IN CONTEXT

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Report of a Work Group on Sluggish Cognitive Tempo: Key Research Directions and a Consensus Change in Terminology to Cognitive Disengagement Syndrome

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Objective: The aim of this work was 2-fold: (1) to evaluate current knowledge and identify key directions in the study of sluggish cognitive tempo (SCT); and (2) to arrive at a consensus change in terminology for the construct that reflects the current science and may be more acceptable to researchers, clinicians, caregivers, and patients.

Method: An international Work Group was convened that, in early 2021, compiled an online archive of all research studies on SCT and summarized the current state of knowledge, noted methodological issues, and highlighted future directions, and met virtually on 10 occasions in 2021 to discuss these topics and terminology.

Results: Major progress has been made over the last decade in advancing our understanding of SCT across the following domains of inquiry: construct measurement and stability; genetic, environmental, pathophysiologic, and neuropsychological correlates; comorbid conditions; functional impairments; and psychosocial and medication interventions. Findings across these domains are summarized, and potential avenues to pursue in the next generation of SCT-related research are proposed. Following repeated discussions on terminology, the Work Group selected "cognitive disengagement syndrome" (CDS) to replace "SCT" as the name for this construct. This term was deemed to best satisfy considerations that should apply when selecting terms for a condition or syndrome, as it does not overlap with established terms for other constructs, is not offensive, and reflects the current state of the science.

Conclusion: It is evident that CDS (SCT) has reached the threshold of recognition as a distinct syndrome. Much work remains to further clarify its nature (eg, transdiagnostic factor, separate disorder, diagnostic specifier), etiologies, demographic factors, relations to other psychopathologies, and linkages to specific domains of functional impairment. Investigators are needed with interests and expertise spanning basic, clinical, and translational research to advance our understanding and to improve the lives of individuals with this unique syndrome.

Key words: attention-deficit/hyperactivity disorder; CDS; cognitive disengagement syndrome; SCT; sluggish cognitive tempo

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The last 2 decades have seen rapidly accumulating interest in sluggish cognitive tempo (SCT), a constellation of behaviors involving excessive daydreaming, mental confusion and fogginess, and slowed behavior and thinking.[1-4] The study of SCT emerged in the mid-1980s when the *DSM-III* specified 2 types of attention-deficit disorder: with hyperactivity (ADD+H) and without hyperactivity (ADD-H).[5] Early studies found that children with ADHD-H had more symptoms of sluggishness, drowsiness, and daydreaming compared to children with ADHD+H.[6,7] Around this same time, factor analytic studies began to provide support for a "sluggish tempo" factor with items that loaded separately from items on an inattention−disorganization factor.[8,9] Much of the initial research that followed focused on the empirical distinction between SCT and attention-deficit/hyperactivity disorder inattention (ADHD-IN); however, as evidence supporting the distinction of SCT from ADHD-IN emerged,[10] investigators have increasingly sought to determine the causes, correlates, and consequences of SCT. Subsequent research has highlighted the association between high levels of SCT symptomatology and impaired global,

social, academic, and occupational functioning. Given these striking negative correlates, there is a need to evaluate the current knowledge on SCT to identify key future directions and avenues for prevention and/or treatment. Research addressing current gaps is also needed to inform whether SCT is best conceptualized as its own disorder, a specifier within ADHD, or a transdiagnostic construct.[11,12] Furthermore, the SCT term has been criticized for being potentially inaccurate, offensive, and pejorative,[12,13] leading to calls for revised nomenclature that reflects the current science and may be acceptable to researchers, clinicians, parents, and patients.

METHOD

To propel the next generation of SCT research and to identify a consensus change in terminology, an international Work Group of 13 active, leading experts in the field of SCT was convened (Supplement 1 and Table S1, available online, provide details). The Work Group compiled an online archive of all research studies on SCT in early 2021, and subcommittees volunteered to summarize each topic, note methodological issues, and highlight future directions for each of the following topics/domains: construct measurement and stability; genetic, environmental, pathophysiologic, and neuropsychological correlates; co-occurring conditions; functional impairments; and psychosocial and medication interventions. The Work Group met virtually on 10 occasions during 2021, with subgroups working on identified domains between meetings (Supplement 2, available online). In this article, we provide an overview of the findings and key directions for future research, followed by the rationale for a proposed change in terminology.

RESULTS AND DISCUSSION: OVERVIEW OF EXISTING FINDINGS AND KEY DIRECTIONS FOR FUTURE RESEARCH

In reviewing each domain of inquiry below, it was readily apparent that some important limitations and future directions were specific to each domain, whereas others were shared across many, if not all, domains. In particular, many studies have used limited measures of SCT symptoms and a mono-method, mono-informant, cross-sectional design, and have been conducted in non-representative or homogeneous samples of school-aged children. These and other cross-cutting limitations and future directions are summarized in Table 1.[14-31] In this section, we provide an overview of existing findings and key directions for future research across domains, starting with measurement and consistency across time, sources, and settings; followed by predictors and etiology of SCT; cognition, neuroimaging, and comorbidity; functional outcomes and impairment; and finally psychosocial and pharmacologic treatment.

