Becker, S. P., & Willcutt, E. G. (2019). 2. *European Child and Adolescent Psychiatry, 28*, 603-613. doi:10.1007/s00787-018-1136-x

Advancing the Study of Sluggish Cognitive Tempo via DSM, RDoC, and Hierarchical Models of Psychopathology

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Disclosures and acknowledgements: Dr. Becker is supported by grants from the National Institute of Mental Health (NIMH; K23MH108603 and R03MH109787) and the Institute of Education Science (IES; R305A160064 and R305A160126). Dr. Willcutt is supported by grants from the National Institute of Child Health and Human Development (NICHD; P50HD27802, R01HD68728; R24HD75460). Drs. Becker and Willcutt report no financial relationships with commercial interests. The content is solely the responsibility of the authors.

Abstract

Sluggish cognitive tempo (SCT) is separable from attention-deficit/hyperactivity disorder (ADHD) and other psychopathologies, and growing evidence demonstrates SCT to be associated with impairment in both children and adults. However, it remains unclear how SCT should optimally be conceptualized. In this article, we argue that multiple models of psychopathology should be leveraged in order to make substantive advances to our understanding of SCT. Both categorical and dimensional approaches should be used, including the Diagnostic and Statistical Manual of Mental Disorders (DSM) nosology, the Research Domain Criteria (RDoC) initiative, and hierarchical models of psychopathology. Studies are needed to determine whether individuals categorized with SCT can be reliably identified and differentiated from individuals without SCT in pathophysiological, neuropsychological, behavioral, and daily life functioning. Studies are also needed to evaluate the validity and utility of SCT as a transdiagnostic and dimensional construct. In considering SCT as a dimensional and potentially transdiagnostic construct, we describe ways in which SCT might be examined within the RDoC framework, including negative valence systems, cognitive systems, and arousal/regulatory systems, as well as within hierarchical models of psychopathology. Conceptualizing SCT within both categorical and dimensional models of psychopathology will help to better understand the causes, developmental pathways, and clinical implications of SCT, both as a construct in its own right and also in relation to other psychopathologies.

Keywords: ADHD; diagnosis; Diagnostic and Statistical Manual of Mental Disorders; psychiatric diagnosis; nosology; psychopathology; Research Domain Criteria; sluggish cognitive tempo; transdiagnostic

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Sluggish cognitive tempo (SCT) has been studied to varying degrees for over four decades, with a sharp increase in interest in the SCT construct over the past 15 years [1,2]. Much of this interest has focused on whether SCT symptoms are empirically distinct from or redundant with attention-deficit/hyperactivity disorder inattention (ADHD-IN) symptoms [3,4]. A recent meta-analysis found that 13 putative SCT items consistently loaded on an SCT factor as opposed to an ADHD-IN factor [5]. These items are displayed in Table 1 and include aspects of inconsistent alertness (e.g., daydreaming, mental confusion or 'fogginess') and slowed behavior (e.g., underactive, lethargic). More recently, SCT has been shown to be statistically distinct from other psychopathology domains including depression, anxiety, and daytime sleepiness [6-11]. Importantly, there is growing evidence that SCT is also related to a range of daily life impairments, including social withdrawal and loneliness, increased emotion dysregulation, poorer sleep quality and increased daytime sleepiness, and lower grades, with most associations between SCT and impairment remaining after controlling for ADHD and other psychopathology symptoms [5,12,13]. Preliminary findings also link SCT to poorer study skills, lowered self-esteem, and increased suicide risk [7,11,14,15]. Given these findings, SCT has been identified as an important construct to be studied in its own right [5,12,13].

Despite the increased research attention devoted to SCT, an important question remains unanswered: *exactly how should SCT be conceptualized?* We believe the extant research base is inadequate for answering this question at this time, yet at least two possibilities have emerged. Barkley has proposed that SCT may be a distinct attention disorder that frequently occurs with, but is nonetheless separate from, ADHD [13,16,17]. Alternatively, it has been suggested that SCT may be a psychopathological dimension or a transdiagnostic process that meaningfully predicts risk and impairment across a range of psychopathologies [2,5]. These two possibilities largely reflect differing approaches to understanding and evaluating the nature and structure of mental health and dysfunction. On one hand, the Diagnostic and Statistical Manual of Mental Disorders (DSM) [18] is a categorical diagnostic system. On the other hand, there is increasing interest in dimensional models that include the National Institute of Mental Health (NIMH) Research Domain Criteria (RDoC) initiative [19-21] as well as hierarchical models of psychopathology [22,23]. The SCT construct is unique in that it is not currently included in any diagnostic nosology and therefore remains largely unfamiliar to researchers, clinicians, and patients alike. As such, SCT is far less subject to the reification of categorical diagnoses that have been included in the DSM for decades [24]. Thus, the SCT construct may be a useful exemplar for examining psychiatric diagnosis as

