

## CHAPTER 15

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# Sluggish cognitive tempo

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### Introduction

There has been longstanding interest in how best to categorize the heterogeneity of children with ADHD (1, 2). In an effort to understand the variability of inattention specifically, there has recently been a resurgence of interest in sluggish cognitive tempo (SCT) (3–6), a set of behavioural symptoms characterized by daydreaming, mental foggy/ness/confusion, and slowed behaviour/thinking. This chapter provides a brief history of the SCT construct and summarizes the rapidly accumulating research examining SCT as distinct from ADHD and other psychopathologies and the extent to which SCT is uniquely related to functioning in various domains of major life activities. We conclude by commenting on issues of terminology and offering key directions for future research.

### History of the sluggish cognitive tempo construct

The first description of attentional problems characterized by underarousal and daydreaming is believed to have appeared in a textbook by Sir Alexander Crichton in 1798 (7). As Barkley (5) observed, Crichton's description of individuals with 'low power', who are generally unsocial and insensible to external objects, may align with current descriptions of SCT. Nevertheless, the scientific study of SCT began in the 1980s when the third edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM) (8) created two subtypes of attention deficit disorder (ADD): one with hyperactivity (ADD+H) and one without hyperactivity (ADD–H). In turn, researchers began to investigate differences between children with ADD+H or ADD–H. A thorough review of this research is beyond the scope of this chapter (see Barkley [5] and Becker et al. [4] for more thorough reviews). It was during this time that researchers first began to evaluate the extent to which a set of symptoms characterized by sluggishness, apathy, lethargy, and being in a world of one's own were part of, or distinct from, inattention as defined in the DSM (9–11). However, when SCT symptoms showed poor predictive power for identifying ADHD in the DSM-IV field trials (12), the study of SCT largely stalled. That is until an influential review by Milich et al. (2) appeared as well as an empirical article by McBurnett and colleagues (13), both published in 2001. These papers emphasized the importance of further study of the SCT construct. In turn, a slow but steady increase in the number of SCT studies began to enter the published literature (4). Specifically, since these two seminal papers were published in 2001, over 50 papers related to SCT have been published, most in just the past 5 years (14). As the findings from these studies have

accumulated (and are reviewed next), the study of SCT has largely shifted away from efforts to identify differences between subtypes of ADHD to examine SCT in its own right, independent from and in contrast to ADHD.

### Sluggish cognitive tempo is distinct from ADHD

It is now clear that SCT symptoms are empirically distinct from symptoms of ADHD. Becker et al. (14) recently conducted a meta-analysis that included factor analytical studies with over 19,000 children and adults, and found strong support for 13 SCT symptoms that loaded consistently on an SCT factor as opposed to an ADHD factor. These 13 symptoms are listed in Box 15.1. Although additional measurement work needs to be conducted now that these 13 items have been identified, it is important to note that parent, teacher, adult self-report, and child self-report ratings scales (15–20), as well as a semistructured clinical interview (21), have been developed that include at least most of these 13 items. Studies using these measures demonstrate that SCT can be reliability assessed, with excellent internal consistency and test–retest reliability, and moderate interrater reliability (14). Importantly, the internal validity of SCT has been identified across a range of sample types (clinical, community, epidemiological), age ranges (spanning ages 3–96 years), and continents (North America, South America, Europe, and Asia) (14).

Additional evidence for the distinctiveness of SCT from ADHD comes from Barkley's two nationally representative studies examining SCT in children (16) and adults (15). As in other studies, Barkley found evidence for an SCT factor that did not load with ADHD. Further, Barkley classified participants as meeting research criteria for SCT and/or ADHD and found only moderate overlap between the SCT and ADHD classifications. In both studies, approximately half of the participants with ADHD were not classified with SCT, and vice versa. These findings led Barkley (15) to conclude that the relation between SCT and ADHD 'is similar to comorbidity between two disorders, such as between anxiety and depression, than one of subtyping within a single disorder or in which subgroups share the same disorder of attention' (p. 987). In line with this conclusion, two recent studies using bifactor modelling found SCT to fall outside of a general ADHD or disruptive behaviour factor (22, 23). SCT and ADHD also appear to have different developmental trajectories, with ADHD hyperactive-impulsive symptoms declining, ADHD inattentive symptoms remaining relatively stable, and SCT symptoms showing a slight increase across childhood and adolescence (24).

