Mechanisms Underlying the Impact of "Cold"/"Hot" Executive Function Deficits on Core Symptoms in Children with ADHD

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Date: 2023-07-05T00:00:00+00:00

Abstract

Attention-Deficit/Hyperactivity Disorder (ADHD) is a neurodevelopmental disorder characterized by attention deficit and/or hyperactivity and impulsivity as core symptoms, which is closely associated with executive function deficits resulting from abnormal prefrontal cortex development. Based on this, the neurocognitive-behavioral developmental pathway proposes that executive function deficits may represent the cognitive-level pathological mechanism underlying the core symptoms of ADHD, wherein "cool" executive function deficits associated with the dorsolateral prefrontal cortex may be the dominant factor contributing to attention deficit core symptoms, while "hot" executive function deficits associated with the ventromedial prefrontal cortex may be the dominant factor contributing to hyperactivity and impulsivity core symptoms. On the one hand, "cool" executive function deficits primarily lead to failures in working memory representation maintenance, insufficient inhibitory control, and difficulties in cognitive switching, which further restrict individuals in sustained attention, selective attention, and attentional shifting. On the other hand, "hot" executive function deficits bring about problems such as delay aversion, abnormal reward processing, and motivational dysregulation, causing failures in behavioral inhibition and making individuals more prone to impulsive choices, thereby manifesting hyperactivity, impulsivity, and other core symptoms. Future research should further examine and refine theoretical models of how "cool" and "hot" executive function deficits influence ADHD core symptoms and provide more empirical evidence at the cognitive-neural level, while also investigating the interactive effects of "cool" and "hot" executive function deficits on ADHD core symptoms from an ecological perspective, and developing personalized, precise, and long-term effective intervention programs for ADHD core symptoms based on executive function.

Full Text

Preamble

Manuscript Number: jz23-076
Title: The Mechanism by Which "Cool"/"Hot" Executive Function Deficits Affect Core Symptoms in Children with ADHD
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Abstract: Attention-deficit/hyperactivity disorder (ADHD) is a neurodevelopmental disorder characterized by inattention and/or hyperactivity-impulsivity as core symptoms, closely associated with executive function deficits stemming from abnormal prefrontal cortex development. Based on the neuro-cognitive-behavioral developmental pathway, we propose that executive function deficits may represent the cognitive-level pathogenesis underlying ADHD core symptoms. Specifically, "cool" executive function deficits related to the dorsal prefrontal cortex may be the dominant factor causing inattention, while "hot" executive function deficits associated with the ventromedial prefrontal cortex may be the primary contributor to hyperactivity-impulsivity. On one hand, "cool" executive function deficits primarily lead to failures in working memory representation maintenance, insufficient inhibitory control, and difficulties in cognitive switching, which further restrict sustained attention, attentional selection, and attentional shifting. On the other hand, "hot" executive function deficits result in problems such as delay aversion, abnormal reward processing, and motivational dysregulation, leading to behavioral inhibition failures and increased likelihood of impulsive decision-making, thereby manifesting as hyperactivity-impulsivity core symptoms. Future research should further examine and refine theoretical models of how "cool" and "hot" executive function deficits influence ADHD core symptoms, provide more empirical evidence at the cognitive-neural level, investigate the interactive effects of these deficits on ADHD symptoms in ecological contexts, and develop personalized, precise, and long-lasting interventions targeting executive functions to alleviate ADHD core symptoms.

Keywords: Attention-deficit/hyperactivity disorder, executive function deficits, core symptoms, prefrontal cortex, mechanism
Classification Code: B845

Attention-deficit/hyperactivity disorder (ADHD) is a neurodevelopmental disorder originating in childhood, characterized by inattention and/or hyperactivity-impulsivity that are inconsistent with developmental level, accompanied by impairments in academic or social functioning (American Psychiatric Association, 2013). The global prevalence rate among children reaches 7.2% (Sayal et al., 2018; Wolraich et al., 2019), while the latest prevalence in China is 6.4% (Li et al., 2022). Notably, 50.9% of children with ADHD experience persistent symptoms into adulthood (Li et al., 2020; Agnew-Blais et al., 2016; Franke et

al., 2018; Sibley et al., 2016), with adverse effects spanning the entire lifespan (Faraone et al., 2015; Forte et al., 2020; Nigg et al., 2020; Retz et al., 2021). Executive function deficits are considered the primary cognitive impairment in ADHD (Kofler et al., 2019; Silverstein et al., 2020), serving not only as sensitive indicators for predicting disease progression, assessment, diagnosis, and intervention, but also as major factors hindering academic performance and social adaptation in children with ADHD. While the etiology and pathogenesis of ADHD remain unclear, increasing research indicates that ADHD core symptoms are closely related to abnormal brain structure and functional connectivity (Faraone et al., 2021; Hoogman et al., 2017). However, the underlying psychological mechanisms through which neural abnormalities lead to core symptoms in children with ADHD remain poorly understood (Hinshaw, 2018; Mueller et al., 2017; Sonuga-Barke et al., 2022), particularly regarding how executive function deficits—fundamental cognitive impairments and endophenotypes associated with abnormal prefrontal development—affect the emergence of different ADHD core symptoms. Therefore, investigating the pathogenesis of ADHD from the perspective of executive function deficits and attempting to build a cognitive bridge between neurodevelopmental abnormalities and core symptoms holds significant scientific and practical value for early identification, diagnosis, and effective clinical intervention of this disorder.

Based on differential prefrontal functions, executive functions can be divided into "cool" and "hot" components (Zelazo & Carlson, 2012; Zelazo & Müller, 2012). "Cool" executive functions primarily involve brain regions such as the dorsolateral prefrontal cortex and ventrolateral prefrontal cortex, focusing on relatively abstract, decontextualized cognitive processing, including working memory, inhibitory control, and cognitive flexibility. In contrast, "hot" executive functions refer to cognitive processing under high emotional/motivational involvement, including delay of gratification and affective decision-making, associated with brain regions such as the ventromedial prefrontal cortex and orbitofrontal cortex (Antonini et al., 2015; Crone & Steinbeis, 2017; Zelazo & Carlson, 2012). The dual-pathway model of ADHD proposes two independent pathways leading to ADHD: a cognitive control pathway related to the mesocortical dopamine system, and a motivational development pathway related to the mesolimbic dopamine reward circuit (Sonuga-Barke, 2002; Sonuga-Barke, 2003; Shen et al., 2020). Although this theory has not explicitly articulated the relationship between executive function deficits and different ADHD core symptoms, it provides valuable insights for considering the pathogenesis of ADHD's two core symptoms from the perspective of "cool" and "hot" executive function deficits. Further evidence suggests that inattention in ADHD children stems more from "cool" executive function deficits, while hyperactivity-impulsivity is primarily associated with "hot" executive function deficits (Castellanos & Aoki, 2016; Shakehnia et al., 2021). Thus, "cool" and "hot" executive function deficits may represent independent pathways leading to ADHD's two core symptoms, resulting in different behavioral manifestations and developmental outcomes (Landis et al., 2021; Mueller et al., 2017; Pauli-Pott et al., 2019). From a

developmental psychopathology perspective, ADHD is a neurodevelopmental disorder in which early neurodevelopmental processes are disrupted through gene-environment interactions, leading to cognitive deficits or impairments that further increase the risk of ADHD, including the manifestation of core symptoms (McLaughlin, 2016; Zelazo, 2020). Therefore, executive function deficits may be the cognitive mechanism through which neurodevelopmental abnormalities lead to ADHD core symptoms. It is necessary to explore from a multi-level neuro-cognitive-behavioral perspective how "cool" and "hot" executive function deficits affect the two core symptoms in children with ADHD, to further elucidate the nature and pathogenesis of ADHD and provide evidence-based support for personalized and precise intervention.

