Variability, quality, and timing: An investigation of the interplay between sleep, cognitive control, and symptoms of attention-deficit/hyperactivity disorder

Ingrid Holmen

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Summary

Author: Ingrid Holmen

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Author statement: This master thesis used data from a pilot study on sleep, affect, and cognitive control, and the design was developed before the author joined the research group. The data were collected by former master's students and a PhD student. The hypotheses and the aim of the thesis were developed by the author in collaboration with supervisors Virginia Conde Ruiz and Margrethe Hansen. Data processing, statistical analyses, and write-up was performed by the author independently.

Supervisor: Virginia Conde Ruiz

Co-supervisors: Margrethe Hansen

Abstract

Sleep has been extensively studied, but its role in cognition remains poorly understood. Research points to a particular interplay between intraindividual variability (IIV) in sleep, chronotype, cognitive control, and symptoms of attention-deficit/hyperactivity disorder (ADHD). Previous findings indicate that cognitive control processes are differentially affected by sleep deprivation and restriction, but the influence of day-to-day variability in sleep is less known. Moreover, evening chronotypes have been associated with higher IIV in sleep and an individual's chronotype can partly explain differences in cognitive performance throughout the day. Lastly, ADHD symptoms have been associated with both cognitive control deficits, higher IIV in sleep, and evening chronotypes. The purpose of this study was to investigate this interplay in a sample of healthy adults. Actigraphy and sleep diaries from 39 participants were used to estimate measures of sleep variability, sleep quality, and sleep timing in a two-week period, while measures of ADHD symptoms were derived from a self-report questionnaire. Performance was estimated for three components of cognitive control with a stop-signal task, a task-switching task, and an n-back task. The study found that evening chronotype and ADHD symptoms were associated with lower sleep quality, but not with each other. Contrary to previous findings, no interaction was observed between the participants' chronotypes and times of testing for cognitive control performance. Post-hoc results indicated that the inattention dimension of ADHD symptomology was associated with lower performance in the task-switching task, but the measure of total ADHD symptoms was not associated with performance on any of the three cognitive control tasks. The study did not find an association

between IIV in sleep and cognitive control performance, but research in larger samples is needed to detect small-magnitude effects. This study serves as an example of how IIV in sleep can be investigated with high ecological validity, and it highlights the need to consider both variability, quality, and timing in investigations of sleep and cognition.

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1. Introduction

Everyone knows that sleep is important for our ability to think; we all have experienced what a lack of it does to our well-being. Sleep affects our mood, our concentration, our memory, our impulse control, our decision making, and many more psychological processes (Alhola & Polo-Kantola, 2007; Killgore, 2010; Monk, 2012; Reynolds & Banks, 2010). On average, we spend one third of our whole life asleep or attempting to sleep (Aminoff et al., 2011), but despite our impressive amount of lived experience, our understanding of sleep is nowhere close to being complete. The biological and psychological purposes of sleep are still under debate in the scientific fields, and it remains unclear how exactly sleep affects the brain and its psychological processes and functions.

One psychological function that has received attention for its complex relationship with sleep is cognitive control. As the name suggests, cognitive control is thought to be the function that allows us to flexibly control how we use our cognition. Research has shown that cognitive control is affected by sleep, but not necessarily in a global and uniform manner (Gevers et al., 2015; Killgore, 2010; Kusztor et al., 2019; Satterfield & Killgore, 2019). Moreover, sleep has also been found to be implicated in attention-deficit/hyperactivity disorder (ADHD), which is a neurodevelopmental disorder that again has connections to cognitive control (Boonstra et al., 2005; Nigg, 2001). The exact nature of this interplay between sleep, cognitive control, and ADHD is poorly understood. Furthermore, the potential effects of day-to-day sleep variability on cognition have been largely overlooked in the research. As a response to these uncertainties, the current study aimed to investigate the interplay between intraindividual variability in sleep, cognitive control, and symptoms of ADHD in a healthy population sample.

1.1. Sleep

For a long time in human history, sleep was thought to be a passive activity during which the brain was ‘shut off’ from its active and alert state (Frank, 2012). However, characterising sleep as a mere pause from wakefulness is highly misleading; sleep is more accurately described as a state of physical calmness and altered vigilance, during which a multitude of physical and mental processes can take place (Harrison, 2012; Peigneux et al., 2012). Sleep has been proposed to serve a multitude of functions in the brain, such as repairing damaged DNA (Zada et al., 2021), removing potentially neurotoxic waste products that accumulate during wakefulness (Xie et al., 2013), and reorganising neurons for memory

consolidation (Siegel, 2001). However, a firm consensus on the function(s) of sleep has yet to be established and the implications of even the most commonly replicated research findings are still under debate (Harrison, 2012). Nevertheless, researchers generally agree that sleep is comprised of two distinct physiological stages that the body cycles between: non-rapid eye movement (NREM) and rapid eye movement (REM; Rowley & Badr, 2012).

The most obvious characteristic of sleep is that it alternates with wakefulness. Human beings and animals have a sleep-wake cycle that is regulated by two important physiological factors: sleep homeostasis and circadian rhythmicity (Dijk & Lazar, 2012). Sleep homeostasis is the balance between sleep and wake states, and it effectively keeps count of whether an organism is getting enough sleep (Dijk & Lazar, 2012). Circadian rhythmicity, also called the circadian clock, is the internal rhythm by which an organism determines the best timing for sleep and wakefulness (Dijk & Lazar, 2012; Roenneberg et al., 2003). In humans, the circadian rhythm is usually synchronised to a cycle of 24 hours by different zeitgebers (Roenneberg, Kuehnle, et al., 2007). Zeitgebers are external/environmental cues that entrain the body's biological rhythms, and light is the most important zeitgeber for the circadian rhythm (Roenneberg, Kumar, et al., 2007). The sleep-wake cycle gives rise to the diurnal behaviour pattern of humans, in which activity is largely preferred to take place during the day and sleep during the night. However, the human sleep-wake cycle is far from fixed; individual differences in sleep homeostasis, circadian rhythm, health, personality, and social lives produce both interindividual and intraindividual variability in sleep (Bei et al., 2016).