**Box 15.1** Sluggish cognitive tempo items

1. Apathetic/unmotivated
2. Daydreams
3. Easily confused
4. In a fog
5. Loses train of thought/cognitive set
6. Lost in thoughts
7. Sluggish
8. Sleepy/drowsy
9. Slow thinking/processing
10. Spacey
11. Stares blankly
12. Tired/lethargic
13. Underactive/slow moving

*Note:* These 13 items were identified in a meta-analysis as having a mean factor loading  $>.70$  on a sluggish cognitive tempo factor; see Becker et al. (14). Data from Becker SP, Leopold DR, Burns GL, Jarrett MA, Langberg JM, Marshall SA, et al. The internal, external, and diagnostic validity of sluggish cognitive tempo: A meta-analysis and critical review. *J Am Acad Child Adolesc Psychiatry*. 2015. Advance online publication. DOI: <http://dx.doi.org/10.1016/j.jaac.2015.12.006>

Little is known regarding the aetiology of SCT, but again, there is emerging evidence for the separateness of SCT from ADHD. The only published twin study to date found SCT to be substantially heritable, although SCT symptoms were less heritable than ADHD and more strongly influenced by shared and non-shared environmental factors (25). The one neuroimaging study of SCT found an association between SCT symptom severity and hypoactivity in the left superior parietal lobe during a cued flanker task, suggestive of deficits in reorientating or shifting of attention (26) that is different from the attention networks implicated in ADHD (27). Although these findings are intriguing, much more research examining the aetiology of SCT is clearly needed. Nevertheless, essentially all available research points to SCT as a set of attentional symptoms distinct from those captured by ADHD.

## Sluggish cognitive tempo in relation to functioning and impairment

In comparison to the number of studies examining the structure of SCT, especially as related to ADHD, far fewer studies have examined SCT in relation to functioning and impairment. Nevertheless, a growing body of research indicates that SCT is associated with poorer adjustment across a range of domains of major life activities, with many associations remaining even after controlling for ADHD (for additional reviews, see [5, 14]).

### Mental health functioning

Multiple studies have documented a significant association between SCT and internalizing symptoms such as anxiety and depression (20, 28–30), although it should be noted that SCT is distinct from both anxiety and depression (18, 31, 32). In considering specific internalizing comorbidities, SCT seems to be more strongly related to depression than to anxiety (16, 31, 33, 34). However, this may be more true in children than in adults (14) and could therefore simply reflect the increased rates of depression as children transition to adolescence and adulthood. In any event, SCT

is clearly more strongly related to internalizing problems than with externalizing problems such as hyperactivity-impulsivity and oppositionality (14). In fact, although SCT and ADHD inattention are themselves strongly correlated, these two attention domains evince differential associations with hyperactive-impulsive and externalizing symptoms when controlling for each other. When controlling for ADHD inattention, SCT is unassociated with, or is *negatively* associated with, externalizing behaviours, whereas ADHD inattention remains strongly positively associated with externalizing behaviours (18–20, 31, 32). Crucially, recent longitudinal studies have found the same pattern whereby SCT predicted greater internalizing behaviours and fewer hyperactive-impulsive and oppositional behaviours up to 2 years later (35–37). Further, children with ADHD who also have high levels of SCT show lower rates of externalizing behaviours such as aggression than other children with ADHD (38, 39).

These findings provide additional compelling evidence for the separation of SCT and ADHD. It would be even more so if additional research supports the hypothesis that SCT falls under the internalizing umbrella of psychopathology whereas ADHD falls under the externalizing domain (40). As Barkley has noted (41), it remains important to identify functioning domains on which SCT may have a negative impact, as well as domains that are unlikely to be impacted by SCT or for which SCT may actually play a buffering role. Future studies should therefore continue to examine SCT in relation to internalizing-salient domains while also testing the hypothesis that SCT would be unrelated to externalizing-salient domains characterized by impulsivity and risk-taking, including substance use, delinquency and antisocial behaviours, speedy and reckless driving, and accidental injuries.

### Academic functioning

Somewhat discrepant findings have been reported in studies examining SCT in relation to academic functioning. Several studies did not find the presence of SCT to predict academic impairment or negatively impact academic achievement after accounting for ADHD (29, 38, 39, 42, 43). This may be due to these studies examining the relation between SCT and academics in samples of children diagnosed with ADHD, which may confound findings specific to SCT. It could also result from using very short measures of SCT comprised of just a few items. In line with this possibility, the most recent studies that have used longer, validated measures of SCT have repeatedly found SCT symptoms to be uniquely associated with poorer academic functioning, even in ADHD samples (30, 33, 44) or after controlling for ADHD symptom severity (17, 18, 32, 34, 45–47). In particular, it appears that SCT is associated with problems in organization and homework completion (16, 39, 44). In terms of academic achievement, SCT may be uniquely associated with lower achievement in mathematics, word reading, and written language (28, 32). Nevertheless, there is some indication even from these recent studies that ADHD is a stronger contributor than SCT to academic problems (16, 18, 19, 48), although more research needs to be done using real world measures of academic functioning and success. Of note, the three studies that have examined overall grade point average (GPA) each found SCT to significantly predict lower GPA after accounting for ADHD symptoms (30, 32, 44). Indeed, two of these studies found ADHD symptoms to no longer remain associated with GPA when SCT was added to the model (30, 44). In sum, additional research is needed to determine the precise relation between SCT and

academic functioning, as well as the mechanisms and developmental progression of this association.