SCT Assessment and Measurement

The assessment of SCT has proceeded in 4 phases to date. Phase 1 (approximately 1988-2008) involved the *ad hoc* selection of SCT items from broadband rating scales (eg, Child Behavior Checklist [CBCL][32]). Phase 2 began with the systematic development of a specific SCT measure by Penny *et al.*,[33] which influenced the development of other SCT-specific measures (for a review, see Becker[14]). Phase 3 began with a meta-analysis of 73 SCT studies from 1985 to 2015 to identify SCT items with the strongest convergent and discriminant validity.[10] The meta-analysis identified 13 SCT items with these properties along with a suggestion to include 3 additional items assessing mental confusion for a total of 16 possible items.[10] A series of studies then evaluated the reliability and validity of these 16 items. Using mother, father, and teacher ratings, 15 items showed strong structural validity, excellent reliability (internal consistency, test−retest, interrater), invariance (across a 1-month interval, sex of rater, community/clinical samples), and independent correlates relative to ADHD symptoms.[14] Similar albeit more variable findings emerged for the self-report measure.[14,34-38]

Phase 4 began with a review of assessment research on SCT from 2009 through 2019,[14] in part to identify a common set of items for future SCT research. Table 2 shows the SCT items with the strongest empirical support from this review. Fifteen of these items are included in the Child and Adolescent Behavior Inventory (CABI) SCT measure from Phase 3, which was identified as an optimal measure for assessing parent- and teacher-rated SCT, with parallel self-report versions also available. Consistent inclusion of a standard, optimal SCT item set across studies will allow the advancement of SCT research based on a common set of items, much as ADHD is currently defined by 18 symptoms. As shown in Table 2, there are 3 potential SCT subdomains (ie, daydreaming, mental confusion, hypoactivity),[14,39-42] although it remains unclear whether these subdomains reliably emerge or consistently relate to distinct outcomeses.[40,42-44] Studies are very much needed to examine whether there are distinct etiologies, developmental trajectories, external correlates, and predictive associations of the specific SCT subdomains. Furthermore, even though earlier, brief measures of SCT correlate strongly with more recent, longer measures of SCT,[45,46] a consistently used item set will also allow for drawing clearer conclusions

TABLE 1 Common Limitations of Existing Sluggish Cognitive Tempo (SCT) Research and Priorities for Future Research

Limitation frequently present in existing SCT studies	Priority for future research and exemplar references
Limited SCT measurement (eg, CBCL items)	Numerous SCT-specific rating scales have now been developed and evaluated and are recommended for use. For a systematic review, see Becker.[14]
Cross-sectional design	Longitudinal research is needed to show temporal prediction of functional outcomes/impairment, in addition to prospective studies examining predictors of SCT. For example, see studies examining the differential impact of childhood SCT and ADHD-IN on adolescent functioning[15] and preschool neuropsychological predictors of school-aged SCT and ADHD-IN symptoms.[16]
Mono-informant methods	Consideration of informant differences, including whether effects are specific to or more pronounced based on source. For example, see studies of impairment in children[17] and adults,[18] and behavioral[19] and pharmacologic[20] intervention effects.
Mono-method studies	Studies are needed that include multiple methods to assess domains of interest (eg, cognition, impairment, comorbidity). In addition, studies of SCT-related impairment are especially needed to incorporate additional measurement approaches that may be more sensitive or have greater ecological validity (eg, school report cards/transcripts,[21] official driving records).
Lack of covariance of ADHD and/or other psychopathology symptoms	It is important to continue and expand covariance of symptoms of other disorders to clarify the unique correlates of SCT, recognizing that covarying for other psychopathologies also has its limitations and should be done thoughtfully and based on the research question being examined.
Focus on school-aged children	Additional studies conducted with very young children, adolescents, and non−college student adults are needed.
Absence of developmental models	Research is needed to understand SCT across the life span in addition to whether SCT-related effects are stronger or weaker in distinct developmental periods. For example, see studies examining temporal nature of SCT from preschool through adolescence[22] and age as a moderator of SCT correlates.[23]
Reliance on ADHD-defined samples	Studies are needed in distinct clinical samples, including individuals with internalizing disorders, sleep disorders, and autism spectrum disorder, among others. For example, see studies examining SCT in sleep-disordered children[24] and youth with autism.[25] Crucially, there are not yet studies of individuals recruited specifically for SCT.
Inattention to possible sex effects	It cannot be assumed that SCT emerges or predicts functioning similarly in male and female individuals. For example, see a study finding SCT may differentially predict social functioning in girls and boys.[26]
Homogenous, non-representative samples	Representative samples are needed, in addition to studies examining the impact of social determinants of health (eg, racism, discrimination, oppression) on SCT and associated outcomes in representative samples. For example, see nationally representative US samples in children[27,28] and adults,[29] and one study examining school climate in the context of SCT.[30]
Primarily US and European samples	Very few studies have been conducted outside of North America (primarily the United States) or Europe (primarily Spain). There is a need to examine SCT across and within diverse cultural contexts.[31]

Note: ADHD = attention-deficit/hyperactivity disorder; ADHD-IN = ADHD inattention; CBCL = Child Behavior Checklist; SCT = sluggish cognitive tempo.

TABLE 2 Sluggish Cognitive Tempo (Cognitive Disengagement Syndrome) Items for Future Research

Daydreaming items
Daydreams
Gets lost in own thoughts
Spaces or zones out
Appears lost in a fog
Stares blankly into space
Mental confusion items
Loses train of thought
Difficulty putting thoughts into words
Forgets what was going to say
Thinking gets mixed up
Easily confused
Thinking is slow
Hypoactivity items
Easily tired or fatigued
Low level of activity (underactive)
Behavior is slow
Drowsy or sleepy during the day

regarding associations of SCT with functional and intervention outcomes.

SCT Across Time, Sources, and Settings

Two studies used latent state−trait models to determine the consistency of SCT across occasions (timepoints), sources (mother, father, primary teacher, and secondary teacher ratings), and settings (home and school contexts). SCT was more trait-like than state-like across 4 assessments from kindergarten to fourth grade,[47] and was more trait-like than state-like across 3 assessments in the first grade.[48] These 2 studies also found SCT to be as trait-like as ADHD-IN and ADHD-HI. Future studies should identify external correlates of trait and state SCT variance.[15] In addition, intensive longitudinal designs could determine whether SCT symptoms are more pronounced at certain times of day (eg, morning)[49] or days of the week (eg, later in the week as insufficient sleep builds up). Together, these types of studies will promote an understanding of the consistency of SCT across varying time intervals, sources, and settings.

