

# FEATURES

## “Rationality”: The Role of Hot Executive Functions in ADHD

Photo from ravil\_s on depositphotos.com.

Jae Dahin Ji '27

(figures courtesy of Natalie Zhang '27)

### Introduction

Attention-deficit/hyperactivity disorder (ADHD) has long been associated with executive dysfunction, a theory that dominates modern research in cognitive neuroscience and clinical-developmental psychology. In recent years, the concept of executive functions (EFs)—modes of higher-order cognition that are essential for adaptive, goal-directed behaviors—has been categorized by experts into two categories: hot EFs, which involve “processing of information related to reward, emotion, and motivation,” and cold (also known as cool) EFs, which concern “purely cognitive information processing” (Salehinejad et al., 2021). The field has historically neglected the role of these hot factors and focused on cognitive skills such as academic achievement, school readiness, and social

behavior (Brock et al., 2009; Peterson and Welsh, 2014). Because ADHD’s most prominent symptoms manifest in impaired time management, mental programming, and working memory, the study of hot EFs in ADHD patients is too often viewed as trivial.

In ADHD research, scientists often conduct analyses using functional MRI (fMRI), which measures brain activity by detecting changes in blood flow and thereby provides insights into brain regions involved in specific cognitive tasks or emotional responses. Evidence from the striato-limbic area, the key neuroanatomical player in regulating emotional processing and motivation, has provided new insights into the impact of hot EFs on ADHD. By weighing reward and emotion processing as major implications of the affective EF system, researchers have found that abnormalities in hot EFs are as relevant as those in cold EFs in individuals with ADHD. The importance of hot EFs in patients with ADHD has been elucidated through experiments investigating numerous neurological processes including reward

anticipation, reward delivery, delay discounting, orbitofrontal cortex activation, and emotional regulation.

## Reward Anticipation

The ventral striatum (VS) plays a crucial role in reward processing, motivation, and reinforcement learning in the brain, and reduced VS activation during reward anticipation is one of the most consistent findings regarding motivation in ADHD patients. Plichta and Scheres (2014) reiterated this in their meta-analysis of eight fMRI studies, and in this review, they discussed the monetary incentive

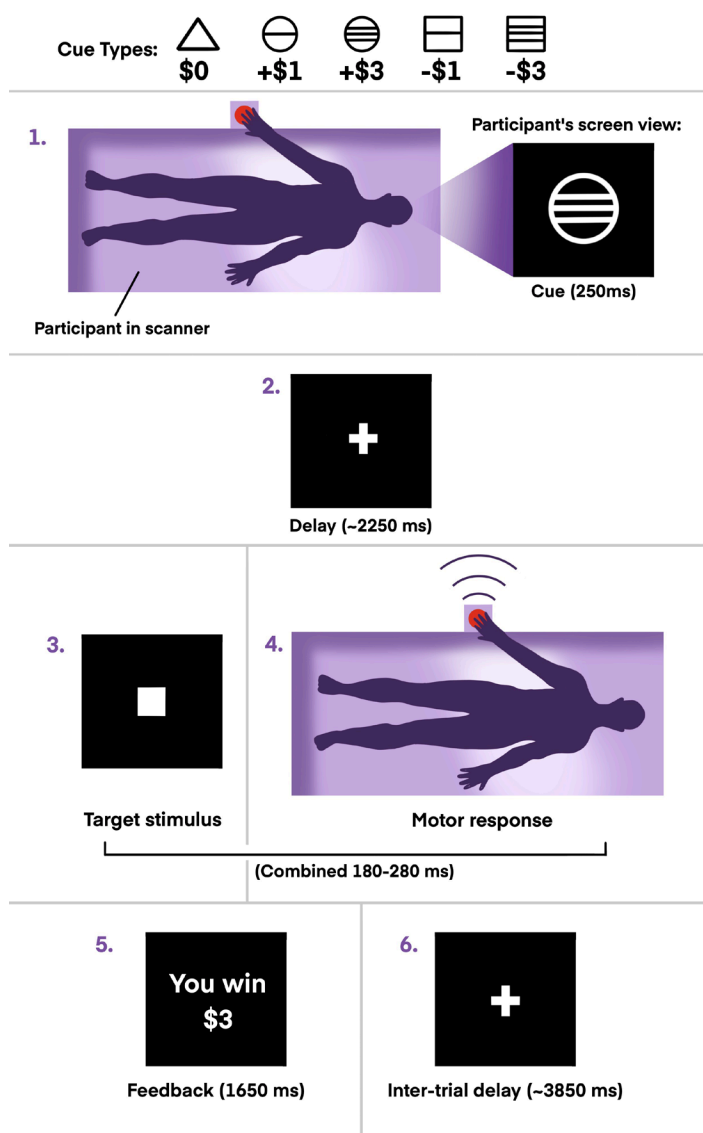
delay (MID) task, a test utilized to examine the brain activity associated with reward anticipation. A typical MID task might involve five phases: (1) presenting a cue; (2) delaying with a crosshair display for a random period of time; (3) flashing a brief target stimulus signaling the opportunity to win money; (4) executing a simple motor response (for example, pressing a button); and (5) providing feedback on the participant's performance via the shortening or lengthening of the response time window (**Fig. 1**). This methodology effectively allows for the examination of both reward (a monetary prize) anticipation and feedback processing (changes in response time). Scheres et al. (2007) found a VS hypoactivation during these two processes in adolescents with ADHD, compared to matched controls through a blood-oxygen-level-dependent (BOLD) fMRI investigation. This may be due to diminished temporal foresight, a hot executive dysfunction typical of ADHD patients (Weissenberger et al., 2021).