### Socioemotional functioning

Studies have repeatedly found a link between SCT and problems/impairment in social functioning (17, 18, 29, 31, 45, 47). One recent study found SCT symptoms to predict increased peer problems across the duration of a school year even after controlling for other psychopathology symptoms and initial levels of peer functioning (49). In terms of specific domains of social functioning, both Bauermeister et al. (28) and McBurnett et al. (19) found SCT to predict poorer social skills in children even after controlling for ADHD severity, though only when teacher ratings were used and not parent ratings. This is perhaps because teachers have ample opportunity to observe children's interactions with peers and the specific types of peer difficulties related to SCT. In a novel computer chat room experiment, SCT symptoms were found to be associated with a poorer perception of subtle social cues and less memory for the chat room conversation (50). Further, whereas children with ADHD are often disliked or actively rejected by peers owing to noxious and annoying behaviours (51), children with SCT are likely to be socially withdrawn and isolated (32, 38, 39, 52). Whether or not this is primarily due to shyness or social disinterest has not yet been specifically examined. It seems likely due to shyness as opposed to lack of interest, since SCT is also associated with parent reports of children's fear/shyness as part of the fight-flight-freeze motivational system (40) and children's own ratings of loneliness (17). Additional studies supporting this conclusion would have clear implications for intervention (e.g. social skills training).

Since SCT is associated with both social problems and depressive symptoms, it is not surprising that it has also been linked to problems in emotion regulation, although findings are not fully consistent across studies. In his nationally representative sample of youth, Barkley (16) found SCT to be significantly associated with children's self-regulation of emotion deficits, but SCT was a much smaller contributor than either ADHD inattention or hyperactivity-impulsivity to these deficits. Conversely, SCT was the strongest predictor of self-regulation of emotion deficits in Barkley's representative sample of adults (15). Does this mean the association between SCT and emotion regulation difficulties grows stronger across development? This is unknown, and no longitudinal studies examining the association between SCT and emotion regulation exist. However, there is some evidence that SCT is in fact associated with poorer emotion regulation in youth. Specifically, Becker et al. (17) found both child- and teacher-rated SCT symptoms to predict poorer child-rated emotion regulation coping (ability to cope with and control emotional experiences appropriately) and lower self-esteem even after controlling for ADHD, anxiety, and depressive symptoms. Likewise, Araujo Jiménez et al. (53) found SCT to be significantly associated with poorer parent-rated emotional control in a sample of children and adolescents with ADHD, even after controlling for both ADHD inattention and hyperactivity-impulsivity. These findings converge with other studies of college students that consistently document a link between SCT and emotion dysregulation (54–56). Interestingly, Willcutt et al. (32) hypothesized that regulation difficulties may account for the association between SCT and social isolation, whereby 'individuals with SCT may become overwhelmed by the rapid flow of complex information that must be processed continuously to successfully navigate social interactions, which may then lead to avoidance of

social situations and subsequent isolation' (pp. 32–3). In line with this possibility, Flannery et al. (54) directly tested and found support for emotion dysregulation mediating the association between SCT and social problems, albeit with a cross-sectional design making longitudinal studies that are better suited for testing indirect and developmental pathways a clear research priority. Also important to distinguish in future research is whether such dysregulation actually reflects greater emotional distress, more emotional lability, or poor self-regulation of elicited emotions.