There is some indication that SCT increases across development. A meta-analysis found a small yet significant association between SCT and age ($r = 0.11$),[10] and 2 longitudinal studies showed a modest increase in SCT with increasing age.[22,50] SCT symptom severity and prevalence shows few differences between children and adults, although individuals with higher levels of SCT appear to be older (ie, retrospective mean age of onset of 13.4 years in the only study examining SCT age of onset[51]) than other clinically referred patients and individuals with ADHD without SCT.[23,27,29] It has not been determined whether SCT worsens with age, vs becoming more apparent and impairing as the environmental expectations for cognitive performance increase; but available evidence suggests that children do not "outgrow" SCT over time.

Although SCT severity may change over time, the construct itself appears invariant across development. Invariance has been shown for both parent and teacher ratings from age 3 years through fifth grade,[50] for teacher ratings from second through fifth grades,[39] for caregivers from preschool to fourth[47] and ninth[22] grades, for parents and teachers across 3 occasions in a 12-month period,[48] and for younger and older adolescents with ADHD.[52] That said, there is a need for more studies evaluating change in and invariance of SCT across wider developmental spans and key developmental transitions (eg, childhood to adolescence, adolescence to adulthood), and studies examining whether possible SCT subdimensions (eg, daydreams, hypoactivity, mental confusion) are similarly invariant and have similar trajectories.

SCT Etiology

Twin Studies of SCT. By comparing the similarity of pairs of identical and fraternal twins, twin studies provide a direct estimate of the extent to which a construct is due to genetic or environmental influences. Although the first twin study of SCT using just 3 CBCL items found relatively modest heritability ($h^2 = 0.28$),[53] subsequent examination of parent ratings of 5 to 7 SCT items in 2 community samples of twins found significant and higher heritability in both samples ($h^2 = 0.55$-0.66).[54] The remaining variance in all 3 samples was explained by nonshared environmental influences and measurement error, suggesting that differences in the reliability of the SCT measures may potentially explain the variability in the heritability estimates.

Molecular Genetic Studies. Only one study attempted to identify the specific genetic mechanisms associated with increased risk for SCT by examining polymorphism frequency in the dopamine transporter (DAT1; SLC6A3) and dopamine D4 receptor (DRD4) genes among children with varied elevations of SCT and/or ADHD.[55] The only significant SCT group difference was increased frequency of the 7-repeat allele of the DRD4 gene among the SCT-only group compared to the ADHD-only group, although this result awaits replication.

Prenatal, Early Childhood, and Medical Risk Factors. SCT elevations were reported in children exposed prenatally to alcohol[56,57] or maternal smoking,[58] pediatric traumatic

brain injury survivors,[59] and youth with spina bifida.[60] Mixed findings were reported in patients with epilepsy[61,62] and survivors of pediatric brain tumors[63,64] or acute lymphoblastic leukemia.[65-67] Thyroid-stimulating hormone levels were weakly but uniquely associated with SCT, but not ADHD, in psychiatrically hospitalized children.[68] Finally, greater iron deficiency in infancy predicted greater SCT and ADHD symptoms in childhood and adolescence.[69]

These results suggest multiple directions for research on the etiology and associated factors of SCT, but nearly all results await independent replication. More research is needed to examine the degree to which medical factors contribute to, exacerbate, or simply co-occur with SCT symptoms, and whether medications prescribed for treatment of other conditions,[70] as well as other prenatal factors such as maternal medication use, play a role.

Psychophysiology Research. Three studies examined psychophysiological indices in association with SCT. Using resting state electroencephalography (EEG) in children with and without ADHD, the ratio of theta to beta waves over the central aspect of the frontal lobe was associated with ADHD symptoms, but not with SCT symptoms.[71] Two studies examined autonomic nervous system activity. Among children with a range of ADHD symptoms, SCT symptoms were significantly associated with greater skin conductance reactivity during a peer rejection task but not an impossible puzzles task, whereas SCT was not related to respiratory sinus arrhythmia during either task.[72] Another study found SCT to be positively related to children's resting-state heart rate variability (HRV) as well as to an increase between resting and warning signal conditions in the standard deviation measure (SD2 nu) derived from a Poincaré plot of HRV.[73] These studies suggest that heightened reactivity of the autonomic nervous system to stress may be associated with SCT. Studies that use other stressor tasks (eg, Trier Social Stress Test) and include physiological assessments alongside behavioral inhibition measures would be informative about possible associations between stress response/reactivity and SCT.

Predictors of SCT

Few studies have explicitly considered multivariate predictors of SCT. Studies examining potential psychopathology correlates as predictors of SCT have found inattention, depression, anxiety, somatic complaints, excessive or reduced sleep, cognitive problems, and autism symptoms to be uniquely associated with SCT.[74-76] Hyperactivity was a significant negative predictor, and externalizing behaviors were not uniquely associated with SCT.[74-76] Studies investigating neuropsychological measures found working memory to be uniquely associated with SCT, and reported mixed findings or weak associations for processing speed, inhibition, and IQ.[77,78] One study examined personality factors and found punishment and reward sensitivity to be differentially associated with SCT and ADHD symptoms, respectively.[79] Race and sex are not uniquely associated with SCT, and results are mixed for socioeconomic indicators.[58,74,77]

However, there is a paucity of longitudinal studies examining factors that contribute to, maintain, or worsen SCT symptoms over time. One of only 2 multivariate, longitudinal studies predicting SCT found slower processing speed at age 5 years predicted both ADHD-IN and SCT teacher ratings in first through third grade, whereas poorer working memory and response inhibition predicted higher ADHD-IN but not SCT.[16] In youth with spina bifida, worse working memory predicted increases in mother-reported SCT, and poorer cognitive shifting abilities predicted increases in teacher-reported SCT over time, whereas attention and planning/organization did not predict increases in SCT.[60]

SCT and Psychosocial Adversity

A handful of studies investigated associations between SCT and psychosocial adversities, finding SCT to be associated with lower income/education,[27,29,50,80] negative family environment,[81] peer victimization,[30] and interpersonal (but not non-interpersonal) trauma exposure.[82] Aside from income/education, all findings await replication.