The fact that the symptom domain of hyperactivity-impulsivity—which is highly associated with hot factors—was negatively correlated with VS response, while that of inattention—which is a primarily cold factor—showed a much more minimal pattern lends further support to a significant relationship between hot EFs and ADHD. Indeed, Ströhle et al. (2008) confirmed that reduced activation of the left VS during reward anticipation “was associated with impulsivity and total ADHD symptom severity” in adult males, a phenomenon that Plichta and Scheres (2014) observed in all ADHD patients.

## Reward Delivery

Not only do individuals with ADHD display decreased VS activity when rewards are anticipated, but they also display increased VS activity when rewards are actually delivered. A 2014 fMRI study using an altered MID task maintained that BOLD responses in the VS increased in the controls but not in the ADHD group during reward anticipation; moreover, upon reward delivery, ADHD patients demonstrated significantly greater BOLD responses in the VS and left dorsal striatum (Furukawa et al., 2014; **Fig. 2**). This provides evidence for the role of hot EFs in ADHD because it is consistent with established findings detailing an ADHD-associated impairment in predictive dopamine signaling. With more research, this could be used as proof that

### 0. Pre-task: Participant learns cue — monetary value associations



**Fig. 1.** Diagram of a MID task (courtesy of Natalie Zhang '27)

the transfer of dopamine release from established reinforcers (i.e., rewards) themselves to the predictive cues after repeated pairings is deficient in ADHD patients, a phenomenon that has been observed in non-human primates and rats (Ljungberg et al., 1992; Pan et al., 2005).

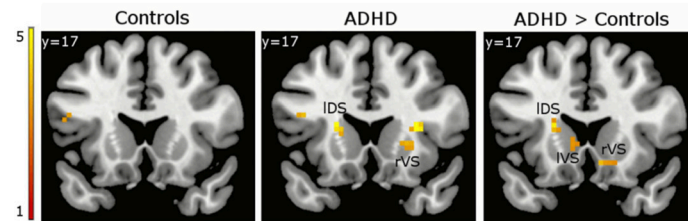


Fig. 2. "Striatal responses to reward delivery in ADHD and Control groups" (Furukawa et al., 2014)

Similarly, increased reward signaling within the superior frontal gyrus and VS was seen in adolescents with ADHD relative to matched controls even when evaluating interference control—a traditionally cold EF—via the Stroop color-word task, which measures the interference between naming the color of a word and reading the word itself and is often used to assess cognitive flexibility and selective attention (Ma et al., 2016). Functional connectivity analyses revealed a hyperconnectivity between the VS and motor regions; the association of connectivity with performance improvement in the controls but not in the ADHD group provides insight into the reduced activation of the VS during feedback processing in the aforementioned MID task studies.