The current recommendation for members of the general population is to sleep 7 hours or more every night (N. F. Watson et al., 2015). A sleep duration of less than 7 hours is labelled sleep restriction or partial sleep deprivation, and when it occurs consistently over longer periods of time it is considered chronic (Banks & Dinges, 2007; Monk, 2012; Reynolds & Banks, 2010). Importantly, sleep restriction has been found to be detrimental to both physical and mental health (Banks & Dinges, 2007; Tai et al., 2022; N. F. Watson et al., 2015) and chronic sleep restriction produces cognitive deficits that are highly similar to the deficits produced by total sleep deprivation (the loss of a full night's sleep; Reynolds & Banks, 2010).

1.1.1. Interindividual variability in sleep

From our everyday lives we are familiar with how some people are morning 'larks' while others are evening 'owls', and how some people seem to need more sleep than others to function well. In addition to these common phenotypes, sleep disturbances such as insomnia

and restless legs syndrome are also examples of interindividual variability in sleep (Espie & Morin, 2012). In fact, interindividual variability can be found in almost every quantifiable and qualitative aspect of sleep. Some of the most notable sleep variables that are studied in sleep research include sleep duration (frequently called total sleep time; TST) sleep quality (SQ), sleep onset latency, bedtime, wake-up time, sleep efficiency (number of awakenings, also called sleep continuity or fragmentation), and duration of awakenings (Ibáñez et al., 2018). The most common methods for measuring and estimating these variables are clinical interviews, questionnaires, sleep diaries, actigraphy (estimation of sleep based on movement activity), and polysomnography (PSG; Ibáñez et al., 2018). The three former methods are subjective methods of measurement, while the two latter are objective. PSG is arguably the gold-standard because it combines several recording methods to give a comprehensive view of bodily functions during sleep, such as brain activity, eye movements, muscle activity, and heart rhythm (Ibáñez et al., 2018). However, actigraphy and sleep diaries alter the sleeper's normal sleep environment to a much lesser degree (Iber et al., 2004), which makes them less intrusive and much more practical and cost-efficient compared to PSG. Actigraphy and sleep diaries have thus become the most common methods of sleep assessment in research conducted on large samples and over long periods of time (Girschik et al., 2012; Ibáñez et al., 2018).

An aspect of interindividual sleep variability that has received special research attention is the natural preference for timing of the sleep-wake cycle. This interindividual preference for timing is related to the so-called 'chronotype' or phase of entrainment (Roenneberg et al., 2003), and it reflects the specific temporal relationships an individual has to zeitgebers, i.e. how their biological rhythms are aligned with external cues (Roenneberg et al., 2003; Roenneberg, Kuehnle, et al., 2007). Chronotype is most frequently estimated as a behavioural tendency towards 'morningness' or 'eveningness' using questionnaires such as the Horne-Östberg Morningness-Eveningness Questionnaire (MEQ; Horne & Östberg, 1976) or the Munich Chronotype Questionnaire (MCTQ; Roenneberg et al., 2003). The MEQ is mostly a subjective measure of chronotype in that it addresses the individual's personal feeling of what the best rhythm of sleep and activity is for them, while the MCTQ is a somewhat more objective measure in that it addresses the specific timings of sleep and activity. Importantly, both measures correlate with key aspects of the sleep-wake cycle, such as sleep onset (SO) and wake-up time (Zavada et al., 2009). Another way to estimate chronotype is to observe an individual's actual sleep and activity habits over time and define

their chronotype based on e.g., the sleep midpoint (SMid) between SO and wake-up time on days when the individual can sleep without constraints (Santisteban et al., 2018).

Interindividual variability in sleep has been consistently linked with interindividual aspects such as age (Ohayon et al., 2004), lifestyle (Dzierzewski et al., 2021), physical and mental health (Baglioni et al., 2016), and occupation and occupational status (Åkerstedt & Kecklund, 2012). The relationships between sleep and these factors seem to be complex and, in many cases, bidirectional.

1.1.2. Intraindividual variability in sleep

In contrast to interindividual variability and intraindividual means (also called individual means; IM), intraindividual variability (IIV) in sleep have received less attention in the research on sleep and its effects (Bei, Seeman, et al., 2017; Shoji et al., 2015; Wiley et al., 2014). Nevertheless, the existence of IIV in sleep is obvious to all those who have experienced a bad night of sleep and desperately looked forward to catching up on it the next night. The same aspects of sleep that vary between individuals can and will also vary within individuals across time, and despite the lack of attention, IIV in sleep is not insignificant (Messman et al., 2021). For example, research shows that the IIV in sleep variables such as duration and fragmentation can be higher than the interindividual variability between IM (Knutson et al., 2007; Mezick et al., 2009; van Hilten et al., 1993).

IIV in sleep is frequently estimated by calculating intraindividual standard deviations (ISD; standard deviation between datapoints belonging to a single person), but it has also been estimated with Bayesian modeling approaches to reduce measurement error (Wiley et al., 2014). Importantly, IIV in sleep is commonly observed alongside sleep restriction and circadian misalignments (i.e. sleeping outside of preferred sleep timing; Costa, 1996; van Leeuwen et al., 2009). In a systematic review (Bei et al., 2016), higher IIV in sleep was found to be associated with an evening chronotype, weight gain, higher body mass index, younger age, physical health conditions, depression symptoms, and stress. In university students, late chronotypes have been associated with higher IIV in sleep measures such as bed time and wake time (Roane et al., 2014), and in insomnia patients it has been associated with higher IIV in bed time and time spent in bed (Bei et al., 2016). The relationship between chronotypes and IIV in sleep is likely to be partly explained by how society is largely structured in ways that suit individuals with earlier chronotypes better (Wittmann et al., 2006).

Although day-to-day variability is an important and unavoidable aspect of sleep, the relationship between IIV in sleep and cognition is far less understood than other aspects of sleep (Bei et al., 2016).

