The Neuropsychological Deficits and Neuropathology of Impulsivity as Manifested in
Attention-Deficit/Hyperactivity Disorder

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Abstract

Two of the core neuropsychological deficits in impulsivity that characterize
Attention-Deficit/Hyperactivity Disorder (ADHD) involve deficient reward
processing and deficient behavioral inhibition. Hence, it is valuable to have a
solid understanding of these deficits and their underlying neuropathology to have
a proper understanding of ADHD. This review provides such an understanding
and concludes by providing implications for differential diagnosis of different
types of ADHD impulsivity.

The importance of impulsivity as a risk factor for the development of antisocial behavior
has been firmly established by evidence that has implicated impulsivity in the etiology of all the
major externalizing disorders[1] of Diagnostic and Statistical Manual of Mental Disorders-5
(DSM-5, American Psychiatric Association, 2013; Beauchaine & McNulty, 2013; Zisner &
Beauchaine, 2015). Hence a basic grasp of the neuropsychological deficits in impulsivity and
their neuropathology is important for understanding how it increases the risk for the development
of antisocial behavior. Impulsivity has a range of definitions that often includes an inability to
control or regulate behaviors and emotions as one element (Dias et al., 2015; Nigg, in press) with
Attention-Deficit/Hyperactivity Disorder (ADHD) being the paradigmatic disorder of behavioral
and emotional impulsivity (Barkley, 2015a; Nigg, in press). Therefore, the neuropsychology and
neuropathology of impulsivity will be discussed primarily in terms of its paradigmatic
manifestation—ADHD. Note that the goal of this article is to focus on the two
neuropsychological deficits which are most clearly and directly linked to ADHD impulsivity and
therefore is not meant to be an exhaustive review of all the neuropsychological deficits and their
underlying neuropathology that can possibly contribute to ADHD impulsivity. For example,
doubtlessly a deficit in attention can indirectly impair the ability to regulate impulse control to
the extent that it impairs the ability to shift focus away from a stimulus that can trigger an
impulsive act (Nigg, in press).

Although ADHD, along with all other conditions in the DSM-5, is considered as a
diagnostic category, there is a strong consensus that ADHD, as well as many other childhood
disorders, is best understood in dimensional rather than categorical terms (Ahmad & Hinshaw,
2015; Nigg & Barkley, 2014; Roberts, Milich, & Barkley, 2015). Namely, the presentations of
ADHD are best understood as representing two distinct, albeit highly-related spectra or
dimensions of inattention/disorganization and hyperactivity/impulsivity on which individuals

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[1] The externalizing disorders are: Oppositional Defiant Disorder, Conduct Disorder, Substance Use Disorder, and
Antisocial Personality Disorder (Beauchaine & McNulty, 2013).

differ (Ahmad & Hinshaw, 2015; Nigg & Barkley, 2014; Roberts et al., 2015). Thus ADHD can be conceptualized as both a diagnostic category and set of behavioral dimensions (Ahmad & Hinshaw, 2015). In this article, ADHD will always designate the hyperactive/impulsive and combined forms of ADHD, as these are most closely associated with the development of antisocial behavior (Ahmad & Hinshaw, 2015; Willcutt et al., 2012).

Neuropsychological Deficits

Theories of ADHD's core neuropsychological deficits can be classified into two major groups. These groups are commonly designated "bottom-up theories" or "top-down theories". Alternately, Nigg's (in press) two process model of impulsivity refers to these theories as Type 1 (i.e., bottom-up) or Type II (i.e., "top-down") regulatory processes and their neural instantiation, respectively. This chapter will adopt the Nigg two process model as the basic framework for discussing the neuropsychology and neurobiology of ADHD because it is arguably the most current, comprehensive model of disturbances in the regulation of impulsivity in ADHD and enjoys widespread research support (Ahmad & Hinshaw, 2015; Arnsten & Rubia, 2012; Castellanos-Ryan & Seguin, 2015; Nigg, in press; Nigg & Barkley, 2014; Willcutt, 2015; Zisner & Beauchaine, 2015). In this model, impulsivity has two major aspects. The first aspect views impulsivity as the result of faulty decision making in which optimal decision making is defined as multi-stage of choosing a particular action among a number of alternative options to achieve the most beneficial outcome. Most of the research focuses on the faulty decision making of impulsive individuals. This defective decision making is thought to result from failures termed *temporal or delay discounting* of rewards in that poor immediate rewards are impulsively chosen over superior but delayed rewards. This aspect of impulsivity involves Type I regulatory processes which refer to processes for automatic handling of information that are *reactive, automatic, and reward motivated.* Impulsivity is caused by deficits in reward processing functions served primarily by bottom-up subcortical structures that are functionally connected to frontal brain areas. The subcortical structures include the basal ganglia, limbic system, thalamus, hypothalamus, and cerebellum.