Future research should examine possible bi-directional processes between SCT and psychosocial adversity (eg, peer victimization, negative family environment). For instance, chronic traumatic experiences are thought to activate physiological hypoactivity, manifesting as lethargy and drowsiness,[83] and, in childhood, may be linked to maladaptive daydreaming.[84] Finally, it is important to understand protective factors that may buffer against the effects of psychosocial adversity on SCT. For example, a positive school climate may mitigate the link between adverse peer relations and SCT.[30]

SCT and Cognition

Neuropsychological Testing. Cognitive findings were recently reviewed in detail elsewhere[85] and largely rely upon psychometric testing of specific domains. Studies have examined more than 15 different psychometrically assessed cognitive constructs and at least 5 or more domains of executive functioning (EF). Psychometric testing has not yet revealed central cognitive deficits underlying SCT. SCT

may have a small but significant negative association with intelligence,[10,86] although evidence is less convincing than for ADHD. SCT is not consistently or strongly associated with 3 major components of EF that are often quite deficient in ADHD, including response inhibition (or interference control), consistent attentional control (as reflected in reaction time variability), and perhaps working memory, although the latter is unclear at the moment.[77,78] Despite its current name, SCT has not been consistently associated with slow response times or processing speed.[85] Although processing speed may be associated with SCT in young children,[23,87] results are mixed among older children and college students.[88-91] Because the 1 longitudinal study showing that slower processing speed predicted SCT ratings was conducted in younger children,[16] studies are needed to examine whether processing speed is distinctly predictive of or associated with SCT in early childhood more so than later in development. The limited research on signal detection, selective information processing, and stimulus (dis)engagement suggest a possible deficit in selective or focused attention, as evident in the orienting and coupling of attention to external events central to task performance (eg, omission errors).[85,92,93]

Daily Life Executive Functioning. Fewer studies, mostly using parent ratings, have examined EF in daily life with children, finding that SCT is associated with daily problems with organization and problem solving, but not consistently with other domains of daily EF.[27,28,94-96] By adulthood, SCT symptoms contribute even more to problems with self-organization/problem-solving, and, perhaps, with time management, with more conflicting results for difficulties with emotional self-regulation.[29,35,97-99]

Mind Wandering. SCT is uniquely associated with self-report ratings of task-unrelated thoughts, daydreaming, and rumination, even after covarying ADHD, depression, and anxiety.[100,101] There seems to be an over-engagement or decoupling of attention to mental representations or cognitive content more generally, as in mind wandering, mind blanking, and daydreaming.[102] Such disengagement from external events could underlie findings concerning impaired daily life EF domains described above, as these EF domains likely require ongoing attention and cognitive engagement with external demands for behavioral performance. This avenue might also connect the findings linking SCT to both rumination[36,101] and depression,[10,15,103,104] which have been hypothesized to reflect deficits in the control of mental content and focus that are central to maladaptive daydreaming.[105]

Future Directions in SCT and Cognition. Future research should examine other cognitive tasks and ratings that may evaluate attentional and cognitive disengagement/decoupling from external events in relation to over-engagement in mind wandering, mind-blanking, and daydreaming, to more fully explore their involvement in the central cognitive mechanisms underlying SCT symptoms.[102] Likewise, the single study of SCT and self-rated temporal processing[106] represents a fruitful avenue for further research on SCT-related cognitive deficits. Although that study used self-ratings, there is substantial research on temporal processing in ADHD that has used tasks evaluating time perception, time estimation, time (re)production, and parent ratings of timing and time management that are worth studying in the context of SCT.

SCT and Neuroimaging

Our understanding of SCT's associations with brain structure and function remains limited and there have been insufficient data for a systematic review or meta-analysis. Across age and imaging modalities, SCT-associated abnormalities appear within either the posterior cortex or areas belonging to the dorsal attention network, a combination of regions thought to support attention and the association between sensory aspects of the environment and motor responses.[107] SCT-related hypoactivity or under-arousal in inferior parietal and parieto-central regions was identified during a cued flanker paradigm[108] and resting EEG,[71] which may be associated with impaired reorienting of attention. In contrast, posterior hyperactivation was found among young adolescents with comorbid ADHD and SCT symptomatology compared to control participants during an inhibitory Go/No-Go task,[109] as well as increased white matter integrity of the corticospinal tract and fornix, theoretically underlying the hypoactive/apathetic and vigilance components of SCT, respectively.[110] Finally, the largest and most comprehensive neuroimaging study of SCT in children found a combination of gray and white matter enlargements in or near the frontal eye fields (part of the dorsal attention network) and bilateral frontal operculum, as well as SCT-related diminished network segregation above and beyond ADHD.[111]

Many opportunities remain for continued progress in understanding the neurobiological bases of SCT. All existing studies except for one study relied on samples of approximately 20 or fewer participants with SCT symptomatology, and because of reporting and analytic differences such as case-control group comparisons without covarying ADHD symptomatology,[109,110] this literature is in a nascent state and should be regarded as preliminary. All of the aforementioned studies also recruited from ADHD-

selected or clinical outpatient samples; no published neuroimaging studies of SCT have yet recruited participants based on elevated SCT, examined adults, or sought to determine the location and extent of surface-based morphometric (ie, cortical surface area, thickness, and gyrification) and subcortical correlates of SCT. Finally, the cerebellum may be a particularly apt structure for SCT-related investigation, given its widespread implications in ADHD and autism, as well as temporal aspects of cognition and movement.