1.2. Cognitive control

The concept of cognitive control refers to a set of high-level cognitive processes that allow an individual to regulate their own thoughts and behaviours in a goal-directed manner (Gazzaniga, 2014). Cognitive control is hypothesised to encompass cognitive abilities such as inhibition, working memory, task-switching, planning, self-monitoring, and self-regulation (Goldstein et al., 2014; Sabhlok et al., 2022). These cognitive control processes, also known as executive functions, exert influence over lower-level processes in the brain like attention and memory, and they are some of the main processes associated with the prefrontal cortex (PFC; Gazzaniga, 2014; Miller & Cohen, 2003; Otero & Barker, 2014). Researchers have suggested that cognitive control originates from representations of task- and goal-relevant information in the PFC (Yeung, 2013). This information can provide bias signals to other brain structures and guide the brain's processing of inputs, internal states, and outputs (Miller & Cohen, 2003). Consequently, cognitive control allows for the flexible processing of information that is key for evolutionary important, high-level cognitive abilities such as planning and monitoring behaviour (Friedman & Miyake, 2017).

Cognitive control is measured and studied using a wide variety of scales, tasks, and batteries. Behaviour rating scales, like the Behavior Rating Inventory of Executive Function (BRIEF; Gioia et al., 2000), have been designed to assess behavioural manifestations of cognitive control and require the individual (or a knowledgeable informant) to rate their own behaviour (Naglieri & Goldstein, 2014). Similarly, self-report scales that more directly address internal cognitive control processes have also been developed. Such self-report scales require the individual to judge their agreement with items such as 'I say things without thinking' and 'I have trouble making decisions' (Nęcka et al., 2012). In research contexts, behavioural tasks and paradigms that target specific or multiple cognitive processes are often preferred. For example, the famous Stroop task (Stroop, 1935) is frequently used to measure inhibition. The Stroop task was designed to make an individual deal with cognitive interference by having them say the colour of the ink in which a colour-name is printed (such as 'red' printed in blue ink) and not the colour-name itself. More complex cognitive tasks such as the Wisconsin Card Sorting Task (WCST; Heaton, 1981), which has the individual classify cards based on different criteria that change over time, can provide a broad measure

of several cognitive control processes (Nęcka et al., 2012). Given the multifaceted nature of cognitive control, test batteries with multiple scales and tasks have also been created in order to measure cognitive control as a broader concept (Naglieri & Goldstein, 2014). However, the validity of cognitive control scales, tasks, and batteries heavily rely on how cognitive control is conceptualised.

1.2.1. Cognitive control processes and frameworks

Despite how far-reaching cognitive control functions are as a whole when studying different aspects of cognition, splitting the general concept of cognitive control into separate and distinct processes has proved challenging. At its time of origin in the 1970s, the term cognitive control was used to describe an executive branch of the attentional system which was responsible for the ability to selectively attend to specific aspects of the environment (Goldstein et al., 2014). Around the same time, Alan Baddeley introduced his model of working memory in which a similar executive component, called the 'central executive', was responsible for information being manipulated in working memory (Baddeley & Hitch, 1974). In the decades since then, the concept of cognitive control has expanded to include many more functions and processes, including verbal reasoning, problem-solving, planning, sequencing, sustained attention, interference control, utilization of feedback, multitasking, and cognitive flexibility (Chan et al., 2008). The commonality amongst these functions is the involvement of internal regulation or control that allows for goal-directed behaviour. The degree to which cognitive control processes interact and share characteristics and how they function on a neurological basis are central questions in current research, and a consensus on how many distinct processes there are and how they are organised has yet to be established (Yeung, 2013).

Nonetheless, one framework of cognitive control that has become popular over the last two decades is the 'unity and diversity' framework proposed by Friedman & Miyake (2017; Miyake et al., 2000). This framework was developed via confirmatory factor analysis approach with the aim to identify commonalities between cognitive control tasks and processes commonly discussed in the cognitive control literature (Friedman & Miyake, 2017). The framework accounts for both common underlying aspects of all cognitive control processes and three distinct core cognitive control components: inhibiting, shifting, and updating. The component of inhibiting is thought to reflect the ability to stop prepotent or automatic responses, whereas the component of shifting is thought to reflect the ability to switch between different tasks/rules. Finally, the component of updating is thought to reflect

the ability to keep and manipulate information in working memory. When a general cognitive control component is included in the framework, all the variance in the inhibiting component is explained by the general component. On the other hand, both the shifting and updating components have additional variance that is too specific to shifting or updating to be explained by the general component (Friedman & Miyake, 2017).

Inhibiting performance is commonly estimated using paradigms such as the stop-signal task (Lappin & Eriksen, 2006; Logan et al., 1984), in which participants must respond when they see a go-signal and refrain from responding (i.e. inhibit the response) if the go-signal is followed by a stop-signal (Verbruggen et al., 2019). Shifting performance is commonly estimated using task-switching tasks (Biederman, 1972), in which participants must flexibly alternate between following two different rulesets while responding to a stream of stimuli. Finally, updating performance is commonly estimated using paradigms such as the n-back task (Kirchner, 1959), in which participants must keep items in their working memory while new items are presented and determine whether the new item matches the item presented n items back (Soveri et al., 2017; Szmalec et al., 2011).

In the literature, the three components of Friedman & Miyake's framework also go by the names inhibitory control, cognitive flexibility, and working memory (Diamond, 2013). The three components have been shown to be moderately correlated with each other, but still separable (Miyake et al., 2000) and have been replicated numerous times (e.g., Fisk & Sharp, 2004; Hull, Martin, Beier, Lane, & Hamilton, 2008; Vaughan, & Giovanello, 2010; Willcutt et al., 2001). However, Friedman and Miyake's framework is not universally agreed upon. Research has also found evidence for a fourth component called conflict monitoring (Egner & Hirsch, 2005; Kane & Engle, 2003; Kerns et al., 2004), which is thought to reflect the ability to monitor and detect conflicts or interference in information processing (Botvinick et al., 2001).

