Mechanisms of "Cold" / "Hot" Executive Function Deficits on Core Symptoms in Children with ADHD: A Postprint

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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 stemming from abnormal prefrontal cortex development. Based on this, the neuro-cognitive-behavioral developmental pathway proposes that executive function deficits may represent the cognitive-level pathophysiological mechanism underlying ADHD core symptoms, wherein "cool"executive function deficits associated with the dorsolateral prefrontal cortex may constitute the dominant factor contributing to attention deficit core symptoms, while "hot" executive function deficits associated with the ventromedial prefrontal cortex may constitute the dominant factor contributing to hyperactivity and impulsivity core symptoms. On the one hand, "cool" executive function deficits primarily lead to failures in maintaining working memory representations, insufficient inhibitory control capacity, and difficulties in cognitive switching, which further constrain individuals' 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, causing failure in behavioral inhibition and making individuals more prone to impulsive choices, thereby manifesting core symptoms of hyperactivity and impulsivity. 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

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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 attention deficits and/or hyperactivity-impulsivity as core symptoms, which is closely related to executive function deficits stemming from abnormal prefrontal development. Based on this, we propose from a neuro-cognitive-behavioral developmental pathway that executive function deficits may represent the cognitive-level pathogenesis underlying ADHD core symptoms. Specifically, "cool" executive function deficits associated with the dorsolateral prefrontal cortex may be the dominant factor causing attention deficit symptoms, while "hot" executive function deficits related to the ventromedial prefrontal cortex may be the dominant factor causing hyperactivity-impulsivity symptoms. On the one hand, "cool" executive function deficits primarily lead to failures in working memory representation maintenance, inadequate inhibitory control, and difficulties in cognitive switching, which further restrict individuals' capacity for sustained attention, attentional selection, and attentional shifting. On the other hand, "hot" executive function deficits bring about problems such as delay aversion, abnormal reward processing, and motivational dysregulation, resulting in behavioral inhibition failure and a greater likelihood of impulsive decision-making, thereby manifesting as hyperactivity-impulsivity core symptoms. Future research should further test 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, examine the interactive effects of "cool" and "hot" executive function deficits on ADHD core symptoms from an ecological perspective, and develop personalized, precise, and long-lasting intervention programs for ADHD core symptoms based on executive function.

Keywords: attention-deficit/hyperactivity disorder, executive function deficit, core symptoms, prefrontal cortex, mechanism

Classification Code: B845

Attention-deficit/hyperactivity disorder (ADHD) is a neurodevelopmental disorder originating in childhood, primarily characterized by attention deficits and/or hyperactivity-impulsivity that are inconsistent with developmental level, accompanied by functional impairment in academic or social domains (American Psychiatric Association, 2013). The global prevalence 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), with 50.9% of children with ADHD experiencing persistent symptoms into adulthood (Agnew-Blais et al., 2016; Franke et al., 2018; Li Tingyu et al., 2020; Sibley et al., 2016), causing harm across the entire lifespan (Faraone et al., 2015; Forte et al., 2019; 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, assessing diagnosis, and evaluating interventions, 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 unclear (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 in children from the perspective of executive function deficits and attempting to build a cognitive bridge between neurodevelopmental abnormalities and core symptoms holds important scientific value for early identification, diagnosis, and effective clinical intervention of this disorder.

Based on differences in prefrontal function, executive function can be divided into "cool"and "hot"components (Zelazo & Carlson, 2012; Zelazo & Müller, 2012). "Cool" executive function primarily involves brain regions such as the dorsolateral prefrontal cortex and ventrolateral prefrontal cortex, focusing on relatively abstract, decontextualized cognitive processing, including subcomponents such as working memory, inhibitory control, and cognitive flexibility. "Hot" executive function refers to cognitive processing with high emotional/motivational involvement, including abilities such as delay of gratification and affective decision-making, and is 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 dopaminergic mesocortical branch, and a motivational development pathway related to the dopaminergic mesolimbic 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 research evidence suggests that attention deficits in children with ADHD primarily stem from "cool" executive function deficits, while hyperactivity-impulsivity is mainly associated with "hot" executive function deficits (Castellanos & Aoki, 2016; Shakehnia et al., 2021). In other words, "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). Moreover, from a developmental psychopathology perspective, ADHD is a neurodevelopmental disorder in which early neurodevelopmental processes are hindered through gene-environment interactions, leading to cognitive deficits or deficiencies that further increase the risk of ADHD, including the emergence of core symptoms and other abnormal behaviors (McLaughlin, 2016; Zelazo, 2020). Thus, executive function deficits may be the cognitive mechanism underlying neurodevelopmental abnormalities that lead to ADHD core symptoms.

The Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) explicitly identifies ADHD as a neurodevelopmental disorder with abnormalities in both brain structure and functional connectivity (Cortese et al., 2021; Hoogman et al., 2019; Hoogman, 2020; Thapar et al., 2017). In particular, developmental delays or deficiencies in the prefrontal cortex of children with ADHD not only constitute the physiological basis for core symptoms but are 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 patients with ADHD show persistent 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., 2016). In addition to structural and functional abnormalities in the prefrontal cortex, abnormal brain network function is also a strong influencing factor for core symptoms in children with ADHD (Cai et al., 2018; Sripada et al., 2014). Specifically, the executive control network centered on the prefrontal cortex and its functional connections 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, core symptoms in children with ADHD are caused by abnormalities in brain structure and function, particularly in the prefrontal cortex and executive control network related to executive function. Meanwhile, as a fundamental cognitive ability, executive function top-down participates in, guides, or coordinates other cognitive processes, playing a decisive role in future academic performance, intentional behavior, and social adaptation (Spiegel et al., 2021; Zelazo, 2015). Deficiencies or deficits in executive function not only directly cause abnormal daily performance and manifestations such as inattention, hyperactivity, and impulsivity; if left unaddressed, these deficits can, in turn, affect brain structural development and functional plasticity, reinforcing neural abnormalities and further exacerbating abnormal behaviors including core ADHD symptoms. Therefore, these neurobiological and cognitive-level evidences indicate that ADHD is a neurodevelopmental disorder, particularly manifested as delayed development of the prefrontal cortex related to executive function and abnormal functional connectivity of the executive control network, leading to underdeveloped executive function (cognitive deficits) and subsequently causing attention deficits and/or hyperactivity-impulsivity core symptoms.