Recent research on the relationship between executive function deficits and ADHD core symptoms has developed rapidly (Groves et al., 2022; Karalunas et al., 2021; Kofler et al., 2019; Shakehnia et al., 2021; Silverstein et al., 2020). However, comprehensive and in-depth analysis of the specific mechanisms through which executive function deficits affect different ADHD core symptoms remains lacking. Based on this, the present paper uses "cool" and "hot" executive functions as the main thread to: (1) explain why executive function deficits may be the cognitive-level pathogenesis of ADHD core symptoms; (2) discuss how executive function deficits affect the two core symptoms in children with ADHD; (3) elaborate on the potential mechanisms through which subcomponent deficits of "cool" and "hot" executive functions affect ADHD core symptoms; and (4) propose future research directions regarding the relationship between executive function deficits and ADHD core symptoms.

2 Executive Function Deficits as Behavioral Characteristics or Pathogenic Mechanisms in ADHD?

Executive function refers to a series of top-down processes that coordinate and control thoughts, behaviors, and emotions to achieve goal-directed outcomes (Diamond, 2013; Zelazo, 2015). Strong evidence indicates that executive function deficits are prevalent in ADHD populations and can serve as clinical diagnostic criteria (Barkley, 1997; Faraone et al., 2015; Kofler et al., 2019; Nigg et al., 2020). Current research on ADHD executive function deficits has primarily focused on behavioral characteristics or developmental patterns, treating these deficits as external behavioral manifestations of ADHD core symptoms while neglecting their critical role in ADHD developmental trajectories. Particularly when explaining the emergence of ADHD core symptoms, it remains unclear whether executive function deficits represent behavioral characteristics or pathogenic mechanisms in children with ADHD. The Research Domain Criteria (RDoC) initiative by the National Institute of Mental Health integrates multiple disciplines (e.g., genetics, neuroscience, psychological behavior) to provide a comprehensive framework for understanding the pathogenesis of psychiatric disorders at psychological and neurobiological levels rather than solely based on behavioral symptoms. Therefore, when comprehensively and systematically

explaining ADHD core symptoms and exploring their pathogenesis, the RDoC framework should be considered in conjunction with neuro, cognitive, and behavioral levels.

Specifically, the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) explicitly states that ADHD is a neurodevelopmental disorder with abnormalities in both brain structure and functional connectivity (Cortese et al., 2021; Hoogman, 2020; Hoogman et al., 2019; Thapar et al., 2017). Particularly, delayed or insufficient development of the prefrontal cortex in children with ADHD not only constitutes the physiological basis for core symptoms but is also closely related to executive function deficits (Shaw et al., 2011; Shaw et al., 2013; Yasumura et al., 2019). Meta-analytic studies have found that ADHD patients show sustained abnormal activation in frontal regions during executive function tasks (including Stop-Signal, Go/No-Go, Stroop, and N-back paradigms) (Hart et al., 2013; Lukito et al., 2020; Norman et al., 2017). Beyond prefrontal structural and functional abnormalities, abnormal brain network function is also a strong influencing factor for ADHD core symptoms (Cai et al., 2018; Sripada et al., 2014). The prefrontal-centered executive control network and its functional connectivity generally show weakened characteristics in ADHD patients and are significantly correlated with the severity of inattention symptoms (Cai et al., 2021; Ryan et al., 2022). From a neurobiological perspective, ADHD core symptoms are caused by abnormal changes in brain structure and function, particularly abnormalities in prefrontal cortex and executive control network function related to executive functions. Meanwhile, as a fundamental cognitive ability, executive function top-down participates in, guides, or coordinates other cognitive processes and plays a decisive role in future academic performance, intentional behavior, and social adaptation (Spiegel et al., 2021; Zelazo, 2015). Insufficient development or deficits in executive function not only directly cause abnormal daily life manifestations and the emergence of inattention, hyperactivity, and impulsivity; if left unaddressed, these deficits can, in turn, affect brain structural development and functional plasticity during growth, thereby reinforcing neural-level abnormalities and further exacerbating ADHD core symptoms. Therefore, evidence from neuro and cognitive levels suggests that ADHD is a neurodevelopmental disorder, particularly manifested in delayed development of prefrontal cortex related to executive functions and abnormal functional connectivity of the executive control network, resulting in insufficient executive function development (cognitive deficits) and subsequently leading to inattention and/or hyperactivity-impulsivity core symptoms.

As shown in Figure 1 [Figure 1: see original paper], from the perspective of the developmental pathway from neural abnormalities to cognitive deficits to behavioral characteristics, executive function deficits may be the cognitive mechanism underlying neural abnormalities leading to ADHD core symptoms, serving as a cognitive bridge between neurodevelopmental abnormalities and core symptoms. In summary, following the RDoC framework and combining developmental psychopathology pathways, we infer that executive function deficits are not only behavioral characteristics of children with ADHD but, more importantly, may

be the pathogenic mechanism at the cognitive level leading to ADHD core symptoms. Why can executive function deficits affect ADHD core symptoms, and do "cool" and "hot" executive function components affect different core symptoms? The following discussion will focus on how executive function deficits affect different core symptoms in children with ADHD.

3 Effects of "Cool" and "Hot" Executive Function Deficits on Different ADHD Core Symptoms

The main core symptoms of ADHD include inattention and hyperactivity-impulsivity (DSM-5, APA, 2013). Inattention typically manifests as difficulty sustaining attention, carelessness, distractibility, and lack of organization, specifically 体现在 difficulty concentrating, vulnerability to interference, difficulty shifting attention, and narrowed attention span. Hyperactivity-impulsivity typically manifests as fidgeting, constant activity, difficulty waiting, and interrupting others, specifically 体现在 difficulty inhibiting behavior, excessive activity, seeking immediate rewards, and emotional dysregulation (see Table 1).

To explain these abnormal behavioral manifestations in children with ADHD, the dual-pathway model proposes two independent pathways leading to ADHD: a cognitive control pathway and a motivational development pathway (Sonuga-Barke, 2002). On one hand, insufficient inhibitory control related to dorsal prefrontal-striatal circuits causes cognitive and behavioral dysregulation in ADHD children. On the other hand, deficits in delay of gratification related to orbitofrontal-striatal reward circuits lead to motivational dysregulation (Shen et al., 2020; Zhu et al., 2018). Although the dual-pathway model has not explicitly articulated the relationship between executive function deficits and different ADHD core symptoms, subsequent researchers generally believe that the two pathways correspond closely to "cool" and "hot" executive function deficits, respectively, with "cool" deficits corresponding to the cognitive control pathway and "hot" deficits corresponding to the motivational development pathway (Zhang et al., 2010; Geurts et al., 2006; Jiang et al., 2018). Thus, the emergence of abnormal behavioral manifestations in ADHD children implies impairment in related cognitive abilities, particularly "cool" and "hot" executive function deficits.

So why can executive function deficits affect different ADHD core symptoms? This question can be addressed by analyzing the neurobiological and cognitive effects of "cool" and "hot" executive function deficits on different ADHD core symptoms. First, "cool" executive function involves brain regions including the dorsolateral prefrontal cortex, ventrolateral prefrontal cortex, and dorsal anterior cingulate cortex, engaging in top-down cognitive control processes. "Hot" executive function is associated with brain regions such as the ventromedial prefrontal cortex, orbitofrontal cortex, and ventral anterior cingulate cortex, primarily involving cognitive processing with motivational or emotional components (Crone & Steinbeis, 2017; Moriguchi, 2022; Zelazo & Carlson, 2012).

Extensive research indicates that ADHD children exhibit "cool" executive function deficits (Barkley, 1997; Kofler et al., 2019; Pievsky & McGrath, 2018). Subcomponents of "cool" executive function, such as working memory, inhibitory control, and cognitive flexibility, have all been shown to relate to ADHD core symptoms (Kofler, Harmon, et al., 2018; Mueller et al., 2017). Moreover, studies have found that "cool" executive function deficits have a greater impact on ADHD inattention core symptoms (Irwin et al., 2021; Shakehnia et al., 2021; Silverstein et al., 2020). For example, working memory deficits lead to carelessness and forgetfulness; inhibitory control failures may cause distractibility and inattention; cognitive flexibility deficits may result in lack of organization and difficulty shifting attention. Additionally, key brain regions involved in "cool" executive function are also core nodes of the dorsal attention network (e.g., dorsolateral prefrontal cortex), participating in intentional attention processing (Lemire-Rodger et al., 2019; Petersen & Posner, 2012). Besides "cool" executive function deficits, abnormalities in the attention network further exacerbate ADHD inattention core symptoms. Therefore, "cool" executive function deficits affect ADHD inattention core symptoms through both insufficient abilities and abnormal neurobiological substrates (dorsal attention network).