envisioned through both categorical (e.g., DSM) and dimensional (e.g., RDoC) frameworks. In this paper, we describe how SCT may be examined within DSM, RDoC, and hierarchical models of psychopathology to make a substantive advance to our understanding of SCT.

Key Considerations for Examining SCT within the DSM Framework

We begin with the DSM framework since it is the current framework used in psychiatric nosology and diagnosis (along with the closely related International Classification of Diseases; ICD), and it is the DSM framework that is often used to define "diagnostic validity" [5]. The DSM (and ICD) uses a categorical approach to psychiatric diagnosis in which a set of descriptive, behavioral symptoms are evaluated to determine whether an individual meets an established diagnostic cutoff (along with other criteria such as symptom duration and functional impairment). As such, an initial starting point is to establish a standard of symptoms that operationalizes the measurement of SCT across studies in order to identify participants meeting an identified cutoff. Significant progress has been made toward this end as empirically-based SCT scales have been developed and validated [7,9,25-28]. Drawing from studies using these and other SCT measures, meta-analytic findings indicated 13 "constructs" to be optimal for defining the SCT construct – at least as separate from ADHD-IN (see Table 1) [5]. Subsequent studies have simultaneously tested these SCT optimal items and it appears that the field is narrowing in on a standard set of SCT symptoms that can be used across studies [11,29,30].

The next step will be for studies to identify a meaningful cutoff to classify ("diagnose") participants with SCT (which Barkley has termed Concentration Deficit Disorder [13,16]). Using nationally representative samples of adults [25] and children [26] in the United States, Barkley has provided preliminary cutoffs (corresponding with the symptom count closest to the 95th percentile) that can be used to categorize participants with SCT. Specifically, Barkley found that at least 3 of 12 symptoms were needed to identify a child with SCT [26], whereas at least 5 of 9 symptoms were needed to identify an adult with SCT [25]. These findings suggest that the best cutoff for classifying SCT may differ across development, or that there are rater differences given that Barkley's child study used parent-report ratings whereas the adult study used self-report ratings. Interestingly, if the optimal cutoff for identifying SCT increases across development – perhaps because SCT itself increases with age [5,31] – this would be in direct contrast to ADHD for which the optimal cutoff actually decreases given the decline in ADHD symptoms across development (particularly hyperactive-impulsive symptoms) [32-34]. In any event, additional studies that use a standard symptom set are clearly needed to evaluate the best cutoff for classifying SCT, including whether the best cutoff differs based on individual characteristics (e.g., sex, age, race), informant (e.g., parent, teacher, self-report), method (e.g., rating scale, interview), or cultural context.

Once individuals can be classified with SCT using a standard symptom set, the next step within the DSM framework is to compare individuals with and without SCT to identify genetic, brain, behavioral, and impairment differences that may guide etiological theories of the disorder and inform interventions targeting SCT symptoms and associated impairments. For example, Barkley found that adults with SCT (with or without ADHD) earned less annual income and also had greater difficulties self-organization/problem-solving than controls or adults with ADHD-only [25]. In Barkley's study of children, children with SCT (with or without ADHD) had parents with lower educational levels and more impairment specifically in the sports domain compared to controls or children with ADHD-only [26]. Children with SCT also had the highest rates of a parent-reported depression diagnosis [26]. These landmark studies exemplify a DSM-esque categorical approach, though they have yet to be replicated.