As shown in Figure 1 [Figure 1: see original paper], the pathway from neural abnormalities to cognitive deficits to behavioral characteristics suggests that executive function deficits may be the cognitive mechanism underlying neurodevelopmental abnormalities that lead 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, this paper infers 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 that leads to the emergence of 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 sections will discuss 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 in children include inattention and hyperactivity-impulsivity (DSM-5, APA, 2013). Inattention typically manifests as difficulty sustaining attention, carelessness, disorganized behavior, and lack of organization, specifically reflected in problems such as poor concentration, susceptibility to distraction, difficulty shifting attention, and narrowed attention span. Hyperactivity-impulsivity typically manifests as fidgeting, constant movement, difficulty waiting, and interrupting others, specifically reflected in problems such as inability to inhibit behavior, excessive activity, pursuit of 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 the one hand, inadequate inhibitory control related to the dorsal executive circuit involving the prefrontal cortex-striatum leads to cognitive and behavioral dysregulation in children with ADHD. On the other hand, deficits in delay of gratification related to the ventral reward circuit involving the orbitofrontal cortex-striatum lead to motivational dysregulation in children with ADHD (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 are closely related to "cool" and "hot" executive function deficits, respectively, with a certain correspondence. "Cool" executive function deficits correspond to the cognitive control pathway, while "hot" executive function deficits correspond to the motivational development pathway (Geurts et al., 2006; Jiang et al., 2018; Zhang Wei et al., 2010). Thus, the emergence of abnormal behaviors in children with ADHD 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 answered by analyzing the effects of "cool" and "hot" executive function deficits on different ADHD core symptoms from neurobiological and cognitive perspectives. First, "cool"executive function involves brain regions including the dorsolateral prefrontal cortex, ventrolateral prefrontal cortex, and dorsal anterior cingulate cortex, which are engaged 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, 2021; Zelazo & Carlson, 2012). Numerous studies have shown that children with ADHD 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 be related to ADHD core symptoms (Kofler, Harmon et al., 2018; Mueller et al., 2017). Moreover, research has 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, insufficient working memory leads to carelessness and forgetfulness; inhibitory control failure may cause distraction and inattention; cognitive flexibility deficits may result in disorganization and difficulty shifting attention. Additionally, the key brain regions involved in "cool" executive function are also core nodes of the dorsal attention network (such as the dorsolateral prefrontal cortex), participating in intentional attention processing (Lemire-Rodger et al., 2019; Petersen & Posner, 2012). In addition to "cool" executive function deficits, abnormalities in the attention network further exacerbate inattention and other core symptoms in ADHD. Therefore, it can be understood that "cool" executive function deficits affect the emergence of ADHD inattention core symptoms through both resulting ability deficits and abnormal neurobiological substrates (dorsal attention network).

Second, children with ADHD also show significant "hot" executive function deficits. "Hot" executive function refers to the ability to flexibly evaluate and make risky decisions under high emotional or motivational involvement, including abilities such as delay of gratification and affective decision-making. Its deficits are often considered related to hyperactivity-impulsivity core symptoms in children with ADHD (Colonna et al., 2022; Dekkers et al., 2016; Shakehnia et al., 2021; Tegelbeckers et al., 2018). For example, children with ADHD have difficulty inhibiting their behavior, pursue immediate rewards, and show emotional dysregulation in daily life, which are likely caused by "hot" executive function deficits (Bunford et al., 2022; Colonna et al., 2022; Petrovic & Castellanos, 2016). The brain regions related to "hot" executive function are also key areas of the reward network (such as the orbitofrontal cortex and ventromedial prefrontal cortex), involved in reward processing and self-emotion regulation (Cubillo et al., 2012; Yang et al., 2019). Research has found that the reward network in children with ADHD shows imbalanced connectivity with other brain regions, particularly decreased functional connectivity with the executive control network and related brain areas, 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 be caused by both ability deficits resulting from "hot" executive function deficits and their abnormal neurobiological substrates.

In summary, based on 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 lead to ability deficits when facing tasks (such as difficulty resisting interference and maintaining memory representations), resulting in attention control failure. "Hot" executive function deficits related to ventromedial prefrontal abnormalities lead to problems such as delay aversion and reward abnormalities, resulting in motivation dysregulation. The abnormal neurobiological substrates of executive function deficits are also key nodes or regions of core brain networks (such as the dorsal attention network and reward network), involved in attention processing and reward processing. Abnormalities in these brain networks further affect individuals' attention control and behavioral inhibition abilities, jointly leading to the emergence of different ADHD core symptoms (as shown in Figure 2 [Figure 2: see original paper]).

4. Mechanisms by Which "Cool" / "Hot" Executive Function Deficits Affect Different ADHD Core Symptoms

The previous section proposed that "cool" and "hot" executive function may be two cognitive pathways leading to different ADHD core symptoms, resulting in 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 dominant factor for hyperactivity-impulsivity core symptoms. The following sections will elaborate on how deficits in subcomponents of "cool" and "hot" executive function affect the emergence of different ADHD core symptoms and their specific mechanisms.

4.1 Mechanisms by Which Subcomponents of "Cool" Executive Function Deficits Affect ADHD Inattention Core Symptoms

"Cool" executive function regulates attention through corresponding abilities to control behavior, making it more adaptive, planned, and focused when solving problems (Zelazo, 2020). If "cool" executive function is deficient, it leads to failures in goal-directed behavior and attention control, specifically manifested in attention maintenance, selection, and allocation. "Cool" executive function typically includes three core subcomponents: working memory, inhibitory control, and cognitive flexibility (Diamond, 2013; Miyake et al., 2000). The following discussion will focus on how these three subcomponents affect the specific mechanisms of ADHD inattention core symptoms.

(1) Working Memory Affects ADHD Inattention Core Symptoms

Working memory refers to a limited-capacity memory system that temporarily stores and processes information during cognitive task execution (Baddeley, 2012). It includes not only an episodic buffer responsible for temporarily storing and integrating memory representation information but also a 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 the one hand, information representation in working memory top-down captures target stimuli, thereby guiding the attention selection process. On the other hand, the central executive system helps ignore or inhibit irrelevant information, selectively allocating resources to target-related stimuli, which facilitates attention selection (Gazzaley & Nobre, 2012; Greene et al., 2015). Additionally, the frontoparietal network is a key region in the working memory system, and its activation can promote the maintenance and processing of target information (Lamichhane et al., 2020; Soto et al., 2013). Studies have found that the frontoparietal network is related to both working memory representation maintenance and attention control processes (Bahmani et al., 2019; Wallis et al., 2015), particularly dorsolateral prefrontal activation is related to attention selection processes in working memory (Panichello & Buschman, 2021; Quentin et al., 2019). Therefore, it is reasonable to infer that working memory deficits in children with ADHD affect attention selection processes through working memory representation and the central executive system.