Second, ADHD children also show significant "hot" executive function deficits. "Hot" executive function refers to the ability for flexible evaluation and risk decision-making under high emotional or motivational involvement, including delay of gratification, reward processing, and emotional regulation. These deficits are considered related to ADHD hyperactivity-impulsivity core symptoms (Colonna et al., 2022; Dekkers et al., 2016; Shakehnia et al., 2021; Tegelbeckers et al., 2018). For example, ADHD children's difficulty inhibiting behavior, seeking immediate rewards, and showing emotional dysregulation in daily life are likely caused by "hot" executive function deficits (Bunford et al., 2022; Colonna et al., 2022; Petrovic & Castellanos, 2016). Brain regions related to "hot" executive function are also key areas of the reward network (e.g., orbitofrontal cortex, ventromedial prefrontal cortex), involved in reward processing and self-emotion regulation (Cubillo et al., 2012; Yang et al., 2019). Studies have found imbalanced connectivity between the reward network and other brain regions in ADHD children, particularly decreased functional connectivity with the executive control network and related brain regions, which may be the neural mechanism underlying hyperactivity-impulsivity core symptoms (Dias et al., 2015; von Rhein et al., 2017). Therefore, ADHD hyperactivity-impulsivity core symptoms may result from both insufficient abilities manifested by "hot" executive function deficits and their abnormal neurobiological substrates.

In summary, combining theoretical and empirical research, this paper proposes that "cool" and "hot" executive function deficits may be two cognitive pathways leading to different ADHD core symptoms. Specifically, "cool" executive function deficits related to dorsolateral prefrontal abnormalities cause insufficient abilities when facing tasks (e.g., difficulty resisting interference, inability to maintain memory representations), leading to attention control failures.

"Hot" executive function deficits related to ventromedial prefrontal abnormalities cause problems such as delay aversion and reward abnormalities, manifesting as behavioral inhibition failures and motivational dysregulation. Meanwhile, the abnormal neurobiological substrates of executive function deficits are also key nodes or regions of core brain networks (e.g., dorsal attention network, reward network) involved in attention processing and reward handling. Abnormalities in these brain networks further affect individuals' attention control and behavioral inhibition abilities, jointly causing the emergence of different ADHD core symptoms of inattention and hyperactivity-impulsivity (as shown in Figure 2 [Figure 2: see original paper]).

4 Mechanisms of "Cool"/"Hot" Executive Function Deficits Affecting ADHD Core Symptoms

The previous section proposed that "cool" and "hot" executive functions may be two cognitive pathways leading to different ADHD core symptoms, producing different behavioral manifestations and developmental outcomes. "Cool" executive function deficits related to the dorsolateral prefrontal cortex may be the dominant factor for ADHD inattention core symptoms, while "hot" executive function deficits related to the ventromedial prefrontal cortex may be the main contributor to ADHD hyperactivity-impulsivity core symptoms. This section will elaborate on how deficits in subcomponents of "cool" and "hot" executive functions affect the emergence of ADHD core symptoms and their specific mechanisms.

4.1 Mechanisms of "Cool" Executive Function Subcomponent Deficits Affecting ADHD Inattention Core Symptoms

"Cool" executive functions regulate attention through corresponding abilities to control behavior, making it more adaptive, planned, and focused when solving problems (Zelazo, 2020). Deficits in "cool" executive functions lead to failures in goal-directed behavior and attention control, specifically manifested in sustained attention, selection, and allocation. "Cool" executive functions typically include three core subcomponents: working memory, inhibitory control, and cognitive flexibility (Diamond, 2013; Miyake et al., 2000). The following discussion will address the specific mechanisms through which these three subcomponents affect ADHD inattention core symptoms.

(1) Working Memory Affects ADHD Inattention Core Symptoms
Working memory refers to a limited-capacity system for temporarily storing and processing information during cognitive task performance (Baddeley, 2012). It includes not only the episodic buffer responsible for temporarily storing and integrating memory representations but also the central executive system responsible for inhibiting irrelevant information and guiding attention to target information, which is closely related to selective attention (Ahmed & de Fockert, 2012; Luck & Vogel, 2013). On one hand, information representations in

working memory top-down capture target stimuli, thereby guiding attentional selection. On the other hand, the central executive system helps ignore or inhibit irrelevant information, selectively allocating resources to target-related stimuli and facilitating attentional selection (Gazzaley & Nobre, 2012; Greene et al., 2015). Additionally, the frontoparietal network is a key region in the working memory system, and its activation promotes the maintenance and processing of target information (Lamichhane et al., 2020; Soto et al., 2014). Research has found that the frontoparietal network is involved in both working memory representation maintenance and attention control processes (Bahmani et al., 2019; Wallis et al., 2015), particularly dorsolateral prefrontal activation related to attentional selection in working memory (Panichello & Buschman, 2021; Quentin et al., 2019). Therefore, it is reasonable to infer that working memory deficits in ADHD children affect attentional selection processes through both working memory representations and the central executive system.

Indeed, research has shown that working memory deficits are significant cognitive impairments in ADHD children and are the strongest predictor of inattention core symptoms (Fosco et al., 2020; Irwin et al., 2021; Ramos et al., 2020). Specifically, ADHD children lack the ability to store and maintain memory representations over time, making them more likely to lose key information needed for task completion and unable to select correct target information. Central executive system deficits make it difficult for ADHD children to inhibit irrelevant information, increasing cognitive control load and limiting top-down attentional control (Jacobson et al., 2011; Kofler, Soto, et al., 2020; Re et al., 2016). In this situation, ADHD children in classrooms easily shift attention to internal thoughts or external stimuli unrelated to classroom content, showing deficits related to selective attention (Rapport et al., 2009). Neuroimaging studies have also found that brain regions with abnormal activation during working memory tasks in ADHD children concentrate in the dorsolateral prefrontal cortex, medial inferior frontal gyrus, cingulate cortex, and parietal lobe, which are also involved in attentional selection and control processes (Ko et al., 2013; Samea et al., 2019). Luo et al. (2019) used a visual working memory paradigm to elicit classic attention processes (including attention selection-maintenance-matching phases), using N2pc (posterior contralateral N2) and CDA (contralateral delay activity) components as indicators. Through simultaneous EEG-behavior analysis, they effectively distinguished attention selection from working memory maintenance processes. Results showed that selective attention and working memory deficits were closely related in ADHD patients; when memory load increased, working memory in ADHD patients was difficult to maintain, leading to decreased attentional selection efficiency (Luo et al., 2019). Overall, the mechanism by which working memory deficits affect ADHD inattention core symptoms is mainly manifested in difficulty maintaining working memory representations and top-down control failures of the central executive system, which are detrimental to attentional selection processes, resulting in abnormal behavioral manifestations such as vulnerability to interference and inattention.