## Delay Discounting

Another small sector of current ADHD research focuses on the deficits to delay discounting in ADHD patients. Odum (2011) defines delay discounting as “the decline in the present value of a reward with delay to its receipt,” and making the conventionally wiser choice between impulsive (smaller but sooner) and self-controlled (larger but later) options is especially challenging for those with the hyperactivity-impulsivity subtype. Although delay discounting tasks measure both cold and hot EFs, they provide explicit evidence for the gravity of the latter in ADHD, as the disorder alters three hot EFs: impaired motivational restraint, decreased tolerance to delays, and poor temporal foresight. Reduced neural activation in ADHD patients lies mostly in

the ventrolateral and dorsolateral prefrontal cortex (PFC), which are involved in both hot and cold EFs such as decision-making, planning, problem-solving, and cognitive control (Ortiz et al., 2015; Rubia et al., 2009). Therefore, there is also a cause to study responses from the hot EF system to delays in rewards among ADHD groups.

## Orbitofrontal Cortex (OFC) Activation

Findings on the altered reward processing networks in ADHD patients are more inconsistent when considering OFC activation. The OFC is involved in decision-making, reward processing, and social behavior, and it integrates sensory information to guide appropriate responses and evaluate the value of stimuli. Several studies found enhanced OFC activation: for example, Ströhl et al. (2008) saw that compared to healthy controls, adult male ADHD patients showed increased activity in the right OFC upon experiencing an experimental “gain” via another MID task. Conversely, other discoveries highlight a distinctly reduced OFC activation during reward delivery. Notably, OFC underactivation was evident in a study of ADHD patients and controls who performed a modified Go/NoGo task, a cognitive test that traditionally evaluates response inhibition, requiring participants to respond to certain stimuli (Go trials) while withholding responses to others (NoGo trials) (Dibbets et al., 2009). Although

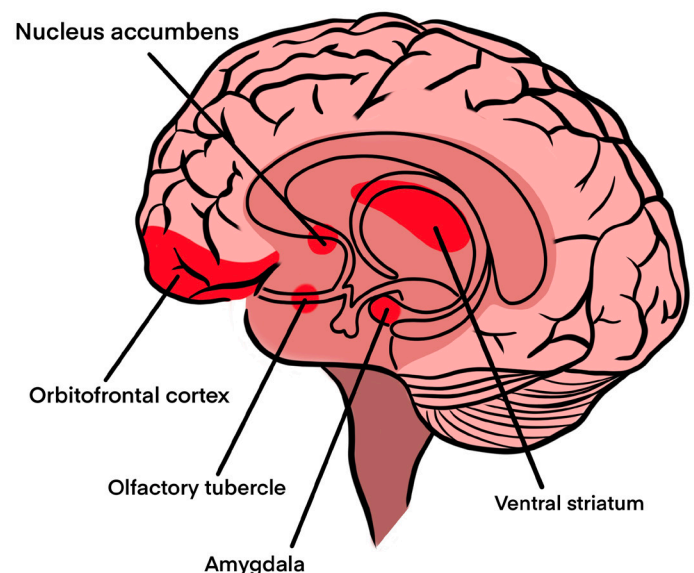


Fig. 3. Neuroanatomical locations of relevant striato-limbic areas (courtesy of Natalie Zhang '27)



researchers did not detect any behavioral signals that would imply feedback-related differences between the two, the ADHD group displayed an undeniable underactivation of the OFC during positive feedback.

In another case, Cubillo et al. (2012) gathered medication-naïve subjects—individuals who had not received any medication for a specific condition or disorder prior to assessment or treatment—with a childhood diagnosis and persistent symptoms of ADHD in adulthood, and Cubillo et al. commented that it is possible to attribute this decreased OFC activation to conduct disorder comorbidity because children with conduct disorder already suffer from an OFC underactivation. However, this hypothesis is still ongoing, and there is no extensive evidence to either support or refute it. These inconsistencies with regard to the OFC also align with its typical irregularity (compared to the VS's relative lack thereof) in reward processing, so the OFC provides no noticeably discernable or valid contribution to this discussion.