In fact, research has shown that working memory deficits are significant cognitive deficits in children with ADHD and have the strongest predictive power for inattention core symptoms (Fosco et al., 2020; Irwin et al., 2021; Ramos et al., 2020). Specifically, children with ADHD lack the ability to store information and maintain memory information representations over time, leading to easier loss of key information needed to complete tasks and inability to select correct target information. Central executive system deficits make it difficult for children with ADHD to inhibit irrelevant information, increasing cognitive control load and limiting top-down attention control (Jacobson et al., 2011; Kofler, Soto et al., 2020; Re et al., 2016). In this situation, children with ADHD in the classroom easily shift their attention to internal thoughts or external stimuli unrelated to classroom content, showing attention selection-related deficits (Rapport et al., 2009). Neuroimaging studies have also found that brain regions with abnormal activation in children with ADHD during working memory tasks are concentrated in the dorsolateral prefrontal cortex, medial inferior frontal gyrus, cingulate gyrus, parietal lobe, and other regions that also participate in attention 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 stages), using posterior contralateral N2 (pcN2) and contralateral delay activity (CDA) components as indicators to effectively distinguish attention selection processes from working memory maintenance processes through simultaneous EEG-behavior analysis. The results found that selective attention and working memory deficits in ADHD patients were closely related; that is, when memory load increased, working memory in ADHD patients was difficult to maintain, leading to reduced attention selection efficiency in tasks (Luo et al., 2019). Overall, the mechanism by which working memory deficits in children with ADHD affect inattention core symptoms is mainly manifested in difficulty maintaining working memory representations and failure of top-down control by the central executive system, which is not conducive to attention selection processes, resulting in abnormal behavioral manifestations such as susceptibility to interference and inattention.

(2) Inhibitory Control Affects ADHD Inattention Core Symptoms

Inhibitory control is the ability to intentionally inhibit strong internal/external interfering information to make correct responses to targets (Aron et al., 2004). According to different interfering stimuli, it can be divided into response inhibition (behavioral-level inhibition ability) and conflict control (interference inhibition, cognitive-level inhibition ability) (Diamond, 2013). Response inhibition refers to inhibiting inappropriate behavioral responses to external interfering information and maintaining attention on target information. Conflict control ability, also called interference inhibition ability, mainly refers to inhibiting conflicting information in the brain while persisting in correct responses (Hung et al., 2018). If inhibitory control is deficient, individuals will have difficulty inhibiting attention or behavioral responses to irrelevant stimuli (such as external distractors or internal irrelevant thoughts), limiting attention sustainability. Most studies have found that children with ADHD have difficulties with inhibitory control. For example, in response inhibition tasks such as Stop-Signal and Go/No-Go, children with ADHD show longer reaction times and higher error rates (Fosco et al., 2019; Hart et al., 2013). In interference control tasks such as Stroop and Simon, children with ADHD show poorer reaction times and accuracy in incongruent trials (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 shown by ADHD in childhood can predict the development of inattention symptoms in adolescence, and insufficient inhibition makes the age-related improvement of attention deficits smaller (DeRonda et al., 2021).

From a neuroimaging perspective, ADHD patients show abnormal activation in the right inferior frontal gyrus, parietal lobe, striatum, and other brain regions during response inhibition tasks (Thornton et al., 2018; van Rooij et al., 2015). Among them, the inferior frontal gyrus participates not only in the initiation and processing of inhibitory signals but is also related to the ventral attention network and temporoparietal junction, responsible for maintaining attention control. The parietal region mainly participates in attention reorientation and task goal maintenance during response inhibition (Cai & Leung, 2011; Fabio & Urso, 2014). Additionally, ADHD patients show lower connectivity between response inhibition networks, which is significantly correlated with the severity of ADHD core symptoms (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 the Go/No-Go task not only showed a significant positive correlation with response inhibition ability but could also predict the severity of inattention deficits in children with ADHD. This provides neural-level evidence for further explaining how response inhibition affects ADHD inattention. In summary, poor behavioral inhibition ability resulting from response inhibition deficits and related attention network impairments can better explain the existence of attention maintenance and concentration problems in children with ADHD.

However, it is worth noting that current research only uses concepts like "inhibition" or "response inhibition" to uniformly represent inhibitory control, without distinguishing between response inhibition and conflict control (interference inhibition) in inhibitory ability. This is insufficiently comprehensive and precise for explaining how inhibitory control deficits specifically affect core symptoms in children with ADHD. 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 have shown that children with ADHD have weaker conflict control ability, making it difficult for them 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 ability, children with ADHD are more susceptible to interference from internal distracting thoughts during task execution and have difficulty maintaining attention. Brain regions mainly activated by conflict control include the dorsal anterior cingulate cortex, dorsolateral prefrontal cortex, parietal region, and dorsolateral frontal control system (Hung et al., 2018). Among them, the dorsolateral prefrontal cortex mainly participates in maintaining and manipulating task-related 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 attention persistence. Given the unique role of conflict control in inhibitory ability, more attention should be paid to exploring how insufficient conflict control affects ADHD inattention core symptoms. Future research should distinguish different types of inhibitory control and conduct more detailed investigations of different inhibitory abilities impaired 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 psychological situations (Diamond, 2013). Cognitive flexibility essentially reflects individuals' abilities in inhibitory control and cognitive shifting, inhibiting dominant but ineffective cues, efficiently reconfiguring resources, and participating in the development of attention shifting ability in children (Dajani & Uddin, 2015; Filippetti & Krumm, 2020). As one of the core components of executive function, children with ADHD 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 classified cognitive flexibility into cognitive domains related to attention deficits and proposed that cognitive flexibility deficits may cause impaired target selection and cognitive control in ADHD patients, leading to inattention core symptoms (Mueller et al., 2017). Luna-Rodriguez et al. (2018) investigated attention shifting ability in ADHD patients during switching tasks and found that increased switching costs might lead to insufficient attention shifting and allocation to different stimuli in ADHD tasks. However, current empirical research on the relationship between cognitive flexibility and ADHD inattention core symptoms is limited. By definition, cognitive flexibility more refers to the ability to flexibly switch cognitive modes according to task demands. If underdeveloped, individuals have difficulty shifting from one task to another, and the effectiveness of attention shifting is challenged, showing problems such as difficulty allocating and shifting attention (Wendt et al., 2018). Therefore, cognitive flexibility deficits in children with ADHD can affect the emergence of inattention core symptoms by influencing attention shifting and allocation. Additionally, considering that cognitive flexibility is built upon the development of working memory and inhibitory control, using more sensitive tools or paradigms to assess cognitive flexibility and distinguish it from other executive function components is important for explaining how cognitive flexibility affects inattention.

