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Sluggish Cognitive Tempo (Concentration Deficit Disorder?): Current Status, Future Directions, and a Plea to Change the Name

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Abstract Symptoms of sluggish cognitive tempo (SCT) have been recognized for nearly 30 years as comprising a semi-independent set(s) of symptoms from the inattentive (IN) and hyperactive-impulsive (HI) symptoms involved in attention-deficit/hyperactivity disorder (ADHD). It has only been within the past decade that research focusing specifically on SCT symptoms and on samples of SCT cases chosen independently from ADHD samples has increased so as to address the question of whether SCT is a distinct condition from ADHD or other disorders. All but two of these studies have focused on children but the two extant large scale studies on adults have replicated those findings. This Commentary highlights not only those findings concerning SCT that appear to be relatively robust, but also those patterns that appear to be emerging yet in need of further research to corroborate their association with SCT, as well as those barely or unexplored areas that may deserve more research. Evidence to date, including the many findings in this special issue, is nearing a critical mass that likely supports the conclusion that SCT is a distinct disorder of attention from ADHD, yet one that may overlap with it in about half of all cases. SCT has unique symptom dimensions and comorbidities from ADHD, probably distinct though lesser domains of impairment and demographic correlates, and perhaps unique cognitive deficits, causes and life course risks. These latter areas, however, are in need of substantially more research as is SCT in adults and treatments specifically designed for cases of SCT. Meanwhile, the name of the condition is premature, implying a known cognitive deficit that is as yet unknown, and is proving derogatory and offensive to patients, leading this author to recommend a change to Concentration Deficit Disorder.

Keywords Sluggish cognitive tempo (SCT) · Attention-deficit/hyperactivity disorder (ADHD) · Symptom dimensions · Comorbidity · Impairment · Etiology

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It is both a privilege and a pleasure to be invited by the Editors of the Journal of Abnormal Child Psychology to summarize the current status and future directions of research on sluggish cognitive tempo (SCT) in view of the results of the excellent collection of research papers on this topic contained in this special section. While space precludes a lengthy discussion of all of the current findings and potential future directions related to this topic, this paper will focus on those issues which the author believes are most important at this time. Although the first description of an attention disorder similar to SCT may have been in the medical textbook by Alexander Crichton (1798), probably the first person to coin the term for a distinct factor of inattention from that seen in ADHD was a graduate student of Caryn Carlson's named R. Neeper (Carlson 1986). Since its inception, the term SCT remains a highly understudied construct and associated pattern of symptoms (and disorder) within the field of clinical psychology and psychiatry—a situation which this special issue was developed to partially address. More than 10,000 articles exist (and more than 4,000 of those have been published just since 2007) on the other attention disorder currently known as attention-deficit/hyperactivity disorder (ADHD). Yet fewer than 50 exist on SCT. This circumstance clearly warrants that substantially more research be directed at all aspects of SCT (vs. ADHD and related disorders as well as typical people) including demographics, correlates, comorbidity, families, and especially etiologies, interventions, and life course risks.

Students now entering the profession could easily make a successful clinical research career specializing in the study of SCT given the paucity of research and the great promise of distinct findings foreshadowed by the results of current research including that contained in this special issue. There will surely be an increased demand for such empirically-based knowledge in view of the increasing clinical referrals occurring now and more rapidly in the near future driven by increased awareness of the general public in SCT. The fact that SCT is not recognized as yet in any official taxonomy of psychiatric disorders will not alter this circumstance given the growing presence of information on SCT at various widely visited internet sites such as YouTube and Wikipedia, among others.

Acknowledging that far more research on SCT needs to be done does not diminish the fact that some very solid findings already exist. Promising patterns of new findings are also becoming evident that I will try to briefly summarize here. Growing evidence demonstrates that SCT is most probably a distinct disorder from ADHD but may overlap with it. Below I highlight some conclusions I believe can be drawn from the evidence base so far along with suggesting some directions for future research.