The second aspect of impulsivity focuses on how impulses are contained, stopped or interrupted and in which an impulsive action is seen as a hasty, stimulus driven action. This aspect of impulsivity is caused by impairments in Type II regulatory processes that are *effortful, deliberate, and goal-motivated* and thus can over-ride stimulus-triggered responses so as to allow regulation of focus and behavior. Impulsivity is caused by deficits in top-down cognitive control/executive functions served primarily by the prefrontal cortex (PFC) and the anterior cingulated cortex (ACC) and their connections to other cortical and subcortical areas[2] (Ahmad & Hinshaw, 2015; Bridgett, Burt, Deater-Deckard, & Edwards, 2015; Castellanos-Ryan & Seguin, 2015; Nigg, in press; Nigg & Barkley, 2014; Zisner & Beauchaine, 2015). These two processes interact continuously throughout development and, indeed, may even be thought of as two aspects of a single system (Nigg, in press). Because Type I processes mature more rapidly than Type II processes with the result that impulsivity is initially more a function of Type I than Type

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[2] The specific cortical and subcortical structures involved in ADHD will be discussed in the section on neuropathology.

II deficits (Nigg, in press; Zisner & Beauchaine, 2015), the Type I deficit in reward processing will be discussed first, followed by a discussion of the Type II deficit in inhibitory control.

Deficient Reward Processing

Beginning with the work of Douglas (1988), a strong consensus has emerged that individuals with ADHD have an undersensitivity to reinforcing stimuli which triggers impulsivity in two major ways (Ahmad & Hinshaw, 2015; Nigg, in press; Willcutt, 2015; Zisner & Beauchaine, 2015). First, this undersensitivity leads to temporal discounting of reward in that individuals with ADHD find the experience in delay of reinforcement so aversive that they tend to impulsively choose immediate rewards even when an alternative option would result in larger reward after a longer delay. Second, this undersensitivity leads to excessive, impulsive reward seeking as individuals with ADHD "experience relatively low hedonic value from pleasurable stimuli and therefore seek more frequent extreme incentives to derive hedonic payoff" (Zisner & Beauchaine, 2015, p. 10). In short, undersensitivity to stimuli that most individuals would find reinforcing results in individuals with ADHD having chronic feelings of anhedonia which they find aversive. To alleviate such feelings, they tend to impulsively seek immediate and frequent reinforcement/gratification.

Deficient Inhibitory Control

In the neuropsychological literature, the regulation of attention, behavior, and emotion is commonly understood within the conceptual framework of an executive functions model (EFs) (Barkley, 2015b). EFs refer to the brain's management (i.e., executive) system which enables self-control over time to accomplish goals (Barkley, 2015b; Brown, 2013). Self-control is required when you "have to concentrate and think, when acting on your initial impulse might be ill advised" (Diamond, 2013, p. 136). Among EF's control functions (there may be as many as six, Willcutt, 2015), there is virtual unanimity that a deficit in one function— response inhibition— is a major cause of impaired self-control in ADHD (Barkley, 2015b; Diamond, 2013; Nigg, in press; Posner, Park, & Wang, 2014; Willcutt, 2015). In Barkley's (2015b, p. 409) conceptualization response inhibition refers to three overlapping yet distinctive processes, the most important of which he defines as "Inhibiting the initial prepotent (dominant) response to an event so as to create a delay in responding." It is the most important because without a delay (stopping) in the prepotent response the possibility of thoughtful, appropriate goal-directed behavior in a situation is greatly reduced if not rendered impossible. EFs such as inhibition form the foundation for reasoning, problem solving, and planning that are crucial to the successful attainment of future goals (Barkley, 2015b; Diamond, 2013) and are of critical importance for successful functioning as they affect every major domain in life (Diamond, 2013).