SCT and Comorbidity

Two large, nationally representative surveys examined parent-reported diagnoses among children with elevated SCT.[27,28] As summarized in Table 3, the most common comorbid disorders (≥5% in both studies) were ADHD, anxiety, autism, depression, language delay, delayed motor skills/coordination, and reading disorder. Importantly, these are likely lower-bound estimates of co-occurrence with SCT, given that both studies used parent report of whether their child had ever received a diagnosis from a clinician.

ADHD. SCT and ADHD-IN are empirically distinct and correlated more strongly (meta-analytic $r = 0.63$ and 0.72 in children and adults, respectively) than SCT and ADHD-HI (meta-analytic $r = 0.32$ and 0.46 in children and adults, respectively).[10] SCT is not associated or is negatively associated with ADHD-HI when covarying ADHD-IN,[10] but frequently co-occurs with all 3 ADHD presentations.[27,28] Epidemiological studies demonstrate elevated SCT symptoms among 25% to 40% of youth with ADHD[27,28,112] and 46% of adults with ADHD.[29]

Externalizing Disorders. Studies show small but significant associations between SCT and a range of externalizing behaviors (meta-analytic $r = 0.21$-0.36),[10] although most of these associations become nonsignificant or even negative when symptoms of ADHD are covaried (for reviews, see Becker and Barkley[3] and Becker *et al.*[10]).

Internalizing Disorders. SCT is strongly related to depression in dimensional analyses of both children and adults (meta-analytic $r = 0.49$-0.50), and associations remain when covarying for ADHD symptoms.[10] SCT appears to be more strongly related to depression than to anxiety.[27,43,113-115] Across informants, 32% to 48% of children with elevated SCT ratings are also elevated on depression ratings.[112] Furthermore, SCT predicts increases in depressive symptoms in children[103] and adolescents.[104] SCT and somatic complaints also appear to be strongly associated.[75,90,116,117]

TABLE 3 Rates of Parent-Reported Comorbid Diagnoses in Children With Elevated Sluggish Cognitive Tempo Symptoms

Diagnosis	Barkley[27] (%)	Burns and Becker[28] (%)
ADHD	27.4	39
Autism	11.1	15.7
Anxiety	11.1	21.3
Depression	7.4	8.8
ODD	1.5	6.9 (combined ODD/CD)
CD	3.0	See ODD above
Bipolar disorder	4.4	2.0
Schizophrenia or psychosis	0.7	2.0
Intellectual disability	8.1	4.9
General developmental delay	(combined with intellectual disability)	6.9
Language delay	15.6	9.8
Delayed motor skills or coordination	12.6	7.8
Reading disorder/ disability	11.9	5.9
Math disorder/ disability	7.4	3.9
Writing disorder/ disability	9.6	2.9
Spelling disorder/ disability	8.1	2.0
Other learning disorders	5.2	Not assessed
Sleep disorder	Not assessed	2.9
Deaf/hard of hearing	2.2	0
Blind/visually impaired	4.4	0

Note: Both studies included a nationally representative US sample and used parent-report of whether their child had ever received a diagnosis of each listed disorder. Barkley[27] included youth aged 6 to 17 years, whereas Burns and Becker[28] included youth aged 4 to 13 years (although very few participants were aged 4 or 13 years). The higher rates of diagnoses reported in Barkley's study may be due to the age range spanning into adolescence. ADHD = attention-deficit/hyperactivity disorder; CD = conduct disorder; ODD = oppositional defiant disorder.

Emotion. Self-reported SCT has been linked to poorer self-reported emotion regulation among children[36,41] and adults[29,97,98,118,119] (for an exception, see Fredrick *et al.*[35]), although inconsistent associations emerge using parent-reported SCT.[27,28,120] Other methodologies (eg, eye tracking, psychophysiology, daily momentary assessment of affect) may be especially fruitful to advance understanding of SCT and emotion. Studies are needed to disentangle whether the association of SCT and emotion dysregulation

reflects greater emotional distress, more emotional lability, poorer emotional clarity/awareness, or poor self-regulation of elicited emotions.[3]

Learning Disabilities. Parent reports from 2 population-based studies suggest that individuals with elevated SCT were more likely to receive diagnoses of reading disability (6%-14%), specific writing disability (11%), or significant language delay (11%-19%) than were individuals without elevated SCT.[27,28] Rates of learning disabilities were highest among groups with elevations of both SCT and ADHD, although neither study administered standardized academic achievement measures to assess learning disabilities directly. Another study showed that a clinic sample with learning disabilities exhibited elevated rates of SCT.[121] Findings for academic functioning are discussed below.