Symptoms

SCT demonstrates a clear separation of symptom dimensional structure (usually via *factor analysis) from the two dimensional structure of ADHD*. While correlated to a low-to-moderate degree with the ADHD symptom dimensions, the two (or more) dimensions of SCT are more highly correlated with each other than with those of ADHD. The relationship between SCT and ADHD dimensions is similar to that found for other dimensions of psychopathology believed to be semi-related yet also mostly distinct from each other, as between anxiety and depression or between oppositionality and ADHD. Also, the SCT dimensions are as closely related to each other as are the two ADHD dimensions to each other. In short, there is greater within-disorder shared variance between symptom dimensions than cross-disorder shared variance. This has been shown in several of the studies in this issue (Becker et al. 2013a; Lee et al. 2013; McBurnett et al. 2013; Willcutt et al. 2013) that replicate a number of past studies on children (Barkley 2013; Garner et al. 2010; Hartman et al. 2004; Penny et al. 2009). The distinctiveness of SCT symptoms from those of ADHD has also been found in direct observational studies of children during testing (McConaughy et al. 2009a) and in school settings (McConaughy et al. 2009b). It has also been evident in a general population representative U.S. sample of adults (Barkley 2012) and a large sample of college students (Becker et al. 2013a). Moreover, SCT symptoms identify a unique group of children even within samples that have ADHD Inattentive Type (Capdevila-Brophy et al. 2012; Marshall et al. 2013). However, findings in the latter type of research have not been as strong or numerous as when SCT groups are selected from community or other samples specifically for SCT first and not for ADHD. In sum, the distinctiveness of SCT symptom dimensions from those of ADHD is now a reliable, well established conclusion.

Of course, there remains the issue of precisely which and how many symptoms should be included in identifying SCT and which should be abandoned as representing excessive overlap with other disorders (e.g., low energy with depression; sleepiness with hypersomnia, etc.). But that situation is no different than the comparable debate that takes place about ADHD diagnostic criteria prior to every new edition of the *Diagnostic and Statistical Manual for Mental Disorders* (DSM) and elsewhere. Though mixed to date, findings from research using at least 10 or more symptoms demonstrate at least two dimensions that seem unique to SCT yet are intercorrelated sufficiently to be combined in this disorder, these being a Daydream/Slow and a Sleepy/Sluggish/Underactive factor (Barkley 2013; Jacobson et al. 2012; Penny et al. 2009). The factor of low initiative/impersistence seems as or more related to ADHD Inattentive symptoms (IN) and thus not of much help in differential diagnosis (Barkley 2013). Thus as in ADHD there is a cognitive-inattentive dimension and a behavioral-motor dimension to SCT yet both are reasonably distinct from those evident in ADHD. Demonstrating the robustness of this semi-independence of SCT from ADHD symptoms has been a very important step forward in the past decade showing that there is a second condition of inattention in the population apart from that seen in ADHD.

There is little if any association of SCT symptoms with the hyperactive-impulsive (HI) symptoms in ADHD when the moderate overlap of SCT with the ADHD IN symptom dimension is statistically controlled as was done by Lee et al. (2013) in this issue and earlier by others (Barkley 2012; Penny et al. 2009). Yet the ADHD IN dimension is positively correlated with that HI dimension. This represents an important demonstration of a double dissociation essential to arguing that SCT is a distinct disorder from ADHD and not a proxy for it or subtype of it.

Comorbidity

Concerning comorbidity, there is no or even a negative association of SCT with oppositional defiant disorder (ODD) as was evident in the paper by Lee et al. (2013) in this issue and several earlier studies (Barkley 2013; Penny et al. 2009). In contrast, ADHD is associated with an 11 times higher risk for ODD (Angold et al. 1999). Here again one finds an important additional double dissociation in research results supporting

the distinctive nature of SCT from ADHD. Moreover, as a consequence of this lack of association with ODD, one is likely to find that SCT also has low or no associations with conduct disorder, substance use disorders, or adult antisocial personality disorder, all of which are linked to varying degrees with ODD. Further evidence for this lack of or even negative association with disruptive behavior disorders comes from the results for the direct observations of disciplinary actions [time outs] received on an inpatient unit noted in the study by Becker et al. (2013b) in this issue. As the authors noted, such disciplinary actions are often instituted for disruptive or aggressive behavior and, as expected, were positively linked to the HI symptoms of ADHD while being negatively associated with SCT symptom severity. This is yet again another demonstration of a double dissociation between SCT and ADHD critical to establishing the separate existence of a disorder of SCT.