Neuropathology

The understanding of the neuropathology of ADHD, as well as other disorders, has evolved from a perspective in which the disorder stems not from circumscribed anomalies in discreet brain regions, but rather from impairments in distributed neural networks (Posner, Park, & Wang, 2014). These impairments have been investigated primarily by two neuroimaging techniques: structural and functional neuroimaging (Ahmad & Hinshaw, 2015). Structural neuroimaging typically consists of magnetic resonance imaging techniques that measure regional and whole brain volume, surface, and surface contour while diffusion tensor imaging is used to

examine white matter structural connectivity. Functional neuroimaging techniques typically consist of positron emission tomography, single photon emission computed tomography, and functional magnetic resonance imaging that assess brain metabolism and infer brain activation by changes in cerebral blood flow.[3]

Neuropathology of Deficient Reward Processing

A compelling model that explains the neuropathology of deficient reward processing in ADHD has been comprehensively articulated by Zisner and Beauchaine (2015). In this model, deficient reward processing because of reward insensitivity is theorized to be caused by a "bottom-up" deficit in the functioning of mesolimbic dopamine circuit. Dopamine (DA) is the monoamine neurotransmitter associated with this circuit that is crucial for the incentive salience component of the reward response (Berridge & Kringelbach, 2015; Zisner and Beauchaine, 2015).[4] Namely, DA motivates the pursuit of rewards by attributing incentive salience/attractiveness to reward-related stimuli thereby prompting a "wanting", a motivation to seek the reward. DA projections that ascend from the midbrain can be divided into three or four neural circuits, with the mesolimbic circuit being the one most closely associated with motivation, incentive salience, and behavioral impulsivity (Zisner & Beauchaine, 2015). Cell bodies in the mesolimbic circuit originate in the ventral tegmental area and project primarily to the ventral striatum, including the nucleus accumbens and the caudate nucleus, and also to the amygdala (Zisner and Beauchaine, 2015). This mesolimbic circuit has commonly been referred to as the brain's reward or pleasure circuit because many diverse pleasures/rewards (e.g., food, sex, addictive drugs, listening to music) activate this network (Berridge & Kringelbach, 2015). Evidence for dysfunction in this circuit in ADHD comes from numerous neuroimaging studies indicating hypo-responding in reward processing tasks in the ventral striatum (Plichta & Scheres, 2014). In addition, this dysfunctional responding of ADHD individuals is further reflected in findings that these individuals also display chronically low tonic (at rest) dopamine levels and blunted phasic dopamine responses to incentives (Zisner & Beauchaine, 2015). Research by Volkow and colleagues (2009) supports that this diminished DA reactivity to incentives may be due to the markedly deficient number of dopamine receptors and transporters in the mesolimbic circuit of individuals with ADHD. This study may also explain the finding of abnormally higher short-ranged connectivity in the reward circuit in children with ADHD as compared with typically developing children (Tomasi & Volkow, 2012). Namely, the lower dopaminergic function in ADHD might cause higher spontaneous activity and increased short range connectivity (Tomasi & Volkow, 2012). Lastly, it should also be noted that although there may be a number of other specific neural circuit profiles of abnormal reward functional connectivity in ADHD which can lead to impulsive decision making (Dias et al., 2015), a dysfunction in the mesolimbic dopamine circuit is the one most directly related to reward insensitivity.

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[3] For the reader interested in a thorough discussion of these techniques which is beyond the scope and space of this chapter, see Wilde, Ayoub, Bigler, Hunter, and Levin (2014).

[4] See Berridge (2007) and Berridge and Kringelbach (2015) for a comprehensive discussion of the causal role of dopamine in the brain's reward system which finds that, contrary to prior thinking, dopamine does not mediate the hedonic impact of a reward (i.e.,"liking") but the incentive salience value of the reward (i.e., "wanting").