Intellectual Disability. Meta-analytic results suggest a small but significant correlation between SCT and lower general intellectual ability (overall $r = 0.24$).[10] In children with autism and/or ADHD, SCT scores did not differ significantly among children with intelligence quotient (IQ) scores, ranging from profound to moderate intellectual disability to average intelligence, although children with above-average intelligence had significantly lower SCT scores.[86]

Autism. Diagnoses of autism spectrum disorder (ASD) are more common among children with elevated parent-reported SCT (13%-16%).[27,28] Several studies have also reported SCT elevations among a significant subset of children with ASD (30%-49%).[25,74,122,123]

Substance Use. The single study on this topic found that greater alcohol but not cannabis use was reported among college students with elevated SCT compared to students with low SCT scores.[124]

Sleep. SCT is uniquely associated with parent-reported sleep problems and daytime sleepiness in youth.[24,52,75,125-128] In college students and adults, SCT is uniquely related to global sleep problems, shorter sleep duration, and increased daytime sleepiness.[35,129-131] Three studies document an association between SCT and self-reported eveningness preference.[128,132,133]

Only 2 studies have examined SCT in relation to objective measures of sleep. No significant relations were found between parent ratings of SCT and 14 polysomnography (PSG) indices of sleep in a population-based sample of children.[75] In adolescents with and without ADHD, self-reported SCT symptoms were uniquely associated with shorter sleep duration and later sleep onset, but not sleep efficiency or wake time, per actigraphy.[128] The only study examining SCT in relation to sleep disorder diagnoses did not find associations with behavioral or psychophysiological insomnia, although other sleep disorder diagnoses (eg, parasomnia, delayed sleep phase syndrome) were not examined.[24]

Future Directions in SCT and Comorbidity. There are fewer studies assessing some disorders that may be associated with SCT, including ASD, LDs, PTSD, substance use disorders, and sleep disorders, and no studies have examined SCT and schizophrenia or other psychotic disorders. The findings from 2 national samples reporting a sizeable minority of children with SCT having received a diagnosis of language disorder or delayed motor skills/coordination[27,28] also warrants further investigation. Studies evaluating rates of comorbidity using systematic interviews to assess comorbidity in individuals with SCT are especially needed. Finally, few studies have used etiologically informed methods or longitudinal designs to explore the etiology of these potential comorbidities.

Functional Outcomes and Impairment

The most decisive support for the external validity of SCT would be provided if SCT leads to significant distress or functional impairment that is sufficiently severe to warrant intervention, independent of symptoms of ADHD, depression, and other disorders that often co-occur with SCT. In particular, as ADHD itself has high rates of comorbidity,[134] it is critical to establish whether SCT is uniquely related to functional outcomes and impairment when ADHD and other psychopathology symptoms are taken into account.

Global Functioning. A meta-analysis indicates that SCT is associated with significant global impairment and lower quality of life in children and adults (pooled $r = 0.40$-0.50).[10] Importantly, numerous studies found that this association remained significant when covarying other psychopathology dimensions.[27,29,90,135,136]

Social Functioning. SCT is associated with global social impairment, with overall medium effect sizes in studies of children and adults (pooled $r = 0.37$-0.38).[10] SCT appears to be most strongly associated with shyness, social withdrawal, and loneliness, and these effects remain significant when other psychopathology symptoms are covaried.[28,90,118,137-140] In contrast, SCT does not appear to be associated with active exclusion or dislike by peers,[90,138] and

studies of SCT and social skills have yielded mixed results.[40,41,141]

Academic Functioning. SCT is associated with informant ratings of overall academic impairment in childhood, with a medium pooled effect size (overall $r = 0.44$).[10] SCT is also associated with lower grades[90,142] and difficulties with study skills and homework motivation and performance.[136,143] Associations between SCT and standardized measures of reading, mathematics, or writing achievement are variable, especially after covarying IQ and/or inattention.[28,92,141,144-149] A longitudinal study found SCT and ADHD-IN to be differentially associated with poorer reading or mathematics impairment, respectively, in adolescence.[15] A limitation of many previous studies is the primary use of untimed measures and those focused on basic academic skills.[150] One recent study suggests that SCT is specifically associated with mathematics and reading fluency even after covarying untimed reading and mathematics achievement,[145] underscoring the need for future studies of other more complex and effortful academic processes such as reading comprehension, mathematics word problems, and expository writing.[15,90,145]

Functional Outcomes and Impairment in Adulthood. Initial studies of a nationally representative sample of adults,[29] adults presenting for an ADHD evaluation,[18] and samples of undergraduate college students[98,118,136] suggest that SCT is uniquely associated with functional impairment. Domains include lower occupational functioning and greater difficulty managing household chores and finances, greater driving impairment, and significant impairment in health-related behaviors and aspects of adult social functioning such as marital relationships and parenting.

Future Directions in SCT and Impairment. Future studies should incorporate additional measurement approaches that may be more sensitive or have greater ecological validity (eg, school report cards/transcripts, official driving records). Studies of social functioning may particularly benefit from the integration of multiple methods to fully characterize the nature of the social difficulties that characterize SCT and the mechanisms that explain these associations.[151] Useful methods could include peer ratings of individuals with SCT and observational coding of social interactions across a range of situations to identify specific areas of difficulty.[152] Relatedly, children with SCT may experience distinct impairment in playing sports[27]; this underscores the need for studies investigating a broader range of functioning domains.

SCT and Psychosocial Treatment

Very few studies have investigated whether psychosocial treatments reduce SCT symptoms and/or associated impairments. One randomized controlled trial (RCT) found that a home-school behavioral intervention for children with ADHD reduced SCT symptoms[131]; another trial of this intervention found higher pre-intervention SCT scores to be weakly associated with poorer treatment response.[132] A pilot trial examining SCT in the context of a summer treatment program found pre-treatment SCT symptoms to predict a less robust response to time out, but not response to other treatment domains examined.[153] In an RCT with middle school students, school-based organization/homework intervention reduced SCT symptoms per parent-report but not adolescent-report.[19] A pilot open trial found that an outpatient behavioral sleep intervention reduced adolescents' SCT symptoms across self, parent, and teacher ratings, with parent and teacher effects maintained at 3 months.[154]

There are many avenues for future research in psychosocial intervention and SCT. One pressing research question is whether SCT moderates treatment response in clinical samples, such as individuals being treated for ADHD, depression, or sleep problems. Evaluating whether individuals with elevated SCT differentially respond to current evidence-based treatments for ADHD (eg, behavioral parent training) or internalizing disorders (eg, cognitive–behavioral therapy [CBT]; mindfulness-based practices) is an important area of future research. Relatedly, although traditional social skills training is often considered ineffective for youth with ADHD, these interventions may improve assertiveness for socially withdrawn youth,[155,156] which may benefit those with SCT.