### Neuropsychological and executive functioning

Only a handful of studies have examined neuropsychological deficits associated with SCT, and most of these studies have used samples (and, thus, neuropsychological test batteries) specific to, or oversampled for, ADHD (28, 32, 43, 57, 58). In general, these studies demonstrate that SCT is unassociated with pervasive neuropsychological deficits, whereas a large literature links ADHD to wideranging neuropsychological impairments (59, 60). In particular, ADHD is clearly associated with deficits in response inhibition, working memory, and response variability. In contrast, SCT does not seem to be associated with any of these deficits when accounting for the contribution of ADHD symptoms (28, 32, 43). However, there is some indication that SCT may be uniquely related to problems with early information processing or selective attention (58). Furthermore, two of the larger studies examining SCT in relation to neuropsychological test performance both found SCT to be related to poorer sustained attention after controlling for ADHD inattention (32, 43). There is mixed evidence in terms of whether or not SCT remains associated with slower motor or processing speed after controlling for ADHD (28, 32, 61). One study found SCT to be associated with variability of spatial memory performance specifically (57). Replication will be needed to draw conclusions regarding any of these findings. It will be especially important for future studies examining SCT in relation to neuropsychological functioning to do so in non-ADHD samples, as well as to use neuropsychological and cognitive tests more specific to the nature of SCT that may not be frequently used in studies of ADHD. In addition, only one study has examined SCT in relation to neuropsychological test performance in adults. This study found neither SCT nor ADHD to predict test performance, which is likely due to this study's use of a college student sample (55). In sum, although more studies are needed, it seems clear that SCT is not associated with substantial or pervasive executive functioning (EF) deficits. This is yet one more indication that SCT is best conceptualized as distinct from ADHD.

When ratings of EF in daily life are used, as opposed to neuropsychological tests of EF, there appear to be different conclusions between studies of youth or adults. This can be seen most clearly in Barkley's national studies (15, 16), which did not suffer from possible ascertainment bias by using previously ADHD defined samples. In the study of children and adolescents, SCT accounted for very little variance across all EF dimensions when controlling for ADHD symptoms in youth, though SCT did contribute somewhat to deficits in self-organization and problem-solving. ADHD inattention was far and away the strongest predictor of EF deficits across all EF dimensions (16). In contrast, in the national study of adults, ADHD inattention remained the strongest predictor of self-management to time and self-motivation EF dimensions, but SCT accounted for the most variance in the self-organization



and problem-solving, self-restraint, and self-regulation of emotion dimensions (15). In line with these studies, Araujo Jiménez (53) found ADHD symptoms to be a stronger predictor than SCT symptoms of most EF dimensions in a sample of youth aged 6–17 years, though SCT did contribute meaningful variance in predicting emotional control, working memory, and planning/organization. Similarly, Becker and Langberg (62) found SCT to be significantly associated with metacognitive EF deficits in a sample of young adolescents diagnosed with ADHD. However, this was only true when parent ratings of SCT were used and not teacher ratings. As hypothesized, SCT was not significantly associated with behavioural regulation EF deficits that are more strongly linked to poor inhibition (62). In line with findings from Barkley's nationally representative study of adults (15), two studies conducted with college students suggest SCT to be more clearly related to EF ratings in adults, with the strongest associations apparent for the organization and problem-solving, self-motivation, and self-regulation of emotion dimensions, with significant, albeit weaker, associations also found for the time management and self-restraint dimensions (55, 56). Once again, longitudinal and developmentally informed studies are needed to evaluate the developmental pathways and mechanisms of the linkage between SCT and daily life EF deficits.

### Functional impairment

In addition to studies examining SCT in relation to aspects of adjustment, several studies have examined SCT in relation to global and specific domains of functional impairment. SCT is related to global impairment (14), though likely to a lesser degree and less pervasively so than ADHD (15, 16). Nevertheless, it is clear that SCT is associated with functional impairment not explained by any overlap with ADHD (15, 16, 19, 32, 56). It is thus not surprising that SCT is also linked to increased stress (63) and a poorer quality of life (64), as well as lower educational attainment and socioeconomic status (14–16). As such, SCT is clearly not benign and so is deserving of both research inquiry and clinical attention.

### Key directions for future research

Throughout this chapter we have pointed out key directions for future research, but it is important to mention a few additional areas (see also 3, 14). First, there is a clear need for additional longitudinal research. Such studies would allow for a better understanding of the developmental course of SCT, longitudinal correlates, and pathways by which SCT is associated with developmental outcomes and functional impairments. Second, and relatedly, the vast majority of extant SCT studies have focused on school-aged children and college students, making research in other developmental periods, as well as in other samples of young adults, a necessity. Third, very little is known regarding the aetiology of SCT, including either biological and/or environmental contributions to the SCT phenotype. In particular, social adversities may be more relevant for the aetiology of SCT than for ADHD (3, 14). Pathological mind wandering is another intriguing possibility for the aetiology of SCT (3, 65). As we have discussed elsewhere (5, 6, 17), it is also possible that different attentional networks are implicated in SCT versus ADHD. Moreover, SCT is only modestly associated with nighttime sleep problems (66–68), but is moderately related to both sleep quality and daytime sleepiness (66, 69). It would be fruitful for studies to evaluate whether similar mechanisms contribute to

SCT and hypersomnia, including use of methodologies such as the multiple sleep latency test and default mode network connectivity.