## Emotion Regulation

In parallel, emotion regulation, a newer topic of interest in 21st-century ADHD research, is another major factor worth investigating to prove the magnitude of hot EFs in afflicted individuals. Upon concluding a longitudinal study with the discovery that individuals whose ADHD persists into adulthood report higher symptoms of emotional impulsivity, Barkley and Fischer (2010) reasoned that emotional impulsivity “[arises] from deficits in the ‘hot’ executive frontal network.” This may be related to insufficient top-down (guided by higher-level factors such as beliefs, expectations, and prior knowledge, influencing perception, attention, and interpretation of information) control over increased bottom-up (guided by processing where information is analyzed based on incoming sensory input, influencing perception and cognition without higher-level guidance) emotional reactivity. This is in accordance with the significant role of the cold executive system in an ADHD brain. Given this, one may predict that ADHD patients will display reduced striato-limbic activation when encountering negatively valenced (having an emotional or evaluative quality) distractors and enhanced activation for positively valenced stimuli.

However, in 2011, Posner et al. examined neural activity in adolescents with and without ADHD

while they performed a working memory task in which fearful faces would be subliminally presented in the midst of other cues. They found that the ADHD patients had a more activated right amygdala (which plays a central role in processing emotions, particularly fear, and in modulating emotional responses and memory consolidation) and greater connectivity between the amygdala and lateral PFC (lPFC) than the control subjects did. These results suggest that amygdala activation during tasks that mimic monetary loss is also abnormally high (Wilbertz et al., 2017), so ADHD patients are at a distinct functional disadvantage in terms of controlling their exaggerated responses to such stimuli even when reward processing and emotion regulation collide.

## Conclusion

Research presents that hot EFs are indispensable to individuals with ADHD, especially when assessing reward processing and emotion regulation in the striato-limbic region. As Cubillo et al. (2012) mention, a caveat is that “very few fMRI studies have tested for neurofunctional deficits during emotion processing in ADHD,” and even fewer have been conducted in adult ADHD. Findings are also more inconsistent as subjects’ ages increase due to limitations that become more pronounced with time, such as “small sample sizes, high rates of comorbidity, long-term medication history, and the need for a retrospective diagnosis of ADHD in childhood.”

Moreover, nearly all of the neuroimaging described was obtained in a region of interest (ROI), so there is a critical lack of whole-brain studies. This is due to several reasons: (1) while whole-brain studies provide a comprehensive view, targeted scans can offer detailed insights into the neural mechanisms underlying ADHD symptoms; (2) whole-brain studies imply several technical and methodological challenges, such as standardizing protocols and gaining access to sophisticated resources; and (3) the heterogeneity of ADHD makes it difficult to pinpoint specific brain regions or networks that are consistently affected across all individuals with ADHD. This calls for an immediate movement toward comprehensive and replicable early intervention studies in younger patients. Furthermore, it may be integral to include those who only experience certain symptoms of ADHD—as opposed to holding a formal diagnosis—in

ADHD studies, as it is important to recognize both the inflexibility of psychological diagnostic criteria and barriers to accessible healthcare. Ultimately, although there are some conflicting judgments in the current literature, the fMRI evidence delineated here—and the surplus that exists outside of it—demonstrates that there is, at the very least, enough of an impetus to warrant more thorough investigations of the dynamic between hot EFs and ADHD.