1.2.2. Cognitive control in attention-deficit/hyperactivity disorder

A subject that is frequently discussed alongside cognitive control is attention-deficit/hyperactivity disorder (ADHD). ADHD is the most common neurodevelopmental disorder and, as the name suggests, it is characterised by markedly increased levels of inattention and/or hyperactivity/impulsivity (Franke et al., 2018). Symptoms of ADHD include impulsiveness, disorganisation, restlessness, and problems with focusing and completing tasks (Surman, 2013). Although ADHD historically has mostly been diagnosed in young children, it is considered a chronic disorder that persists into adulthood (Surman, 2013)

and research indicates that between 2.5 and 5% of the adult population has ADHD (Ginsberg et al., 2014; Simon et al., 2009; Steinhausen, 2009). Since the research on ADHD has been heavily skewed towards children and adolescents much less is known about the developmental trajectory and nature of ADHD in adulthood. However, ADHD has been associated with a wide range of negative life outcomes, such as poor academic performance, unemployment, financial problems, higher mortality, and divorce (Franke et al., 2018). The precise origin and neurological nature of ADHD is still under debate; no single risk factor can explain ADHD, but potential causal factors include genes, pre- and perinatal risks, and psychosocial and environmental factors (Steinhausen, 2009; Thapar et al., 2013).

One of the most commonly replicated research findings in the ADHD literature is that individuals with ADHD have impaired cognitive control (Antshel et al., 2014; Boonstra et al., 2005; Nigg et al., 2005; Pennington & Ozonoff, 1996; Willcutt et al., 2005). For example, ADHD has been associated with poor performance relative to healthy controls in tasks associated with in response inhibition (Schachar et al., 2000), task-switching (Luna-Rodriguez et al., 2018), and working memory (McInnes et al., 2003). Moreover, two meta-analyses (Pennington & Ozonoff, 1996; Willcutt et al., 2005) have reported that ADHD is associated with weaknesses in a large set of cognitive control tasks, with the strongest and most consistent effects being found in tasks that involve inhibition, working memory, and planning.

Consequently, ADHD has frequently been described as a disorder of cognitive control, and several researchers have even suggested that a primary deficit in cognitive control is the cause of the core ADHD symptoms (Barkley, 1997; Castellanos & Tannock, 2002; Schachar et al., 2000). According to this hypothesis, impairments to fundamental cognitive control processes lead to deficits in higher-level cognition and consequently become the origin of the symptomatology of hyperactivity, distractibility, and impulsivity that characterises ADHD. A main argument to support this hypothesis is that lesions to the PFC, the designated ‘seat’ of cognitive control, can lead to behavioural symptoms that are very similar to ADHD symptomatology (Carlin et al., 2000; Fuster, 2015; Miller & Cohen, 2003). However, there are also research findings that cannot be explained by this causal hypothesis. For example, the observed variability in ADHD symptoms is much larger than the observed variability in cognitive control performance and although correlations between symptoms and performance are significant, they are rarely large in magnitude (Willcutt et al., 2005).

Moreover, other researchers have hypothesised that a cognitive control deficit could be the distinguishing factor between different subtypes of ADHD rather than the primary causal factor of the disorder (Chhabildas et al., 2001; Nigg, 2001). ADHD is a relatively

heterogeneous disorder and research suggests it has two subtypes characterised by two distinct symptom dimensions: inattention and hyperactivity/impulsivity (Willcutt et al., 2012). Studies show that poor cognitive control performance is mainly associated with the inattention subtype and not with the hyperactivity/impulsivity subtype (Brocki et al., 2010; Chhabildas et al., 2001; Martel et al., 2011; Nigg et al., 2005; Sabhlok et al., 2022). Additionally, cognitive control deficits are more strongly associated with covert inattention symptoms such as forgetfulness, distractedness, and trouble with organization than overt inattention symptoms such as inappropriate talkativeness and interruption (Sabhlok et al., 2022). In summary, if a central cognitive control deficit has causal role in ADHD like researchers have suggested, it would seem that such a deficit has stronger links to inattention than hyperactivity/impulsivity. However, evidence from the current literature does not give a clear image of the causal relationship between ADHD and cognitive control deficits and it is not possible to rule out bidirectional effects (Willcutt et al., 2005).

Importantly, associations between ADHD symptoms and poor cognitive control performance have also been found in non-clinical samples of healthy adults (Arabacı & Parris, 2020; Das et al., 2015; Elisa et al., 2016; Silverstein et al., 2020). However, as few studies on this subject have been conducted, the relationship between mild and/or subclinical ADHD symptoms and cognitive control performance is still unclear. For example, findings are mixed when it comes to cognitive control differences between the two ADHD symptom dimensions of inattention and hyperactivity/impulsivity (Das et al., 2015; Silverstein et al., 2020).

1.3. The relationship between sleep and cognitive control

A poor night's sleep has immediate effects on our ability to focus and think clearly, and there is much research to show how wide-ranging the effects of poor sleep, sleep restriction, and sleep deprivation are (Killgore, 2010). For example, total sleep deprivation is detrimental to attention, working memory, long-term memory, and decision-making (Alhola & Polo-Kantola, 2007). The PFC may be particularly susceptible to sleep deprivation effects compared to other brain regions (Killgore, 2010) and cognitive control performance has also been found to be impaired by sleep deprivation (Gosselin et al., 2005; Martella et al., 2011; Nilsson et al., 2005; Qi et al., 2010; Tsai et al., 2005). Research indicates that more basic cognitive functions, such as cognitive control processes, are more vulnerable to the negative effects of sleep deprivation than more complex cognitive functions, such as judgement and decision-making (Harrison & Horne, 2000; Lim, 2010; Wickens et al., 2015). However, there

are also findings that indicate that cognitive control processes are differentially affected by sleep deprivation (Jackson et al., 2013; Killgore, 2010). More automatic processes, such as response inhibition and performance monitoring, seem to be more resilient to negative effects than processes that are more cognitively demanding, such as information integration (Gevers et al., 2015; Kusztor et al., 2019). This view is supported by research on attention, which has found that negative effects following sleep deprivation can be attributed to impairments in the dynamic allocation of attention rather than impairments in simple sustained attention (Chua et al., 2017; Honn et al., 2019; Whitney et al., 2017).