Another way in which the DSM framework may be useful is in refining current diagnostic categories. The study of SCT largely emerged as a possible way to identify children with a "pure" inattentive presentation of ADHD [2,35], and while research has not generally supported this possibility [4], it important to note that most studies examining this possibility were conducted before validated rating scales existed. Even so, there is some indication that among children with ADHD, those who also display SCT symptoms are less aggressive, more withdrawn, and display more internalizing symptoms than other children with ADHD [36-38]. The first neuroimaging study to focus on SCT, conducted by Fassbender and colleagues, found that among adolescents diagnosed with ADHD, the added presence of SCT was associated with hypoactivity in the left superior parietal lobe to cues during a flanker task, perhaps indicating impaired reorienting or shifting of attention [39]. A recent medication trial of children with ADHD found that children with higher SCT (and sluggish/sleepy symptoms specifically) had less improvement in their ADHD symptoms across doses of methylphenidate (MPH) and were also more likely to be MPH nonresponders or placebo responders compared to other children with ADHD [40]. Interestingly, research suggests that atomoxetine reduces SCT symptoms in youth with ADHD [41,42], which aligns with other studies indicating atomoxetine to be effective for youth with ADHD and co-occurring internalizing symptoms [43,44] These findings together suggest that SCT as a possible specifier among children with ADHD is an area worth further examination, particularly if an SCT specifier can inform treatment decisions. In addition, there has recently been some renewed interest in a possible ADHD restrictive inattentive presentation [45], and children with this phenotype may have a different neuropsychological and attentional profile than other children with ADHD, as well as different brain functioning and genetic markers (e.g., presence of the DRD4-7 repeat allele) [46,47]. Integrating SCT within studies of ADHD restrictive inattentive presentation would be advantageous as part of overall efforts to better understand and categorize the phenotypic heterogeneity of ADHD.

Key Considerations for Examining SCT within the RDoC Initiative

The RDoC initiative proposes a radical shift away from the DSM in the way psychiatric diagnoses are conceptualized. Within the RDoC framework (currently a research rather than clinical enterprise), historical distinctions between DSM-defined disorders may be ignored, and instead a “bottom up” approach grounded in neuroscience methods is used to identify biologically-based constructs (i.e., negative valence systems, positive valence systems, cognitive systems, social processes, arousal and regulatory systems) and units of analysis (i.e., genes, molecules, cells, circuits, physiology, behavior, self-report) that are likely to cut across current diagnostic categories [19-21,48]. The long-term goal of the RDoC initiative is to identify biosignatures that in tandem with behavioral symptoms can be used to inform psychiatric nosology and clinical management – precision medicine for psychiatry [20,49].

The RDoC framework takes an explicitly dimensional approach. Although dimensional models of psychopathology have been advocated long before the arrival of the RDoC initiative [50-52], the explicitly dimensional framework espoused by the RDoC initiative ensures that dimensional models will receive even more attention and scrutiny. No study has directly tested whether SCT is best conceptualized as dimensional or dichotomous, though given findings for most other psychopathologies it is likely that such research will find SCT to have a dimensional rather than taxonic latent structure. In any event, extremes at one or both ends of the normative distribution may be considered pathological within the RDoC framework, with a key priority for the RDoC initiative being the identification of “tipping points” that mark a (potentially nonlinear) transition to severe pathology and associated impairments [19,21]. Mild and transient psychopathology is also captured in this dimensional approach, with tipping points more easily identified and adjusted based on individual characteristics (e.g., age, sex) and methodological factors (e.g., informant) [52].

RDoC may provide a useful approach to understand SCT for several reasons. SCT remains a relatively understudied construct, particularly at levels of analysis beyond the behavioral manifestation of SCT and related impairment. Therefore, studies that examine RDoC constructs across multiple levels of analysis in relation to SCT may provide important data to inform the development of a comprehensive theoretical model of SCT. Further, these results may help to clarify the relations between SCT and other dimensions of psychopathology by identifying which RDoC dimensions are related to both SCT and other disorders that covary with SCT, and which are uniquely associated with SCT. For example, while individual differences in frontal lobe functioning play a role in most mood and anxiety disorders, one recent study proposed that specific relative differences in left frontal lobe activity may be uniquely associated with RDoC constructs that are linked to specific symptom dimensions, such

as the correlation between anhedonia and depression and excessive approach motivation and mania [53]. A similar approach may be a fruitful way to disentangle the shared and unique correlates of SCT and correlated measures of ADHD inattention and internalizing symptoms, which may then inform understanding of the placement of SCT in comprehensive theoretical models of psychopathology that, ultimately, can be used to guide treatment decisions. This aligns with a key priority of the RDoC initiative, which "explicitly focuses on the complex overlapping multidimensionality of mental illness" [54, p. 76]. Research to date indicates that SCT is in a unique position to inform advancements in this area given its clear links to both ADHD and internalizing (i.e., heterotypic symptom presentations) and the absence of diagnostic reification that may place assumptions on precisely how and where SCT should fit in the RDoC matrix.