Finally, it is important to examine whether the presence of SCT symptoms predicts or moderates treatment response to currently established evidence-based interventions for ADHD as well as other psychiatric disorders (e.g. anxiety, mood, sleep disorders). It is likewise important to evaluate interventions that might be efficacious for treating SCT, including both pharmacological and psychosocial interventions. Initial evidence indicates that atomoxetine may effectively reduce SCT symptoms (70), and selective serotonin reuptake inhibitors may also be effective given the strong association between SCT and internalizing problems such as anxiety and depression (5). In terms of psychosocial interventions, Pfliffer and colleagues (71) found evidence-based parent- and teacher-mediated behavioural interventions to reduce SCT symptoms in children with ADHD predominantly inattentive type. Given SCT's associations with internalizing problems (especially depression) and social withdrawal, we have also hypothesized that cognitive-behavioural treatment (CBT), social skills training, and behavioural activation may be effective for treating SCT (3, 5, 14, 72). However, each of these possibilities has yet to be examined empirically. Of course, it will be important to do so in samples that are not restricted to individuals diagnosed with ADHD but instead use a broader sampling strategy (e.g. including individuals with anxiety, depression, and/or sleep disorders) or are selected based on SCT specifically.

### A comment on terminology

Before concluding, we believe one additional point regarding terminology is warranted. In coauthoring this chapter, we hope it is clear that we (SPB and RAB) agree on the vast majority of conclusions that can be drawn regarding the current state of SCT, as well as the very clear need for additional research that can ultimately shed light on the aetiology, course, and clinical implications of SCT. Nevertheless, we also acknowledge one area of professional disagreement; namely, the best term to be used to describe this constellation of symptoms.

Barkley has advocated moving away from the SCT terminology and suggests the term concentration deficit disorder (CDD) (3, 5, 41, 73) for various reasons, 'not the least of which is that SCT can be viewed by the public as pejorative, derogatory, or frankly offensive' (5) (p. 435). It is important to note that Becker agrees that the SCT term is far from optimal and 'shares with Barkley the overarching concerns he has for the SCT label' (17) (p. 1038). In fact, this is why Becker avoided the SCT term when naming his youth self-report scale, calling it the Child Concentration Inventory (17). However, Becker has also expressed concerns that it may be 'premature to use terminology suggesting that the SCT construct is a diagnostic entity (e.g. Concentration Deficit Disorder)' (14). Just as the SCT label may be perceived as pejorative and offensive, premature use of the term 'disorder' may create confusion for families and professionals while also drawing unfavourable media attention that detracts from the important value of SCT research (74, 75). Further, terminology suggestive of a 'disorder' casts SCT in a dichotomous light, and Becker believes it is important to consider other options, especially the possibility that SCT is best viewed as a transdiagnostic construct as opposed to a categorical diagnosis (4, 76). This latter possibility aligns with the broader shift in psychiatry as outlined

by the United States National Institute of Mental Health research domain criteria (RDoC) initiative (77, 78). Thus, Becker would currently favour a term that focuses more on ‘symptoms’ as opposed to ‘diagnosis’ and is actively considering possible alternatives as research continues to emerge, even though he has so far continued to use the SCT terminology.

However, as Barkley has appropriately noted, ‘the mere fact that SCT has existed for 35 years and so should continue to be used out of mere historical tradition is not a convincing reason to retain a term that research participants and other consumers likely will find offensive’ (41). Becker agrees fully with this point, but for him the issue is one of timing: a new term is needed but much more remains to be learned before we can settle on an ideal term. There is no easy solution to this dilemma, as the introduction of a new term earlier may likely spur the very research needed to determine an even better term. Barkley has clearly acknowledged that he is not wedded to his proposed CDD terminology (41). He suggests here that perhaps the term ‘syndrome’ might be a better modifier of ‘concentration deficit’ so as not to convey official sanctioning of it as a recognized clinical disorder. Where does this leave us? We both agree that we need a new term that: 1) accurately describes the constellation of symptoms and state of the science; and 2) is not off-putting to either the scientific community or general population. We hope we can find consensus around such a term in the near future. In the meantime, we are certainly in agreement that there is a crucial need for much more research on SCT/CDD, and we hope our colleagues will join us in pursuing this theoretically and clinically important area of study.