Total sleep deprivation is not the only way sleep can affect cognition, however; other important aspects of sleep that have been found to relate to cognition in healthy people are sleep restriction, SQ and sleep efficiency. Importantly, short term sleep restriction has significant negative effects on cognition (Lowe et al., 2017). For example, Saksvik-Lehouillier et al. (2020) found that sleep restriction negatively influenced reaction time and accuracy on a cognitive control task, while Lo et al. (2016) found that several nights of sleep restriction gradually deteriorated sustained attention, working memory, and cognitive control. Furthermore, poor self-reported SQ has been associated with impaired cognitive control performance (de Almondes et al., 2016; Nebes et al., 2009), and higher sleep continuity, i.e. fewer awakenings during sleep, is associated with improved cognitive control performance (Wilckens et al., 2014).

1.3.1. The effects of intraindividual sleep variability on affect and cognition

Although the primary focus in sleep and cognition research has been sleep deprivation and IM, a growing body of findings shows that it is highly necessary to also consider IIV in sleep. Most of these studies have focused on affect rather than cognition. For example, Bei et al. (2017) measured the sleep of 146 adolescents using actigraphy during a 15-day vacation. Since it was a vacation-period, the adolescents were relatively free to choose when and for how long they wanted to sleep. When comparing the actigraphy sleep measures to a variety of measures from questionnaires, the authors found that higher IIV in sleep latency and in time spent in bed were associated with more negative mood. This association was mediated by the adolescents perceived SQ, which suggests that higher IIV in sleep can lead to lower SQ and consequently more negative mood. Additionally, the effects of the IIV measures were larger in magnitude than the effects found for the IM measures. Similar associations between IIV in sleep, SQ, and negative mood have also been found for adolescents and adults during more constrained sleep periods (Bei et al., 2016; Fuligni & Hardway, 2006). Another study by

Lemola et al. (2013) found that higher sleep duration IIV was related to poorer subjective well-being. Similar to the previously stated findings, this study also found that this relationship was partially mediated by subjective SQ and that sleep duration IM had no significant effect.

It is fair to assume that lowered mood and well-being could influence cognitive performance, but not much is known about the direct relationship between IIV in sleep and cognitive performance as such. Although cognitively demanding attentional processes have previously been found to be negatively affected by sleep deprivation, Barclay et al. (2020) found that neither IM nor IIV in sleep duration significantly predicted performance in an attention task with several different outcomes measures. To the author's knowledge, no studies have investigated IIV in sleep and cognitive control.

1.3.2. The effects of chronotype on cognition

The research on circadian rhythms to date also suggests that individual chronotypes are an important factor in understanding cognitive performance (Goel et al., 2013; Schmidt et al., 2007). The chronotype denotes an individual's natural preferred timing for sleep as well as their preferred timing of activity during the day (Roenneberg et al., 2003). Research has shown that cognitive performance varies throughout the day (Holding et al., 2021; Matchock & Toby Mordkoff, 2009; Ngo et al., 2018), and chronotype is likely to influence when an individual will be in their optimal state to perform well (Schmidt et al., 2007). For example, Facer-Childs et al. (2018) found that individuals with earlier chronotypes had their optimal performance window shortly after their entrained wake-up time, whereas individuals with later chronotypes had their optimal performance window approximately 5-7 hours after their entrained wake-up time. When tested in the morning, individuals with later chronotypes performed significantly worse than individuals with earlier chronotypes on both a sustained attention test and in a cognitive control task that targeted working memory. Salehinejad et al. (2021) replicated this finding by showing that motor learning and cognitive control performance (measured in an n-back task and a Stroop task) was higher when participants were tested at a time aligned with their chronotype compared to a time that was non-aligned, i.e., the evening for morning chronotypes and vice versa.

Similar misalignment effects between chronotype and cognitive performance have also been found by Chellappa et al. (2018) and Martínez-Pérez et al. (2020); in both studies, individuals performed worse on a sustained attention task when the testing took place during a non-optimal time of day. Additionally, Chellappa et al. (2018) found that training

improvements to cognitive throughput, information processing, and visual-motor performance were lost when training took place during non-optimal times of the day. Moreover, Martínez-Pérez et al. (2020) found that optimal test-time had a significant effect on performance in an inhibition task, but when dividing the sample by chronotypes, this effect only remained significant for the evening-chronotypes group and not the morning-chronotypes group.

1.3.3. The relationship between attention-deficit/hyperactivity disorder, intraindividual sleep variability, and chronotype

Although our understanding of the relationship between IIV in sleep, chronotype, and cognitive control is incomplete, there is a large amount of evidence for the involvement and convergence of all three concepts in ADHD. As previously explained, cognitive control deficits are commonly found in individuals with ADHD, but another well-established finding is that individuals with ADHD have abnormal sleep patterns (Coogan & McGowan, 2017; Hvolby, 2014). ADHD has been consistently associated with delayed circadian rhythmicity and later/evening chronotypes (Coogan & McGowan, 2017; Durmuş et al., 2017). In line with this, ADHD is associated with longer sleep latency, lower sleep efficiency, and later wake-up time (Snitselaar et al., 2013; van Veen et al., 2010; Ziegler et al., 2021). Furthermore, both children, adolescents, and adults with ADHD have higher IIV in their sleep patterns compared to control groups (Langberg et al., 2019; van Veen et al., 2010; Ziegler et al., 2021).

Research on interventions and biologicals markers suggest that the predisposition towards eveningness in ADHD is caused by facets of inner, biological time-keeping rather than social or entrained time-aspects (Coogan & McGowan, 2017; Korman et al., 2017; McGowan et al., 2016). A study by Sandra Kooij & Bijlenga (2014) found that adults with ADHD have higher sensitivity to light, which could suggest that ADHD involves changes to the photoreceptive system that in turn affect circadian rhythmicity. Moreover, researchers have suggested that disruptions to the circadian rhythm could be a subserving factor to the symptomology of ADHD and that shifting misaligned circadian rhythms in the right direction could lessen symptom severity (Baird et al., 2012; Hvolby, 2014; Korman et al., 2018).

Importantly, the interplay between sleep, chronotype, and ADHD is not only relevant in clinical populations; recent research has found significant associations between sleep, chronotype, and ADHD symptoms in healthy young adults as well (McGowan et al., 2016).