(2) Inhibitory Control Affects ADHD Inattention Core Symptoms
Inhibitory control refers to the ability to intentionally suppress strong internal/external interfering information to make correct responses to targets (Aron et al., 2004). Based on different interfering stimuli, it can be divided into response inhibition (behavioral-level inhibition) and conflict control (interference inhibition, cognitive-level inhibition) (Diamond, 2013). Response inhibition refers to suppressing inappropriate behavioral responses to external interfering information and maintaining attention on target information. Conflict control, also called interference inhibition, mainly refers to suppressing conflicting information in the brain while maintaining correct responses (Hung et al., 2018). In other words, if inhibitory control is deficient, individuals will have difficulty suppressing attention or behavioral responses to irrelevant stimuli (e.g., external distractors, internal irrelevant thoughts), limiting attentional sustainability. Most studies have found that ADHD children have difficulties with inhibitory control. For example, in response inhibition tasks like Stop-Signal and Go/No-Go, ADHD children show longer reaction times and higher error rates (Fosco et al., 2019; Hart et al., 2013). In interference control tasks like Stroop and Simon, ADHD children show poorer reaction times and accuracy under incongruent conditions (Borella et al., 2013; Mullane et al., 2009). The close relationship between inhibitory control and ADHD inattention core symptoms is widely recognized (Janssen et al., 2018; Mueller et al., 2017). Behavioral studies have found that response inhibition deficits in childhood predict the development of inattention symptoms in adolescence, and insufficient inhibition makes the age-related improvement of inattention symptoms less pronounced (DeRonda et al., 2021). From a neuroimaging perspective, ADHD patients show abnormal activation in the right inferior frontal gyrus, parietal lobe, and striatum during response inhibition tasks (Thornton et al., 2018; van Rooij et al., 2015). The inferior frontal gyrus is involved not only in initiating and processing inhibitory signals but also in maintaining attentional control through connections with the ventral attention network and temporoparietal junction. The parietal region mainly participates in attentional reorienting and task goal maintenance during response inhibition (Cai & Leung, 2011; Fabio & Urso, 2014). Additionally, ADHD patients show lower connectivity within response inhibition networks, which is significantly correlated with ADHD core symptom severity (van Rooij et al., 2015). Cai et al. (2021) found that effective connectivity between the dorsal anterior cingulate cortex and ventrolateral prefrontal cortex evoked by Go/No-Go tasks not only significantly correlated with response inhibition ability but also predicted the severity of inattention deficits in ADHD children. This provides neural-level evidence for further explaining how response inhibition affects ADHD inattention. In summary, poor response inhibition and associated attention network impairments can better explain why ADHD children have difficulty maintaining attention and concentrating.

However, it is worth noting that current research uses concepts like “inhibition” or “response inhibition” to represent inhibitory control uniformly, without distinguishing between response inhibition and conflict control (interference

inhibition), which is insufficiently comprehensive and precise for explaining how inhibitory control deficits specifically affect core symptoms. Conflict control (interference inhibition) as an active control ability at the cognitive level plays an important role in processing unreasonable information in the brain, especially in overcoming cognitive conflicts. Some studies suggest that ADHD children have weaker conflict control abilities, making them unable to maintain and protect task goals from interference when facing subconscious or consciously induced cognitive conflicts (Borella et al., 2013; Mueller et al., 2017). In other words, due to lack of cognitive conflict control, ADHD children are more vulnerable to interference from internal distracting thoughts and have difficulty maintaining attention during task execution. Brain regions mainly activated during conflict control include the dorsal anterior cingulate cortex, dorsolateral prefrontal cortex, parietal regions, and dorsolateral frontal control system (Hung et al., 2018). The dorsolateral prefrontal cortex mainly participates in maintaining and manipulating task-relevant information, while the dorsal anterior cingulate cortex plays a greater role in regulating behavioral adaptation and persistence (Kolling et al., 2016; Sheth et al., 2012). Therefore, conflict control may also be a key factor in maintaining attentional persistence. Given the unique role of conflict control in inhibitory ability, more attention should be paid to how conflict control deficits affect ADHD inattention core symptoms. Future research should distinguish different types of inhibitory control and conduct more detailed investigations of different inhibitory ability impairments in ADHD inattention.

(3) Cognitive Flexibility Affects ADHD Inattention Core Symptoms
Cognitive flexibility, also called shifting, refers to the ability to quickly and effectively switch between different mental contexts (Diamond, 2013). Cognitive flexibility essentially reflects individuals' abilities in inhibitory control and cognitive switching, suppressing dominant but ineffective cues, efficiently reconfiguring resources, and participating in the development of attentional shifting abilities (Dajani & Uddin, 2015; Filippetti & Krumm, 2020). As a core component of executive function, ADHD children have cognitive flexibility deficits and show reduced activation in the left inferior frontal gyrus, prefrontal cortex, and bilateral anterior insula during cognitive flexibility tasks (Roshani et al., 2020; Rubia et al., 2010). When summarizing impaired cognitive domains in ADHD, researchers have categorized cognitive flexibility into domains related to inattention impairment and proposed that cognitive flexibility deficits may cause impaired goal selection and cognitive control, leading to inattention core symptoms (Mueller et al., 2017). Luna-Rodriguez et al. (2018) investigated attentional shifting abilities in ADHD patients during switching tasks and found that increased switching costs might lead to insufficient attentional shifting and allocation to different stimuli in ADHD. However, empirical research on the relationship between cognitive flexibility and ADHD inattention core symptoms is limited. By definition, cognitive flexibility refers more to the ability to flexibly switch cognitive modes according to task demands. If underdeveloped, individuals have difficulty shifting from one task to another, challenging the effective-

ness of attentional shifting and showing behavioral problems such as difficulty allocating and shifting attention (Wendt et al., 2018). Therefore, ADHD children's cognitive flexibility deficits can affect their inattention core symptoms by influencing attentional shifting and allocation. Additionally, considering that cognitive flexibility is built upon working memory and inhibitory control development, using more sensitive tools or paradigms to assess cognitive flexibility and distinguishing it from other executive function components is important for explaining how cognitive flexibility affects inattention.

(4) Verifying Mechanisms from a Causal Manipulation Perspective
The previous sections preliminarily explained how "cool" executive functions affect ADHD inattention core symptoms from cognitive and neurobiological perspectives, but lack support from causal manipulation viewpoints. The following will provide empirical evidence from cognitive (or near-causal) and neuromodulation perspectives to verify the specific mechanisms through which "cool" executive functions affect inattention core symptoms.

Cognitive interventions primarily improve ADHD children's attention performance by directly training different components of "cool" executive functions. Working memory training has been confirmed as a promising intervention for improving ADHD children's inattention and academic difficulties (Kofler, Wells, et al., 2020; Wiest et al., 2022). Central Executive Training (CET) is a computer-based intervention targeting working memory deficits in ADHD children. Studies have found that CET not only significantly improves working memory and inhibitory control scores but also durably improves academic performance. Further analysis revealed that academic improvement was achieved primarily by enhancing attention behavior in the classroom (Kofler, Sarver, et al., 2018; Singh et al., 2022). Researchers also used Central Working Memory Training (CWMT) to intervene in ADHD children's working memory and found that ADHD children receiving CWMT showed working memory improvement, enhanced attentional persistence and selectivity, and improved academic performance (Ackermann et al., 2018; Gray et al., 2012). Additionally, executive function training related to inhibitory control and cognitive flexibility can also significantly improve ADHD attention performance. For example, Kofler et al. (2020) conducted a four-week computer-based cognitive training using Go/No-Go tasks for 8-12-year-old ADHD children and found sustained improvement in attention ability post-intervention. AKL-T01 (Akili Interactive Labs-T01) is an adaptive digital intervention program based on a game interface, adapted from inhibitory control and cognitive flexibility tasks. After four weeks of AKL-T01 cognitive intervention, parent-reported and objective behavioral indicators of attention in ADHD children significantly improved (Davis et al., 2018; Kollins et al., 2020). In summary, cognitive interventions manipulate different executive function components such as working memory, inhibitory control, and cognitive flexibility, and examine changes in ADHD children's attention ability before and after intervention, thereby providing causal (or near-causal) evidence to support and verify the mechanisms through which "cool" executive functions

affect ADHD inattention core symptoms.

