Advances in Neurodevelopmental Disorders (2022) 6:184–195
https://doi.org/10.1007/s41252-022-00244-y

ORIGINAL PAPER

Wrong Brains at the Wrong Time? Understanding ADHD Through the Diachronic Constitution of Minds

Mads J. Dengsø[1]

Accepted: 5 February 2022 / Published online: 14 March 2022

Abstract

Objectives The purpose of this theoretical analysis of current research on ADHD is to provide an account integrating executive functional profiles with its broader structural neurodevelopmental profile.

Methods Comparative theoretical analyses between executive functional deficit disorder models of ADHD and results from default mode network fMRI data. This was followed by an analysis of the temporal profile of ADHD and phase synchronous neural assemblies.

Results Comparative analyses suggest disparities within executive functional deficit disorder models and discontinuities between executive functional and structural profiles of ADHD. Analysis of the temporal signature of ADHD provides a potential avenue for integrating different profiles by means of anchoring executive functions within inherent diachronic neurocognitive organization.

Conclusions The analyses provided suggest that executive functional deficits in ADHD arise from much broader idiosyncrasies, rooted within the inherent diachronic organization of neurocognitive function, and whose challenges must be understood in conjunction with socio cultural environmental factors.

Keywords ADHD · Diachronic mind · Cognitive science · Executive function · Phase synchronization

ADHD is among the most common neurobehavioral disorders in the world (Remschmidt, 2005). The current psychiatric profile of ADHD has been recognized for well over 50 years and has remained stable in terms of diagnostic criteria and prevalence for the last three decades (Polanczyk et al., 2014; Swanson et al., 1998; see also Amaral, 2007, p. 1612; Barkley, 2015a; Barkley & Peters, 2012). Yet for all that, our dominant account of ADHD is in tension with findings from recent neuroimaging studies.

Most accounts conceptualize ADHD as an executive function deficit disorder (Barkley, 2015c). Accordingly, we should understand ADHD in terms of deficits in executive functioning. This view of the explanatory basis of ADHD has since met with challenges from neuropsychological studies (Willcutt, 2015) and findings from resting-state neuroimaging research (Castellanos & Proal, 2012). Most saliently, this shows that there is a discontinuity between the executive functional profile of ADHD and its performance-independent neural basis as shown by resting-state activity.

A more general criticism has been raised against the application of medical model approaches to neurodevelopmental conditions including ADHD (Bertilsdotter Rosqvist et al., 2020). Medical model approaches interpret normative divergencies in terms of internally pathological deficits. Alternatives instead appeal to the responsibility of institutions in producing or preventing challenges for people with neurodevelopmental conditions. Despite the deliberately neurological focus of this paper, an implication will be precisely that the pathological status of ADHD cannot be attributed to individual brain function but must be outlined in terms of broader socio-cultural interactions. Indeed, it will be shown that conceptualizing ADHD in terms of executive function deficits is inadequate—even from a neurological perspective. But neither is the intention to rule out a role for executive functioning in characterizing a neurological component of ADHD. The error is not the recognition of a neural component but mistaking this for a full picture.

The goal of this paper is to remedy the discontinuity between ADHD as understood through its executive

✉ Mads J. Dengsø
mjd972@uowmail.edu.au

[1] Faculty of Arts, Social Sciences & Humanities, University of Wollongong, Wollongong (New South Wales), Australia

functional profile and its general neural profile. In other words, it seeks to understand executive function deficits in ADHD as just one expression of a less straightforwardly pathological phenomenon. It therefore seeks to integrate the manifestation of the executive functions with the inherent temporal structure of the brain. This will serve to provide a theoretical basis for understanding ADHD in its executive functional aspect without reducing the whole of ADHD to explicit manifestations of behavioral deficit. A downstream result will be both a deeper understanding of ADHD and of executive function. It will be argued that what can be measured as executive function deficits in ADHD arises from a heightened connectivity in the inherent temporal organization of brain. This heightened connectivity does, however, not warrant talk of any straightforward intrapersonal functional deficit. In other words, the neural basis of ADHD is not sufficient for pathology or deficit. This is by no means to render challenges for individuals with ADHD inconsequential. Rather, talk of deficit and pathology in ADHD must take account of the mismatches occurring in situations constituted across individuals *and* their socio-cultural context.

The Executive Function Deficit Disorder Model of ADHD

ADHD is a universal condition whose prevalence in all measured populations vary between an average 5–7% in children and 3–5% in adults with geographical variation likely resulting from methodological differences (Polanczyk et al., 2007). It ranges among the most common and most studied neurodevelopmental disorders in children, adolescents, and adults alike (Barkley, 2015a). The main characteristics of ADHD are persistent and maturationally inconsistent patterns of inattention and/or hyperactivity disruptive of social and vocational activities (American Psychiatric Association, 2013, p. 59). In accordance with these characteristics, ADHD has been correlated with a wide array of personal, social, academic, and occupational problems across all age groups (Aduen et al., 2018; see also Barkley, 2015b; Bernfort et al., 2008; Brandt & Fischer, 2017; Loe & Feldman, 2007).

ADHD as an Executive Function Deficit Disorder

Despite a vast body of research, the delineation of a neural basis for ADHD is beset by several difficulties. The condition is at once resistant to classification by a single persistent characteristic and by multiple subtypes. The former because individual presentations of ADHD are highly heterogenous (Wåhlstedt et al., 2009). The latter because the predominating presentation may change over time. This is one reason for why the use of ADHD "subtypes" has been abandoned for the term "predominant presentation" (Roberts et al., 2015). Indeed, presentations change both with maturation and with mature development. It may even change depending on the time of day (Roberts et al., 2015). Furthermore, individuals with ADHD exhibit a near ubiquitous comorbidity, with over 80% of children, adolescents, and adults with ADHD also meeting criteria for other disorders (Pliszka, 2015). It is therefore not surprising to see some researchers argue that ADHD should be understood as a multiple deficit disorder encompassing several (individually insufficient) deficits (Willcutt, 2015). Nonetheless, one overarching class of deficit has become a focal point for research.

Executive function deficit has been correlated with ADHD by a wide range of research (Biederman et al., 2004). This includes psychometric tests (Willcutt, 2015, p. 393), rating scales (Solanto, 2015), and lesional data (Barkley, 2015c, p. 406; Fuster, 2015, p. 202), as well as correlations between executive function and the diagnostic criteria of ADHD (Barkley, 2015c, p. 408). The culmination is that "executive dysfunction has in recent years come to be regarded by many in the field as a defining characteristic of ADHD in both children and adults" (Solanto, 2015, p. 256). As a result, executive function deficit is seen as both central to ADHD and to be the root cause of its behavioral manifestation. As articulated here,

> [t]heorists and clinical scientists have long speculated that problems with EF [executive function], or the cross-temporal organization of behavior specifically and self-regulation more generally, are at the heart of this disorder [ADHD] and give rise to the more superficial and surface symptoms represented in clinical diagnostic criteria. (Barkley, 2015c, p. 406)

In short, the prevailing literature has come to understand ADHD as an *executive function deficit disorder* (henceforth EFDD). This EFDD model for ADHD suggests that research and treatment should focus on executive function. So, what is executive function?