No intervention has been developed specifically for individuals with SCT. This is in part because of the limited understanding of core factors that contribute to or exacerbate SCT symptoms, as well as mechanisms linking SCT to impairment, which can inform treatment development. Based on findings of functional impairments, however, treatment development could incorporate current evidence-based interventions including behavior activation, CBT, sleep hygiene/CBT for insomnia, social skills training, time management/organizational training, and mindfulness-based practices, depending on individual needs. Developmental factors will also be important to consider. For example, social skills and parent management training will likely be beneficial for younger children,[157] whereas cognitive restructuring and mindfulness-based interventions may be appropriate for adolescents and adults. Attention checks (possibly with rewards for younger children), training to bolster organization, time management, and

planning skills, and sleep interventions may be beneficial across development. Finally, families of children with SCT report wanting at least some degree of parent involvement while also noting a willingness for treatment to be provided in the school setting.[49]

SCT and Pharmacologic Treatment

Methylphenidate, lisdexamfetamine, and atomoxetine may be helpful in addressing SCT symptoms. SCT symptom ratings improved with methylphenidate in an open-label trial of children with ADHD, although response varied somewhat by informant.[20] Among adults with ADHD, SCT symptoms improved with lisdexamfetamine, with moderately large effect sizes that are largely independent of changes in ADHD symptoms.[158] Similarly, atomoxetine improved SCT symptom ratings independent from changes in ADHD symptoms[159] in an RCT of children with ADHD and/or dyslexia.[160] Beneficial effects of atomoxetine on SCT symptoms (including sluggishness/sleepiness) are notable given its side effect profile, with 15% to 17% reporting somnolence with atomoxetine (vs 2%-4% with placebo) in controlled trials in ADHD.[161,162]

Alternatively, SCT may moderate psychotropic medication response in pediatric ADHD. An RCT of children with ADHD suggested that increased SCT Sluggish/Sleepy, but not Daydreaming, scores were linked to methylphenidate non-response and diminished dose-response for inattention symptoms.[163] A subsequent open-label trial similarly found higher pre-treatment SCT Daydreaming symptoms and SCT Total symptoms to be associated with diminished methylphenidate effects on teacher-rated inattention and total ADHD symptoms, respectively.[20] In contrast, a naturalistic study of children with ADHD-IN did not find CBCL-defined SCT symptoms to moderate methylphenidate response.[164]

Further study of the moderating effects of SCT dimensions on stimulant and non-stimulant response is needed, as well as replication of main effects in samples selected for elevated SCT rather than ADHD. Data are needed regarding medication effects on SCT-related neuropsychological and neurobiological processes. In addition, investigation of the effects of psychotropic medications addressing SCT comorbidities may be fruitful. Alternative potential medications include bupropion and viloxazine; although both are primarily used to treat depression,[165,166] bupropion shows utility as an off-label treatment of inattention,[167] and viloxazine is Food and Drug Administration (FDA) approved for pediatric ADHD treatment.[168] In addition, modafinil, FDA approved for the treatment of narcolepsy and associated daytime sleepiness,[169] is a logical candidate to address the sluggish/sleepy aspects of SCT and may target SCT-associated abnormalities in the dorsal attention network.[107,111,170] Finally, guanfacine may ameliorate signal detection errors in SCT, consistent with evidence for reduced errors of omission in ADHD.[171,172] However, side effects of α-agonists such as guanfacine and clonidine include high prevalence of somnolence or sedating effects,[173,174] necessitating dose adjustments or changes in time of administration. Furthermore, clonidine may exacerbate SCT-related deficits in attentional orienting.[175]

Concluding Comments on Key Future Directions

There are clearly many avenues to pursue in the next generation of SCT-related research. In addition to considerations that cut across domains (Table 1), there are important ways to advance the existing research base in each domain included herein. Together, research in these domains will be necessary to build theoretical, etiological, and developmental models of SCT, in addition to advancing prevention and intervention efforts.

RESULTS AND DISCUSSION: RATIONALE AND PROPOSED CHANGE IN TERMINOLOGY

The original term, sluggish cognitive tempo (SCT), was initially used in the mid-1980s in a dissertation by a graduate student, Ronald Neeper, working at the University of Georgia with Benjamin Lahey and Caryn Carlson.[8,176] Those early studies explored new and possibly better symptoms for the identification of children with ADHD than were included in the *DSM* of that era. Results of the project serendipitously identified a set of symptoms that formed a dimension distinct from the symptoms that defined ADHD (inattention, hyperactive-impulsive). That dimension was labeled as SCT to capture the aspects of staring, daydreaming, spaciness, mental confusion, slow movement and hypoactivity, among others that loaded on this unique dimension. In the interim, the term has been used in research studies, clinical practice, rating scales, and book chapters, in some trade media, as well as on the Internet. Several problems have arisen that now warrant its replacement.[12,13] First, the term is considered derogatory or demeaning and hence potentially offensive to patients and their families. In particular, "sluggish" can be perceived as derogatory or implying laziness or low intelligence,[12,177] and a qualitative study found almost half of parents of children with elevated SCT symptoms had a negative reaction to the SCT term.[49] In addition, "SCT" is likely incorrect, given that subsequent research reviewed earlier has not typically supported slow cognitive processing or tempo as a reliable feature of this condition, much less as its central deficit.