There is a greater association of SCT with internalizing symptoms than is the case for ADHD symptoms. This association remains even when the IN symptoms of ADHD are statistically removed as was evident in several papers in this issue (Lee et al. 2013; Willcutt et al. 2013). Indeed, when the inverse is done such that SCT symptoms are statistically removed, the IN dimension of ADHD may show no relationship to internalizing symptoms (Lee et al. 2013; Penny et al. 2009) or even to ratings of social problems (Becker et al. 2013b). Moreover, where the relationship of SCT to ODD is negative, the relationships of SCT to anxiety and depression are positive (Lee et al. 2013); a pattern that is different than that seen in ADHD where the relationships to both ODD and internalizing symptoms are both positive. SCT was found here to predict each of these internalizing dimensions (anxiety, depression) even after controlling for the overlap of the latter dimensions with each other (Becker et al. 2013b). Furthermore, the link of SCT to depression remains even after controlling for parental internalizing dimensions as was done by Becker et al. (2013b) in this issue.

Demographics

Although the body of evidence is not yet robust enough to make definitive statements, *emerging patterns of results suggest that the demographic correlates associated with SCT are different from those evident in ADHD.* For example, my own studies have found little or no evidence of substantial sex differences in SCT symptoms in children (Barkley 2013) or adults (Barkley 2012) whereas males demonstrate significantly greater ADHD symptoms in childhood and adolescence in these and many other studies. SCT was also not associated with age whereas ADHD symptoms in these studies, specifically the HI dimension, declined significantly with age across childhood (Barkley 2012). In the study of children, I (Barkley 2013) found that children with SCT were older than those with ADHD, implying a somewhat later age of onset for the former symptoms. This lack of association of SCT with age and sex was also evident here in the study by Lee et al. (2013) who found no sex differences and no effect of age on teacher ratings and only a very small relationship of those demographics in parent ratings. There is also no association of SCT with ethnicity whereas the HI symptoms of ADHD appear to demonstrate such a relationship (higher in Hispanic-Latino groups) (Barkley 2012, 2013). Earlier studies also found no such relationships of SCT with age, sex, and ethnicity (Garner et al. 2010; Jacobson et al. 2012). Surprisingly, in contrast to ADHD, my own national surveys of children (Barkley 2013) found a greater association of SCT with lower parent education, lower income, and a greater likelihood of having a parent out of work due to disability. In the survey of adults (Barkley 2012), those having SCT likewise had less education and income, and in the group comorbid for SCT+ADHD, there was a greater risk that the adult was out of work due to disability. Worth future study is whether such findings of greater social adversities in SCT contribute to its linkage with anxiety and especially depression that are known to have social adversities or stressors as correlates or contributors.

Impairment

Of course, having a distinct set of symptoms and demographic correlates by themselves are not supportive of concluding that a new disorder of SCT has been identified. Disorders require that there be evidence of impairment or harm to or adverse consequences for the individual from those symptoms (American Psychiatric Association 2013). We can think of symptoms as the cognitive and behavioral expressions of a disorder while impairment represents the consequences that flow from such symptoms. The body of evidence concerning impairments in SCT is small and somewhat mixed depending on the domain of impairment examined. Nevertheless, some areas of distinct impairments seem to be emerging.