As befits a neural basis for as heterogenous a condition as ADHD, executive function is a meta-construct encompassing several different mental abilities (Gilbert & Burgess, 2008). Precisely how the abilities or constructs specified by executive function are distinguished varies widely from one account to the next. Indeed, this is itself a known source of trouble (see Barkley, 2012, pp. 17–22). Nonetheless, executive functions are consistently defined in terms of mental abilities needed to sustain problem-solving towards a goal (Barkley, 2015c). Specifically, executive function underlies deliberate actions over short or long intervals, coordination, and social interaction. As will be seen in "Resting-State Imaging Data," it is especially closely correlated with temporal awareness. On most accounts, executive function is understood in terms of a set of abilities or "mental tools"

employed for sustained and adaptive problem-solving (Barkley, 2015c, p. 410; see also Barkley, 2012; Willcutt, 2015, p. 392).

The EFDD model of ADHD understands executive function deficits as constituting the neurocognitive basis from which arise the many heterogeneous challenges that make up its diagnostic criteria (Barkley, 2012, 2015c). Impacts to functioning cascades across several levels of functional integration from developmental self-regulation of behavior and time management to long-term planning and social skill. All challenges may be traced back to a dysfunction of the mental tools of executive function. Accordingly, when we understand ADHD as a disorder of executive function—and we understand executive function as a set of mental abilities for problem-solving—ADHD is constituted by deficits in our mental problem-solving abilities.

Thus far, it should be clear that ADHD is both a widely heterogeneous disorder, and that it is reliably correlated with executive function. A different matter, however, is whether these kinds of executive functional problem-solving mental abilities are exhaustive of the neurocognitive component of ADHD (Castellanos et al., 2006). As well-grounded as this conception is, there is also well-grounded reasons for remaining doubtful as to whether the EFDD model adequately captures the neurocognitive basis of ADHD.

Resting-State Imaging Data

The executive function-based view of ADHD has met with some issues from neuroimaging studies. Recent developments within neuroimaging and ADHD research have provided two new insights about the neural basis of ADHD. Firstly, the neural substrate of ADHD is not restricted to networks and regions tied to executive functioning, such as the prefrontal and striatal networks (Castellanos & Proal, 2012). Secondly, the manifestation of ADHD in executive function deficits likely constitute only the tip of the iceberg that comprises the intrinsic neural substrate of ADHD (Castellanos & Aoki, 2016).

Resting-state functional magnetic resonance imaging (or R-fMRI) allows for the measurement of neural connectivity patterns outside of functional task performance. The idea is that R-fMRI accordingly provides a functional map of intrinsic connectivity patterns between different regions of the brain because the elucidated activity and interconnection is independent of the subject performing executive functional tasks. Such measurements have indicated that ADHD involves neural networks well beyond those correlated with executive functioning (Castellanos & Proal, 2012; Proal et al., 2011)—prompting calls for the development of a broader neurocognitive profile. ADHD pertains to neural phenomena more subtle and pervasive than those specified by executive function deficits. Some of the authors' introductory remarks are highly relevant here.

> Prefrontal striatal circuits underpin executive function, and dysfunction in such processes has long been considered an important neuropsychological correlate of ADHD. This model has been largely supported by an ever-increasing number of structural and functional imaging studies, but divergent evidence such as the involvement of occipital or temporal cortex has tended to be ignored because of the initially reasonable assumption that unexpected results probably represent false positives. However, accumulating evidence suggests that the prefrontal-striatal model of ADHD should be extended to include other circuits and their interrelationships from the perspectives of systems neuroscience. We suggest that formulation of a more inclusive brain model of ADHD is facilitated by the new paradigm of resting-state functional magnetic resonance imaging (R-fMRI), which is increasingly revealing the intrinsic functional architecture of the brain. (Castellanos & Proal, 2012, p. 17).

In addition to executive function deficits, this research indicates dysregulated or aberrant interactions within and among large-scale neural systems as the neural basis of ADHD (Castellanos & Proal, 2012, p. 18). This neural basis is neither exhausted nor specified by its manifestation in executive function deficits. Instead, they seem to correlate ADHD with disruptive large-scale network connectivity in children and adolescents (Castellanos & Aoki, 2016; see also Bos et al., 2017; dos Santos et al., 2014; Qian et al., 2019). The indication is that the neural basis of ADHD does not pertain to localized or functionally specified neural areas, but to pervasive and structural principles of large-scale brain function. This will be unpacked in greater detail in the development of a positive account of the neural basis of ADHD. For present purposes, the point to be shown here and in the remainder of the section is that the EFDD model is under increasing pressure from accumulating neuroscientific evidence and from further difficulties to be outlined shortly.

These findings from R-fMRI research pose a rather serious challenge to EFDD models of ADHD both in terms of its neural topology and functional basis. In terms of neural topology, the structural basis of the condition exceeds the neural regions underlying executive function. And in terms of its functional basis, the intrinsic functional connectivity described by resting-state imaging is not accounted for by its manifestation in executive function deficits. If this is right, then the EFDD model likely constitutes an oversimplification of the neural profile of ADHD both in terms of structure and function. At this point, it might be objected that the purpose of a model was never to provide an exhaustive

description, and that simplification by no means must prove pernicious for either theory or practice.

Theoretical and Practical Implications

The conceptualization of ADHD in terms of executive function deficits has implications both for understanding and treatment. In the first instance, as the R-fMRI research shows, executive function deficits describe only part of the neural basis of ADHD. This has already been a source of discordance between interpretations of ADHD based on respectively psychometric tests and rating scales of executive function. The theoretical issues here are twofold. Firstly, different types of executive function measurements are inconsistent. Secondly, either measurement presuppose an executive functional basis. I will outline each in turn.

