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Developmental pathways to attention-deficit/hyperactivity disorder and disruptive behavior disorders: Investigating the impact of the stress response on executive functioning

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HIGHLIGHTS

- Integrated research areas to examine a proposed pathway to ADHD and DBD
- Non-optimal stress results in sub-optimal cognitive performance
- Atypical stress response is associated with symptoms of ADHD, DBD, and aggression
- Atypical response likely linked to deficits in executive functioning

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ABSTRACT

A current theory suggests multiple pathways to the onset of attention-deficit/hyperactivity disorder (ADHD) and comorbid oppositional defiant disorder or conduct disorder, proposing that heterogeneous factors lead to various patterns of behavior, cognitive impairments, and even physiological signs which are categorized as ADHD and comorbid disorders. This review focused on one proposed pathway to the onset of ADHD and ODD/CD in order to examine how low physiological arousal, as indicated by atypical hypothalamic–pituitary–adrenal axis and sympathetic adrenomedullary functioning, might be associated with cognitive impairment. First, the cognitive deficits associated with ADHD and disruptive behavior disorders were reviewed. In order to understand the atypical response, studies of the typical stress response and its relationship to cognition, particularly executive functioning, were then examined. Finally, this review summarized findings of an atypical stress response among children with ADHD and ODD/CD. Review of the literature led to the conclusion that the theorized pathway may be improved by taking into account the effects of stress on executive functioning given that an atypical stress response would likely be associated with impairment in this area. Future research directions needed to advance our understanding of the relationship between low arousal, ADHD, and ODD/CD were highlighted.

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1. Introduction

Since its formation as a diagnostic category in the 1960s and subsequent evolution from "Hyperkinetic Reaction of Childhood" to attention-deficit/hyperactivity disorder (ADHD), ADHD has been the subject of much scrutiny and extensive research (Barkley, 2006). Recent findings suggest that ADHD, currently defined as a childhood disorder of inattention and hyperactivity/impulsivity, is associated with an array of neurological, cognitive, and behavioral impairments (Nigg, 2006; Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005). Although a number of etiological mechanisms have been proposed, no clear pathway to ADHD has yet been established (Nigg, 2006, 2012; Sonuga-Barke, 2005). Multiple pathway models provide the best current theoretical models of how the ADHD symptom profile and associated comorbidities, such as disruptive behavior disorders (DBDs), which include conduct disorder and oppositional defiant disorder, arise (Castellanos, Sonuga-Barke, Milham, & Tannock, 2006; Nigg, Goldsmith, & Sachek, 2004; Sonuga-Barke, 2005). The constellation of symptoms associated with ADHD and the lack of a single neuropsychological or biological profile for the disorder suggests that a single model of ADHD development is unlikely to explain its onset in most cases (Fair, Bathula, Nikolas, & Nigg, 2012; Nigg, Willcutt, Doyle, & Sonuga-Barke, 2005; Sonuga-Barke, 2005).

Nigg et al. (2004) multiple pathway construal of disorder development is based upon the idea that particular temperaments early in life give rise to disordered behaviors (e.g., ADHD and comorbid disorders). In the first of these pathways, Nigg et al. (2004) theorizes that early negative temperament (i.e., anger) results in later problems in regulation that then may lead to ADHD-C (ADHD-Combined type per DSM-IV) and comorbid DBD along with minimal executive dysfunction, particularly in executive attention (i.e., being able to maintain attention in a goal-directed fashion through the use of working memory) (Nigg & Huang-Pollock, 2003). A second pathway is posited to lead to primary deficits in executive functioning as well as possible DBD comorbidity through early dysfunction in regulatory abilities (Nigg et al., 2004) The third and final pathway proposed to lead to conduct disorder and comorbid ADHD posits a developmental trajectory focused on low arousal or withdrawal related behaviors associated with subsequent serious conduct problems (i.e., aggression and late onset of conduct-related behaviors) as well as low physiological arousal/diminished anxiety (Nigg et al., 2004). Although Nigg and colleagues speculate that three different pathways could lead to the development of ADHD and comorbid DBDs, only the latter of the three is of especial interest in this review.

This review will focus on the low arousal pathway to ADHD development, a pathway that bears similarity to the theory of ADHD proposed by Barkley (1997) which was built in part upon Quay's theory (1997) first published in 1988. Quay's theory stated that the central deficit in ADHD was an underactive behavioral inhibition system (BIS), which is a motivational system associated with responding to punishment, lack of reward, and fear/anxiety provoking stimuli and the physiological stress response. In testing this theory, others have noted that if the BIS is impaired, individuals with ADHD would be expected to exhibit deficient HPA axis functioning as well as deficits in response inhibition, which should lead to deficits in executive functioning more broadly (e.g., King, Barkley, & Barrett, 1998; Randazzo, Dockray, & Susman, 2008; van West, Claes, & Deboutte, 2009). Although Nigg and colleagues' low arousal pathway and Barkley's theory of response inhibition share a proposed dysfunctional biological mechanism, the two theories appear to differ in the hypothesized outcomes of this mechanism; Barkley's theory proposes a central outcome of deficient response inhibition and executive functioning whereas Nigg's theory proposes primarily conduct problems and aggression.

The intent of this review is to determine whether low physiological arousal should be considered a central component of a pathway to ADHD leading to primary executive functioning deficits as proposed by Barkley or, as theorized by Nigg, does low arousal not necessarily lead to these deficits. In order to fulfill this aim, the current review will examine cognitive deficits associated with ADHD and DBDs, the typical stress response, the impact of stress on executive functioning, and the nature of the stress response among those with ADHD and comorbid DBDs. This review will also synthesize findings and suggest future directions.

2. ADHD & disruptive behavior disorders: deficits in executive functioning

2.1. ADHD and executive functioning

One line of research to be evaluated when examining these pathways is that of cognitive deficits among children with ADHD. The constellation of cognitive deficits attributable to ADHD points to impairment in executive functioning as one of the main deficits associated with the disorder. Executive functions, also known as goal-directed behaviors, govern an important array of cognitive abilities including one's ability to plan and organize information, to retain information for a brief period of time, to switch between tasks, and to maintain task focus (Barkley, 1997; Willcutt et al., 2005). Among the executive functions, spatial working memory, response inhibition, and vigilance showed the most profound and widely replicated deficits (Willcutt et al., 2005). Findings of impaired working memory, defined as one's ability to temporarily store and process information needed for goal-directed decision-making, among children with ADHD have been corroborated by a meta-analysis of studies comparing working memory performance between children with ADHD and children without the disorder (Martinussen, Hayden, Hogg-Johnson, & Tannock, 2005).

Despite convincing evidence of executive functioning deficits, particularly working memory impairment, within the ADHD population, these deficits are not found across all individuals with the disorder (Nigg et al., 2005). As postulated by Nigg et al. (2004), executive functioning deficits may not be a significant component of the pathway to ADHD development in all cases. More recent research suggests that the specific types of executive functioning deficits, when present, vary within the population of individuals with ADHD in a similar fashion to the variation within the typically developing population (Fair et al., 2012). For example, some individuals exhibit deficient working