Among the most robust findings concerning impairment, research shows that *SCT is associated with distinct types of social impairment even after controlling for the overlap with ADHD* (Barkley 2012, 2013; Penny et al. 2009). The nature of the social problems linked to SCT differs from the ones evident in ADHD (aggressive, emotional, disruptive behavior leading to rejection) (Mikami et al. 2007). In SCT, as shown in several articles in this issue (Becker et al. 2013b; Marshall et al. 2013; Willcutt et al. 2013) and in prior direct observational research (Mikami et al. 2007), the problems are those of social withdrawal or isolation. This linkage of SCT to social withdrawal remains even after statistically removing ADHD symptoms as well as those of ODD, CD, generalized anxiety

disorder, major depressive disorder, and even IQ as shown in the studies herein. And while SCT and the IN dimension of ADHD contribute to variance in social problems and apparently peer neglect, their contributions are independent or additive, not redundant (Willcutt et al. 2013). Similarly, the study by Becker et al. (2013b) in this issue showed that the positive association of SCT with general social problems was apparently not due to disruptive social problems, given the association noted above with significantly lower rates of discipline in inpatient children. Such relationships of SCT to social withdrawal as were evident in several studies in this issue remained even after controlling for demographic factors and comorbidity. These results are quite consistent with earlier ones demonstrating that SCT, whether studied independently of ADHD or within ADHD groups, is linked to social withdrawal, reticence or frank social anxiety. Thus SCT contributes unique variance to certain areas of social impairment independent of other disorders including ADHD.

An interesting question to explore is the causal pathways or relationships among SCT, anxiety, depression, and social problems. Which originates first and then links to the others? A case can be made for all possible hypotheses here in that the daydreaming or mind wandering nature of SCT may lead to social difficulties that then foster an increase in anxiety or depressive symptoms. Or SCT may represent a more ruminative mind that is prone to anxiety and depressive symptoms which then lead to social problems. Inversely, it may be that greater symptoms of anxiety or depression lead to either social withdrawal or to SCT symptoms with either of these latter problems then leading to the other. This hypothesis does not seem as feasible as the others given extant findings that while SCT is linked to anxiety and depression, the correlations are moderate and thus they share only a minority of their variance. Moreover, this hypothesis would require high levels of anxiety or depression be present in all cases of SCT if that is the starting point for the latter disorder. That does not seem to be the case. Barkley (2013) found that only a minority of SCT children had a prior professional diagnosis of depression (as reported by parents) even though that percentage was significantly elevated compared to ADHD and control groups.

The association of SCT with unique difficulties in academic functioning is emerging but not as well established as that for social withdrawal given a history of mixed results (see Langberg et al. 2013, this issue). In my own national surveys, SCT was not as strongly linked as ADHD to academic or work related impairment in children (Barkley 2013) but was actually more impairing in adult self-reports of educational and occupational impairment than was ADHD (Barkley 2012). In both studies, when comorbid, SCT+ ADHD disorders were additive, resulting in far more severe impairment and more domains of impairment than either disorder alone. Some of this inconsistency in findings may be due to the fact that some earlier studies of this issue selected their samples for ADHD first and then within such samples examined those high and low in SCT symptoms. This can contaminate any findings for SCT with those known to be related to ADHD. Even so, when symptoms of ADHD are statistically removed, significant findings concerning academic functioning seem to show the same pattern as was evident for social problems discussed above. That is, SCT appears to add unique variance to the prediction of academic problems (Barkley 2013) and may make unique contributions to written language and reading, organization problems, and homework specifically beyond the contribution of ADHD IN symptoms. This pattern was evident in a number of studies in this special issue (Langberg et al. 2013; Marshall et al. 2013; Willcutt et al. 2013). Difficulties with math performance may also be more evident in SCT than in ADHD, although this finding, too, requires replication to be considered reliable (Bauermeister et al. 2012). Yet not all studies find so convincing a relationship between SCT and academic impairment (Watabe et al. 2013).

I believe that the weight of the evidence suggests that SCT is associated with unique impairments in social functioning in comparison to ADHD. More research is needed to verify that it also is linked to unique forms of academic impairment as these are not as strongly established as are the social difficulties. It is clear that the IN symptoms seen in ADHD are more contributory to academic performance problems than is the form of inattention seen in SCT (Barkley 2012, 2013) yet SCT does seem to create impairment beyond what can be accounted for by its overlap with ADHD IN (Barkley 2013; Bauermeister et al. 2012; Langberg et al. 2013; Willcutt et al. 2013). Apparently, by adulthood, the contribution of SCT to educational and occupational impairment may exceed that of ADHD (Barkley 2012). Far more research on the nature of the specific impairments associated with SCT is in order but a pattern of evidence is emerging that the problems associated with SCT may be unique from ADHD to some degree, even if of a far lower general magnitude.