Methods for measuring the interrelation of ADHD and executive function include normed rating scales of executive function and neuropsychological tests. Psychometric tests consist of measurements of executive functional performances within a laboratory setting while rating scales consist of self-ratings of executive function specifically developed for the assessment of ADHD (Solanto, 2015). However, psychometric tests and rating scales of executive function show little correlation with each other and significant incongruity on key issues concerning the relation of ADHD and executive functional deficits (Toplak et al., 2013). In particular, rating scales demonstrate a consistent correlation between ADHD and executive function deficits (Barkley, 2015c) whereas psychometric tests demonstrate some but no consistent correlation (Willcutt et al., 2005; see also Willcutt, 2015). As such, results from psychometric tests and rating scales dissent on the issue of whether executive function deficit is a necessary feature of ADHD. EFDD proponents contend that the disparity between tests and ratings simply reflects shortcomings of psychometric tests of executive functions in relating to the real circumstances of individuals with ADHD (Barkley, 2012, pp. 9–13; see also Barkley & Murphy, 2011; Solanto, 2015; Weyandt & Gudmundsdottir, 2015). Another perceived fault of psychometric tests is that by denying the necessary connection between ADHD and executive function deficits, these tests seem to contradict their own basis as measures of executive function (Barkley, 2015c).

This latter criticism raises the following concern. Both methods focus exclusively on executive functioning. What they do not measure is whether or not executive function deficits themselves constitute the only determining factor. That is, psychometric tests and rating scales do not so much prove that executive function deficits are a decisive factor in ADHD as they simply presuppose it. On the one hand, this makes psychometric tests and rating scales of executive functioning excellent for purposes of measuring and identifying *which* executive function deficits may be correlated with ADHD. But on the other, it simultaneously renders such measurements effectively mute on the question of whether executive function deficit *is* the only decisive factor.

Concerning the practical consequences of the EFDD model of ADHD, recent accounts have argued that the application of medical models to neurodevelopmental disorders such as ADHD and autism spectrum disorder generate a harmfully misunderstood view of these conditions (Rosqvist et al., 2020). Medical models identify conditions by emphasizing functional defects in terms of individual and internally localized deficits. Given its identification of ADHD solely in terms of its manifestation of behavioral deficits, the EFDD constitute a clear-cut medical model approach to ADHD. However, the diagnostic criteria of ADHD are inextricably tied to manifestations within vocational, educations, and social institutions: challenges for individuals with ADHD occur in relation to contextual interactions with normative institutions adapted to the needs of neurotypical individuals (Boorse, 2009). This irreducibly social aspect of neurodevelopmental disorders like ADHD calls for more research dedicated to the extrapersonal and interactive aspects of ADHD. But neither is it to disparage any investigation of the role of neural executive functional capacities as components of cognition.

What makes this particular application of the medical model all the more striking is that an exclusive EFDD model is not supported by the neuroscience either. Not only is the whole condition reduced to the individual neural manifestation; but this component of neural manifestation is, in turn, equated with performative deficit. This creates a potentially pernicious view of challenges in ADHD as arising internally within neurocognitive mechanisms in the individual. In one stroke, the relational mismatch between the non-neurotypical individual and a neurotypical socio-institutional context is asymmetrically attributed as a deficit of the individual—who's very atypicality is then subsequently profiled as just this reductive aspect of (executive) functional *deficit*. But this resulting picture (of individuals suffering from straightforward brain-deficits) misrepresents both the condition of ADHD *and* its neural component.

Executive Function and Temporal Awareness

ADHD is defined as a dimensional disorder (Roberts et al., 2015). This means that diagnostic criteria characterizing ADHD (such as inattention and impulsivity) are also common within healthy populations. Problems like struggling with attention can be perfectly normal but produce impairment at high levels of frequency and severity—indeed, being overly fixated can itself become a problem. Some authors even suggest that ADHD and autism spectrum disorder may reflect inverse disproportionalities in neural responses to

unexpected stimuli (Gonzales-Gadea et al., 2015). In accordance with the large-scale patterns from resting-state imaging presented in "Theoretical and Practical Implications," the dimensional character of ADHD suggests that its basis is not some functional breakdown, but rather a disproportional or excessive configuration of functions which exist in normal populations as well. In this regard, what constitutes disproportionality or potentially harmful excess is contingent upon situational context. That is, executive functional impacts in individuals with ADHD may be less a matter of dysfunction and more of idiosyncratic functioning. This section will explore one potential avenue for understanding executive function by way of a pervasive function: namely temporal awareness.

Temporal Awareness, Executive Function, and ADHD

Neuroimaging and behavioral research alike have correlated ADHD with specific deficits in temporal processing (Smith et al., 2002). Individuals with ADHD register impaired performance on psychometric tests across all measured temporal scales. These are generally distinguished into three timescales of increasing duration: the timing of actions, the ability to perceive time, and capacity for long-term planning (Rubia, 2006). ADHD affects executive functional temporal processing across all measured parameters (Rubia, 2006, p. 228). One review of time deficits in ADHD remarks on this special relation as follows.

> The most prominent deficits in executive functions in ADHD are in tasks of motor response inhibition, working memory and sustained attention. However, there is also consistent evidence that ADHD patients have cognitive deficits in the timing domain, including impairments in motor timing, time perception and temporal foresight. Furthermore, there is emerging evidence that ADHD patients have abnormalities in the underlying neurofunctional networks that mediate these timing functions, (Noreika et al., 2013, p. 237)

The review quoted here goes on to suggest that "timing impairments are integral to ADHD, demonstrating that ADHD is at least in part a disorder of abnormal temporal processing" (Noreika et al., 2013, p. 261).

Additionally, this interrelation pertains in no lesser degree to research explicitly focused on executive function. Temporal processing is indispensable to executive function (Noreika et al., 2013, p. 236). Indeed, advocates of the EFDD model have described executive function itself as a temporal capacity, describing ADHD in corresponding terms of time blindness.

> [ADHD] is also a disorder of time management specifically, in that the individual manifests an inability to regulate his or her behavior relative to time and future welfare, as well as that of others at his or her developmental level. This creates a sort of temporal myopia, or time blindness, in which the individual responds to or prepares only for events that are relatively imminent, not the ones that lie further ahead in time and for which others of their age are preparing so as to be ready for their eventual arrival. (Barkley, 2015c, p. 422).

Executive functions constitute the ability to inhibit and control behavior over longer timespans. This allows us to anticipate sequences of events of larger timescales. In other words, executive functions increase our temporal scope, and reciprocally, this temporal capacity determines our ability for deliberate executive self-control. A cognitive system's executive function and its temporal capacity are therefore doubly correlated: the increase in one is correlated with an increase in the other; and a decrease in one is correlated with a decrease in the other. Executive functions are intertwined with the awareness of time unfolding over short or long periods of time. But how should we characterize this relation?

The Detachment Model of Temporal Awareness

One common way is to conceptualize temporal awareness as itself an executive function. Cognitive science often conceptualizes temporal awareness as an executive functional "mental tool" for detachment from our current "sensorimotor reality" (Pezzulo, 2008). I will call this view *the detachment view of temporal awareness* because it views temporal awareness as being correlated with a capacity for detachment from present input.