TABLE 4 Considerations in Deriving the Change in Sluggish Cognitive Tempo Terminology

Terms considered	Inclusionary justification	Exclusionary justification
Sluggish		3, 7
Cognitive	4	
Tempo		2, 7
Daydreaming		5, 9
Mind wandering		5, 9
Staring	2, 6	4
Concentration		5, 9
Attention		2, 5
Disengaged	4	
Decoupled (/ing)	4	6
Slow		3, 7
Underactive	2, 4, 6	5
Under-arousal		2, 5
Hypoactivity	2, 4	5
Syndrome	1, 4	
Deficit		5
Disorder	1	2, 8
Maladaptive	1	2
Constraint of thought		2, 6

Note: Numerous principles were considered when evaluating possible terminology, with each principle able to be considered either inclusionary or exclusionary. Principles used in evaluating possible terminology are as follows:

1. Indicative of impairment when present at high levels
2. Observable and avoids unsubstantiated claims about the construct
3. Pejorative or demeaning terminology
4. Broadly describes the constellation of symptoms without being overly general
5. Overlaps with extant psychology/psychiatry literatures, terminology, and idioms, thus causing unnecessary confusion
6. Face valid and accessible by general public
7. Empirically shown to be inaccurate
8. Implies official designation as an established clinical condition
9. Risks over-pathologizing normative behaviors

In deliberations concerning replacement terminology, the Work Group considered a variety of terms that might best capture the several symptom dimensions that have been reliably identified with this construct (Supplement 3, available online, provides additional details). Table 4 lists these various terms and the advantages and disadvantages of each as a possible replacement term. Following repeated discussions of these terms, Work Group members selected "cognitive disengagement syndrome" (CDS) as most appropriate, given that it seemed to best satisfy the conditions that should apply in the selection of such terms for a condition or syndrome, as listed in the footnote in Table 4. It should be noted, however, that although the group agreed to recommend CDS, it was challenging to arrive at a term that was not overlapping with established terms for other constructs, was not offensive, and reflected the current state of the science, and it was agreed that the term will likely continue to evolve as the science advances.

CDS refers to a set of developmentally inappropriate and persistent behaviors (symptoms) that is best characterized as follows: (1) cognitive symptoms involving the disengagement or decoupling of attention and conscious or effortful mental processing from the ongoing external context, as reflected in difficulties with staring, daydreaming, mental confusion, or fogginess, withdrawal, and sleepy appearance; and (2) motor symptoms involving hypoactivity as manifested in underactivity, periods of passive or sedentary movement, and slow, reduced, or delayed motor movements. We note that although motor symptoms are not represented in the CDS term, this is because a briefer, more accessible term was preferred by the Work Group, not because the motor symptoms are viewed as less primary or pronounced than the cognitive symptoms. This constellation of symptoms is considered to be a syndrome because of the higher co-occurrence (intercorrelation) or coherence of these symptoms with each other and the interrelatedness of its dimensions relative to their relationship with symptoms/dimensions of other psychopathologies (ie, internal validity) and unique association and prediction with functional outcomes when covarying other psychopathologies (ie, external validity).

Additional research is needed to clarify how best to conceptualize CDS. There are several possibilities, including CDS being viewed as having transdiagnostic importance (including but not limited to ADHD),[11] important for understanding ADHD heterogeneity,[178] or its own psychiatric disorder.[12] Although a conclusion regarding these possibilities cannot currently be made based on the existing evidence (see Table 1 for limitations of existing research, and discussion of diagnostic validity in Becker *et al.*[10]), we believe that it is nevertheless time to change the terminology even as empirical evidence continues to accumulate that can inform such possibilities. In addition, studies examining CDS in various ways are needed to directly inform whether CDS is best conceptualized as a transdiagnostic factor, diagnostic specifier, or distinct disorder. Here, the different conceptualizations of chronic irritability may serve as a useful example as it is currently considered a specifier for ODD in the *International Classification of Diseases, 11th*

Revision (*ICD-11),* categorized as disruptive mood dysregulation disorder (DMDD) in the *DSM-5,* and conceptualized as a possible subtype of ADHD[179] or important transdiagnostic factor across a wide range of disorders.[177] It would be beneficial for research on CDS to examine similar possibilities *prior* to making determinations of how to best conceptualize CDS, to avoid some of the challenges and critiques that have arisen in classifying chronic irritability within DMDD in the *DSM-5*.[180,181] Relatedly, as the science continues to advance, there will also be a need to develop a tentative definition and criteria for identifying individuals with CDS. In addition to establishing the presence of developmentally inappropriate symptoms (Table 2), other criteria that should be considered include duration of symptoms that have lasted for at least 6 months and that result in impairment (ineffective functioning or distress) in one or more major life activities. As research accumulates, the CDS term can be re-evaluated, and qualitative and participatory action research on the CDS term itself would be highly informative. In the meantime, we encourage all researchers and clinicians interested in the study of this construct to join us in transitioning from using "SCT" to using "CDS" instead. To maintain continuity with existing SCT literature and to allow for the time that it will take before the CDS term gains widespread use, and given that some measures currently have SCT in their titles, it would be beneficial to use both "cognitive disengagement syndrome" and "sluggish cognitive tempo" as key words and on first use in text (eg, "cognitive disengagement syndrome [CDS], previously termed sluggish cognitive tempo").

In conclusion, this is an exciting time for the scientific study of CDS (SCT). Available evidence supports the bifurcation of attention syndromes from one of primarily ADHD-related symptoms to a second involving CDS (SCT). To experts in the field, it is evident that CDS has reached the threshold of recognition as a distinct syndrome. Still, there is much more work to be done in further clarifying its nature, etiologies, demographic factors, relations to other psychopathologies, and linkages to specific domains of functional impairment. Many directions remain ripe for future study. Investigators are needed with interests and expertise spanning basic, clinical, and translational research to advance our understanding of CDS and to improve the lives of individuals with this unique syndrome.

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