One question that was raised in the findings above and earlier by Lahey (2001) is *whether SCT simply represents the attention problem associated with and thus serves as merely a proxy for other established dimensions of psychopathology, especially anxiety and/or depression.* After all, symptoms such as lethargy, low energy, and sleepiness may signal depression while those related to daydreaming and staring might be evident in anxious states or even post-traumatic stress disorder. Though possible, this seems unlikely to me as most childhood cases of SCT are not found to have anxiety or depression diagnoses (Barkley 2012). However, my survey was limited by the use of parent reports of prior professional diagnoses and not direct clinical diagnostic evaluations conducted in the project. Several papers here speak to this issue, however, and support the view that SCT is distinct from internalizing disorders even if comorbid with them. For

instance, the study by Willcutt et al. (2013) showed that a separate dimension of SCT can be found in their factor analysis that is distinct from anxiety and depression symptoms and that none of the SCT symptoms have significant cross-loadings on the anxiety or depression dimensions. Other papers showed that while SCT was correlated with those dimensions of psychopathology, the correlations are such that the dimensions do not share a majority of variance with each other. Thus they are not mere proxies for each other.

Cognitive Deficits

While evidence for the specific and unique cognitive dysfunction(s) that may underlie SCT is lacking, prevailing evidence, especially from rating scales, is suggesting that *SCT is not primarily a disorder of executive functioning (EF) as manifested in daily life activities or on most EF tests* (Barkley 2012, 2013; Bauermeister et al. 2012; Willcutt et al. 2013). In contrast, ADHD is a serious and pervasive EF disorder across dimensions of EF ratings in which the vast majority of cases place in the bottom 7 % of the typical population, such as in time management, self-organization, problem-solving, self-restraint, the self-regulation of emotion, and self-motivation (Barkley 2012, 2013; Barkley and Murphy 2010) or in ratings of behavioral regulation and metacognition (Becker and Langberg 2013). ADHD also is often, though not always, associated with deficits on neuropsychological tests of EF, particularly inhibition, working memory, and response variability (Frazier et al. 2004; Hervey et al. 2004; Willcutt et al. 2005). There is a small contribution of SCT to omission errors on continuous performance testing as shown in this issue, implying it is some type of attention disorder but not like the one evident in ADHD. This dissociation of SCT from EF deficits in daily life implies that the cognitive dysfunctions underlying SCT symptoms are not like those involved in ADHD; thus nor is SCT a subtype of ADHD.

While McBurnett et al. (2013) in this issue found some items related to the EF component of working memory to be a useful additional dimension of SCT symptoms beyond those usually found in community studies, some caution concerning this opinion is in order. That reservation arises from the fact that the sample used in that study was a large group of children with ADHD Inattentive Type referred for a psychosocial treatment program. Such a sampling procedure can lead to a rather heterogeneous group, only a subset of whom can be expected to be SCT cases and none purely so. Moreover, others may well be subthreshold variants of ADHD Combined Type that fall just shy of the necessary six HI symptoms, as McBurnett et al. noted. These cases are likely better construed as mild versions of ADHD-C. The same problem afflicts the Langberg et al. (2013) study in this issue which also used ADHD referrals as a starting point for analyzing the academic correlates of SCT. As noted above, drawing samples of SCT from patients referred for ADHD can result in contamination of SCT cases with findings linked more to ADHD symptoms or make it seem as if SCT is more strongly linked to ADHD and hence may be a subtype of it. While the disorders do overlap such that SCT cases can be found within ADHD samples, the comorbid cases are far more impaired than either group alone (Barkley 2012, 2013) and have many of the same correlates of ADHD only samples, such as pervasive impairments across settings and EF deficits on rating scales that are not evident in SCT only samples (Barkley 2012, 2013).