Neuromodulation regulates cortical excitability by stimulating brain regions related to "cool" executive functions, such as the dorsolateral prefrontal cortex (DLPFC), changing brain function and thereby improving executive function and inattention core symptoms in ADHD patients. A recent large-scale randomized double-blind experiment found that using transcranial direct current stimulation (tDCS) with anodal stimulation of the right DLPFC and cathodal stimulation of the left DLPFC for more than four weeks significantly improved inattention symptoms in ADHD adults (Leffa et al., 2022). Nejati et al. (2021) applied single-session anodal tDCS to the right DLPFC in 9-10-year-old ADHD children and found that activating the right DLPFC helped improve inhibitory control, with stimulation effects positively correlated with ADHD severity (Nejati et al., 2021). Additionally, a three-week (15 sessions) transcranial magnetic stimulation (TMS) study of 62 ADHD adults found that stimulating the right PFC significantly improved inattention and working memory, with enhanced activation in attention-related brain regions post-intervention during working memory tasks. That is, stimulating the right PFC can enhance attention-related brain activity and improve attentional behavioral performance (Bleich-Cohen et al., 2021). Furthermore, studies have found that six weeks of high-frequency stimulation of the right DLPFC in 7-12-year-old ADHD children helped improve both inattention and hyperactivity-impulsivity core symptoms (Cao et al., 2019). In summary, using neuromodulation techniques to stimulate DLPFC and other regions in ADHD patients can improve "cool" executive function task performance and effectively ameliorate inattention clinical symptoms, further providing neural-level evidence from a causal manipulation perspective for the mechanisms through which executive function deficits affect inattention. Currently, neuromodulation technology for treating ADHD patients is in a rapid development stage, but there are still no unified standards for stimulation sites, treatment parameters (frequency/intensity), or stimulation duration, with considerable variability in clinical effects (Westwood et al., 2021). Most studies have found that neuromodulation of DLPFC significantly affects "cool" executive functions in ADHD patients, but less attention has been paid to how improvements in "cool" executive functions ameliorate inattention. Notably, TMS or tDCS can also directly stimulate core nodes of structural or functional connections to induce changes in "downstream" neural activity, including cortical and subcortical brain network functional connectivity (Sydnor et al., 2022). That is, stimulating an individual's DLPFC also activates or inhibits other cortical or subcortical brain networks to varying degrees, jointly affecting behavioral performance. From this perspective, we can further reveal the cognitive-neural mechanisms through which stimulating "cool" executive function-related brain regions improves attention.

4.2 Mechanisms of "Hot" Executive Function Subcomponent Deficits Affecting ADHD Hyperactivity-Impulsivity Core Symptoms

"Hot" executive function refers to the ability for flexible evaluation and risk decision-making under high emotional or motivational involvement, including delay of gratification, reward processing, and emotional regulation. Deficits lead to behavioral control failures and motivational dysregulation in emotional or social contexts, specifically manifested as delay aversion, seeking immediate rewards, and motivational dysregulation. Researchers increasingly recognize that "hot" executive function deficits may be the dominant factor for ADHD hyperactivity-impulsivity core symptoms. The following discussion addresses how abnormalities in different subcomponents of "hot" executive function affect ADHD hyperactivity-impulsivity core symptoms.

(1) Delay of Gratification Affects ADHD Hyperactivity-Impulsivity Core Symptoms Delay of gratification refers to the decision-making orientation of voluntarily forgoing immediate satisfaction for more valuable long-term goals, and the self-control demonstrated during the waiting process (Mischel et al., 1989). Temporally, delay of gratification includes at least two stages: "making a choice" and "persisting with the choice" (Ren et al., 2015). The former involves value evaluation and decision-making among options, while the latter involves resisting temptation and executing intended behavior. The famous "marshmallow test" measures children's delay of gratification ability (Mischel et al., 1989). In such experiments, children can choose immediate rewards (e.g., a marshmallow) or wait for a period to receive double rewards. Delay of gratification helps children weigh immediate versus future benefits, make optimal choices, and maintain behavior consistent with their choice to maximize benefits. ADHD children show poor delay of gratification ability, which is considered closely related to their impulsive behavioral manifestations (Sonuga-Barke et al., 2010; Van Dessel et al., 2019). Examining the two stages, ADHD children in the "making a choice" stage tend to assign lower value to future benefits, show obvious aversion to waiting time, and make more impulsive, reckless choices. Even when making advantageous choices, ADHD children in the "persisting" stage often cannot resist immediate temptation, fail at behavioral inhibition, and show impulsive behaviors like "changing their mind midway." Delay of gratification is ubiquitous in daily life, such as waiting in lines or doing homework before playing. However, due to insufficient delay of gratification ability, ADHD children show difficulty waiting and aversion to delay in daily situations (Utsumi et al., 2016; Van Dessel et al., 2019).

Existing evidence supports these views. A meta-analysis of 22 studies found that ADHD children and adolescents overall showed higher delay discounting in delay discounting tasks (Patros et al., 2016), with ADHD children more likely to choose immediate satisfaction and show stronger impulsive behavior (Martinelli et al., 2017). Additionally, researchers found that ADHD individuals experience more subjective negative emotions during delay waiting, showing obvious delay

aversion (Rosch & Mostofsky, 2016). Neuroimaging studies further found that delay aversion is related to hyperactivation in brain regions associated with negative emotion processing (e.g., amygdala), with stronger responses as delay time increases (Van Dessel et al., 2018; Van Dessel et al., 2020). Besides amygdala hyperactivation, ADHD children also show excessive spontaneous neural activity (low-frequency oscillations) during waiting, which is related to impulsive decision-making characteristics (Hsu et al., 2015). The "persisting" stage of delay of gratification more reflects individuals' self-control ability for goal-directed behavior, involving brain regions such as dorsolateral prefrontal cortex, ventrolateral prefrontal cortex, and posterior parietal lobe, which are also related to impulsive behavior (Dalley & Ersche, 2019). Failure of self-control in ADHD children not only affects their ability to persist but also leads to more externalizing problem behaviors. In summary, poor delay of gratification leads ADHD children to prefer immediate satisfaction and show delay aversion in the "making a choice" stage, and to fail at resisting conflicts from immediate temptation and behavioral control in the "persisting" stage. These two factors jointly affect the emergence of impulsive behavior in ADHD.

(2) Reward Processing Affects ADHD Hyperactivity-Impulsivity Core Symptoms Increasingly, researchers consider abnormal reward processing as an important factor affecting ADHD hyperactivity-impulsivity core symptoms (Bunford et al., 2022; Kallweit et al., 2021; Tegelbeckers et al., 2018). Reward processing can be divided into two stages: reward anticipation and outcome response (Rademacher et al., 2010). On one hand, reward anticipation refers to an individual's waiting and craving state for upcoming potential rewards. In life, once individuals notice reward-predictive stimuli, they assign higher expected outcome value and promote goal-directed behavior. Impulsive behavior is often driven by strong reward anticipation, with such individuals having higher reward sensitivity (Xiong & Sun, 2017; Dawe et al., 2004). Especially, ADHD children have higher reward sensitivity to certain stimuli (e.g., money) (Fosco et al., 2015; Tripp & Wickens, 2012), making them more likely to ignore threat cues and adverse consequences, unable to inhibit drive caused by reward anticipation, and producing impulsive behavior. On the other hand, the outcome response stage refers to evaluating obtained rewards and monitoring whether behavior achieves task goals. In this stage, individuals establish behavioral associations with reward cues based on perceived reward feedback (Yang et al., 2015). However, ADHD children often show abnormal responses to reward outcomes. For example, ADHD patients assign higher value to "salient" rewards like money and power, while losing interest in lower "return" rewards like smiles or beautiful scenery (Demurie et al., 2011; Gonzalez-Gadea et al., 2016). This dysregulated reward response makes individuals dependent on "distorted" reward associations; once "salient" reward cues appear, ADHD individuals show more urgent seeking responses (i.e., impulsivity). Over time, most reward responses become weakened, with only "higher value" reward stimuli producing sufficient signals. Without

intervention, this abnormal reward processing mechanism leads ADHD individuals to continuously pursue more stimulating risky activities (e.g., speeding, gambling) and is key to the development and maintenance of substance abuse and addiction (Grimm et al., 2021). Therefore, ADHD children often show motivation dysregulation driven by reward anticipation and failure in reward cue-behavior associations, making them constantly seek more frequent external stimulation, which can explain why ADHD children are more hyperactive and impulsive than their peers.