## References

- Barkley, R. A., & Fischer, M. (2010). The unique contribution of emotional impulsiveness to impairment in major life activities in hyperactive children as adults. *Journal of the American Academy of Child & Adolescent Psychiatry*. 49(5), 503–513.
- Brock, L. L., Rimm-Kaufman, S. E., Nathanson, L., & Grimm, K. J. (2009). The contributions of 'hot' and 'cool' executive function to children's academic achievement, learning-related behaviors, and engagement in kindergarten. *Early Childhood Research Quarterly*. 24(3), 337–349.
- Cubillo, A., Halari, R., Smith, A., Taylor, E., & Rubia, K. (2012). A review of fronto-striatal and fronto-cortical brain abnormalities in children and adults with Attention Deficit Hyperactivity Disorder (ADHD) and new evidence for dysfunction in adults with ADHD during motivation and attention. *Cortex*. 48(2), 194–215.
- Dibbets, P., Evers, L., Hurks, P., Marchetta, N., & Jolles, J. (2009). Differences in feedback- and inhibition-related neural activity in adult ADHD. *Brain and Cognition*. 70(1), 73–83.
- Furukawa, E., Bado, P., Tripp, G., Mattos, P., Wickens, J. R., Bramati, I. E., Alsop, B., Ferreira, F. M., Lima, D., Tovar-Moll, F., Sergeant, J. A., & Moll, J. (2014). Abnormal striatal BOLD responses to reward anticipation and reward delivery in ADHD. *PLoS ONE*. 9(2), e89129.
- Ljungberg, T., Apicella, P., & Schultz, W. (1992). Responses of monkey dopamine neurons during learning of behavioral reactions. *Journal of Neurophysiology*. 67(1), 145–163.
- Ma, I., Van Holstein, M., Mies, G. W., Mennes, M., Buitelaar, J., Cools, R., Cillen, A. H. N., Krebs, R. M., & Scheres, A. (2016). Ventral striatal hyperconnectivity during rewarded interference control in adolescents with ADHD. *Cortex*. 82, 225–236.
- Odum, A. L. (2011). Delay discounting: I'm a k, you're a k. *Journal of the Experimental Analysis of Behavior*. 96(3), 427–439.
- Ortiz, N., Parsons, A., Whelan, R., Brennan, K., Agan, M., O'Connell, R., Bramham, J., & Garavan, H. (2015). Decreased frontal, striatal and cerebellar activation in adults with ADHD during an adaptive delay discounting task. *Acta Neurobiologiae Experimentalis*. 75(3), 326–338.
- Pan, W.-X., Schmidt, R., Wickens, J. R., & Hyland, B. I. (2005). Dopamine cells respond to predicted events during classical conditioning: Evidence for eligibility traces in the reward-learning network. *The Journal of Neuroscience*. 25(26), 6235–6242.
- Peterson, E., & Welsh, M. C. (2014). The development of hot and cool executive functions in childhood and adolescence: Are we getting warmer? *Handbook of Executive Functioning*. pp. 45–65).
- Plichta, M. M., & Scheres, A. (2014). Ventral-striatal responsiveness during reward anticipation in ADHD and its relation to trait impulsivity in the healthy population: A meta-analytic review of the fMRI literature. *Neuroscience & Biobehavioral Reviews*. 38, 125–134.
- Posner, J., Nagel, B. J., Maia, T. V., Mechling, A., Oh, M., Wang, Z., & Peterson, B. S. (2011). Abnormal amygdalar activation and connectivity in adolescents with attention-deficit/hyperactivity disorder. *Journal of the American Academy of Child & Adolescent Psychiatry*. 50(8), 828–837.e3.
- Rubia, K., Halari, R., Christakou, A., & Taylor, E. (2009). Impulsiveness as a timing disturbance: Neurocognitive abnormalities in attention-deficit hyperactivity disorder during temporal processes and normalization with methylphenidate. *Philosophical Transactions of the Royal Society B: Biological Sciences*. 364(1525), 1919–1931.
- Salehinejad, M. A., Ghanavati, E., Rashid, M. H. A., & Nitsche, M. A. (2021). Hot and cold executive functions in the brain: A prefrontal-cingular network. *Brain and Neuroscience Advances*. 5, 239821282110077.
- Scheres, A., Milham, M. P., Knutson, B., & Castellanos, F. X. (2007). Ventral striatal hypo-responsiveness during reward anticipation in attention-deficit/hyperactivity disorder. *Biological Psychiatry*. 61(5), 720–724.
- Ströhle, A., Stoy, M., Wrase, J., Schwarzer, S., Schlagenhauf, F., Huss, M., Hein, J., Nedderhut, A., Neumann, B., Gregor, A., Juckel, G., Knutson, B., Lehmkuhl, U., Bauer, M., & Heinz, A. (2008). Reward anticipation and outcomes in adult males with attention-deficit/hyperactivity disorder. *NeuroImage*. 39(3), 966–972.
- Weissenberger, S., Schonova, K., Büttiker, P., Fazio, R., Vnukova, M., Stefano, G. B., & Ptacek, R. (2021). Time perception is a focal symptom of attention-deficit/hyperactivity disorder in adults. *Medical Science Monitor*. 27.
- Wilbertz, G., Delgado, M. R., Tebartz Van Elst, L., Maier, S., Philipsen, A., & Blechert, J. (2017). Neural response during anticipation of monetary loss is elevated in adult attention deficit hyperactivity disorder. *The World Journal of Biological Psychiatry*. 18(4),