(4) Verifying the Mechanism of "Cool" Executive Function Affecting ADHD Core Symptoms from a Causal Manipulation Perspective

The previous sections preliminarily explained how "cool" executive function affects ADHD inattention core symptoms from cognitive and neural perspectives, but lack support from a causal manipulation perspective. The following will provide empirical evidence from cognitive intervention and neuromodulation (or near-causal) perspectives to further verify the specific mechanisms by which "cool" executive function affects inattention core symptoms.

Cognitive intervention mainly improves attention performance in children with ADHD by directly training different components of "cool" executive function. Among them, working memory training has been confirmed as a promising intervention measure for improving inattention and academic difficulties in children with ADHD (Kofler, Wells et al., 2020; Wiest et al., 2022). Central Executive

Training (CET) is a computer-based intervention training targeting working memory deficits in children with ADHD. Research has found that CET training can not only significantly improve working memory and inhibitory control scores but also persistently improve academic performance. Further analysis found that the improvement in academic performance was mainly achieved by improving 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 working memory in children with ADHD and found that children with ADHD who received CWMT training showed improved working memory, enhanced attention 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 attention performance in ADHD. For example, Kofler et al. (2020) conducted a Go/No-Go computer-based cognitive training with children with ADHD aged 8-12 and found that attention ability in children with ADHD continued to improve after the 4-week intervention. AKL-T01 (Akili Interactive Labs-T01) is an adaptive digital intervention program based on a game interface, mainly adapted from inhibitory control and cognitive flexibility tasks. After 4 weeks of AKL-T10 cognitive intervention, parent-reported and objective behavioral indicators of attention in children with ADHD were significantly improved (N. Davis et al., 2018; Kollins et al., 2020). In summary, cognitive intervention manipulates different executive function components such as working memory, inhibitory control, and cognitive flexibility, and examines changes in attention ability in children with ADHD before and after intervention, thereby providing causal (or near-causal) evidence to support and verify the mechanism by which "cool" executive function affects inattention core symptoms.

Neuromodulation regulates cortical excitability by stimulating the dorsolateral prefrontal cortex (DLPFC) related to "cool" executive function, causing changes in brain function and thereby improving executive function and alleviating 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 4 weeks could significantly improve inattention symptoms in adults with ADHD (Leffa et al., 2022). Nejati et al. (2021) applied single-session anodal stimulation to the right DLPFC of children with ADHD aged 9-10 and had them complete Flanker, Go/No-Go and other tasks before and after stimulation. The results found that activating the right DLPFC helped improve inhibitory control ability, and the stimulation effect was positively correlated with ADHD severity (Nejati et al., 2021). Additionally, a study applying high-frequency stimulation to the right DLPFC of children with ADHD aged 7-12 for 6 weeks found that it 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 their "cool" executive function task performance and effectively alleviate clinical symptoms of inattention. This further provides neural-level evidence from a causal manipulation perspective for the mechanism by which executive function deficits affect inattention. Currently, the application of neuromodulation techniques in treating ADHD patients is in a rapid development stage, but there are still no unified standards for stimulation brain region location, treatment parameters (frequency/intensity), and stimulation duration, and clinical effects show great variation (Westwood et al., 2021). Most studies have found that neuromodulation techniques stimulating DLPFC can improve "cool" executive function, but how the improvement of "cool" executive function after intervention improves inattention still receives little attention. It is worth noting that TMS or tDCS can also directly stimulate core nodes of structural or functional connections to cause indirect changes in neural activity in "downstream" areas, including cortical and subcortical brain network functional connectivity (Sydnor et al., 2022). That is, stimulating an individual's DLPFC will also activate or inhibit other brain cortical or subcortical networks to varying degrees, jointly affecting individual behavioral performance. From this perspective, the cognitive neural mechanism by which stimulating "cool" executive function-related brain regions improves individual attention can be further revealed.

4.2 Mechanisms by Which Subcomponents of "Hot" Executive Function Deficits Affect ADHD Hyperactivity-Impulsivity Core Symptoms

"Hot" executive function refers to the ability to flexibly evaluate and make risky decisions under high emotional or motivational involvement, including delay of gratification, reward processing, and emotional regulation. If "hot" executive function is deficient, it leads to behavioral control failure and motivation dysregulation in emotional or social contexts, specifically manifested as delay aversion, pursuit of immediate rewards, and motivation dysregulation. Researchers have gradually recognized that "hot" executive function deficits may be the dominant factor for hyperactivity-impulsivity core symptoms in children with ADHD. The following sections will specifically discuss how abnormalities in different subcomponents of "hot" executive function affect hyperactivity-impulsivity core symptoms in children with ADHD.

(1) Delay of Gratification Affects ADHD Hyperactivity-Impulsivity Core Symptoms

Delay of gratification ability refers to a choice orientation that willingly forgoes immediate satisfaction for more valuable long-term goals, and the ability to demonstrate self-control during the waiting process (Mischel et al., 1989). From a temporal dimension, delay of gratification includes at least two stages: "making a choice" and "maintaining the choice" (Ren Tianhong et al., 2015). The former involves the process of value evaluation and choice-making when facing options, while the latter involves resisting temptation and executing intended behavior. The famous "marshmallow experiment" is used to measure children's delay of gratification ability (Mischel et al., 1989). In this experiment, children can choose to receive a reward (such as a marshmallow) immediately or wait for a period to receive double the reward. Delay of gratification ability can help children weigh immediate versus future benefits, make the most advantageous choice, and maintain behavior consistent with the choice after selection to obtain maximum benefits. Children with ADHD show poor delay of gratification ability, which is often considered closely related to their impulsive abnormal behavioral manifestations (Sonuga-Barke et al., 2010; van Dessel et al., 2019). Examining the two stages of delay of gratification, children with ADHD in the "making a choice" stage often assign lower value to future benefits and show obvious aversion to waiting time, making more impulsive and reckless choices. Even when making advantageous choices, children with ADHD in the "maintaining the choice" stage often cannot resist immediate temptation, fail in behavioral inhibition, and show impulsive behaviors such as "midway regret." Delay of gratification is common in daily life, such as waiting in line, doing homework before playing, etc. However, due to insufficient delay of gratification ability in children with ADHD, they show difficulty waiting and aversion to delay in daily situations (Utsumi et al., 2016; van Dessel et al., 2019).