On the detachment view of temporal awareness, temporally extended capacities constitute a set of mental tools—a special cognitive faculty. By special faculty I mean that temporal awareness is here viewed as functionally distinct from other neurocognitive functions. In contrast to perceptual engagement with our surroundings, our temporal capacities are then characterized by the ability to *dis*-engage from perception and representationally attend to non-present points in time. Whereas some implicit anticipation may be sufficient for immediate perceptual coordination, autonomous and higher cognitive capacities, it is argued, require a perceptually detached form of anticipatory coordination. In these cases, anticipatory coordination combines disengagement from the here-and-now with representational extrapolations forward and backward in time (Pezzulo, 2008, p. 207). Our temporal awareness is accordingly constituted by the ability to detach from current input and represent past and future states. Indeed, organisms' capacities for representing larger and larger spans of time—and corresponding detachment from

present sensory input—are in this way often taken to constitute the mark of cognitive sophistication par excellence (see, for instance, Corcoran et al., 2020). This view creates a disjunction between perceptually detached temporal capacities, exemplified by executive function, and direct perceptual processes.

Parceling neurocognitive function into executive and pre-executive function is theoretically productive—for better and for worse. On the one hand, it allows us to clearly delineate between sophisticated cognitive processes and reflexive functions. As such, research on executive function carefully distinguishes between executive and "pre-executive" functions. One such example from an advocate of the EFDD model reads:

> [Pre-Executive level] brain functions are responsible for the stimulus–response, moment-to-moment, and largely unconscious or automatic activities of the organism as it goes about sustaining its life in its natural habitat. This level can be fruitfully regarded as the "automatic" level of human activity often described in models of self-regulation. (Barkley, 2012, p. 76)

Compared with the representational detachment model of temporal awareness:

> anticipatory capabilities [beyond the immediately perceptible world] are considered by cognitive scientists a presupposition for autonomous mental life, since they permit to cognitive agents to *disengage* from their sensorimotor loops and to conceive and pursue their goals. (Pezzulo, 2008, p. 214, italics in original).

Both accounts introduce a separation between, one the one hand, functions that are direct and automatic and, on the other hand, functions that are indirect and deliberate. While this kind of delineation is often helpful, it also risks obfuscating the continuity between each type of function. In particular, it makes it hard to see how temporally indirect and deliberate executive functions can arise from out of oppositely temporally direct and automatic functions. How do disengaged and executive capacities arise out of engaged and automatic ones? Specifically, for present purposes, how do executive functional profiles, such as can be seen in temporal discounting, emerge from the kinds of basic connectivity patterns outlined by resting-state imaging research? Without accounting for the continuity of these different neurocognitive "strata" runs the risk of oversimplifying neurocognitive profile by identifying them with only one level of cognitive function. What is needed is a way to understand the continuity between automatic and deliberate functions—between executive functional connectivity patterns and performance-independent connectivity patterns. The detachment view, however, only exacerbates the discontinuity between these functions.

As an alternative to this notion of temporal awareness, a final excursion before returning to the question of ADHD will serve to show that the awareness of temporality neither requires a specially dedicated executive function nor representational detachment.

The Diachronic Organization Model of Temporal Awareness

Our understanding of neurocognitive function has in recent decades shifted towards a network-based approach which emphasizes the organizing role of global dynamics across large-scale neural systems (Fuster, 2006). On this "systems neuroscientific" approach, the functions of more localized neural systems are constrained by more globally distributed neural systems.

> Nowadays, there is overwhelming evidence that all representations in the brain are distributed. Perceptions, memories, and even emotions are represented in a distributed manner; hence, a deeper understanding of the mechanisms underlying distributed processing is a central question for neuroscience. (Deco et al., 2012, p. 10)

System neuroscience understands neurocognitive functions by way of large-scale cross-regional neural assemblies (Menon, 2012). Instead of having their function specified by their proximity to other local systems, these large-scale neural systems which underlie neurocognitive function are functionally organized by way of shared *temporal* dynamics (Daffertshofer & van Wijk, 2012). In short, the neural substrates of neurocognitive function are organized by temporal signature (Engel, 2010, p. 231). So how does this work?

It works by way of temporal organization. The temporal signatures which determine these large-scale neural assemblies are made up of neural oscillations. Neurons fire at periodic intervals corresponding to certain oscillational frequencies. For instance, neurons that oscillate in delta waves fire at a frequency of 1–4 Hz, while ones that oscillate in theta waves fire at a frequency of 4–8 Hz. Large groups of neurons fall into oscillational coherence by what is called phase synchronization (Varela et al., 2001). Phase synchronization patterns that interconnect large-scale neural assemblies are hypothesized to constitute the structural basis for neurocognitive functions. Of especial relevance to the question of executive function deficits, phase synchronization patterns are modulated by arousal and attention, and may be decisive in explaining cognitive abnormalities (Varela et al., 2001, p. 236). Moreover, studies into the emergence of cortical circuits for central executive functions have indicated correlation between theta phase synchronization and executive function, and between beta phase synchronization and attentional control (Mizuhara & Yamaguchi, 2007, pp.

232, 242). In short, neurocognitive functions do not support temporality: on the contrary, temporal organization supports neurocognitive function (see Buszáki, 2006 and 2019, for a comprehensive account of the temporal organization of neural and neurocognitive function).

At this fundamental level, neurocognitive functions no more need to process the time by which they are organized than they need to process the space they take up. In this sense, contrary to the representational detachment view, temporality is not an input which the brain needs to process but is rather an organizational principle of how brains work. Instead of accounting for temporality via executive or representational functions, neurocognitive (and executive) functions are determined by way of the brain's inherent temporal organization. But can this intrinsic diachronicity also account for executive and temporally extended functions? Even if phase synchronization accounts for coordination in perception, the appeal to representational detachment arises exactly from the concern that such perceptual processes cannot account for fully autonomous (or executive) coordination (Pezzulo, 2008, p. 213). How can the phase synchronous organization of neurocognitive functions account for the full spectrum of executive functional capacities?

Phase synchronization constitutes neurocognitive functions across several scales of integration ranging from intervals of instants, moments, or longer. Different integrational scales have been categorized by three timescales, including an elementary timescale of (10–100) milliseconds, an integration timescale of seconds, and a narrative timescale more than seconds (Varela, 1999; see also Gallagher, 2017a, pp. 8, 143–148; Gallagher, 2020, p. 29; Gallagher, 2017b). The first is exemplified by subconscious perceptual and cognitive activity; the second by consciously aware and deliberate activity in the living present; and the third by still longer intervals, such as long-term planning and durations greater than the living present. These three (elementary, integrational, and narrative) timescales of phase synchronization correspond to the three levels of temporal processing highlighted by studies of the neural correlates of timing functions (Rubia, 2006) and of impacts on temporal awareness in ADHD (Noreika et al., 2013)—timing of actions, the ability to perceive time, and capacity for long-term planning. For each timescale of executive function, there is a corresponding phase synchronization timescale.