In view of the numerous studies attesting to EF deficits in ADHD, particularly working memory, this finding in the study by McBurnett et al. (2013) may be an artifact of recruitment that may not replicate in community samples from which SCT cases were directly sampled. The relationship to or independence of SCT from ADHD will only be definitively tested if samples of each are recruited through community or general clinical samples and not from clinical referrals having ADHD.

Even so, perhaps consistent with the McBurnett et al. (2013) finding, I did observe in large population based samples of both adults (Barkley 2012) and children (Barkley 2013) that SCT did have a very modest but significant relationship with an organization and problem solving dimension of an EF rating scale (5 % of variance shared) even after statistically controlling for both ADHD symptom dimensions. This EF dimension likely reflects items similar to the working memory factor found by McBurnett et al. (2013). Becker and Langberg (2012) likewise found a smaller contribution of SCT to the metacognitive factor on the Behavior Rating Inventory of Executive Functioning than that found for ADHD IN symptoms. This link of SCT to EF-like problems was also evident in the study by Langberg et al. (2013) here but only for parent reported organizational problems. In contrast, only ADHD IN symptoms were related to teacher reported organizational problems. It is possible that problems with certain aspects of working memory may be related to or possibly secondary to the cognitive SCT daydreaming dimension. But such working memory/organizational problems hardly compare to the more severe and pervasive EF deficits so evident in ratings of daily life in children and adults with ADHD (Barkley 2012, 2013).

Though currently under-studied, some research does suggest that SCT may have a unique set of cognitive dysfunctions from ADHD. For instance, a deficit in early information processing or selective attention may exist in SCT that is not characteristic of ADHD (Huang-Pollock et al. 2005). Likewise, slower motor speed has been linked to SCT in some studies (Adams et al. 2010; Garner et al. 2010), as might be expected from its behavioral symptom profile, but not in others (Bauermeister et al. 2012). Skirbekk and colleagues (2011) found that variability of spatial memory performance

was specifically linked to SCT but not ADHD even after controlling for IQ, ADHD inattention, and other variables. These results suggesting a distinct cognitive deficit in SCT apart from that evident in ADHD remain to be replicated but offer some promise that future research may find such distinctions.

The Overlap of SCT with ADHD

Evidence indicates that SCT can overlap with ADHD but also exists independent of it indicating a pattern of partial comorbidity, such as that which is evident between anxiety and depression. The possibility of such overlap was automatically ruled out when the historical precursors of SCT, known as ADD without hyperactivity and later as the Inattentive Type of ADHD, were viewed as subtypes of ADD or ADHD. Yet the semi-independence of the symptom dimensions of SCT from those of ADHD, as demonstrated in numerous papers in this special issue as well as many earlier studies discussed above, argue for at least a partial separation of the disorders that are based upon those symptom dimensions. This is precisely what has been found in my two recent national surveys that covered the age range from 5 to 89 years (Barkley 2012, 2013). In the study of U.S. children (Barkley 2013), more than half (59 %) of those cases that had SCT also had ADHD. The overlap was mainly with those ADHD DSM-IV subtypes having significant IN symptoms rather than with the HI-Type. These results are in keeping with those of earlier studies in children (Garner et al. 2010; Penny et al. 2009; Skirbekk et al. 2011). Conversely, 39 % of the children qualifying for ADHD (any type) also qualified for SCT, which is also consistent with prior studies of children (Garner et al. 2010; Hartman et al. 2004). In the survey of U.S. adults (Barkley 2012), I found that 5.8 % of the sample met criteria for high SCT symptoms and that about half (54 %) of them had ADHD. However, nearly half did not, indicating that SCT can occur independently of ADHD in U. S. adults. Similarly, about half of the cases having ADHD (46 %) also met criteria for SCT. When viewed in the context of the totality of findings on SCT, these results were interpreted as indicating a pattern of partial comorbidity between two distinct attention disorders rather than as evidence of SCT being a subtype of ADHD and hence sharing the same attention deficit.