Related evidence supports the above view. A meta-analysis summarizing 22 studies found that children and adolescents with ADHD overall showed higher delay discounting in delay discounting tasks (Patros et al., 2016), with children with ADHD more inclined to choose immediate satisfaction and showing more impulsive behavior (Martinelli et al., 2017). Additionally, researchers found that during the delay waiting process, children with ADHD often experience more subjective negative emotions and obvious aversion to delay (Rosch & Mostofsky, 2016). Neuroimaging studies further found that delay aversion is related to overactivation in brain regions related to negative emotion processing (such as the amygdala), with stronger responses as delay time increases (van Dessel et al., 2018; van Dessel et al., 2020). In addition to amygdala overactivation, children with ADHD also show excessive spontaneous neural activity (low-frequency oscillations) during waiting, and this excessive activity is related to impulsive decision-making behavioral characteristics (Hsu et al., 2015). The "maintaining choice" stage more reflects individuals' ability for self-control of goal-directed behavior, involving participation of brain regions such as the dorsolateral prefrontal cortex, ventrolateral prefrontal cortex, and posterior parietal lobe, which are also related to impulsive behavior (Dalley & Ersche, 2019). The failure of self-control in children with ADHD not only affects their ability to persist but also brings more externalizing problem behaviors. In summary, poor delay of gratification ability leads children with ADHD to show a tendency for immediate satisfaction and aversion to delay in the "making choice" stage, and inability to inhibit conflicts caused by immediate temptation and behavioral control failure in the "maintaining choice" stage. These two aspects jointly affect the emergence of impulsive behavior in ADHD.

(2) Reward Processing Affects ADHD Hyperactivity-Impulsivity Core Symptoms

Increasingly, researchers believe that abnormal reward processing is also an important factor affecting hyperactivity-impulsivity core symptoms in children with ADHD (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 the one hand, reward anticipation refers to an individual' s state of waiting and desire for upcoming potential rewards. In life, once an individual notices reward-predictive stimulus cues, they will assign higher expected outcome value to the stimulus and promote goal-directed behavior. The emergence of impulsive behavior is often driven by strong reward anticipation, with such individuals having higher reward sensitivity (Dawe et al., 2004; Xiong Suhong & Sun Hongjie, 2017). Particularly, children with ADHD have higher reward sensitivity to certain stimuli (such as money) (Fosco et al., 2015; Tripp & Wickens, 2012). When facing such reward anticipation, individuals are more likely to ignore threat cues and adverse consequences, unable to inhibit the drive caused by reward anticipation, and produce impulsive behavior. On the other hand, the outcome response stage refers to the process of evaluating obtained rewards and monitoring whether behavior has achieved task goals. In this stage, individuals establish behavioral associations with reward cues based on perceived reward feedback (Yang Ling et al., 2015). However, children with ADHD often have abnormal responses to reward outcomes. For example, patients with ADHD consider "salient" rewards such as money and rights to have higher value, but lose interest in other "low-return" rewards such as smiling faces and 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 shows more urgent seeking responses (i.e., impulsivity). Over time, most reward responses are weakened, and only "higher value" reward stimuli can produce sufficient signals. Without intervention, this abnormal reward processing mechanism will lead individuals with ADHD to continuously pursue more stimulating risky activities (such as speeding, gambling), which is also key to the development and maintenance of substance abuse and addiction processes (Grimm et al., 2021). Therefore, children with ADHD often show motivation dysregulation driven by abnormal reward anticipation and failure in reward cue-behavior association, making children continuously seek more frequent external stimulation, which can also explain why children with ADHD are more hyperactive and impulsive than their peers.

Currently, a large number of studies have shown that abnormal reward processing in ADHD has a neurological basis, namely structural and functional abnormalities in the brain reward circuit (von Rhein et al., 2017; Zhu et al., 2018). Previous studies have consistently found that during monetary reward processing tasks, patients with ADHD show insufficient activation in the ventral striatum during reward anticipation (Plichta & Scheres, 2014; Scheres et al., 2007; van Hulst et al., 2017), and this reduced anticipation of reward cues leads ADHD to prefer immediate rewards. Studies have also found that the orbitofrontal cortex of ADHD patients is overactivated during reward anticipation, and the degree of activation is significantly positively correlated with hyperactivity-impulsivity core symptoms. It is further inferred that due to orbitofrontal overactivation, individuals with ADHD cannot correctly encode expected outcome value, resulting in more maladaptive behaviors (Tegelbeckers et al., 2018). Additionally, the most common reward abnormality in ADHD is increased delay discounting, i.e., the tendency to choose smaller immediate rewards over larger delayed rewards (Jackson & MacKillop, 2016; Marx et al., 2021; Yu & Sonuga-Barke, 2020). Under delay discounting tasks, studies have found abnormal functional connectivity (imbalance) between the reward network and frontoparietal network in children with ADHD, which affects the emergence of impulsive behavior and may also be the neural mechanism behind hyperactivity-impulsivity core symptoms (Dias et al., 2015). ADHD also shows strong negative emotions toward delayed rewards. Higher delay discounting and delay aversion (negative emotions) work together to exacerbate individuals' 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 individuals with ADHD through higher 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 children with ADHD and is considered an important clinical feature and potential treatment target (Barkley, 2015; Graziano & Garcia, 2016). Faraone et al. (2019) explicitly proposed in an expert consensus that emotional dysregulation in ADHD is mainly manifested in two aspects: emotional impulsivity (EI) and deficient emotional self-regulation (DESR). Emotional impulsivity refers to individuals showing abnormally high reactivity to emotional stimuli, leading to rapid escalation of emotional responses to stimuli, reaching impaired levels. Deficient emotional self-regulation refers to the inability to manage emotional experiences and control behavioral manifestations, resulting in abnormally slow return of activated emotional responses to baseline levels (Barkley, 2015; Faraone et al., 2019). Emotional dysregulation is considered a core component of ADHD and functional impairment, but as part of "hot" executive function, emotional dysregulation may more significantly affect the emergence of hyperactivity-impulsivity behaviors in children with ADHD. Specifically, children with ADHD have strong emotional reactivity, often manifested as irritability and tendency to produce negative emotions (Colonna et al., 2022; Liu et al., 2019). It is worth noting that when facing emotional stimuli, in addition to showing strong emotional impulsivity, individuals with ADHD show large and sustained increases in physiological arousal. Studies measuring autonomic nervous system indicators have found that children with ADHD show greater sympathetic dysregulation (i.e., enhanced electrodermal index) in negative emotion-induced 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, not only producing more impulsive behavior but also requiring "more behavior" to release excess energy. The most intuitive behavioral manifestation of emotional reactivity is impulsive behavior. Current research often treats emotional reactivity and impulsive behavior as one entity or confuses them (Faraone et al., 2019), without detailed discussion of how emotional reactivity, especially high emotional impulsivity, specifically affects the emergence of hyperactivity-impulsivity core symptoms in ADHD. Meanwhile, physiological indicators can provide more reliable and ecological data for emotional reactivity in children with ADHD and can be applied in future research to explore the relationship and mechanism between emotional dysregulation and hyperactivity-impulsivity in children with ADHD.