Numerous studies indicate that abnormal reward processing in ADHD has a neurobiological basis: structural and functional abnormalities in the brain reward circuit (von Rhein et al., 2017; Zhu et al., 2018). Previous research consistently found that during monetary reward processing tasks, ADHD patients show insufficient activation in the ventral striatum during reward anticipation (Plichta & Scheres, 2014; Scheres et al., 2007; Van Hulst et al., 2017), and this decreased anticipation of reward cues leads ADHD individuals to prefer immediate rewards. Studies also found that the orbitofrontal cortex is hyperactivated in ADHD patients during reward anticipation, with activation levels significantly positively correlated with hyperactivity-impulsivity core symptoms. It is speculated that orbitofrontal hyperactivation prevents ADHD individuals from correctly encoding expected outcome value, leading to more maladaptive behaviors (Tegelbeckers et al., 2018). Additionally, the most common reward abnormality in ADHD children is increased delay discounting, i.e., preferring smaller immediate rewards over larger delayed rewards (Jackson & MacKillop, 2016; Marx et al., 2021; Yu & Sonuga-Barke, 2020). Under delay discounting tasks, studies found abnormal functional connectivity (imbalance) between the reward network and frontoparietal network in ADHD children, which may be the neural mechanism behind hyperactivity-impulsivity core symptoms (Dias et al., 2015). ADHD individuals also show strong negative emotions toward delayed rewards, with higher delay discounting and delay aversion (negative emotions) jointly exacerbating impulsive behavioral manifestations (Van Dessel et al., 2018). Based on the above cognitive and neural evidence, it can be inferred that abnormal reward processing mainly affects hyperactivity-impulsivity levels in ADHD individuals through high reward anticipation drive and dysregulated reward outcome responses.

(3) Emotional Dysregulation Affects ADHD Hyperactivity-Impulsivity Core Symptoms Emotional dysregulation is common and persistent in the daily lives of ADHD children and is considered an important clinical feature and potential treatment target (Barkley, 2015; Graziano & Garcia, 2016). Faraone et al. (2019) explicitly stated in a consensus statement that ADHD emotional dysregulation is mainly manifested in two aspects: emotional impulsivity (EI) and deficient emotional self-regulation (DESR). Emotional impulsivity refers to abnormally high reactivity to emotional stimuli, leading to rapid escalation of emotional responses to impaired levels. Deficient emotional self-regulation refers to the inability to manage emotional experiences

and control behavioral manifestations, causing abnormally activated emotional responses to return to baseline levels significantly slower (Barkley, 2015; Faraone et al., 2019). Some researchers view emotional dysregulation as a core symptom and functional impairment of ADHD, but as part of "hot" executive function, emotional dysregulation more likely affects the emergence of hyperactivity-impulsivity behaviors in ADHD children. Specifically, ADHD children have strong emotional reactivity, often showing irritability and negative emotions (Colonna et al., 2022; Liu et al., 2019). Notably, when facing emotional stimuli, besides showing strong emotional impulsivity, ADHD individuals show large and sustained increases in physiological arousal. Studies measuring autonomic nervous system indicators found that ADHD children show greater sympathetic dysregulation (i.e., enhanced skin conductance) during negative emotion induction states and greater parasympathetic dysregulation (i.e., enhanced respiratory sinus arrhythmia) during emotion regulation processes (Morris et al., 2020; Musser et al., 2011). This process of rapid emotional change may involve more energy accumulation, leading not only to more impulsive behaviors but also requiring "more behavior" to release excess energy. The most direct behavioral manifestation of emotional response is impulsive behavior, with current research often viewing emotional response and impulsive behavior as integrated or conflated (Faraone et al., 2019), without detailed discussion of how emotional response, especially high emotional impulsivity, specifically affects the emergence of hyperactivity-impulsivity core symptoms. Meanwhile, physiological indicators can provide more reliable and ecological data for ADHD children's emotional responses and can be applied in future research to explore the relationship and mechanisms between emotional dysregulation and hyperactivity-impulsivity.

ADHD emotional dysregulation is considered caused by functional abnormalities in the amygdala, ventral striatum, and orbitofrontal cortex, which bottom-up affect other cognitive processing (Christiansen et al., 2019; Shaw et al., 2014). For example, emotional dysregulation makes ADHD individuals unable to regulate negative emotions brought by delayed rewards, amplifies emotional experiences, and leads to more impulsive behavior. Lugo-Candelas et al. (2017) also found that ADHD children are less effective than peers in allocating attention and cognitive control in emotional contexts, and emotional regulation failure may exacerbate both inattention and hyperactivity-impulsivity core symptoms. Therefore, ADHD children with emotional dysregulation typically show high emotional reactivity and poor emotional regulation, making them more prone to maladaptive behaviors in daily life or when facing emotional events. More importantly, emotional dysregulation exists more in daily life, with laboratory-induced emotional responses and regulation being partial. Researchers have used ecological momentary assessment to capture emotional fluctuation changes in ADHD children's daily lives, finding that ADHD children have higher emotional variability (i.e., intense emotional fluctuations), which causes more severe functional impairment (Rosen et al., 2015; Walerius et al., 2018). However, research examining the dynamic relationship between ADHD children's sponta-

neous emotional fluctuations and core symptoms from an ecological perspective is still lacking.

(4) Verifying Mechanisms from a Causal Manipulation Perspective
The previous sections mentioned that "hot" executive function deficits affecting ADHD hyperactivity-impulsivity core symptoms can be explained by: (a) poor delay of gratification ability leading to preference for immediate satisfaction and difficulty persisting; (b) abnormal reward processing caused by abnormal activation of the brain reward network, leading to motivational dysregulation and cue-behavior association failure; and (c) negative effects of emotional dysregulation manifested as emotional impulsivity and deficient emotional self-regulation. The following will provide empirical evidence from causal (or near-causal) manipulation perspectives to support and verify these specific mechanisms.

In recent years, mindfulness practice has been considered an effective way to train "hot" executive functions, emphasizing non-judgmental awareness and experience of present physical and mental states (Kabat-Zinn, 1994). Mindfulness combines focused attention training (e.g., letting go of attention) and emotion regulation training (e.g., cognitive reappraisal, acceptance of negative emotions), improving "hot" executive functions by reducing stress and negative emotion awareness levels and promoting reshaping of healthy reward mechanisms (J. Davis et al., 2018; Garland, 2021; Tang et al., 2015). More essentially, mindfulness training can significantly improve impulsive behavior by reducing impulsive drive from emotional or motivational involvement and enhancing self-control ability (Yang & Zeng, 2023). Mindfulness training can also improve awareness and focus levels, reducing motor impulsivity (i.e., inappropriate or unhelpful overt actions) through top-down control systems and emotion regulation (Franco et al., 2016; Ron-Grajales et al., 2021). It can be concluded that mindfulness training can improve abilities related to "hot" executive functions, thereby helping reduce hyperactivity and impulsivity in ADHD patients. A meta-analysis systematically reviewed the intervention effects of mindfulness training on ADHD and found that mindfulness-based interventions significantly reduced hyperactivity-impulsivity and other clinical symptoms in ADHD individuals, with relatively obvious improvement in inattention as well (Cairncross & Miller, 2020). Researchers conducted an eight-week mindfulness intervention for 11-15-year-old ADHD children and their parents and found that parent-rated hyperactivity-impulsivity levels significantly decreased, with effects lasting at least eight weeks (Van der Oord et al., 2012). Intervention studies on ADHD adults also found that mindfulness meditation can improve emotion regulation strategies and impulse control levels (Mitchell et al., 2017). Notably, mindfulness training can also cause changes in brain activation patterns and functions (Tang et al., 2015). For example, mindfulness training reduces amygdala activation during negative emotional picture processing but enhances functional connectivity between the amygdala and ventromedial frontal cortex, meaning reduced emotional arousal and enhanced emotional control ability (Kral et al., 2018). After mindfulness training, brain region activation involved in reward an-

ticipation processing also significantly weakens when individuals face addiction reward cues, reducing impulsive choices (Kirk et al., 2019). The above neuroimaging evidence indicates that mindfulness training can improve abnormal brain activation and functional connectivity related to "hot" executive functions, thereby improving hyperactivity-impulsivity levels. This provides near-causal manipulation experimental evidence for verifying how "hot" executive functions affect ADHD hyperactivity-impulsivity core symptoms.