The Nature of SCT

Remaining to be answered is the issue of just what is the nature of the mental dysfunction(s) in SCT? As Lee et al. (2013) noted in this issue and McBurnett et al. (2013) as well both here and even earlier (McBurnett et al. 2001), there is a clear need to select items that are not mere proxies for depression or sleep problems. It is possible that SCT represents a dysfunction in the focus/execute component of attention in Mirsky's (1996) model of attention components or in the vigilance component as some have conjectured here as being a Primary Disorder of Vigilance. It is also possible that SCT is a form of hypersomnia or arousal disorder given that some dimensions of SCT identified in past research include symptoms of sleepiness, low arousal or energy, or drowsiness (Penny et al. 2009). A very interesting hypothesis is that SCT may represent a form of pathological mind wandering (Adams et al. 2010). Past research suggests that mind wandering is commonplace and advantageous under certain conditions. It arises when a primary task being performed demands little EF capacity and thus allows the contemplative or problem-solving capacity of the EF system to focus on more salient personal concerns. The latter then becomes a secondary task that is engaged while the individual performs the relatively automatic actions toward familiar goals (primary task) in the environment (Smallwood and Schooler 2006). When poorly regulated, however, mind wandering can lead to adverse effects on performing EF tasks (perhaps due to reduced meta-awareness or self-monitoring of goal pursuit, diminished working memory capacity available for pursing the external goals, etc.) (Smallwood and Schooler 2006). It can also adversely impact academic performance (Smallwood et al. 2007). One could also speculate that SCT could arise from a ruminative/obsessional disorder perhaps being a milder variant of obsessive compulsive disorder. Excessive and recurrent focusing on maladaptive thoughts might well lead to an attentional problem resembling SCT. Or is SCT an amotivational syndrome in which the person lacks not only energy but also initiative or self-motivation? Though possible this seems unlikely given that research has not linked SCT to deficits in self-motivation as reflected on EF rating scales in children or adults once the overlap with ADHD symptoms is statistically removed (Barkley 2012, 2013). Similar findings in several studies in this issue would also argue against this idea given that symptoms related to lack of task persistence, initiative, or motivation were as or more related to ADHD as to SCT and thus not useful items for discriminating between them.

Etiology

Abundantly clear in the extant literature is the crying need for research into the etiologies of SCT. Only a handful of studies exist on this topic, such as the study here by (Moruzzi et al. 2013) on the heritability of SCT. Even that study is limited by the very short list of symptoms employed to identify SCT that raise concerns about its reliability of measuring SCT and also generality of its findings. Of interest in their findings is that while SCT is substantially heritable and shares approximately half of its genetic contribution with that of ADHD, it is less

heritable and may involve a greater contribution of unshared or unique environmental factors than is seen in ADHD. That is consistent with another study showing that SCT may be associated with prenatal alcohol exposure (Graham et al. 2012) and with the treatment emergent side effects, lower IQ, and lower academic achievement in acute lymphoblastic leukemia (Reeves et al. 2007). The discussion of demographic factors linked to SCT above also suggests that there may be a greater role for social adversities to contribute to it than may be the case for ADHD. But this evidence is only suggestive, and causal pathways here could prove to be in the opposite direction than that suggested here or even be bidirectional. Therefore, like ADHD, SCT is likely to have multiple etiologies that largely fall in the realm of neurobiological and genetic factors but perhaps less strongly so than is seen in ADHD. Studies using neuro-imaging as well as more behavioral genetic and molecular genetic studies are needed to differentiate SCT from other disorders, especially from ADHD. In doing so, researchers must take care to be aware of the comorbidity of SCT with ADHD as failure to study cases of exclusively one or the other will be contaminated with ADHD related results.

Treatment

There is also a great need of research on treatments that specifically target SCT symptoms rather than just assuming that those therapies for ADHD can be applied equally as well to SCT. Given the distinctive nature of SCT from ADHD symptom dimensions evident above as well as it's emerging differences in domains of impairment, treatments need to be designed expressly for the symptoms and impairments linked to SCT. A commendable step in this direction was the design and evaluation by Pfiffner and colleagues of a joint home and school package of behavioral strategies aimed specifically at the symptoms and academic problems likely to exist in children having high SCT symptoms (within the context of ADHD Inattentive Type)(Pfiffner et al. 2007). The results showed considerable success and offer an exemplar for the study of other therapies for SCT.