Emotional dysregulation in ADHD is considered to be caused by functional abnormalities in the amygdala, ventral striatum, and orbitofrontal cortex, which bottom-up affect other cognitive processing processes (Christiansen et al., 2019; Shaw et al., 2014). For example, emotional dysregulation makes it difficult for individuals with ADHD to regulate negative emotions brought by delayed rewards and amplifies emotional experiences, thereby showing more impulsive behavior. Lugo-Candelas et al. (2017) also found that children with ADHD are less effective than peers in allocating attention and cognitive control in emotional contexts, and failure in emotion regulation may exacerbate the manifestation of inattention and hyperactivity-impulsivity core symptoms in children with ADHD. Therefore, children with ADHD who have emotional dysregulation usually show high emotional reactivity and poor emotional regulation ability, making them more likely to show maladaptive behaviors in daily life or when facing emotional events. More importantly, emotional dysregulation exists more in daily life, while laboratory-induced emotional reactivity and regulation have limitations. Some researchers have used ecological momentary assessment to capture emotional fluctuation changes in children with ADHD in daily life and found that children with ADHD have higher emotional variability (i.e., intense emotional fluctuations), and this variability brings more severe functional impairment (Rosen et al., 2015; Walerius et al., 2018). However, there is still a lack of research from an ecological perspective exploring the dynamic relationship between spontaneous emotional fluctuation states and core symptoms in children with ADHD.

(4) Verifying the Mechanism of "Hot" Executive Function Affecting ADHD Core Symptoms from a Causal Manipulation Perspective

The previous sections mentioned that explanations for "hot" executive function deficits affecting hyperactivity-impulsivity core symptoms in children with ADHD mainly include: (a) poor delay of gratification ability related to self-control, leading to a tendency for immediate satisfaction and difficulty persisting in waiting; (b) abnormal reward processing caused by abnormal activation of the brain reward network, leading to motivation dysregulation and cue-behavior association failure; and (c) negative effects of emotional dysregulation manifested as emotional impulsivity and emotional regulation difficulties. The following sections will further provide empirical evidence from a causal (or near-causal) manipulation perspective to support and verify the specific mechanisms by which "hot" executive function affects hyperactivity-impulsivity deficits.

The orbitofrontal cortex (OFC) is an important brain region for "hot" executive function. Due to its location in the cerebral cortex, 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 have used tDCS or TMS techniques for neuromodulation of the OFC and found that stimulating the OFC can improve individuals' emotional experiences and risk decision-making behaviors (Howard et al., 2020; Ouellet et al., 2015; Yang et al., 2017). One study applied single-session tDCS stimulation to the DLPFC and OFC of 24 normal individuals and found that stimulating the OFC significantly reduced individuals'risk-taking behavior and risk decision-making, i.e., improved individuals' "hot" executive function (Nejati et al., 2018). Although the OFC plays an important role in "hot" executive function deficits in ADHD patients, current research on neuromodulation of the OFC is still limited. One study using tDCS with cathodal stimulation of the DLPFC and anodal stimulation of the OFC found that the most obvious improvement effect after stimulation was on cognitive flexibility in children with ADHD (Nejati et al., 2020). This study has not directly explored whether stimulating the OFC can improve "hot" executive function performance in children with ADHD, nor has it considered the impact of OFC stimulation on hyperactivity-impulsivity core symptoms in ADHD. 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 their "hot" executive function task performance but also effectively alleviate hyperactivity-impulsivity clinical symptoms.

In recent years, mindfulness practice has been considered an effective way to train "hot" executive function, mainly emphasizing individuals' conscious experience and awareness of their present physical and mental states with a non-judgmental attitude (Kabat-Zinn, 1994). Mindfulness combines focus training (such as letting go of attention) and emotion regulation training (such as cognitive reappraisal, accepting negative emotions, etc.), reducing individuals' stress and negative emotional awareness levels, and promoting the reshaping of benign reward mechanisms to improve individuals' "hot" executive function (J. Davis et al., 2018; Garland, 2021; Tang et al., 2015). More essentially, mindfulness training can significantly improve individuals' impulsive behavior, and its mechanism is also to reduce the impulsive drive brought by emotional or motivational involvement, thereby enhancing self-control ability (Yang Zhenzhi & Zeng Hong, 2023). Additionally, mindfulness training can improve awareness and focus levels, and through top-down control systems and emotion regulation, reduce individuals' motor impulsivity (i.e., untimely or unhelpful overt motor behavior) (Franco et al., 2016; Ron-Grajales et al., 2021). Mindfulness training can improve "hot" executive function-related ability levels, thereby helping to reduce hyperactivity and impulsivity behaviors in ADHD patients. A meta-analysis systematically reviewed the intervention effects of mindfulness training on ADHD and found that mindfulness-based intervention training could significantly reduce clinical symptoms such as hyperactivity-impulsivity in ADHD individuals, while also showing significant improvement in inattention (Cairncross & Miller, 2020). Researchers conducted an eight-week mindfulness intervention with children with ADHD aged 11-15 and their parents and found that parent-rated hyperactivity and impulsivity levels in children significantly decreased, with effects lasting at least eight weeks (van der Oord et al., 2012). Intervention studies on adults with ADHD also found that mindfulness meditation can improve individuals' emotion regulation strategies and impulse control levels (Mitchell et al., 2017). It is worth noting that mindfulness training can also cause changes in brain activation patterns and functional changes (Tang et al., 2015). For example, mindfulness training reduces amygdala activation when individuals process negative emotional pictures, 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, individuals show significantly reduced activation in brain regions involved in reward anticipation processing when facing addiction reward cues (Kirk et al., 2019), reducing individuals' impulsive choices. The above neuroimaging evidence indicates that mindfulness training can improve abnormal activation and functional connectivity in brain regions related to "hot" executive function, thereby improving abnormal behavioral levels of hyperactivity-impulsivity in individuals. This provides near-causal manipulation experimental evidence for verifying that "hot" executive function affects hyperactivity-impulsivity core symptoms in ADHD.