The decisive difference here is that higher timescales of phase synchronization do not require any appeal to special detached faculties. Instead of understanding temporally extended executive functional capacities by way of special mental faculties which create a detachment away from more immediate engagement, we can understand them as phase synchronous activity patterns instantiated across longer timescales. When phase synchronous structures at the narrative timescale constrain the activity of synchronizations at the integrational or elementary timescales, these constraints are facilitated not by way of detachment but simply by "higher" phase synchronous structures being themselves retained across longer intervals of time.

It all comes down to interactions between different integrational levels of the intrinsic temporal structure of neurocognitive function. Phase synchronous structures at higher levels of integration allow for more temporally extended control by constraining the activity of phase synchronous structures at lower levels of integration. For example, a long-term phase synchronous structure may reinforce or suppress certain types of short-term phase synchronous structures. In this sense, long-term phase synchronous structures may be seen as exerting a top-down influence over short-term phase synchronous structures. At the behavioral level, this top-down influence manifests itself as the kinds of entrainment of behavioral trajectories associated with executive control and executive functions. Importantly, however, this top-down influence of reinforcement and suppression does not need to rely upon special representational detachment or distal intentions. Instead, we can appeal to the inherent synergy or discord between rhythmic patterns of different types of phase synchronous structures. Solely by strength of their differential rhythmic patterns, some phase synchronous structures will have a suppressive or a reinforcing effect upon other phase synchronous structures. Like the conductor of a musical orchestra, the rhythmic structure of long-term phase synchronous structures imposes a constraining influence by setting a background structure for short-term phase synchronous structures to fall into. Conversely, accumulative short-term phase synchronous structures, as well as other concurrent long-term phase synchronous structures, may cause the long-term background structure to change or break altogether.

From this perspective, temporally distant goals do not rely on the addition of distal intentions (for a comprehensive account, see Hutto & Myin, 2013; see also Stepp & Turvey, 2010). I call this *the diachronic organization model of temporal awareness* because it implements temporality by way of the already existent temporal extension of neurocognitive functioning. Just as neural functions are constituted by spatiotemporally distributed neural assemblies, cognitive processes are better described in terms of trajectories than in terms of special detached states (Kirchhoff, 2015; Kirchhoff & Kiverstein, 2019, pp. 104–108; see also Spivey, 2008). Finally, this way to conceptualize "higher" neurocognitive functions such as executive function provides a way to conceptualize the executive functional manifestation of ADHD in a way that retains continuity with its basis in the structural connectivity outlined by resting-state imaging data.

The Temporal Signature of ADHD

In order to demonstrate the continuity between the executive functional and structural profile of ADHD, I will start by briefly outlining a key feature of the topographical profile of ADHD. Topographically, there is a significant correlation between ADHD and maturational lag in cortical thickness. Indeed, the large-scale disruptive connectivity found by resting-state imaging research bears a striking resemblance to absent maturational neural modularity (Janssen et al., 2017; see also Sripada et al., 2014; Vaidya, 2011). By "modularity" is meant the degree of functional separation between different neural networks. This indicates that "[a]bnormalities in brain network maturation play a significant role in ADHD" (Qian et al., 2019, p. 2). In normal neural development, different neural networks become more pronounced and correspondingly more clearly functionally segregated with maturation. Networks gradually become defined and demarcated as cortical thickness grows. This maturational increase in cortical thickness is, in turn, correlated with more stable neurocognitive patterns. (This is part of why old habits die hard: neural networks become developmentally shaped into stabilized patterns; but it is also part of why infants are such swift learners.) Higher levels of cortical thickness are correlated with higher degrees of functional separation between neural networks whereas lower levels are correlated more functional interconnectivity.

Lower Cortical Thickness and Hyper-Connectivity

Comparisons with age-matched control groups indicate that children with ADHD have a significantly higher correlation between the executive control network and the salience network, with children with more compulsive presentations of ADHD exhibiting comparative hyperconnectivity in the anterior default-mode network (Qian et al., 2019). A general suggestion from this is that ADHD may be correlated with age-comparatively low neural modularity and a corresponding heightened interconnectivity between neural assemblages involved with observation and with executive function.

As with the stubbornness or flexibility of habits, these interactions are ambivalent: their value or harm depends on the context. On the one hand, connections between different networks of neural assemblies are crucial for forming new connections in learning. But, on the other hand, this same openness may also contribute to increased interruptions of neurocognitive processes constituted across longer timescales. So how does this relate to the diachronic organization model of temporal awareness presented in the previous section? Phase synchronization allows us to understand the continuity between this heightened connectivity and the executive functional manifestation of ADHD. Simply put, the executive functional profile of ADHD may be traced back to this heightened connectivity.

Heightened connectivity corresponds to heightened volatility to interruptions—which poses a disproportional challenge to phase synchronous assemblies of longer temporal duration. This higher tendency towards interruptions of temporally longer phase synchronous assemblies translates to an increased volatility in (temporally longer) executive functional tasks, such as temporal discounting. Heightened connectivity between different neurocognitive networks means that different long-term phase synchronous structures are more likely to interact, causing a proportionally higher likelihood of changes in long-term phase synchronous structures.

The difference here is that, instead of classifying the neurobehavioral profile of ADHD in terms of deficit to a special executive functional faculty, we can understand it in terms of a tendential volatility within a much broader neurocognitive profile. In other words, when we take into account the continuity of executive functional capacities with the underlying diachronic organization of neurofunction, we get a much more encompassing, and more nuanced picture of the neurobehavioral profile of ADHD—and one which takes seriously the contribution of neurofunctional idiosyncrasy in ADHD without reducing it to its manifestation in task-dependent functional deficits. This broader perspective corresponds with both the behavioral and developmental profile of ADHD. In the first instance, ADHD's characterization by inattention and impulsivity corresponds to the potentially disruptive desynchronization brought on by heightened neurofunctional connectivity brought on by lower maturational cortical thickness. On the developmental profile, the relation between higher levels of connectivity and maturational lag corresponds to the developmental variation in predominant presentations of ADHD, and with the prevalence of a maturational diminishing of severity in symptoms. Instead of drawing a line dividing general default-mode functioning from special executive capacities, we can trace a line *from* executive function to the inherent neurocognitive structure of the brain. In this way temporal phase synchronization becomes the common denominator, or mutual structural principle, bridging executive function and performance-independent connectivity patterns demonstrated in resting-state imaging data. Each represents the inherent temporal organization of the mind at different scales of integration. This also creates a decisive shift in how we understand executive function.

