled processes and include lateral prefrontal cortex, posterior parietal cortex, medial prefrontal cortex, rostral anterior cingulate cortex, and the hippocampus and surrounding medial temporal lobe region.

Luu, Tucker and Stripling (2007) also outlined that neuro imaging studies have suggested there may be direct neural correlates of the reduced demands for controlled processes, as evidenced by decreased demands on brain activity resulting from increasing practice with task performance.

Given that frontal lobe activity is thought to be particularly important to goal representations and providing control-related outputs (Miller & Cohen, 2001), it is theoretically important that both meta-analysis of fMRI studies and new experiments have suggested a specific decrease in frontal lobe activity (bilateral dorsal frontal, left ventral prefrontal, anterior cingulate cortex,

left insular regions) as participants become more practiced in task performance (Chein & Schneider, 2005).

The issue that the prefrontal cortex may be the only locus for top-down signal and controlled processes is open to doubt. As Miller, Erickson & Desimone (1996) point out, the prefrontal (PF) cortex is associated with a wide range of "executive'' functions critical for complex behaviour, such as problem solving, planning, selecting action, and working memory. Consistent with an "executive'' role in brain function are the extensive interconnections between the PF cortex and many other brain regions (Pandya & Yeterian, 1990; Pandya, Barnes, 1987; Cavada & Goldman-Rakic, 1989; Preuss & Goldman-Rakic, 1989; Webster, Bachevalier & Ungerleider, 1994). It should be noted, however, that the prefrontal cortex is unlikely either to be the only region involved in top-down control or to act alone. For example, some studies implicate frontostriatal loops in top down control of attention (Robbins & Rogers, 2000). Also in the view of Bonelli & Cumming (2007), the recent neuroanatomical, neuropsychological, and functional imaging literature, has made it increasingly clear that subcortical structures are also intimately involved in regulating higher cerebral processes that control cognition, decision-making, the planning of complex behavioural strategies, and neuropsychiatric symptoms (Houk, 2001; Seger, 2006). The frontal-subcortical circuitry provides a unifying framework for understanding the behavioural changes that accompany neurodegenerative disorders (Litvan, 2001). In the past three decades, a number of significant advances have been made in our understanding, not only of the neuroanatomy, but also of the neurophysiology and chemoarchitecture, of the frontalsubcortical circuits (Mayberg, 2001). Paralleling this new understanding, an increasingly broad spectrum of neuropsychiatric phenomenology is recognized as being interpretable in the context of frontal-subcortical circuit dysfunction.

Basal ganglia functional connectivity, based on a recent meta-analysis of 126 positron emission tomography (PET) and fMRI imaging publications, showed that patterns of functional connectivity between the cortex and the striatal nuclei are broadly consistent with the predictions of this classical parallel loop model. The function of the frontal lobes as an integrator of information, related both to the external sensory and internal limbic worlds and its role in motivation and appropriate motor response, make this region and its subcortical connections critically important to an understanding of both normal and disordered psychomotor functions. Cortical areas that are closely connected functionally appear to send

converging projections into adjacent regions of the striatum (August, Rothenberger, Baudewig, Roessner & Dechent, 2015). Information derived from the cortex is recombined at the striatal level to form small, functionally specialized domains.

In the same view of cortical-subcortical circuit, Lum, Conti-Ramsden, Page and Ullman (2012) point out that working memory is supported by multiple neural structures (D'Esposito, 2007). Prefrontal cortex, in particular dorsolateral prefrontal cortex, plays an important role in the central executive and attentional processes posited by Baddeley and Cowan (Curtis & D'Esposito, 2003; Wager & Smith, 2003). The basal ganglia also seem to play a role in these executive/attentional working memory functions (McNab & Klingberg, 2007; O'Reilly & Frank, 2006). An interesting proposal is that the connections from the basal ganglia to prefrontal cortex act as a gating system allowing information held in working memory to be updated with relevant information from long-term memory or from the environment (Frank, Loughry & O'Reilly, 2001; McNab & Klingberg, 2007). The storage of the information held in working memory seems to depend at least in part on Broca's area and left posterior parietal cortex for verbal information, and right parietal and occipital cortex for visuo-spatial information (Gathercole, 1999; Smith & Jonides, 1998).

Evidence has also been presented to suggest that procedural memory subserves the learning and use of rule-governed aspects of grammar, across syntax, morphology and phonology (Ullman & Pierpont, 2005). As we see above, learning in procedural memory is slower than in declarative memory; it proceeds gradually, as stimuli are repeated and skills practiced. However, once this knowledge has been acquired, skills can be executed rapidly. Although the neural bases of procedural memory are less well understood than those of declarative memory, evidence suggests that this system is supported by a network of brain structures that includes the basal ganglia, cerebellum and portions of frontal cortex, including premotor cortex and posterior parts of Broca's area (e.g., BA 44) (Gabrieli, 1998; Knowlton, Mangels, & Squire 1996; Robertson, Tormos, Maeda, & Pascual-Leone, 2001; Ullman, 2004; Ullman & Pierpont, 2005).