The OFC is located in the ventromedial area and is closely connected to the limbic system and reward network. However, it is still unclear how "hot" executive function deficits affect abnormal behavioral manifestations in children with ADHD through brain network and functional connectivity abnormalities. Considering that neuromodulation techniques can cause changes in other brain regions or brain networks by stimulating core nodes of cortical structure and function connections, future research can further adopt neuromodulation techniques to manipulate "hot" executive function and provide neural-level causal evidence for the mechanism affecting hyperactivity-impulsivity core symptoms in ADHD.

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

With the deepening of executive function research, there has been a growing trend of attention to the interrelationship between "cool" and "hot" executive functions in recent years (Zelazo, 2020). Researchers have found that "cool" and "hot" executive functions do not exist independently; there are dynamic interactions between them through top-down (cortical-limbic) and bottom-up (limbic-cortical) pathways (Moriguchi, 2021; Nejati et al., 2018; Zhu et al., 2018). On the one hand, "cool" executive function can regulate individuals' perception of reward value when facing reward anticipation, and can also control emotional responses and establish correct behavioral associations during outcome feedback, playing a key role in top-down cognitive control (Kryza-Lacombe et al., 2022; Luna, 2009). For example, stronger "cool" executive function can protect children and adolescents in environments full of temptation and threat, control emotional responses and motivational drives, make correct choices, and reduce environmental harm. On the other hand, "hot" executive function participates in value evaluation and risk decision-making, which directly affects goal-oriented "cool" executive function performance, playing a bottom-up emotion-evoking role. Better-developed "hot" executive function 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 motivational contexts (Ma et al., 2016). It can be seen that the emergence of certain behaviors is the result of the interactive coordination of "cool" and "hot" executive functions.

However, children with ADHD show abnormalities in both "cool" and "hot" executive functions, and there may be interactive effects that jointly affect the emergence of core symptoms. 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 abnormal behaviors in children with ADHD. However, whether these deficits can directly trigger hyperactivity-impulsivity behaviors also depends on the participation of "cool" executive function. That is, if children with ADHD have insufficient "cool" executive function, they cannot control the abnormal emotional responses and unreasonable behaviors brought by "hot" executive function deficits, thus showing more hyperactivity and impulsivity. For example, children with ADHD already have poor delay of gratification ability and are more inclined to immediate satisfaction. If combined with insufficient inhibitory control, they cannot resist conflicts caused by immediate temptation and show more impulsive behavior, which is often a common scenario in daily life. Additionally, attention control processes require the participation of "cool" executive function and consume a large amount of cognitive resources. Due to underdeveloped "hot" executive function, children with ADHD are more susceptible to strong drives from automated emotional factors, making individuals' attention more easily shifted to external stimuli. Even if an individual' s "cool" executive function is well-developed, if "hot" executive function is deficient, the individual still has difficulty controlling strong emotional drives and inhibiting automated processing, thus experiencing attention control problems. For example, in situations requiring attention concentration (classroom), children with ADHD not only have difficulty concentrating attention due to "cool" executive function deficits but may also be more easily driven by motivational or emotional cues due to "hot" executive function deficits, making it difficult to resist tempting distractors and attention being interfered with. From a neurobiological perspective, developmental imbalance or impairment between brain regions related to "cool" and "hot" executive functions may also lead to the emergence of core symptoms and other abnormal behaviors in children with ADHD. For example, the dual systems theory used to explain individual impulsivity suggests that imbalance between the cognitive control system (mainly dorsolateral prefrontal cortex, anterior cingulate cortex) and the socio-emotional system (mainly orbitofrontal cortex, amygdala) leads to impulsive behavior in adolescents (Shulman et al., 2016; Steinberg et al., 2008). Patients with ADHD show deficiencies in both systems (Capri et al., 2020), and studies have also found abnormal functional connectivity between the prefrontal cortex and amygdala or striatum networks in children with ADHD, which may be the reason for maladaptive behavior and impulsivity (Dias et al., 2015).

In summary, the interactive effects of "cool" and "hot" executive functions in children with ADHD jointly affect the emergence of inattention and hyperactivity-impulsivity core symptoms. Specifically, "cool" executive function deficits not only directly affect attention processing in children with ADHD 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 hyperactivity and impulsivity. "Hot" executive function deficits not only lead to motivation dysregulation and reward abnormalities but also bring more erroneous goal-directed behavior and automated processing due to bottom-up "dysregulation," thereby affecting individuals' 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 core symptoms in children with ADHD. Although this issue has received widespread attention from researchers, the mechanisms by which executive function deficits affect the emergence of ADHD core symptoms have not yet been fully elucidated. Based on existing theoretical and empirical research, this paper explains from a neuro-cognitive-behavioral developmental pathway why "cool" and "hot" executive function deficits can affect different ADHD core symptoms and attempts to build a cognitive bridge between neurodevelopmental abnormalities and core symptoms in children with ADHD. Furthermore, it proposes that "cool" and "hot" executive functions may be two cognitive pathways leading to different ADHD core symptoms, with "cool" executive function being the dominant factor for inattention and "hot" executive function being the dominant factor for hyperactivity-impulsivity. More importantly, this paper elaborates on the mechanisms and specific pathways by which subcomponents of "cool" and "hot" executive function deficits affect different ADHD core symptoms, providing research ideas for exploring the cognitive-neural mechanisms leading to ADHD core symptoms. However, some issues still require investigation, and future research can examine the following four aspects:

First, the theoretical mechanism by which "cool" and "hot" executive function deficits affect different ADHD core symptoms needs further testing and refinement. The pathogenesis of ADHD in children is still unclear. Exploring 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 the pathogenesis of ADHD at the cognitive level. Although previous studies have explored correlations between executive function deficits and ADHD core symptoms, there is still a lack of an integrated theoretical model explaining the cognitive-neural mechanisms by which executive function deficits affect different ADHD core symptoms. The previous sections systematically discussed the effects of "cool" and "hot" executive function deficits on different ADHD core symptoms and possible cognitive mechanisms. The validity of these mechanisms still needs support from empirical research. From a cognitive mechanism perspective, the emergence of ADHD inattention and hyperactivity-impulsivity core symptoms is mainly caused by executive function developmental deficits or deficiencies, and each subcomponent of "cool" and "hot" executive functions has unique and important roles in the pathways affecting ADHD core symptoms. Therefore, future research should incorporate executive function deficits into large-scale epidemiological investigations of ADHD, systematically explore the impact weights of "cool" and "hot" executive function deficits on different ADHD subtypes, and determine the explanatory power of each executive function subcomponent for different core symptoms of inattention and hyperactivity-impulsivity. Based on this, a cognitive model of executive function deficits affecting ADHD core symptoms should be constructed to provide scientific criteria for early screening and diagnosis of children with ADHD. Additionally, examining the impact of executive function developmental changes on ADHD core symptoms from a child development perspective is also very important. Existing cross-sectional studies have difficulty explaining 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 in childhood, they may have different impacts on ADHD core symptoms (O' Toole et al., 2018). Therefore, future research can also combine longitudinal tracking designs to reveal whether developmental changes in executive function can predict or affect the severity of ADHD core symptoms in children, testing and refining the cognitive mechanism by which executive function deficits affect ADHD core symptoms from a developmental perspective.