1.4. The current study

As outlined in the previous sections, one of the major gaps in our understanding of sleep and cognition concerns the interplay between IIV in sleep, chronotype, and cognitive control. This interplay, moreover, seems to be relevant in ADHD and has been shown to play a role in the presence and severity of ADHD symptoms in healthy young adults as well (McGowan et al., 2016). Research has shown that cognitive control processes could be differentially affected by sleep (Gevers et al., 2015; Kusztor et al., 2019), but the influence day-to-day sleep variability is less known. On the other hand, chronotype can partly explain observed differences in cognitive performance throughout the day (Schmidt et al., 2007; Valdez et al., 2012) and later/evening chronotypes are associated with higher IIV in sleep (Bei et al., 2016). In relation to ADHD symptoms, there seems to be a particular co-occurrence of cognitive control deficits together with higher IIV in sleep and later/evening chronotypes (Das et al., 2015; McGowan et al., 2016).

The aim of the current study was to explore the relationship between these concepts in a healthy population sample and to investigate how IIV in sleep could affect the three core cognitive control components outlined by the unity and diversity framework (inhibiting, updating, and shifting; Friedman & Miyake, 2017; Miyake et al., 2000). To address these aims, the current study utilised measures of sleep from actigraphy and sleep diaries, measures of cognitive control performance from three computerised tasks, and measures of ADHD symptoms derived from a self-report questionnaire (the Attention Deficit/Hyperactivity Problems scales from Achenbach System of Empirically Based Assessment – Adult Self-Report). Based on these measures, the current study had 5 hypotheses:

H1. 'Later/evening chronotypes will have higher intraindividual variability in total sleep time and lower average sleep quality compared to earlier/morning chronotypes.' This hypothesis is based on the previous findings that individuals with later/evening chronotypes have higher IIV in sleep (Bei et al., 2016) and lower SQ (Juda et al., 2013; Tokur-Kesgin & Kocoglu-Tanyer, 2021) compared to individuals with earlier/morning chronotypes.

H2. 'ADHD symptoms will be associated with higher intraindividual variability in sleep measures, later/evening chronotype and lower average sleep quality.' This hypothesis is based on the previous findings that individuals with ADHD have higher IIV in sleep (Langberg et al., 2019; van Veen et al., 2010; Ziegler et al., 2021), later/evening chronotype (Coogan & McGowan, 2017; Durmuş et al., 2017), and lower SQ (Snitselaar et al., 2013; van Veen et al., 2010; Ziegler et al., 2021). Importantly, associations with chronotype and SQ also exist for ADHD symptoms in healthy adults (McGowan et al., 2016).

H3. 'Cognitive control performance will be lower for individuals with later/evening chronotypes tested in the morning, and vice versa for individuals with earlier/morning chronotypes.' This hypothesis is based on the previous findings that cognitive performance is affected by time of testing and chronotype (Chellappa et al., 2018; Facer-Childs et al., 2018; Martínez-Pérez et al., 2020; Salehinejad et al., 2021).

H4. 'ADHD symptoms will be associated with lower performance on all three cognitive control tasks.' This hypothesis was based on the previous findings that ADHD symptoms are strongly associated with cognitive control deficits (Das et al., 2015; Silverstein et al., 2020).

H5. 'Associations between sleep and cognitive control performance will be lower in the task measuring "inhibiting" than the tasks measuring "updating" and "shifting".' This hypothesis is based on the findings that cognitive control processes are differentially affected by sleep, with more automatic processes (inhibiting) being less affected than more demanding processes (updating and shifting; Chua et al., 2017; Gevers et al., 2015; Honn et al., 2019; Jackson et al., 2013; Kusztor et al., 2019; Whitney et al., 2017).

2. Materials and Methods

2.1. Participants

44 healthy adult participants were recruited for the current study via posters at the University of Oslo and online via social media. All participants had normal or corrected-to-normal vision and no history of psychological and neurological disorders. Participants provided their written consent after receiving a thorough explanation of the purpose and implications of the study. Participants were compensated with a 500NOK universal gift card upon completion of their last data collection session. One participant withdrew from the study, one was excluded due to missing sleep data, one was excluded due to sleep deprivation, and two were excluded because of intake of substances known to affect sleep (melatonin and alcohol). A total of 39 participants were included in the final analyses. The participants in the final sample were between the ages of 20 and 35 (M = 26.03, SD = 3.12), and 27 participants were female.

2.2. Design

The data utilised in the current study was collected as part of a pilot project investigating the relationship between sleep, affect, and cognitive control. This project collected data from participants in three sessions: two test-sessions for cognitive control tasks and questionnaires with electroencephalography (EEG) and electromyography (EMG) recordings, and one session for magnetic resonance imaging (MRI). The two test-sessions were spaced two weeks apart and contained the same cognitive tasks and questionnaires. Objective and subjective data used for sleep estimation were collected with actigraphy and sleep diaries in the two-week period between the test-sessions. The MRI session took place within this two-week period or shortly after. The current study analysed questionnaire data and behavioural data from cognitive control tasks collected in the first test-session, and sleep data collected in the two-week period between the test-sessions.

2.3. Materials

2.3.1. Cognitive control tasks

The three cognitive control tasks included in the current study were chosen to measure the three core components of cognitive control outlined by the unity and diversity framework: inhibiting, shifting, and updating (Friedman & Miyake, 2017; Miyake et al., 2000). Inhibiting performance was estimated with a stop-signal task (SST), shifting performance with a

number-letter task-switching task (NLTST), and updating performance with a n-back task (NBT).

The cognitive control tasks were conducted on a Dell Precision T5500 computer (Dell, Inc., Texas, USA). The tasks were programmed with PsychToolbox-3 (version 3.06.16) and presented using MATLAB (v2019.1). The task stimuli were presented on an EIZO FlexScan S2411W monitor (EIZO, Inc.) with a resolution of 1920x1200 and a refresh rate of 60 Hz. Participants' responses were recorded using a Cedrus SuperLab RB-740 response pad (Cedrus Corporation, 2006). To avoid potential training effects, the current study only used behavioural data from the cognitive control tasks collected in the first EEG session. MATLAB (v2021.2) was used to extract the variables of interest from the behavioural data.