Neuropsychological and imaging studies indicate that ADHD is associated with alterations in prefrontal cortex (PFC) and its connections to striatum and cerebellum. Lesions to the PFC produce a profile of distractibility, forgetfulness, impulsivity, poor planning, and locomotor hyperactivity.

In addition to commonly reported disturbances in frontal and other cortical regions (Castellanos et al., 2002; Sowell et al., 2003; Shaw et al., 2007), the pathogenesis of ADHD is thought to involve anatomical and functional alterations of the basal ganglia and hippocampus, including the learning and memory functions that those regions support. The overall volumes of each of the basal ganglia nuclei (caudate, putamen, and Globus pallidus), for example, are reduced in ADHD relative to healthy comparison youth, but volumes of the putamen are particularly small (Sobel et al., 2010; Qiu, Tan, Zhou, Khong, 2008). The volume reductions are scattered throughout the spatial extent of each of the three basal ganglia nuclei, but they most prominently affect the ventral portions of these nuclei (Sobel et al., 2010), regions that anatomically connect and functionally interact with the limbic system, including the orbitofrontal cortex, amygdala, and nucleus accumbens (Nakano, Kayahara, Tsutsumi, & Ushiro, 2000).

Presumably, striatal connections to these limbic regions help to guide reinforcement-based learning (Pasupathy & Miller, 2005), and therefore striatal disturbances could account for the difficulties that ADHD youth have with delaying gratification and with selecting inappropriate behaviours for a given environmental context. Additional reductions in volume are located in the anterodorsal portions of these nuclei; portions that support habit learning and that connect with association cortices, including the frontal cortex, to support executive functioning. In addition, there is a correlation between smaller basal ganglia volumes and reduced activation of the basal ganglia in individuals with ADHD across a wide range of tasks (Cubillo, Halari, Smith, Taylor, & Rubia, 2012; Hart, Radua, Nakao, Mataix-Cols, & Rubia, 2013; Scheres, Milham, Knutson, & Castellanos, 2007) and across all ages, from early childhood to adulthood.

Striatal based learning are impaired in ADHD and may affect the neural loops based on it. Cortico-striato-thalamo-cortical (CSTC) loops are a series of parallel neural circuits projecting from the cortex to the striatum and thalamus, and then back to the cortex again (Alexander, DeLong & Strick, 1986; Alexander and Crutcher 1990; Maia et al. 2008; Wang et al. 2011). Multiple lines of evidence, including human neuroimaging studies and studies of animal models, suggest that impulsivity involves functional and anatomical abnormalities within the CSTC loops, particularly the cognitive and limbic CSTC loops (Casey et al., 1997; Cardinal et al., 2004; Marsh et al., 2009). Taken together, rs-fcMRI studies suggest atypical connectivity in both the cognitive and limbic CSTC loops in ADHD. Such findings lend support to a neuropsychological model of ADHD termed the “dual pathway model” (Sonuga-Barke, et al.

2008). This neuropsychological model suggests that for some children with ADHD, the principal neurocognitive deficit may reside in neural substrates underlying executive functions (such as the cognitive CSTC loop). These models are in line with the hypothesis of a deficit in automatic processes in ADHD people. Other recent research suggests that executive abilities emerge from cortico-cortical interactions between interlaminar prefrontal microcircuits, the posterior parietal cortex, and cortico-striatal-thalamo-cortical circuits (Opris et al., 2013). These prefrontal microcircuits also play a key role in the perception- to-action cycle that integrates relevant information about the environment and then environment, and then selects and enacts behavioural responses (Opris & Casanova, 2014).

The Cumulative and Emergent Automatic Deficit model (CEAD)

The analysis of the automatic and controlled models based on normally developing people in which we considered the impossibility to separate automatic and controlled factors, the new models on the conditional automaticity and attentional sensitization model of unconscious cognition and the literature on the underlying neural structures led us to deduct that EF weaknesses are neither necessary nor sufficient to explain all cases of ADHD.

In a detailed meta analysis, Willcutt et al. (2007) concludes that moderate effect sizes and lack of universality of EF deficits among individuals with ADHD lead us to believe that the difficulties with EF appear to be an important component of the complex neuropsychology of ADHD (Willcutt et al., 2005).

Combining evidence coming from both cognitive and neurophysiological research, it follows that a subtle deficit in the attention of ADHD subjects (as evidenced by lower processing speed in codify information) may cumulate and become more prominent, resulting in a deficit in automatic processes. A little deficit in attention can progressively be amplified in acquiring more complex concepts and use it in an easy way. So, these deficits in automatic processes can lead to a deficit in controlled processes in an emergent way (Fabio, 2009).

Each learning process can be seen as an internal map facilitating recognition through tags and smaller building-block schemes, and facilitating actions by linking triggers and actions (Holland, 1995). Physiologically schemes are distributed electro-chemical networks embedded in connections between neurons. As learning proceeds, there may be one or more schemes that evolve; in fact, it is possible that the schemes compete during the initial stages of learning, and a single dominant scheme eventually takes over. The cumulation of learning creates an

emergent process, a chunk, that is an automatized process and can be used by controlled processes in an efficient way because it reduces the amount of information to be held in memory (Fabio, 2009).