Unlike the standard approach to executive function as a discontinuous faculty based on representational detachment, the mind's inherent temporal structure shows how executive

functional capacities may be accounted for without incurring an unhelpful discontinuity between profiles. The neural substrate of executive function has been said to constitute the highest stage of integration in the perception–action cycles by which we navigate our environment (Fuster, 2015, p. 3). And we may understand executive function as just that: not as a special faculty or a set of mental tools supervening upon perception in the manner of anticipatory representational detachment, but as an expansion of existing—inherently temporally extended—perceptual capacities.

A Heterogeneous and Relational Condition

Heightened connectivity and the resulting increase in volatility to desynchronization is constituted at the level of the implicit temporal structure of neurocognitive functioning. It pertains to the temporal organization of brain function. For this reason, the challenges pertaining to the neural basis of ADHD may be understood in terms of heightened connectivity between different phase synchronization assemblies and a corresponding heightened volatility to interruption in the brain's implicit temporal structure.

This explanation is highly commensurable with the heterogeneous, overlapping, and fluctuating character of ADHD which otherwise poses a challenge for a comprehensive explanatory basis. The answer to why ADHD has various presentations—and whose presentations vary across several timescales—is just that ADHD is not a disorder of any one special faculty or specified functional deficit but a condition pertaining to the implicit temporal organization constitutive of cognitive functions as such. The common ground between the heterogeneous manifestations of ADHD is located not just at the level of executive function but of inherent temporal organization. The diachronic organization view of temporal awareness hence not only facilitates continuity between executive functional manifestations and general connectivity patterns in ADHD: it also provides a more comprehensive understanding of the heterogeneous character of ADHD.

Finally, heightened connectivity, as may be ascribed to individual persons with ADHD, is not inherently pathological. Desynchronization is an integral part of normal phase synchronization and is crucial to our ability to respond to new perceptual information. In other words, the change or breaking of long-term phase synchronous patterns are crucial for our ability to respond and adapt to new information. However, a heightened disposition can also become a burden for the ability to maintain attention. Whether a heightened connectivity provides an asset to adaptivity or a burden to attentional resilience will vary according to what the situation calls for. The perceptual sensitivity brought on by a heightened connectivity can be a blessing or a curse depending on the environmental context we find ourselves in. This is why social and institutional factors are indispensable to any talk of challenges in ADHD.

What is pathological and non-pathological in this regard is determined by the whole situation involving both individuals and their respective environments. What this means is that the pathological status of ADHD is partly dependent on social context. All of this suggests that, although we may talk of a special neural signature of ADHD, we would be mistaken to talk about a special neural pathology. Deficits in ADHD are inextricably tied to social contexts. Although this implication deserves much more attention than can be given in the present paper, I will leave with a few points deserving of further study given the importance of social context for the status of ADHD.

Firstly, phase synchronous stability varies throughout maturation, from person to person, and throughout our daily routines. In this sense, it is not clear that defining ADHD in terms of pathology will be consistent across different situations, since its challenges will change with maturation, social contexts, and even the time of day. Secondly, the interaction between different phase synchronous structures characteristic of the neural basis of ADHD is also a key factor for many beneficial traits, such as imaginative innovation. As such, defining the cognitive profile of ADHD in terms of pathology due to challenges with conforming to normative social context begs the question of which side of the interaction is responsible for this mismatch. Lastly, it is not clear that the kind of atypicality defining ADHD simply presents a net-negative for group-performances. While we may define cognitive diversity in terms of pathological deviation away for a single "healthy" populational norm, there may be inherent strengths at the environmental level to having a more diverse population (Chapman, 2021). Studies indicate that cognitively diverse groups outperform cognitively uniform groups. As such, even if we adopt a view of cognitive profiles based upon their ability to successfully perform functional tasks, a more open-minded social dynamic that makes room for cognitive diversity may not only be its own reward—but simultaneously may improve performative achievements as well.

The neural basis of ADHD in heightened connectivity precedes and exceeds its manifestation in executive function deficits. For this reason, executive function deficits should not be taken as the neural (or general) basis of ADHD. ADHD, on this view, is not restricted to executive function, but has its basis in the inherent temporal organization of brain function. This reconfiguration permits integrating executive functions within a broader scope in accordance with the findings of resting-state imaging data. Lastly, while the neurobehavioral profile of ADHD, consisting of a heightened sensitivity to incoming stimuli, may be statistically correlated with challenges within certain socio-cultural contexts, it would be incorrect to ascribe the pathological state

of the individual neural component rather than the broader context involving both brain body and the broader social environment.

Acknowledgements Thanks to Michael Kirchhoff, Glenda Satne, Dan Hutto, and Nick Brancazio for helpful comments on earlier drafts of this paper. Thanks to Ian Robertson and Thomas Netland for support and encouragement.

Funding Open Access funding enabled and organized by CAUL and its Member Institutions.

Availability of Data and Material Not applicable.

Code Availability Not applicable.

Declarations

Ethics Approval Not applicable.

Conflict of Interest The authors declare no competing interests.

References

Aduen, P. A., Day, T. N., Kofler, M. J., Harmon, S. L., Wells, E. L., & Sarver, D. E. (2018). Social problems in ADHD: Is it a skills acquisition or performance problem? *Journal of Psychopathology and Behavioral Assessment, 40*(3), 440–451.

Amaral, O. B. (2007). Psychiatric disorders as social constructs: ADHD as a case in point. *American Journal of Psychiatry, 164*(10), 1612–1612.

Barkley, R. A. (2012). *Executive functions: What they are, how they work, and why they evolved*. Guilford Press.

Barkley, R. A. (2015a). History of ADHD. In R. A. Barkley (Ed.), *Attention-deficit hyperactivity disorder: A handbook for diagnosis and treatment* (pp. 3–50). The Guilford Press.

Barkley, R. A., & Murphy, K. R. (2011). The nature of executive function (EF) deficits in daily life activities in adults with ADHD and their relationship to performance on EF tests. *Journal of Psychopathology and Behavioral Assessment, 33*(2), 137–158.

Barkley, R. A., & Peters, H. (2012). The earliest reference to ADHD in the medical literature? Melchior Adam Weikard's description in 1775 of "attention deficit" (Mangel der Aufmerksamkeit, attentio volubilis). *Journal of Attention Disorders, 6*(8), 623–630.

Barkley, R.A. (2015b). Etiologies of ADHD. In R.A. Barkley (Ed.), *Attention-deficit hyperactivity disorder: A handbook for diagnosis and treatment* (4th ed., pp. 356–390). The Guilford Press.