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# **Attention Deficit Hyperactivity Disorder**

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# Foreword

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# Contents

Abbreviations *xv*

Contributors *xi*

## SECTION 1

### Introduction

- 1 Development of the concept** 3  
Eric Taylor
- 2 ADHD in the twenty-first century: biology, context, policy, and the need for integrative perspective** 9  
Stephen P. Hinshaw and Richard M. Scheffler

## SECTION 2

### Aetiology and pathophysiology

- 3 ADHD genetics** 19  
Kate Langley
- 4 Conceptualizing and investigating the role of the environment in ADHD: correlate, cause, consequence, context, and treatment** 25  
Edmund Sonuga-Barke and Gordon Harold
- 5 Gene–environment interactions** 35  
Barbara Franke and Jan K. Buitelaar
- 6 Brain volumes and intrinsic brain connectivity in ADHD** 57  
Kerstin Konrad, Adriana Di Martino, and Yuta Aoki
- 7 ADHD brain function** 64  
Katya Rubia
- 8 Insights from neuroanatomical imaging into ADHD throughout the lifespan** 73  
Philip Shaw and Eszter Szekely
- 9 Neurophysiology** 82  
Daniel Brandeis, Sandra K. Loo, Grainne McLoughlin, Hartmut Heinrich, and Tobias Banaschewski

- 10 Cognitive functioning in ADHD: inhibition, memory, temporal discounting, decision-making, timing, and reaction time variability** 94  
David Coghill, Maggie Toplak, Sinead Rhodes, and Nicoletta Adamo

- 11 Emotional dysregulation and ADHD** 103  
Celine Ryckaert, Jonna Kuntsi, and Philip Asherson

- 12 Neuropsychological functioning and ADHD: a developmental perspective** 118  
Sarah O'Neill, Jeffrey M. Halperin, and David Coghill

## SECTION 3

### Epidemiology

- 13 Epidemiology** 131  
Guilherme V. Polanczyk

## SECTION 4

### Clinical presentation

- 14 Current diagnostic criteria: DSM, ICD, and future perspectives** 139  
Luis Augusto Rohde, Christian Kieling, and Giovanni Abrahão Salum
- 15 Sluggish cognitive tempo** 147  
Stephen P. Becker and Russell A. Barkley
- 16 Sex differences in ADHD** 154  
Corina U. Greven, Jennifer S. Richards, and Jan K. Buitelaar
- 17 Quality of life and impairment in ADHD** 161  
Melissa Mulraney and David Coghill
- 18 Adult ADHD and employment** 170  
Marios Adamou
- 19 Adult ADHD: clinical presentation and assessment** 178  
Philip Asherson, Josep Antoni Ramos-Quiroga, and Susan Young

## SECTION 5

**Comorbidity**

- 20 Conduct disorder in ADHD** 193  
Anita Thapar and Stephanie van Goozen
- 21 Irritability, disruptive mood, and ADHD** 200  
Melissa Mulraney, Argyris Stringaris, and Eric Taylor
- 22 Comorbidity: depression and anxiety** 206  
Cristal Oxley and Argyris Stringaris
- 23 ADHD and substance misuse** 215  
Timothy Wilens, Nicholas Carrellas, and Joseph Biederman
- 24 Autism spectrum disorder** 227  
Sven Bölte, Luise Poustka, and Hilde Geurts
- 25 Intellectual impairment and neurogenetic disorders** 235  
Emily Simonoff
- 26 Influence of tics and/or obsessive-compulsive behaviour on the phenomenology of coexisting ADHD** 247  
Aribert Rothenberger, Andreas Becker, Lillian-Geza Rothenberger, and Veit Roessner
- 27 Developmental coordination disorder** 254  
Christopher Gillberg, Elisabeth Fernell, I. Carina Gillberg, and Björn Kadesjö
- 28 ADHD and communication disorders** 261  
Rosemary Tannock
- 29 ADHD and reading disorder** 273  
Erik G. Willcutt
- 30 ADHD and sleep** 280  
Melissa Mulraney, Emma Sciberras, and Michel Lecendreux
- 31 The relationship of ADHD to obesity and asthma** 289  
Samuele Cortese and Marcel Romanos

## SECTION 6

**Clinical assessment** 295

- 32 Children and adolescents: assessment in everyday clinical practice** 297  
Marina Danckaerts and David Coghill
- 33 ADHD in adults; assessment issues** 307  
Sandra Kooij, Philip Asherson, and Michael Rösler