Although it is possible that SCT could map cleanly onto a single RDoC domain, it is more likely that individual differences in multiple RDoC constructs could contribute independently or interactively to the development of the complex constellation of behaviors that defines SCT. For example, a recent theoretical model of suicide informed by RDoC underscored the need to integrate findings implicating heightened fear-potentiated startle response, attentional bias toward negatively valenced information, and reduced fear of death [55]. In this review we illustrate the potential application of RDoC to SCT by examining negative valence systems, cognitive systems, and arousal/regulatory systems, three domains that show the most promising potential links with SCT in initial research or theoretical models. As a starting point for examining SCT within the RDoC framework, studies may simultaneously include multiple units of analysis across all three of these constructs so that the relative, unique, and even interactive contributions of RDoC constructs to SCT (or SCT subdimensions or even individual items) can be elucidated. Once such effects have been identified, research can proceed to uncover more nuanced, mechanistic theories, with implications for potential prevention efforts that curtail the rise of SCT (and its associated impairments) at the most sensitive periods in development. To reach this goal, it is important to note that while initial studies may establish *links* between various RDoC constructs and their interactions in relation to SCT, the long-term goal will be to determine when abnormality in the constructs themselves *give rise to* SCT and related symptoms [54].

Negative valence systems. Within the negative valence systems domain, SCT may be most clearly included in studies investigating the loss, potential threat ('anxiety'), and sustained threat constructs. SCT is associated with apathy, amotivation, psychomotor retardation, and withdrawal [5,10,56], which are all components of the loss construct (defined in the RDoC as "a state of deprivation of a motivationally significant con-specific, object, or situation" [57]). Might SCT be in part caused by elevated sensitivity to even mild loss and a tendency to

ruminate or “get stuck” when such events occur? Although not directly tested, it has been hypothesized that SCT is associated with rumination, which itself has a large literature base and is included as a loss construct in the RDoC matrix [14]. Rumination has been identified as a model example of a potential transdiagnostic mechanism that predicts not only depression but also anxiety, alcohol abuse, disordered eating, and self-harm behaviors [58]. Similarly, SCT predicts (and may bi-directionally relate to) a range of psychopathologies and impairments, including depression, anxiety, ADHD, learning difficulties, and sleep problems. A starting point could be to evaluate the behavioral association between SCT and rumination, as well as whether similar or different mechanisms give rise to rumination and SCT. In particular, serotonergic functioning (e.g., 5-HTTLPR), disruptions in cortico-limbic circuits (and the dorsolateral prefrontal cortex [DLPFC] specifically [59,60]), default mode network connectivity, and heart rate variability should be examined [58,61-63]. Interestingly, despite potential similarities between rumination and SCT, “the majority of studies have focused on rumination solely within the context of a current major depression disorder diagnosis” [58, p. 9] just as “almost all studies of SCT have been conducted in community/school-based samples or samples of children with ADHD” [5, p. 175]. We return to this point below in considering how SCT may advance models of the covariation of internalizing and externalizing dimensions of psychopathology.

The clearest evidence to date linking SCT to the potential and sustained threat constructs of the negative valence systems domain comes from initial studies demonstrating SCT to be associated with punishment sensitivity in children [64] and increased behavioral inhibition system (BIS) sensitivity in adults [65]. These are important preliminary findings since the RDoC positive and negative valence systems constructs were in part informed by the fields of child temperament and adult personality [21]. Still, these findings will certainly need to be replicated and extended to other units of analyses within these constructs. For example, it is unknown if SCT relates to attentional bias to threat as assessed by the dot-probe task, but this seems both likely and important since the prospective association between behavioral inhibition and social withdrawal is found only among children who display attentional biases to threat [66].