The orbitofrontal cortex (OFC) is an important brain region for "hot" executive functions. Due to its location in the cortical area, it is often considered a key target for neuromodulation stimulation for emotion regulation and risk decision-making. The OFC participates in complex decision-making processes such as reward processing and risk perception and plays an important role in negative emotion regulation (Ernst et al., 2002; Stalnaker et al., 2015). Some researchers using tDCS or TMS to modulate the OFC found that OFC stimulation can improve emotional experience and risk decision-making behavior (Howard et al., 2020; Ouellet et al., 2015; Yang et al., 2017). One study applied single-session tDCS to the DLPFC and OFC of 24 normal individuals and found that OFC stimulation significantly reduced risk-taking and risk decision-making, i.e., improved "hot" executive functions (Nejati et al., 2018). Although the OFC plays an important role in "hot" executive function deficits in ADHD patients, current neuromodulation research targeting the OFC is still limited. One study using tDCS with cathodal stimulation of DLPFC and anodal stimulation of OFC found the most obvious improvement effect on cognitive flexibility in ADHD children post-stimulation (Nejati et al., 2020). This study has not directly examined whether OFC stimulation can improve "hot" executive function performance in ADHD children, nor has it considered the impact of OFC stimulation on ADHD hyperactivity-impulsivity core symptoms. Based on existing research and theoretical foundations, it can be speculated that stimulating OFC and other "hot" executive function-related brain regions in ADHD patients can not only improve "hot" executive function task performance but also effectively improve hyperactivity-impulsivity clinical symptoms. Additionally, "hot" executive function-related brain regions are located in ventromedial areas and are closely connected with the limbic system and reward network, but how "hot" executive function deficits affect ADHD children's abnormal behavioral manifestations through brain network and functional connectivity abnormalities remains unclear. Considering that neuromodulation technology can cause changes in other brain regions or networks through stimulation of cortical structural and functional connections, future research can further use neuromodulation technology to manipulate "hot" executive functions and provide neural-level causal evidence for mechanisms affecting ADHD hyperactivity-impulsivity core symptoms.

4.3 Interactive Effects of "Cool" and "Hot" Executive Function Deficits on ADHD Core Symptoms

With deepening executive function research, recent trends show increasing attention to the interrelationship between "cool" and "hot" executive functions (Zelazo, 2020). Researchers have found that "cool" and "hot" executive functions are not independent but have dynamic interactions both top-down (cortical-limbic) and bottom-up (limbic-cortical) (Moriguchi, 2022; Nejati et al., 2018; Zhu et al., 2018). On one hand, "cool" executive functions can regulate individuals' perceived value of rewards during reward anticipation, control emotional responses during outcome feedback, and establish correct behavioral associations, playing a key top-down cognitive control role (Kryza-Lacombe et al., 2022; Luna, 2009). For example, stronger "cool" executive functions can protect children and adolescents in tempting and threatening environments by controlling emotional responses and motivational drives, making correct choices and reducing environmental harm. On the other hand, "hot" executive functions participate in value evaluation and risk decision-making, which directly affects goal-directed "cool" executive function performance, playing a bottom-up emotion-induced role. Better-developed "hot" executive functions can help individuals evaluate value reasonably, avoid risks, and enhance self-control ability. For example, children and adolescents show better "cool" executive function task performance in reward and motivation-driven contexts (Ma et al., 2016). Thus, certain behaviors emerge from the interactive coordination of "cool" and "hot" executive functions.

However, ADHD children show abnormalities in both "cool" and "hot" executive functions, and there may be interactive effects jointly affecting core symptom emergence. From a behavioral perspective, reward abnormalities and emotional dysregulation brought by "hot" executive function deficits are important factors for the occurrence and persistence of hyperactivity-impulsivity in ADHD children. But whether these deficits can directly trigger hyperactive-impulsive behavior also depends on "cool" executive function involvement. That is, ADHD children's insufficient "cool" executive functions make them unable to control abnormal emotional responses and unreasonable reward anticipation (motivational tendencies) brought by "hot" executive function deficits, thus showing more hyperactive-impulsive behavior. For example, ADHD children inherently have poor delay of gratification and prefer immediate satisfaction; if combined with insufficient inhibitory control, they cannot resist conflicts from immediate temptation, showing more impulsive behavior—a common scenario in daily life. Additionally, attention control processes require "cool" executive function involvement and consume substantial cognitive resources. Due to insufficient "hot" executive function development, ADHD children are more susceptible to strong drives from automatic emotional factors, making attention more easily shifted to external stimuli. Even with well-developed "cool" executive functions, if "hot" executive functions are deficient, individuals still have difficulty controlling powerful emotional drives and inhibiting automatic processing, leading to

attention control problems. For example, in situations requiring attention concentration (classroom), ADHD children not only have difficulty concentrating due to "cool" executive function deficits but may also be more driven by motivational or emotional cues due to "hot" executive function deficits, making it difficult to resist tempting distractors and causing attention interference. From a neurobiological perspective, developmental imbalance or impairment between brain regions related to "cool" and "hot" executive functions may also lead to ADHD core symptoms and other abnormal behaviors. For example, the dual systems theory explaining individual impulsivity suggests that imbalance between the brain cognitive control system (mainly dorsolateral prefrontal cortex, anterior cingulate cortex) and socio-emotional system (mainly orbitofrontal cortex, amygdala) leads to adolescent impulsive behavior (Shulman et al., 2016; Steinberg et al., 2008). ADHD patients show deficits in both systems (Capri et al., 2020), and studies have found abnormal functional connectivity imbalances between prefrontal cortex and amygdala or striatum networks in ADHD children, which may be the cause of maladaptive behavior and impulsivity (Dias et al., 2015).

In summary, the interaction between "cool" and "hot" executive function deficits in ADHD children jointly affects the emergence of inattention and hyperactivity-impulsivity core symptoms. Specifically, "cool" executive function deficits not only directly affect attention processing in ADHD children but also make it difficult for individuals to control emotional responses and inhibit unreasonable behaviors due to top-down "loss of control," thus showing more hyperactive-impulsive behavior. "Hot" executive function deficits not only cause motivational dysregulation and reward abnormalities but also bring more incorrect goal-directed behavior and automatic processing due to bottom-up "dysregulation," thereby affecting attention processing.

5 Future Research Directions

ADHD is a neurodevelopmental disorder, and executive function deficits related to prefrontal abnormalities may be the cognitive mechanism underlying ADHD core symptoms in children. Although this issue has received widespread attention, the mechanisms through which executive function deficits affect ADHD core symptoms have not been fully elucidated. Based on existing theoretical and empirical research, this paper explains why "cool" and "hot" executive function deficits can affect different ADHD core symptoms from a neuro-cognitive-behavioral developmental pathway perspective, attempting to build a cognitive deficit bridge between neurodevelopmental abnormalities and core symptoms in ADHD children. Additionally, it proposes that "cool" and "hot" executive functions may be two cognitive pathways leading to different ADHD core symptoms, with "cool" executive functions being the dominant factor for inattention and "hot" executive functions being the dominant factor for hyperactivity-impulsivity. More importantly, this paper elaborates on the mechanisms and specific pathways through which subcomponent deficits of "cool" and "hot" exec-

utive functions affect different ADHD core symptoms, providing research ideas for exploring the cognitive-neural mechanisms underlying ADHD core symptoms. However, several issues require further investigation, and future research can examine the following four aspects:

First, theoretical models of how "cool" and "hot" executive function deficits affect different ADHD core symptoms need further testing and refinement. Previous research on ADHD pathogenesis has been unclear, and examining the cognitive-neural mechanisms leading to different ADHD core symptoms from the perspective of "cool" and "hot" executive function deficits makes it possible to explain ADHD pathogenesis at the cognitive level. Although previous studies have explored correlations between executive function deficits and ADHD core symptoms, an integrated theoretical model explaining the cognitive-neural mechanisms through which executive function deficits affect different ADHD core symptoms is still lacking. This paper systematically discussed the effects of "cool" and "hot" executive function deficits on different ADHD core symptoms and their potential cognitive mechanisms, but the validity of these mechanisms remains to be supported by empirical research. From a cognitive perspective, the emergence of ADHD inattention and hyperactivity-impulsivity core symptoms is mainly caused by deficits or insufficient development of executive functions, with each subcomponent of "cool" and "hot" executive functions playing unique and important roles in affecting ADHD core symptoms. Therefore, future research should incorporate executive function deficits into large-scale ADHD epidemiological investigations, systematically examining the impact weights of "cool" and "hot" executive function deficits on different ADHD subtypes and the explanatory rates of executive function subcomponents for inattention versus hyperactivity-impulsivity core symptoms. Based on this, a cognitive model of executive function deficits affecting ADHD core symptoms can be constructed to provide scientific criteria for early screening and diagnosis of ADHD children. Additionally, examining the impact of executive function developmental changes on ADHD core symptoms from a child development perspective is crucial. Existing cross-sectional studies cannot explain whether the emergence or age-related improvement of ADHD core symptoms is related to executive function development, especially since "cool" and "hot" executive functions have different developmental trajectories during childhood, which may have different impacts on ADHD core symptoms (O'Toole et al., 2018). Therefore, future research can also combine longitudinal designs to reveal whether developmental changes in executive functions can predict or affect the severity of ADHD core symptoms in children, testing and refining the cognitive mechanisms through which executive function deficits affect ADHD core symptoms from a developmental perspective.

Second, further exploration of the neural substrates and cognitive-neural mechanisms through which "cool" and "hot" executive functions affect ADHD core symptoms is needed. From a neural perspective, ADHD core symptoms are mainly closely related to structural and functional abnormalities in the prefrontal cortex, but the cognitive deficits and mechanisms behind these abnormal

neural substrates are still in the exploratory stage. Whether structural changes or functional connectivity in brain regions related to "cool" and "hot" executive functions are associated with ADHD core symptoms, and whether related abnormal neural substrates can predict ADHD core symptom severity, remain to be resolved. Notably, the prefrontal cortex does not work independently but has close functional connections with subcortical structures and other brain networks. For example, pyramidal neurons in the prefrontal cortex project signals to the striatum, then to the thalamus, and finally back to the cortex, forming a cortico-striatal-thalamo-cortical loop structure that functions as a whole (Jiang et al., 2018; Zhu et al., 2016). Therefore, neural circuit abnormalities centered on the prefrontal cortex may provide a bridge for understanding how executive function deficits affect different ADHD core symptoms (Zhu et al., 2018). Additionally, brain regions involved in "cool" and "hot" executive functions are also core nodes of neural networks such as the attention network and reward network. Deficiencies in these regions not only limit executive function development but also cause abnormal activation in related brain networks through functional connectivity, jointly affecting ADHD core symptom emergence. Future research can further explore whether specific brain regions activated in ADHD children under different executive function tasks and their functional connectivity with other brain networks are abnormal, striving to identify the neural substrates and network models through which "cool" and "hot" executive function deficits affect different ADHD core symptoms. Meanwhile, most research in the field on ADHD neural substrates is based on recording and correlational perspectives, lacking causal (or near-causal) manipulation evidence to support the effects of "cool" and "hot" executive function deficits on different ADHD core symptoms and their cognitive-neural mechanisms. Given that neuromodulation technology can cause indirect changes in downstream brain structures and network functional connectivity through cortical stimulation, future research can also integrate neuromodulation technology into brain imaging studies to verify cognitive-neural models of executive function deficits affecting ADHD core symptoms from a causal manipulation perspective.

Third, the effects of "cool" and "hot" executive function deficits on ADHD core symptoms should be examined from an ecological perspective. Research has found obvious individual heterogeneity in executive function deficits in ADHD children (Bunger et al., 2021; Kofler et al., 2019). Specifically, ADHD children's executive function scores show significant differences between laboratory task-based tests and parent/teacher rating scales, with only about 52% of ADHD children showing executive function deficits on laboratory tasks, while over 90% are reported by parents/teachers to have executive function deficits (Sjöwall & Thorell, 2019). Moreover, clinicians rely more on parent/teacher-reported scale scores for diagnosis because they better reflect ADHD children's clinical manifestations in daily life (DSM-IV, 2013). This also indicates that current laboratory tasks have insufficient ecological validity and poorly reflect individuals' abnormal manifestations in real life (Barkley & Murphy, 2011; Bunger et al., 2021). Especially, existing laboratory tasks mostly focus on "cool" executive functions,

with less attention to how "hot" executive functions under high emotional or motivational involvement affect ADHD core symptom emergence. Meanwhile, motivational and emotional drive cues are everywhere in daily life, requiring not only "hot" executive function involvement but more importantly the interaction between "cool" and "hot" executive functions (Zelazo, 2020). For example, difficulty resisting distractors results from the struggle between inhibitory control and temptation drive in ADHD; when tempting distractors overcome control ability, individuals interrupt attentional persistence and show inattention. That is, even with well-developed "cool" executive functions, if "hot" executive functions are insufficient and motivational drive is too strong, abnormal behavioral manifestations are still unavoidable (Yang & Zeng, 2023). However, ADHD children have abnormalities in both "cool" and "hot" executive functions, and their interaction will prompt more executive function deficit-related abnormal behavioral manifestations in daily life. Existing research largely ignores this view and one-sidedly examines the relationship between ADHD children's executive function deficits and core symptoms. Therefore, future research should explore from an ecological perspective how the interaction between "cool" and "hot" executive function deficits affects the emergence of core symptoms in ADHD children's daily lives, and attempt to answer why there is individual heterogeneity in ADHD children's executive function deficits.

Finally, based on "cool" and "hot" executive function deficits, develop psychological intervention and neuromodulation programs with long-term effects on ADHD core symptoms. Theoretically, interventions targeting executive function deficits have important clinical value, as they not only improve ADHD core symptoms and social functions but may also have lasting effects (Karr et al., 2018). However, current executive function intervention programs do not significantly improve ADHD-related core symptoms or normalize ADHD children's problem behaviors. The reason for this low efficiency may be that these interventions only focus on abilities themselves without considering the relationship between executive function subcomponent deficits and ADHD abnormal behavioral manifestations, greatly reducing far-transfer effects of interventions (Kofler, Wells, et al., 2020). The cognitive-neural mechanisms proposed earlier have clarified the effects of "cool" and "hot" executive function deficits on different ADHD core symptoms, suggesting that future research can start from cognitive interventions to improve inattention core symptoms by manipulating ADHD children's "cool" executive functions, or reduce hyperactivity-impulsivity symptoms by training "hot" executive function-related abilities. Meanwhile, future research can use neuromodulation technology to examine whether single or multiple TMS/tDCS sessions targeting DLPFC reduce inattention and other abnormal behaviors by improving "cool" executive functions, or whether stimulating OFC and other brain regions can improve behavior and emotion control abilities by enhancing "hot" executive functions. Notably, ADHD children's executive function subcomponent deficits show strong individual differences. For example, for inattention symptoms, some ADHD children may result from working memory representation difficulties preventing correct target selection,

while others may relate to insufficient inhibitory control making it difficult to resist external interference. Without individual-level differentiation, intervention targets are difficult to match individual ability deficits and developmental levels, greatly limiting the effectiveness and long-term nature of core symptom improvement. Therefore, based on clarifying ADHD children's pathogenesis, future research should develop personalized interventions and precise treatments for ADHD core symptoms based on "cool" and "hot" executive function deficits from both cognitive intervention and neuromodulation perspectives, striving to achieve far-transfer effects of interventions.

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