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Editorial: Neural Correlates of Sluggish Cognitive Tempo: Biological Evidence of a Distinct Clinical Entity?

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Attention-deficit/hyperactivity disorder (ADHD) is a heterogeneous disorder encompassing a wide array of clinical presentations, levels of impairment, etiologies, and neurobiological correlates. Despite this well-known heterogeneity, most research into the pathophysiology of ADHD has relied on comparisons between typically developing youth and those with the disorder (or perhaps further stratifying by *DSM*-defined ADHD subtypes). Although informative, this approach assumes a level of pathophysiologic homogeneity that belies the large and growing body of literature underscoring diverse neurobiological and neuropsychological profiles subsumed under the umbrella of this complex syndrome.¹ For example, deficits in executive functions are characteristic of ADHD, yet meta-analysis suggests that these deficits are neither necessary nor sufficient to define all cases² and that substantial overlaps in levels of executive functioning exist between children with ADHD and their typically developing peers.¹ Likewise, neuroimaging research points to distinct neural circuits underlying different clinical presentations of ADHD, suggesting that, within the diagnosis of ADHD, the neural circuits that are most salient to the disorder can differ from one child to the next.³ Refining our understanding of ADHD subtypes not only on the basis of symptoms, but also by incorporating neurobiology, physiology, and neurocognitive profiles, could help propel the field toward greater specificity in diagnosis and treatment.

Sluggish cognitive tempo (SCT) provides a candidate phenotype that could help define a clinical entity that in current practice is commonly subsumed under ADHD. SCT symptoms are often observed in children with ADHD,^{4–6} yet despite this co-occurrence, a growing body of research suggests that SCT symptoms, such as seeming to be “in a fog,” daydreaming, and being sleepy or lethargic, might be distinct from those of ADHD.^{7,8} Furthermore, SCT symptoms are stable over time⁷ and are independently associated with social impairment, lower self-esteem, and difficulties with emotion regulation.⁸ Currently, the SCT construct lacks a definition sufficiently well agreed upon to constitute a diagnosis. For example, there is no consensus on a symptom set for SCT, although a meta-analysis has pointed to 13 items that could provide a starting point.⁷ Furthermore, only a few studies have examined biological factors related to SCT. Improving our understanding of the biological underpinnings of SCT could provide additional evidence to help determine whether the construct warrants consideration as a distinct clinical entity or diagnosis.

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In their current article, Camprodon-Rosanas *et al.*⁹ examined associations between SCT symptoms and brain morphometry, functional connectivity, and white matter microstructure in a large sample (N = 178) of children (8–12 years old) from the general population. Perhaps the most striking finding was that as SCT symptoms increased, volumes within the frontal lobe increased, including the right prefrontal and premotor cortices. Furthermore, similar to studies of ADHD,^{10–12} SCT symptoms were associated with altered functional connectivity of the brain's default mode network (DMN), a neural circuit implicated in inattention and mind-wandering.¹³ More specifically, less pronounced anti-correlations between a key node of the DMN and nodes of a task-positive network were found. These decreases in anti-correlations between the DMN and task-positive nodes persisted after accounting for ADHD symptoms, suggesting that these neural correlates of SCT are distinct from those of ADHD. These findings raise important questions about the relation between the DMN and attentional symptoms. For example, several studies of ADHD have reported decreased anti-correlations between the DMN and task-positive networks,^{10–12} yet in these studies SCT symptoms were not measured. Thus, the results from Camprodon-Rosanas *et al.* invite future studies to examine whether anti-correlations between the DMN and task-positive nodes are in fact related to ADHD, as has been reported, or whether they are better explained by the presence of SCT symptoms.

The results from Camprodon-Rosanas *et al.* build on emerging evidence of brain differences related to SCT. To date, only 1 prior neuroimaging study has examined SCT in a small sample (n = 16) of adolescents with ADHD and typically developing adolescents (n = 13).¹⁴ Results suggested that teens with ADHD had decreased brain activity in the superior parietal lobe while preparing to respond on a cued Flanker task. Within the ADHD group, increased SCT symptoms were associated with decreased activity in the same brain region over and above the contribution of inattentive symptoms (hyperactive/impulsive symptoms were not considered). This study is important because it demonstrated that SCT symptoms play a distinct role in neural alterations in ADHD. Camprodon-Rosanas *et al.* further these results by demonstrating neural correlates of SCT in a general population, thereby providing further support SCT as a distinct clinical entity.

Contrary to several studies associating ADHD with smaller volumes of the frontal lobes,^{15–17} the current study associated SCT symptoms with larger frontal volumes. This finding highlights a potential pitfall of pooling dissimilar attentional symptoms under the umbrella of “ADHD.” For example, because their relations with frontal lobe volumes are in opposing directions, grouping children with ADHD with and without SCT into 1 “ADHD” category and comparing them with typically developing children might obscure important pathophysiologic features of attentional symptoms. In other words, homogenizing clinical presentations that are in fact heterogeneous risks could mask meaningful neurobiological and neurocognitive correlates. Indeed, this could explain some of the inconsistent findings, and small effect sizes, reported in neurobiological and genetic studies of ADHD.¹⁸

In the current study, SCT was measured using 4 parental report items on the Child Behavior Checklist.¹⁹ Future research could benefit from the use of a more sensitive instrument, such as the SCT scale,²⁰ and from measurements of SCT taken in more than 1 setting, for example, including a teacher report in addition to a parental report. Furthermore, the current

study followed the recommendations of the FP7-ERC Ethics Review Committee and used a 1.5-T magnetic resonance imaging scanner for this study to decrease childhood exposure to magnetic fields. Future studies could provide more detailed information about neural substrates using a more powerful magnet. In addition, longitudinal studies of brain structure and function could help define neurodevelopment trajectories related to SCT symptoms and might test whether these trajectories are distinct or convergent with those of ADHD.

As evidence accumulates for SCT as a meaningful biological subtype of neurodevelopmental disorders, it is worth-while to consider the impact of its name. In addition to conveying a pejorative tone, SCT implies a slower rate of cognitive processing. Although some research has provided empirical support for decreased processing speed in SCT,^{5,21} findings have not been consistent.^{6,22,23} Because there is no consensus that SCT is characterized by decreased processing speed on neuropsychological testing, and because the term could appear disparaging, choosing another name might be sensible. Russell A. Barkley proposed *concentration deficit disorder* as a term that would be more accurate and less offensive.²⁴

The present study takes an important step forward in the study of disorders of attention by providing evidence of neural correlates of SCT in a large population-based sample. Furthermore, this work sets the stage for new research that can further explore and characterize SCT, ultimately helping the field determine whether this construct should be classified a distinct clinical entity.

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