## SECTION 7

**Interventions**

- 34 Long-term outcomes in the Multimodal Treatment study of Children with ADHD** 315  
James M. Swanson, L. Eugene Arnold, Peter S. Jensen, Stephen P. Hinshaw, Lily T. Hechtman, William E. Pelham, Laurence L. Greenhill, C. Keith Conners, Helena C. Kraemer, Timothy Wigal, Benedetto Vitiello, Glen R. Elliott, Howard B. Abikoff, Betsy Hoza, Jeffrey H. Newcorn, Karen Wells, Marc Lerner, Brooke S. G. Molina, Jeffery N. Epstein, Elizabeth B. Owens, James Waxmonsky, Desiree W. Murray, Margaret H. Sibley, John T. Mitchell, Arunima Roy, Annamaire Stehli for the MTA Cooperative Group
- 35 Behavioural interventions for preschool ADHD** 333  
David Daley and Saskia Van der Oord
- 36 Cognitive-behavioural treatment in childhood and adolescence** 340  
Manfred Döpfner and Saskia van der Oord
- 37 Behavioural therapy (adolescent/adult)** 348  
Alexandra Philomena Lam and Alexandra Philipsen
- 38 Cognitive training approaches for ADHD: can they be made more effective?** 358  
Edmund Sonuga-Barke and Samuele Cortese
- 39 Neurofeedback** 366  
Martin Holtmann, Björn Albrecht, and Daniel Brandeis
- 40 Nutritional intervention for ADHD** 373  
Jan K. Buitelaar, Nanda Rommelse, Verena Ly, and Julia J. Rucklidge
- 41 ADHD treatment: psychostimulants** 379  
Alessandro Zuddas, Tobias Banaschewski, David Coghill, and Mark A. Stein
- 42 Non-stimulants in the treatment of ADHD** 393  
Ralf W. Dittmann, Alexander Häge, Juan D. Pedraza, and Jeffrey H. Newcorn
- 43 ADHD and transitions to adult mental health services** 402  
Chris Hollis
- 44 ADHD and school** 408  
Christine Merrell and Kapil Sayal

**SECTION 8****Clinical management****45 Organizing and delivering treatment for ADHD** 417

David Coghill and Marina Danckaerts

**46 Treatment in adult ADHD** 426

Philip Asherson and Josep Antoni Ramos-Quiroga

**47 The next steps: future clinical and research developments in ADHD** 437

David Coghill, Alessandro Zuddas, Luis Augusto Rohde, and Tobias Banaschewski

**Index** 445





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# Abbreviations

ACC	anterior cingulate cortex	DAMP	deficits in attention, motor control + perception
ACG	anterior cingulate gyrus	DAWBA	development and wellbeing assessment
ADD	attention deficit disorder	DCD	developmental coordination disorder
ADHD-RS	ADHD rating scale	DESR	deficient emotional self-regulation
AERS	Adverse Event Reporting System	DIVA 2.0	Diagnostic Interview for ADHD for adults
AHI	apnea–hypopnea index	DLMO	dim light melatonin onset
AIM	ADHD plus impairments in mood	DMDD	disruptive mood dysregulation disorder
AMHS	adult mental health services	DMN	default mode network
APA	American Psychiatric Association	DN	default network
ARI	affective reactivity index	DPFC	dorsolateral prefrontal cortex
ASD	autism spectrum disorder	DSM	<i>Diagnostic and Statistical Manual of Mental Disorders</i>
ATX	atomoxetine	DTI	diffusion tensor imaging
BADDS	Brown Attention Deficit Disorder Scale	DZ	dizygotic
BDNF	brain-derived neurotropic factor	EAGG	European ADHD Guidelines Group
BMI	body mass index	EC	effortful control
BOLD	blood oxygen level-dependent	EDS	excessive daytime sleepiness
BPD	borderline personality disorder	EEG	electroencephalogram/electroencephalography
BPT	behavioural parent training	EF	executive functioning
CAADID	Conners Adult ADHD Diagnostic Interview	EIS	emotional impulsiveness scale
CAARS	Conners Adult ADHD Rating Scale	EMA	European Medicines Agency
CAI	computer-assisted instruction	EMG	electromyogram/electromyography
CAMHS	child and adolescent mental health services	EMR	electronic medical record
CAPA	Child and Adolescent Psychiatric Assessment	EPSC	excitatory postsynaptic current
CBCL	childhood behaviour checklist	ERA	English and Romanian Adoptees study
CBT	cognitive–behavioural therapy/treatment	ERN	error-related negativity
CD	conduct disorder	ERP	event-related potential
CDD	concentration deficit disorder	ES	effect size
CDI	child-directed intervention	ESM	experience sampling method
CELF	Clinical Evaluation of Language Fundamentals	ESSENCE	early symptomatic syndromes eliciting neurodevelopmental clinical examinations
CEN	central executive network	FAS	fetal alcohol syndrome
CGAS	Child Global Assessment Scale	FDA	Food and Drug Administration
CGH	comparative genome hybridization	fMRI	functional magnetic resonance imaging
CGI	Clinical Global Impressions	FR	familial risk index
CHMP	Committee for Medicinal Products for Human Use	GABA	gamma-aminobutyric acid
CHP	Challenging Horizons Programme	GCTA	genome-wide complex trait analysis
CI	confidence interval	GREML	genomic-relationship-matrix restricted maximum likelihood
CLAS	Child Life and Attention Skills	GWAS	genome-wide association studies
CNV	contingent negative variation	GXR	guanfacine extended-release
CNV	copy number variant	HKD	hyperkinetic disorder
CoT	children-of-twins	HRQoL	health-related quality of life
CSD	current source density	ICA	independent component analysis
CWPT	classwide peer tutoring	ICD	<i>International Classification of Diseases</i>
CXR	clonidine extended-release		
DA	dopamine		