In ADHD the chunks are not well structured because of the inefficient access of outside and inside information (Rucklidge & Tannock, 2002; Shanahan et al., 2006; Willcutt et al., 2001a, 2001b, 2005b; Bush et al., 1999; Casey et al., 1997; Doyle, 2006; Pliszka et al., 2006; Rubia, et al., 2005; Vaidya et al., 2005). So these ineffective chunks delay the access to automatization and can't be efficiently used by controlled processes. The logic is that if basic processes are not well automatized, they will result in a high cognitive load and compete for limited resources used by executive functions. Chunking and transfer from earlier learning facilitate the development of each new level (Ulman, 2005). More complex forms of learning are based on automatization of previous ones. When this chunking, that is prerequisite for higher or new learning has not been fully automatized, the capacity of the new learning will be impaired. Weakness in automatization creates still greater weakness in later stages. In this study we explain this process through the Cumulative and Emergent Automatic Deficit model (CEAD model). It is similar to the Cumulative Automatic Deficit Hypothesis (CADH) of Ulman (2005) that proposes that learning problems in ADHD can be largely explained by abnormalities of brain structures underlying procedural memory and in particular, portions of frontal/basal-ganglia circuits (especially the caudate nucleus and the region around Broca's area) and the cerebellum. According to the CADH, these abnormalities should lead to impairments of various domains and functions that depend on these structures. Most importantly, procedural memory itself is predicted to be impaired, leading to deficits in other non-procedural functions that depend, at least in part, on such structures, including working memory. Unlike procedural memory deficits, which the CADH considers to be core deficits, impairments of other functions that depend on the same brain structures might or might not be observed, depending on the extent and nature of the underlying brain abnormalities (e.g., since different but parallel and anatomically proximate frontal/ basal-ganglia circuits may underlie procedural and working memory) (Fabio, 2015).

Flor and Dooley (1999) more in depth explain that the brain is able to chunk several distinct but interrelated ideas into a unique idea, and this produces access and processing time. Chunking is a very important process and may lead to significant gains in task performance. As seen in the above presented model, assuming the A level as an automatized subroutine means

performing a chunking operation in an emergent way. As we have seen before, the weakness in processing speed in ADHD has been well documented (Rucklidge & Tannock, 2002; Shanahan et al., 2006; Willcutt et al., 2001a, 2001b, 2005b; Bush et al., 1999; Casey et al., 1997; Doyle, 2006; Pliszka et al., 2006; Rubia, et al., 2005; Vaidya et al., 2005) and this delay in speed can explain the delay in automatization that leads to a load in controlled processes. Also McCabe, Roediger, McDaniel, Balota & Hambrick (2010) proposed that the development of automatic inhibitory control mechanisms may be involved in the development of intentional inhibitory control. Specifically, the normal development of automatic inhibitory control mechanisms of attention may serve as the cognitive precursors to intentional inhibitory control mechanisms that are assumed to develop later (Aksan & Koschanska, 2004). So, there is a lot of evidence supporting the dependence of intentional inhibitory control mechanisms and vice versa.

Conclusion

In this study the current status of research on a neurological and neuropsychological bases of automatic and controlled processes was presented.

Two questions were posed. The first was whether the prefrontal cortex is the only locus for top-down signal and controlled processes; the second was if it is the only factor responsible for ADHD deficits. The analysis of automatic and controlled models based on normally developing subjects showed that it is impossible to separate automatic and controlled factors, furthermore the neural structures of the cortico-subcortical neural loops led us to conclude that there is an inextricable relationship between controlled and automatic processes and that the prefrontal cortex is not the only locus for top-down signals. With reference to the second question we demonstrated that, since the strict relationship between controlled and automatic processes, EF weaknesses are neither necessary nor sufficient to explain all cases of ADHD. The model here presented, the cumulative emergent automatic deficit model, proposes that learning problems in ADHD can be largely explained by abnormalities of the input phase that lead to inaccurate automatic processing, again, leading to the deficit in controlled processes. In this pathway the lack of automatization is linked to the fact that in the ADHD learning process the chunking is weakened and so it is not available for more complex learning.

Some important directions for future studies can emerge: the first is that if the cumulative deficit is the basis of the rationale for ADHD, then compensatory education can reverse

deprivation effects and carry performance levels up to national-norm expectations; thus more potent interventions along the lines discussed will be necessary (Deutsch, 2010). So, it would seem reasonable to conclude that if automatization of the level of underlying abilities determines a decline in performance, an improvement of these skills through an enrichment program at the preschool and kindergarten levels may be helpful in arresting or reversing the cumulative deficit (Deutsch, 1967). The second is that we have to change the precise type of cognitive rehabilitation: the focus has to be changed on automatization abilities and not only be based on executive function rehabilitation.

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