Second, further exploration of the neural basis and cognitive-neural mechanisms by which "cool" and "hot" executive function deficits affect ADHD core symptoms is needed. From a neural perspective, ADHD core symptoms in children are mainly closely related to structural and functional abnormalities in the prefrontal cortex, but the cognitive deficits and mechanisms behind this abnormal neural basis are currently in the exploration 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 bases can predict the severity of ADHD core symptoms, remain to be addressed. It is worth noting that the prefrontal cortex does not work independently; it 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 cortical-striatal-thalamic-cortical loop structure that functions as a whole (Jiang et al., 2018; Zhu et al., 2016). Therefore, abnormal neural loop structures centered on the prefrontal cortex may provide a bridge for understanding the impact of executive function deficits on 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. If the above brain regions are deficient, it not only limits executive function development but also causes abnormal activation in related brain networks through functional connectivity, both acting on the emergence of ADHD core symptoms. Future research can further explore whether specific brain regions activated in children with ADHD under different executive function tasks and functional connectivity with other brain networks are abnormal, striving to identify the neural basis and network models by which "cool" and "hot" executive function deficits affect different ADHD core symptoms. Meanwhile, most research in the field on the neural basis of ADHD is based on recording and association perspectives, lacking causal (or near-causal) manipulation empirical evidence to support the impact of "cool" and "hot" executive function deficits on different ADHD core symptoms and their cognitive-neural mechanisms. Given that neuromodulation techniques can cause indirect changes in "downstream" brain structures and network functional connectivity by stimulating the cortex, future research can also integrate neuromodulation techniques into brain imaging studies, verifying the cognitive-neural model of executive function deficits affecting ADHD core symptoms from a causal manipulation perspective.

Third, examine the impact of "cool" and "hot" executive function deficits on ADHD core symptoms in children from an ecological perspective. Research has found obvious individual heterogeneity in executive function deficits in children with ADHD (Bunger et al., 2021; Kofler et al., 2019). Specifically, there are significant differences between laboratory task-based and parent/teacher rating scale scores for executive function in children with ADHD. Only about 52% of children with ADHD show executive function deficits on laboratory tasks, while parents/teachers report executive function deficits in over 90% of children with ADHD (Sjöwall & Thorell, 2019). Moreover, clinicians rely more on parent/teacher-reported rating scale scores for diagnosis because they better reflect the clinical manifestations of children with ADHD in daily life (DSM-IV, 2013). This also indicates that the ecological validity of current laboratory tasks is insufficient, making it difficult to truly reflect individuals' abnormal manifestations in daily life (Barkley & Murphy, 2011; Bunger et al., 2021). Especially existing laboratory tasks mostly focus on "cool" executive function, with less attention to how "hot" executive function with high emotional or motivational involvement affects the emergence of ADHD core symptoms. Meanwhile, motivational and emotional-driven cues are everywhere in daily life, requiring not only the participation of "hot" executive function but also more involvement of the interaction between "cool" and "hot" executive functions (Zelazo, 2020). For example, difficulty resisting distractors is the result of a struggle between inhibitory control and temptation drive in ADHD. When tempting distractors overcome control ability, individuals will interrupt attention persistence and show inattention. That is, even if an individual' s "cool" executive function is well-developed, if "hot" executive function is deficient and motivational drive is too strong, abnormal behavioral manifestations are still difficult to avoid (Yang Zhenzhi & Zeng Hong, 2023). However, children with ADHD have abnormalities in both "cool" and "hot" executive functions, and their interaction will prompt children with ADHD to show more abnormal behaviors related to executive function deficits in daily life. Existing research largely ignores the above viewpoints and one-sidedly views the relationship between executive function deficits and core symptoms in children with ADHD. Therefore, future research should examine from an ecological perspective how the interaction between "cool" and "hot" executive function deficits affects the emergence of core symptoms in daily life in children with ADHD, and attempt to answer the question of individual heterogeneity in executive function deficits in children with ADHD.

Finally, develop psychological intervention and neuromodulation programs with long-term effects on ADHD core symptoms in children based on "cool" and "hot" executive function deficits. Theoretically, interventions targeting executive function deficits have important clinical value, as they not only improve ADHD core symptoms and social function but may also have lasting improvement effects (Karr et al., 2018). However, the improvement effects of existing executive function intervention programs on ADHD-related core symptoms are not significant, or it is difficult to "normalize" the problem behaviors of children with ADHD. The reason for this low efficiency may be that these intervention targets only focus on abilities themselves, without considering the relationship between executive function subcomponent deficits and ADHD abnormal behavioral manifestations, making the far-transfer effects of interventions greatly reduced (Kofler, Wells et al., 2020). The cognitive-neural mechanisms proposed in the previous sections have clarified the effects of "cool" and "hot" executive function deficits on different ADHD core symptoms, suggesting that future research can start from cognitive intervention to improve inattention core symptoms by manipulating "cool" executive function in children with ADHD, or train abilities related to "hot" executive function to reduce hyperactivity-impulsivity symptoms. Meanwhile, future research can also use neuromodulation techniques to examine whether single or multiple sessions of TMS/tDCS stimulation of DLPFC reduce inattention and other abnormal behaviors by improving "cool" executive function, or whether stimulating OFC and other brain regions can increase behavior and emotion control ability by improving "hot" executive function. It is worth noting that executive function subcomponent deficits in children with ADHD have strong individual differences. For example, for inattention symptom manifestations, some children with ADHD may be caused by working memory representation difficulties that prevent correct target information selection, while others may be related to insufficient inhibitory control that makes it difficult to resist external interfering stimuli. If not distinguished at the individual level, intervention targets are difficult to match individual ability deficits and developmental levels, greatly limiting the effectiveness and long-term nature of improving their core symptoms. Therefore, based on clarifying the pathogenesis of ADHD in children, future research should achieve personalized intervention and precise treatment for ADHD core symptoms in children based on "cool" and "hot" executive function deficits from two main lines of cognitive intervention and neuromodulation, striving to achieve long-lasting intervention effects.

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Note: Figure translations are in progress. See original paper for figures.