ID	intellectual disability	PLMD	periodic limb movement disorder
IFC	inferior prefrontal cortex	PSG	polysomnography
IFG	inferior frontal gyrus	PUFA	polyunsaturated fatty acids
IPC	inferior parietal cortex	QoL	quality of life
iPSC	induced pluripotent stem cells	RAI	resource allocation index
ITT	intention to treat	RCT	randomized controlled trial
K-SADS	Kiddie-Schedule of Affective Disorders	RD	reading disorder
LDX	lisdexamphetamine	RDoC	research domain criteria
LI	language impairment	REM	rapid eye movement
LMIC	lower- and middle-income countries	rGE	gene–environment interaction
LNCG	local normative comparison group	RLS	restless leg syndrome
MAP	mindful awareness practice	ROI	region of interest
MAS	mixed amphetamine salts	RR	relative risk ratio
MBCT	mindfulness-based cognitive therapy	RTV	response time variability
MBD	minimal brain dysfunction	SCID-5	Structured Clinical Interview for DSM-5
MDD	major depressive disorder	SCN	suprachiasmatic nucleus
MEG	magnetoencephalography	SCP	shared care protocol
MEP	motor-evoked potential	SCP	slow cortical potential
MET	motivational enhancement therapy	SCT	sluggish cognitive tempo
MI	motivational interviewing	SD	standard deviation
MMN	mismatch negativity	SDB	sleep-disordered breathing
MND	minor neurological dysfunction	SDQ	Strengths and Difficulties Questionnaire
MPFC	medial prefrontal cortex	SES	socioeconomic status
MPH	methylphenidate	SFC	superior frontal cortex
MRI	magnetic resonance imaging	SFT	solution-focused treatment
MRR	mortality rate ratio	SICI	short-interval intracortical inhibition
MSLT	multiple sleep latency test	SMA	supplementary motor area
MTA	Multimodal Treatment study of children with ADHD	SMD	severe mood dysregulation
MZ	monozygotic	SMD	standardized mean difference
NCLB	No Child Left Behind	SMR	sensorimotor rhythm
NE	negative emotionality	SNAP	Swanson, Nolan, and Pelham rating scale
NE	norepinephrine	SNP	single nucleotide polymorphism
NIDA	National Institute of Drug Abuse	SNRI	serotonin-norepinephrine reuptake inhibitors
NIMH	National Institute of Mental Health	SPCD	social pragmatic communication disorder
NIRS	near-infrared spectroscopy	SPECT	single-photon emission computed tomography
NMDA	N-methyl-D-aspartate	SSD	speech sound disorder
NREM	non-rapid eye movement	SSRI	selective serotonin reuptake inhibitor
NSCH	National Survey for Children's Health	SSRT	stop signal reaction time
OCB	obsessive-compulsive behaviour	SST	social skills training
OCD	obsessive-compulsive disorder	SUD	substance use disorder
ODD	oppositional defiant disorder	SWA	slow-wave activity
OEST	other early standard therapy	tACS	transcranial alternating current stimulation
OFC	orbitofrontal cortex	TAU	treatment as usual
OR	odds ratio	TBR	theta to beta power ratio
OSA	obstructive sleep apnea	TCA	tricyclic antidepressant
PAS-ADD	Psychiatric Assessment Schedule for Adults with Developmental Disabilities	TD	tic disorders
PCC	posterior cingulate cortex	TDC	typically developing control
PCIT	parent–child interaction therapy	tDCS	transcranial direct current stimulation
PD	pharmacodynamic	TMS	transcranial magnetic stimulation
PDD	pervasive development disorder	ToM	theory of mind
PDI	parent-directed intervention	VAN	ventral attention network
PET	positron emission tomography	VBM	voxel-based morphometry
PFC	prefrontal cortex	VNTR	variable number of tandem repeats
PK	pharmacokinetic	WCC	weak central coherence
		WHO	World Health Organization