Cognitive systems. Within the cognitive systems domain, SCT may clearly be examined within the attention construct, a “range of processes that regulate access to capacity-limited systems, such as awareness, higher perceptual processes, and motor action” [67]. One starting point can be found in Posner’s model of attention, which is widely accepted and, crucially, has itself been validated across different units of analysis [68-72]. Within this model, alerting, orienting, and executive control networks carry out different functions that involve different brain structures and neurotransmitters [68-72]. It has been hypothesized that SCT may be a behavioral

manifestation of reduced efficiency in orienting [7,16]. Although a group-based design in the vein of DSM can certainly be used to test this hypothesis (see above), studies using the RDoC framework would take a different approach by using a unit of analysis, rather than SCT or ADHD group, as the independent variable. For instance, an RDoC study interrogating the attention construct might broadly recruit all children presenting to an ADHD or behavior disorders clinic, which would thus include children with a range of both ADHD and SCT severity, as well as children from general pediatric practices or schools to capture the full range of attentional functioning. Then, network efficiency on a task such as the Attention Network Test (ANT) could be the independent variable, irrespective of how children were recruited or whether they met criteria for either ADHD or SCT. Associations between ANT performance and attention symptom severity (including both ADHD and SCT) can be evaluated in an effort to identify whether attention network efficiencies are differentially related to specific behavioral symptoms irrespective of current conceptualization. Ultimately, research in this vein may identify underlying mechanisms of heterogeneous attention symptoms.

Arousal and regulatory systems. Arousal and circadian rhythm constructs may also have relevance for SCT, for several reasons. Arousal, defined by the RDoC framework as “a continuum of sensitivity of the organism to stimuli, both external and internal” [73], has been identified as a key component in etiological models of both ADHD and depression [74,75]. As described by Hegerl and Hensch [74], unstable arousal (e.g., vigilance in brain arousal) or underarousal, often measured by the electroencephalogram (EEG), is an important correlate of ADHD inattentive symptoms. In contrast, tonic hyperarousal is theorized as a contributor to affective symptomatology as well as social withdrawal specifically [74]. This has been shown to be clinically relevant, as higher brain arousal has been found in responders compared to nonresponders of antidepressant medication treatment [76]. How might these diverging hypotheses regarding the role of arousal inform the study of SCT, particularly since SCT is strongly associated with both ADHD inattention and depression? One avenue might be to simultaneously examine both arousal and sleep in relation to SCT, ADHD, and depression. Daytime sleepiness is pronounced in ADHD [77] and has also been linked to SCT [8,78], though the association between sleepiness and SCT has yet to be examined with objective methods such as the multiple sleep latency test. Nevertheless, the association between daytime sleepiness and SCT suggests that underarousal may be at least one mechanism or biomarker of SCT. Yet withdrawal is also a clearly established correlate of SCT [10,36,38,56], pointing to the alternative possibility of hyperarousal being implicated. We propose that considering SCT in arousal models of psychopathology will not only inform our understanding of SCT – is SCT caused by underarousal or hyperarousal or perhaps dysregulation in arousal? – but also inform our understanding of these other psychopathologies and

their covariation. By considering multiple RDoC constructs (e.g., arousal and circadian rhythm) in reference to multiple psychopathologies (e.g., SCT, ADHD, depression), it may become clearer how to best conceptualize the relations among psychopathologies with both overlapping and unique symptoms. Whether or not SCT is a key piece of this puzzle will be unknown until it is included in studies aiming to elucidate such patterns and processes.

A recent study by Fair and colleagues [79] is informative in considering another way in which models (and measures) of arousal may inform SCT and broader models of psychopathology. Using graph theory and community detection, the investigators found distinct neuropsychological subgroups that were strikingly similar across both ADHD and non-ADHD groups. This is itself an important finding since, as the authors note, investigators using the DSM framework are "generally obliged to conduct their analysis as if typically developing comparison populations represent a monolithic group" [79, p. 6769]. Particularly germane to our focus herein on SCT, among both the ADHD and comparison youth there was a subset (16-18%) characterized by weak signal detection (*d*-prime) – the sensitivity to the difference between a target (signal) and a nontarget (noise) that is interpreted as an indicator of low (suboptimal) arousal [79]. Interestingly, these youth did not display other neuropsychological impairments [79], just as SCT has not been convincingly associated with neuropsychological deficits [5]. Are these youth characterized by weak signal detection the same youth who display elevated SCT? In considering these and other findings, Nigg [80] suggested four possible neurobiological routes to ADHD, including a "breakdown in bottom-up attentional capture" (p. 9) route consistent with a restrictive inattentive presentation of ADHD as well as SCT and low arousal. The inclusion of SCT in studies seeking to inform heterogeneity among children with and without attention problems could further advance the characterization of more homogeneous profiles and inform neurobiological models of classifications and, interestingly, point to areas of possible convergence and cross-fertilization between the DSM and RDoC models (see discussion above regarding SCT within efforts to refine the heterogenous ADHD phenotype).