2.3.1.1. Stop-signal task (SST). The SST consisted of five blocks with 68 go-trials and 22 stop-trials in random order (except for the first 10 trials of each block which were always go-trials). The task started with a short training block with 20 trials with feedback provided after each trial. The go- and stop-signal stimuli were blue and orange arrows indicating the direction (left or right) of the required go-response. Responses were made via button-presses with the left or right thumb depending on the direction of the stimuli. The colours of the go- and stop-signals were counterbalanced across participants; participants saw either a blue go-signal followed by an orange stop-signal or vice versa. The total number of trials for all five blocks was 450 and the probability of the stop-signal appearing after the go-signal was .24.

Each block began with a presentation of a black fixation cross with a duration that varied randomly between 700 and 1200 ms. In each trial, a go-stimulus (blue or orange arrow that pointed either left or right) was presented for 100 ms. Stop-trials were trials in which the go-stimulus was followed by a stop-stimulus. The stop-signal delay (SSD) had a range of 100 to 600 ms and was initially set to 250 ms. The SSD was adjusted according to a standard adaptive tracking procedure (described in Verbruggen et al. (2019)) so that it increased or decreased by 50 ms following successful and unsuccessful stops respectively. This approach to adjusting the SSD has previously been found to yield a stopping accuracy of approximately 50% (Rubia et al., 2003, 2007). The response window was 1000 ms from the presentation of the go-stimulus, and this was followed by an inter-trial interval with a black fixation cross that varied randomly between 700 and 1200 ms. After each block, participants received feedback on their performance in the block; if their average response time was longer than 600 ms they were instructed to be faster and if their stopping accuracy was below 40% they were instructed to be more accurate.

The primary inhibitory performance measure estimated from the SST in the current study was the stop-signal reaction time (SSRT). The SSRT is an estimate of response inhibition latency, and it was estimated using the integration method described by Verbruggen et al. (2019). SSRT was labelled inhibiting score in the analyses, and a higher inhibiting score was interpreted as a lower inhibiting performance.

2.3.1.2. Number-letter task-switching task (NLTST). The NLTST consisted of four blocks with 60 trials, in which participants had to repeat a ruleset in 160 trials and switch ruleset in 80 trials. The stimuli consisted of a combination of numbers and letters. The letters were five vowels and five consonants, and the numbers were odd and even numbers from 0 to 9. In each trial, a number and a letter were presented together on screen in one of two colours: blue or yellow. The task had a number condition and a letter condition. In the number condition participants were instructed to respond to the number presented, while in the letter condition they were instructed to respond to the letter presented. Each condition was assigned a colour so that when the stimuli were presented participants had to consider the colour of the stimuli to determine which condition they should respond according to. E.g., 'If the text is yellow: Press the left-most button for vowel and the right-most button for consonant, (A, E, I, Y, U) vs. (B, C, D, F, G).' and 'If the text is blue: Press the left-most button for even number and the right-most button for odd number, (0, 2, 4, 6, 8) vs. (1, 3, 5, 7, 9).'. A trial in which the stimuli colour was the same as in the previous trial was a repeat trial, while a trial in which the stimuli colour changed from the previous trial was a switch trial.

Each block began with a presentation of which colour corresponded to which ruleset and was followed by 15 practice trials in which participants should respond according to the instructions they had seen. In each trial, the stimuli were presented for 1200 ms and the response window was equal to the stimulus presentation. The inter-trial interval varied randomly between 1300 and 1700 ms. After each block, participants received feedback on their performance in the block; if their average response time was longer than 850 ms they were instructed to be faster and if their response accuracy was below 60% they were instructed to be more accurate.

The primary shifting performance measure estimated from the NLTST in the current study was the switch-cost. The switch-cost was the difference in mean reaction time between repeat trials and switch trials. The switch-cost was labelled shifting score in the analyses, and a higher shifting score was interpreted as a lower shifting performance.

2.3.1.3. N-back task. The NBT had two conditions: 1-back and 3-back. In both conditions, the stimuli were letter sequences that contained 25 random letters from A to Z presented on screen one by one. The task consisted of five blocks and each block had four letter sequences, two for each condition. In the 1-back condition participants had to respond when a letter was the same as the letter that was presented one letter ago, while in the 3-back condition they had to respond when a letter was the same as the letter that was presented three letters ago. For example, if the sequence was X – Y – Y – X participants should respond on the third letter in the 1-back condition and on the fourth letter in the 3-back condition. In both the 1-back and 3-back condition, each sequence of 25 letters had eight target letters that participants should respond to.

Each letter sequence began with the presentation of which condition participants should respond according to (e.g., 'Press the button when the letter on the screen is the same as the letter presented three letters before'). In each trial, the stimulus letter was presented on screen with a duration of 1000 ms and the response window was equal to the stimulus presentation. Between each trial a black fixation cross was presented with a duration that jittered between 800 and 1200 ms. After each block, participants were given feedback on their performance in the block; if their average response time was over 800 ms they were instructed to respond faster and if their response accuracy on the target letters were below 90% they were instructed to be more accurate.

The primary updating performance measure estimated from the NBT in the current study was the condition-mean difference. The condition-mean difference was the difference in mean reaction time between 1-back condition trials and 3-back condition trials. The condition-mean difference was labelled updating score in the analyses, and a higher updating score was interpreted as a lower updating performance.

2.3.2. Actigraphy

Actigraphy is a widely used, non-invasive, and objective method of estimating sleep/wake patterns in humans by tracking movements. In the current study, actigraphy was used to estimate participants' sleep/wake patterns in the two-week period between test-sessions. Actigraphy watches of the type Actiwatch Spectrum Plus (Philips Respironics Inc, Murrysville, PA, USA) were used. The watches recorded gross motor activity and light exposure, and they collected data at 15-second epochs for the two-week period. The data from the actigraphy watches were processed with the manufacturer's software (Philips Actiware 6) and the default threshold for sleep/wake detection was used. For quality assurance, three

people involved in the project (including the author of the current study) worked together to manually check the sleep/wake estimates provided by the software and corrected estimates that seemed erroneous based on activity levels and light exposure together with information from the participants' sleep diaries. The sleep measures from the actigraphy data were bedtime, get-up time, time spent in bed, total sleep time (TST), sleep latency, sleep efficiency, number of awakenings, and total duration of awakenings. Furthermore, the actigraphy data was used to calculate two continuous timing-related sleep variables: sleep onset (SO) and sleep midpoint (SMid). In the present study's statistical analyses, the only actigraphy measures utilised were TST, SO, and SMid.