Neuropathology of Deficient Inhibitory Control

There is a remarkable convergence in neuroscience studies that the neuropathology of deficient inhibitory control involves structural and functional impairments in several neural circuits. The prefrontal cortical structures (PFC) and the anterior cingulate gyrus provide top down regulatory control through connections with posterior (parietal) cortical and subcortical structures[5] (Arnsten & Rubia, 2012; Barkley, 2015c; Castellanos-Ryan & Sequin, 2015; Nigg, in press; Posner et al., 2014; Rubia, Alegria, Brinson, 2014). The prefrontal cortex comprises several functional substructures of the brain's frontal lobes. The main substructures are the dorsolateral PFC; ventromedial PFC; ventrolateral PFC; the orbitofrontal PFC; and the inferior frontal cortex (Arnsten & Rubia, 2012; Castellanos-Ryan & Seguin, 2015). Each of these substructures are hypothesized to be somewhat differentially involved in top down regulatory control (see Arnsten & Rubia, 2012, for a thorough discussion). In sum, "the PFC is positioned to orchestrate all aspects of behavior" (Arnsten & Rubia, 2012, p. 357).

Evidence for impairment in these circuits in ADHD comes from numerous structural and functional neuroimaging studies. Structural imaging studies, beginning with Castellanos and colleagues (2002), have found that children with ADHD have significantly smaller overall brain volumes, including the PFC and ACC, than comparison children (Ahmad & Hinshaw, 2015; Rubia et al., 2014). More recently, diffusion tensor imaging (DTI) studies of ADHD have found widespread altered white matter in multiple brain regions indicative of impaired connectivity in these circuits, with the most common finding pertaining to the fronto-striatal circuitries (Ahmad & Hinshaw, 2015). In addition, studies have found cortical thickness (grey matter) abnormalities in subcortical limbic regions such as the insula, amygdala, and thalamus (Rubia et al., 2014). Because there are extensive connections between these regions and the PFC and ACC, these abnormalities provide additional evidence for impairments in the fronto-striatal circuitries (Rubia et al., 2014). Furthermore, functional imaging studies in various task-based studies[6] of ADHD have found reduced responding (e.g., hypoactivation as indicated by reduced blood flow) in these circuits thus implicating hypoconnectivity and therefore deficient functioning (Ahmad & Hinshaw, 2015; Castellanos-Ryan & Sequin, 2015; Hong et al., 2014; Nigg, in press; Posner et al., 2014; Rubia et al., 2014).

Lastly it should be noted that although most studies of the neuropathology of ADHD have implicated the prefrontal and striatal regions and their interconnections, i.e., the "frontal-striatal model of ADHD," impairments in other brain regions and their connections are also implicated in ADHD (Shenton, Kubicki, & Makris, 2014). For example, recently Hong et al. (2014) using DTI showed altered white matter connectivity involving 23 regions (in particular the cerebellum.

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[5] The subcortical structures include the: basal ganglia; limbic system (amygdala, hippocampus, cingulate gryus); thalamus; hypothalamus; cerebellum.

[6] For example, one commonly used task is the **Go/No-Go** task (Castellanos-Ryan & Sequin, 2015) in which stimuli are presented in a continuous stream and participants perform a binary decision on each stimulus. One of the outcomes requires participants to make a motor response (**go**), whereas the other requires participants to withhold a response (**no-go**).

Conclusion

This review discussed two of the core neuropsychological deficits and their underlying neuropathology that contribute to the development of impulsivity as manifested in its extreme form in ADHD. The identification of such deficits is indicative of the progress that is being made in addressing the "perennial and seemingly intractable nosological problem for ADHD" (Nigg, 2015, p. 10) of establishing subtypes anchored in neural circuitry as has been proposed by the NIMH initiative on Research Domain Criteria (Insel, et al., 2010). And thus, for example, it may be possible to differentiate highly impulsive ADHD children in terms of whether or not the primary problem is involving a Type I processing problem affecting reward sensitivity or a Type II processing problem affecting response inhibition. Recent research by Nigg and colleagues (2015) illustrates that such clinical applications may not be too far off from realization.

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