Key Considerations for Examining SCT within Hierarchical Models of Psychopathology

Although the RDoC initiative is a relatively new dimensional model, it is strongly informed by other dimensional and transdiagnostic models that have a large and robust literature base [22,23,81,82]. These models of psychopathology have clear points of agreement with the RDoC initiative but also key points of departure [22,23] and are also likely to be useful toward advancing our understanding of SCT. Indeed, it is possible that SCT is not best conceptualized within the RDoC matrix but may nevertheless have validity and utility as a dimensional, transdiagnostic construct. A growing body of research indicates that psychopathology is hierarchically structured with a general psychopathology factor (*p* factor) contributing to higher-order externalizing

and internalizing dimensions that subdivide into more specific dimensions [22,23,83,84]. In considering SCT, an intriguing pattern of findings has emerged in studies examining how SCT and ADHD-IN each relate to other psychopathology symptoms. Specifically, SCT and ADHD-IN are themselves strongly correlated, yet when controlling for the other, SCT is consistently associated with increased internalizing symptoms whereas ADHD-IN is consistently associated with increased externalizing symptoms [3,5,12,13]. Furthermore, several studies have found that SCT is *negatively* associated with externalizing behaviors when controlling for ADHD-IN [6,27,28,85]. These findings suggest that the simultaneous consideration of these different attention dimensions (SCT and ADHD-IN) may help understand the separation and covariation of internalizing and externalizing psychopathologies.

As *p* factor models advance, it is also worth considering whether inattention – broadly defined – falls under the externalizing or internalizing dimensions. Historically, ADHD-IN has been considered alongside ADHD-HI as an externalizing behavior, and this conceptualization does have some empirical support (though several studies did not include separate ADHD dimensions) [86-89]. However, inattention was not included in a number of studies examining the structure of psychopathology [90-93], and attention problems are not included as part of either the broad internalizing or externalizing dimensions of the Achenbach assessment system (e.g., Child Behavior Checklist [CBCL]) [94]. One study using the Achenbach measures found support for including attention problems on the latent externalizing factor, but also found that attention problems were more indicative than other externalizing behaviors of a general factor [95]. Another study by Noordhof et al. [96] used the CBCL in a sample of adolescents and did not include attention problems as either an internalizing or externalizing indicator, but instead included attention problems as a separate dimension. Findings supported this decision and the investigators concluded that “the CBCL-scale attention problems is not captured by the externalizing nor the internalizing domain” [95, p. 584]. Similarly, although Carragher and colleagues [88] found ADHD to load on an externalizing factor in a large sample of adults, “relatively speaking it was the weakest indicator of the seven syndromes examined…Future structural research should investigate whether ADHD loads onto other latent factors in addition to externalizing liability” (p. 1314). Studies investigating inattention within hierarchical models should take care to use carefully defined SCT and ADHD-IN measures. For instance, the CBCL attention problems scale includes several ADHD-IN items but also SCT items (e.g., “confused or seems to be in a fog”, “daydreams or gets lost in his/her thoughts”) and even hyperactive-impulsive items (e.g., “can’t sit still, restless, or hyperactive”, “impulsive or acts without thinking”) [94], and this symptom and construct heterogeneity could likely impact how this specific “attention problems” scale loads on broader internalizing vs. externalizing factors.