Barkley, R.A. (2015c). Executive functioning and self-regulation viewed as an extended phenotype: Implications of the theory for ADHD and its treatment. In R.A. Barkley (Ed.), *Attention-deficit hyperactivity disorder: A handbook for diagnosis and treatment* (4th ed., pp. 405–434). The Guilford Press.

Bernfort, L., Nordfeldt, S., & Persson, J. (2008). ADHD from a socio-economic perspective. *Acta Pædiatrica, 97*(2), 239–245. https://doi.org/10.1111/j.1651-2227.2007.00611.x

Bertilsdotter Rosqvist, H., Chown N., & Stenning, A. (2020). Introduction. In Bertilsdotter Rosqvist, H., Chown N., & Stenning, A. (Eds.), *Neurodiversity studies: a new critical paradigm*. Routledge.

Biederman, J., Monuteaux, M. C., Doyle, A. E., Seidman, L. J., Wilens, T. E., Ferrero, F., Morgan, C. L., & Faraone, S. V. (2004). Impact of executive function deficits and attention-deficit/hyperactivity disorder (ADHD) on academic outcomes in children. *Journal of Consulting and Clinical Psychology, 72*(5), 757–766. https://doi.org/10.1037/0022-006X.72.5.757

Boorse, C. (2009). Disability and medical theory. In D. Ralston & J. Ho (Eds.), *Philosophical reflections on disability*. Springer.

Bos, D. J., Oranje, B., Achterberg, M., Vlaskamp, C., Ambrosino, A., de Reus, M. A., van den Heuvel, M. P., Rombouts, S. A. R. B., & Durston, S. (2017). Structural and functional connectivity in children and adolescents with and without attention deficit/hyperactivity disorder. *Journal of Child Psychology and Psychiatry, 58*(7), 810–818. https://doi.org/10.1111/jcpp.12712

Brandt, L., & Fischer, G. (2017). Adult ADHD is associated with gambling severity and psychiatric comorbidity among treatment-seeking problem gamblers. *Journal of Attention Disorders, 23*(12), 1383–1395. https://doi.org/10.1177/1087054717690232

Buszáki, G. (2006). *Rhythms of the brain*. Oxford University Press.

Castellanos, F. X., & Aoki, Y. (2016). Intrinsic functional connectivity in attention-deficit/hyperactivity disorder: A science in development. *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging, 1*(3), 253–261. https://doi.org/10.1016/j.bpsc.2016.03.004

Castellanos, F. X., & Proal, E. (2012). Large-scale brain systems in ADHD: Beyond the prefrontal–striatal model. *Trends in Cognitive Sciences, 16*(1), 17–26. https://doi.org/10.1016/j.tics.2011.11.007

Castellanos, F. X., Sonuga-Barke, E. J., Milham, M. P., & Tannock, R. (2006). Characterizing cognition in ADHD: Beyond executive dysfunction. *Trends in Cognitive Sciences, 10*(3), 117–123. https://doi.org/10.1016/j.tics.2006.01.011

Chapman, R. (2021). Neurodiversity and the social ecology of mental functions. *Perspectives on Psychological Science*. https://doi.org/10.1177/1745691620959833

Corcoran, A. W., Pezzulo, G., & Hohwy, J. (2020). From allostatic agents to counterfactual cognisers: Active inference, biological regulation, and the origins of cognition. *Biology & Philosophy, 35*(3), 1–45.

Daffertshofer, A., & van Wijk, B. C. M. (2012). Transient motor behavior and synchronization in the cortex. In M. I. Rabinovich, K. J. Friston, & P. Varona (Eds.), *Principles of brain dynamics* (pp. 233–259). MIT Press.

Deco, G., Jirsa, V., & Friston, K. J. (2012). The dynamical and structural basis of brain activity. In M. I. Rabinovich, K. J. Friston, & P. Varona (Eds.), *Principles of brain dynamics* (pp. 9–26). MIT Press.

dos Santos Siqueira, A., Biazoli Junior, C. E., Comfort, W. E., Rohde, L. A., & Sato, J. R. (2014). Abnormal functional resting-state networks in ADHD: Graph theory and pattern recognition analysis