Likewise, one cannot just assume that medications that have proven effective for ADHD will prove so for SCT without directly evaluating those medications with cases specifically selected for having SCT. Stimulant medications, such as methylphenidate, have not proven to be quite as effective for the Inattentive Type of ADHD (Milich et al. 2001), in which many SCT cases exist implying that these medications may not be so useful for managing SCT. But this is not direct evidence for or against the efficacy of such medications for SCT specifically as such cases of SCT were not studied in these papers. I am aware of just one study to date that has evaluated an ADHD medication for treating SCT symptoms specifically (Wietecha et al. 2011). It found that the norepinephrine reuptake inhibitor *atomoxetine* was effective at reducing SCT symptoms even after statistically controlling for the overlap of those symptoms with those of ADHD. Far more research needs to be done in this area not only evaluating ADHD medications like the stimulants, atomoxetine, or guanfacine XR but also other medications that may have some effectiveness for attention problems, such as the anti-narcoleptic modafinil, or anti-anxiety and anti-depressive medications, given the significant relationship of anxiety and depression to SCT.

Given that SCT is partially distinct from ADHD, one also cannot just assume that treatments that proved ineffective for ADHD, such as cognitive behavioral therapy (CBT; Abikoff 1985) or social skills training (SST; Antshel and Remer 2003), would be ineffective for SCT. Especially since SCT does not appear to be a disorder of EF, as discussed above, then EF deficits would not be expected to interfere with the efficacy of such treatments as they might do in children with ADHD. In view of the efficacy of both of these forms of intervention with internalizing disorders and the significant linkage of SCT to those disorders, one might reasonably expect CBT or SST to be worth exploring for the management of the cognitive and social problems occurring in SCT.

A Plea to Change the Name – Concentration Deficit Disorder?

Because we do not yet know the answer to the question of the underlying cognitive dysfunction in SCT, *the label sluggish cognitive tempo seems premature, likely misleading, and certainly derogatory.* My colleagues and I (Saxbe and Barkley 2013) are finding the term to prove offensive to those it so characterizes and their families when the condition is explained to them. And clinical professionals to whom I have lectured on this topic also note the pejorative nature of the term, smacking as it does of connotations of mental slowness, slow learning or wittedness, or frank mental retardation. So can we please change the name of SCT to something less offensive or misleading? Although Primary Disorder of Vigilance (PDV or PVD) may be less offensive, even that term conveys an understanding of the cognitive nature of SCT that does not currently exist. It would seem to be as premature as is SCT. The term ADD (attention deficit disorder) was also suggested by Diamond (2005) and Adams et al. (2010) but its problem is that it is the historically antecedent term for ADHD and thus creates unnecessary confusion in terminology. It also implies that SCT shares the same attention disorder as ADHD or is a subtype of ADHD, which growing research suggests is not the case. *Concentration Deficit Disorder (CDD)* seems to me to be the best option at this time for various reasons: (a) it keeps the focus of the label on an

attention problem yet makes it distinct from ADHD; (b) it is not offensive or pejorative to patients and family members as is SCT; and (c) it does not imply we know more than we do about the underlying cognitive dysfunction, as do the terms SCT and PDV. For now, to facilitate literature searches, authors could refer to SCT by both terms, as in Concentration Deficit Disorder (aka sluggish cognitive tempo), in their research papers while using the term CDD in discussions with patients and their families and other caregivers (teachers, partners). Making the shift in terminology will prove far easier now than it will be a decade from now after much more research is published on SCT/CDD as is clearly likely to be the case after this special issue.

Conclusion

In closing, let me reiterate the point made above that there are clearly rich veins of research topics to be mined in the study of SCT sufficient to make entire careers for clinical scientists looking for an area of research specialization. The early findings in this growing field of study are quite encouraging of SCT being a distinct disorder from yet at times overlapping with ADHD. It is hoped that the papers published in this special issue along with the small but growing body of other research on SCT will serve to strongly encourage just such future research and associated careers into the nature, cognition, comorbidities, correlates, etiologies, and life course risks for SCT as well as treatments for this condition.

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