To further advance our understanding of attention problems broadly, and SCT and ADHD-IN specifically, studies should evaluate SCT within hierarchical models of psychopathology. There is reason to believe that SCT will be first-order dimension on the higher-order internalizing factor since (a) SCT is more strongly associated with internalizing symptoms than with externalizing symptoms (with the exception of ADHD-IN) [5], (b) SCT does not fall within a bi-factor umbrella of either ADHD or a general disruptive behavior factor [97-99], (c) SCT is linked to *greater* withdrawal and isolation and *lower* aggression and externalizing behaviors [6,10,36,38,56], (d) SCT may parallel depression in increasing across the transition from childhood to adolescence and adulthood [5,31], and (e) SCT is associated with increased punishment and BIS sensitivity [64,65]. Should empirical support for our hypothesis be found, one set of attentional difficulties (SCT) may be subsumed under the internalizing dimension whereas another set of attentional difficulties (ADHD-IN) may be subsumed under the externalizing dimension, even though these two attention dimensions are themselves strongly correlated (*r*s = 0.63 and 0.72 in children and adults, respectively [5]). In ways resembling the model examined by Noordhof et al. [96], a broader attention problem construct may be examined as a possible bridge that theoretically and empirically links the higher-order internalizing and externalizing dimensions. Studies in this vein could greatly inform our current understanding of the covariation of these higher-order dimensions, as well as models of homotypic and heterotypic continuity of psychopathology [23,84]. Toward this end, although a standard symptom set for assessing SCT is itself an important goal, it may be easier for investigators to freely examine its measurement – both at the construct level and at the individual symptom level. There may also be separate dimensions within SCT that are important to examine in terms of etiology, course, and associations with impairment and treatment response [27,40,100-104]. In agreement with Lahey and colleagues [23], the overarching SCT construct may be too large a “grain size” for understanding its psychobiology since it includes a range of cognitive, psychomotor, and motivational components while at the same time the SCT construct may be too small if not examined and understood within higher-order models of psychopathology.

Conclusion

SCT is not currently defined as a psychiatric disorder and has never been included in any previous diagnostic models. However, this possibility has been proposed [13,16] at just the same time that the field of psychiatry engages in an important debate about the strengths and weaknesses of the categorical DSM nosology, the dimensional approach adopted by the RDoC initiative, and hierarchical models of dimensional psychopathology. Although the categorical DSM approach has been the *modus operandi* in psychiatry for over three decades, a number of nagging issues with the DSM and other categorical approaches remain unsettled. It is

clear that the categorical DSM system does not carve nature at its joints, though categorical diagnoses in the DSM have been prematurely and inaccurately treated as “natural kinds” [21,24]. Indeed, Barkley’s nationally representative studies of SCT in children and adults [25,26] were landmark studies but nevertheless underscore three long-standing issues in categorical-based diagnosis that will likely apply to SCT just as they do to most current DSM-based diagnoses: categorical demarcation of a likely dimensional construct, within-diagnosis heterogeneity, and excessive comorbidity [21,24,105-108]. Likewise, although the RDoC initiative seeks to rectify some key issues within the DSM system, it is important to note that the RDoC initiative has been met with its own set of critiques, including an underdeveloped conceptual framework, an overemphasis on biological units, neglect of the psychometric weaknesses and measurement error of some methods, inadequate consideration of developmental factors and socio-cultural contexts, and a disconnect between research and clinical practice [109-114]. At the current time, there is no perfect approach to studying psychopathology. For SCT research to advance, we agree with Kraemer [110] that “valid diagnosis of mental health disorders, like that of physical health disorders, is a developing process based on accumulating evidence, not a fixed goal, and should use all resources available: dimensional and categorical, DSM and RDoC” (p.1164). We would add that other dimensional models should also be considered alongside the RDoC framework. We propose that the study of SCT will be greatly advanced by critically and creatively conceptualizing SCT within different models of psychopathology.

Conflict of Interest Statement: Neither author has any conflict of interest.

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Table 1

Sluggish cognitive tempo (SCT) items with meta-analytic support as distinct from ADHD inattention

SCT Item
Sluggish
Tired/lethargic
Slow thinking/processing
Loses train of thought/cognitive set
Sleepy/drowsy
Spacey
In a fog
Underactive/slow moving
Daydreams
Lost in thoughts
Stares blankly
Easily confused
Apathetic/unmotivated

Note. The meta-analysis required the item to have a primary factor loading on an SCT factor (mean factor loading ≥.70 across studies) (see ref. [5]). ADHD = attention-deficit/hyperactivity disorder.