of fMRI data. *Biomed Research International*. https://doi.org/10.1155/2014/380531
Engel, A. K. (2010). Directive minds: How dynamics shapes cognition. In Stewart, J., Gapenne, O., & Di Paolo, E.A. (Eds.), *Enaction: Towards a new paradigm for cognitive science*, 219–243.
Fuster, J. M. (2006). The cognit: A network model of cortical representation. *International Journal of Psychophysiology, 60*(2), 125–132.
Fuster, J.M. (2015). *The prefrontal cortex* (5th ed). Academic Press.
Gallagher, S. (2017a). *Enactivist inverventions*. Oxford University Press.
Gallagher, S. (2017b). The past, present and future of time-consciousness: From Husserl to Varela and beyond. *Constructivist Foundations, 13*(1), 91–97.
Gallagher, S. (2020). *Action and interaction*. Oxford University Press.
Gilbert, S. J., & Burgess, P. W. (2008). Executive function. *Current Biology, 18*(3), R110–R114.
Gonzalez-Gadea, M. L., Chennu, S., Bekinschtein, T. A., Rattanazzi, A., Beraudi, A., Tripicchio, P., Moyano, B., Soffita, Y., Steinberg, L., Adolfi, F., Sigman, M., Marino, J., Manes, F., & Ibanez, A. (2015). Predictive coding in autism spectrum disorder and attention deficit hyperactivity disorder. *Journal of Neurophysiology, 114*, 2625–2636. https://doi.org/10.1152/jn.00543.2015
Hutto D, & Myin E. (2013). *Radicalizing enactivism: Basic minds without content*. MIT Press.
Janssen, T. W. P., Hillebrand, A., Gouw, A., Geladé, K., Van Mourik, R., Maras, A., & Oosterlaan, J. (2017). Neural network topology in ADHD; evidence for maturational delay and default-mode network alterations. *Clinical Neurophysiology, 128*(11), 2258–2267. https://doi.org/10.1016/j.clinph.2017.09.004
Kirchhoff, M. D. (2015). Extended cognition & the causal-constitutive fallacy: In search for a diachronic and dynamical conception of constitution. *Philosophy and Phenomenological Research, 90*(2), 320–360.
Kirchhoff, M.D., & Kiverstein, J. (2019). *Extended consciousness and predictive processing: A third-wave view*. Routledge.
Loe, I. M., & Feldman, H. M. (2007). Academic and educational outcomes of children with ADHD. *Ournal of Pediatric Psychology, 32*(6), 643–654.
Menon, V. (2012). Functional connectivity, neurocognitive networks, and brain dynamics. In M. I. Rabinovich, K. J. Friston, & P. Varona (Eds.), *Principles of brain dynamics* (pp. 27–48). MIT Press.
Mizuhara, H., & Yamaguchi, Y. (2007). Human cortical circuits for central executive function emerge by theta phase synchronization. *NeuroImage, 36*(1), 232–244.
Noreika, V., Falter, C. M., & Rubia, K. (2013). Timing deficits in attention-deficit/hyperactivity disorder (ADHD): Evidence from neurocognitive and neuroimaging studies. *Neuropsychologia, 51*(2), 235–266.
Pezzulo, G. (2008). Coordinating with the future: The anticipatory nature of representation. *Minds & Machines, 18*, 179–225.
Pliszka, S.R. (2015). Comorbid psychiatric disorders in children with ADHD. In R.A. Barkley (Ed.), *Attention-deficit hyperactivity disorder: A handbook for diagnosis and treatment* (4th ed., pp. 140–168). The Guilford Press.
Polanczyk, G., de Lima, M. S., Horta, B. L., Biederman, J., & Rohde, L. A. (2007). The worldwide prevalence of ADHD: A systematic review and metaregression analysis. *American Journal of Psychiatry, 164*(6), 942–948.
Polanczyk, G., Willcutt, E. G., Salum, G. A., Kieling, C., & Rohde, L. A. (2014). ADHD prevalence estimates across three decades: An updated systematic review and meta-regression analysis. *International Journal of Epidemiology, 43*(2), 434–442.
Proal, E., Reiss, P. T., Klein, R. G., Mannuzza, S., Gotimer, K., Ramos-Olazagasti, M. A., Lerch, J. P., He, Y., Zijdenbos, A., Kelly, C., Milham, M. P., & Castellanos, F. X. (2011). Brain gray matter deficits at 33-year follow-up in adults with attention-deficit/hyperactivity disorder established in childhood. *Archives of General Psychiatry, 68*(11), 1122–1134.
American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders (5th ed.)*. https://doi.org/10.1176/appi.books.9780890425596
Qian, X., Castellanos, F.X., Uddin, L.Q., Loo, B.R.Y., Liu, S., Koh, L.H., Poh, X.W.W., Fung, D., Guan, C., Lee, T.S., Lim, C.G., & Zhou, J. (2019). Large-scale brain functional network topology disruptions underlie symptom heterogeneity in children with attention-deficit/hyperactivity disorder. *NeuroImage: Clinical*, 21, 101600.
Remschmidt, H. (2005). Global consensus on ADHD/HKD. *European Child & Adolescent Psychiatry, 14*(3), 127–137. https://doi.org/10.1007/s00787-005-0439-x PMID: 15959658.
Roberts, W., Milich, R., & Barkley, R.A. (2015). Primary symptoms, diagnostic criteria, subtyping, and prevalence of ADHD. In R.A. Barkley (Ed.), *Attention-deficit hyperactivity disorder: A handbook for diagnosis and treatment* (4th ed., pp. 51–80). The Guilford Press.
Rosqvist, H. B., Chown, N., & Stenning, A. (Eds.). (2020). *Neurodiversity studies: A new critical paradigm*. Routledge.
Rubia, K. (2006). The neural correlates of timing functions. In J. Blicksohn & S. Myslobodsky (Eds.), *Timing the future: The case for a time-based prospective memory* (pp. 231–238). World Scientific Publishing Company.
Smith, A., Taylor, E., Warner Rogers, J., Newman, S., & Rubia, K. (2002). Evidence for a pure time perception deficit in children with ADHD. *Journal of Child Psychology and Psychiatry, 43*(4), 529–542.
Solanto, M.V. (2015). Executive function deficits in adults with ADHD. In R.A. Barkley (Ed.), *Attention-deficit hyperactivity disorder: A handbook for diagnosis and treatment* (4th ed., pp. 256–266). The Guilford Press.
Spivey, M. (2008). *The continuity of mind*. Oxford University Press.
Sripada, C. S., Kessler, D., & Angstadt, M. (2014). Lag in maturation of the brain's intrinsic functional architecture in attention-deficit/hyperactivity disorder. *Proceedings of the National Academy of Sciences of the United States of America, 111*(39), 14259–14264.
Stepp, N., & Turvey, M. T. (2010). On strong anticipation. *Cognitive Systems Research, 11*(2), 148–164.
Swanson, J. M., Sergeant, J. A., Taylor, E., Sonuga-Barke, E. J., Jensen, P. S., & Cantwell, D. P. (1998). Attention-deficit hyperactivity disorder and hyperkinetic disorder. *Lancet, 351*(9100), 429–433.
Toplak, M. E., West, R. F., & Stanovich, K. E. (2013). Practitioner review: Do performance-based measures and ratings of executive function assess the same construct? *Journal of Child Psychology and Psychiatry, 54*(2), 131–143.
Vaidya, C. J. (2011). Neurodevelopmental abnormalities in ADHD. In C. Stanford & R. Tannock (Eds.), *Behavioral neuroscience of Attention Deficit Hyperactivity Disorder and its treatment* (pp. 49–66). Springer.
Varela, F. J. (1999). The specious present: A neurophenomenology of time consciousness. In J. Petitot, F. J. Varela, B. Pachoud, & J.-M. Roy (Eds.), *Naturalizing phenomenology: Issues in contemporary phenomenology and cognitive science* (pp. 266–314). Stanford University Press.
Varela, F. J., Lachaux, J. P., Rodriguez, E., & Martineire, J. (2001). The brainweb: Phase-synchronization and long-range integration. *Nature Reviews Neuroscience, 2*, 229–239.
Wåhlstedt, C., Thorell, L. B., & Bohlin, G. (2009). Heterogeneity in ADHD: Neuropsychological pathways, comorbidity and symptom domains. *Journal Abnormal Child Psychology, 37*(4), 551–564.

Weyandt, L.L., Gudmundsdottir, B.G. (2015). Developmental and neuropsychological deficits in children with ADHD. In R.A. Barkley (Ed.), *Attention-deficit hyperactivity disorder: A handbook for diagnosis and treatment* (4th ed., pp. 116–139). The Guilford Press.

Willcutt, E. G., Doyle, A. E., Nigg, J. T., Faraone, S. V., & Pennington, B. F. (2005). Validity of the executive function theory of attention-deficit/hyperactivity disorder: A meta-analytic review. *Biological Psychiatry, 57*(11), 1336–1346.

Willcutt, E.G. (2015). Theories of ADHD. In R.A. Barkley (Ed.), *Attention-deficit hyperactivity disorder: A handbook for diagnosis and treatment* (4th ed., pp. 391–403). The Guilford Press.