2.3.3. Consensus Sleep Diary

Sleep diaries are a subjective method of measuring sleep in humans by asking individuals to track their own sleeping habits (Ibáñez et al., 2018). In the current study, the Consensus Sleep Diary (Carney et al., 2012) was used to measure participants sleep/wake patterns in the two-week period between test-sessions alongside the actigraphy measurements. The Consensus Sleep Diary was developed to be a standardised version of the many different sleep diaries previously used in sleep research (Carney et al., 2012), and it asks people to fill out sections both before and after sleep. The Consensus Sleep Diary includes questions about both quantitative and qualitative aspects of sleep, as well as questions about factors that could affect sleep, such as dietary intake and medication. Some of the most notable sleep measures acquired by the sleep dairy are bedtime, get-up time, sleep latency, number of awakenings, sleep duration, sleep quality (SQ), and sleep efficiency. In the current study's statistical analyses, only the SQ measure was used. Participants rated their subjective SQ every morning following sleep on a scale of 1 ('very poor'), 2 ('poor'), 3 ('fair'), 4 ('good'), and 5 ('very good').

2.3.4. Achenbach System of Empirically Based Assessment – Adult Self-Report

The Achenbach System of Empirically Based Assessment – Adult Self-Report (ASEBA-ASR; Achenbach & Rescorla, 2003) is a well-validated questionnaire that measures different aspects of adaptive functioning and problems (Achenbach et al., 2017). The ASEBA taxonomy of questionnaires stem from factor-analytic analyses of child and adolescent psychopathology (Achenbach, 1966) and the ASEBA-ASR consists of 120 items that address different aspects of adaptive functioning and problems. The items address the participant's

thoughts, feelings and behaviour over the last 6 months, and the participant is tasked with rating how applicable the items are on a scale of 0 = not true, 1 = somewhat true and 2 = very true. The items map onto 8 syndrome scales that relate to adult psychopathology (Achenbach & Rescorla, 2003).

In the current study, only the scale of Attention Deficit/Hyperactivity Problems was of interest in order to measure ADHD symptoms. This scale has previously been used to measure ADHD symptoms in healthy adults in cognitive control research (Arntsberg Grane et al., 2014; Robinson et al., 2022). Examples of items on this scale include 'I am too forgetful' and 'I have trouble concentrating or paying attention for long', and it had 13 items total. The total score of the participants' responses on these 13 items was calculated using the accompanying software (ASEBA-PC; minimum score = 0 and maximum = 26). Additionally, total scores were calculated for two subscales of attention deficit/hyperactivity problems: inattention and hyperactivity-impulsivity. For example, 'I fail to finish things I should do' was an item on the inattention subscale, and 'I feel restless or fidgety' was an item on the hyperactivity-impulsivity subscale. The inattention subscale had seven items (minimum score = 0 and maximum = 14), whereas the hyperactivity-impulsivity subscale had six items (minimum = 0 and maximum = 12). In the current study's statistical analyses, the total score on the attention deficit/hyperactivity problems scale was used as a measure of ADHD symptoms, and the total scores on the two subscales were used as measures of inattention and hyperactivity-impulsivity symptoms. A higher score was interpreted as more severe symptoms.

2.4. Procedure

The full project consisted of three data collection sessions: two test-sessions for EEG recording and one session for MRI. Prior to the first test-session, participants were given information about the study procedure and exclusion criteria via email. At the beginning of the first test-session participants were given the same information again and signed a written informed consent form after they had gone through the exclusion criteria checklist.
Next, preparations for the EEG recording were performed and the EEG cap and EMG electrodes were placed on the participant. While EEG and EMG were recorded, participants completed four cognitive control tasks: the SST, the NLTST, a Stroop task (Stroop, 1935), the NBT, and an additional resting-state task. The tasks were completed in the listed order and the full duration of the EEG/EMG preparation and cognitive control task was approximately two and half hours. Afterwards, participants had the opportunity to wash the gel used for EEG out

of their hair before they completed six questionnaires on a computer. The questionnaires were the Pittsburgh Sleep Quality Index (PSQI; Buysse et al., 1989), the Epworth Sleepiness Scale (Johns, 1991), the Positive Affect and Negative Affect Schedule (PANAS; Watson et al., 1988), the ASEBA-ASR (Achenbach & Rescorla, 2003), the Emotion Regulation Questionnaire (ERQ; Gross & John, 2003) and a dietary/health questionnaire (Turconi et al., 2003). In total, the six questionnaires took approximately 30-40 minutes to complete. Finally, participants received an actigraphy watch and a sleep diary and were instructed to use them for the two-week duration between the first and second test-sessions. The second test-session was scheduled to the same timepoint as and was identical to the first regarding the EEG recording, cognitive control task, and questionnaires. At the end of the second test-session the actigraphy watches and sleep diaries were collected from participants. In total, the test-sessions lasted approximately three hours each. The MRI session took place at the Intervention Centre at Oslo University Hospital within the two-week period between test-sessions or shortly afterwards. The MRI session consisted of structural MRI scans and a resting-state functional MRI scan, and it lasted for approximately 40 minutes.

2.5. Data processing

All IIV variables were calculated in units of intraindividual standard deviation (ISD). The minimum number of datapoints required for calculating participants' IM and IIV in sleep variables was set to 10 days. This criterion only affected the SQ variable, for which 32 out of 39 participants had minimum 10 entries in the sleep diary. The two timepoint-related sleep variables, namely SO and SMid, were calculated in units of 'hours relative to midnight' (hrtm; midnight = 0 hrtm, 1 hour before midnight = -1 hrtm, and 1 hour after midnight = 1 hrtm). A binary chronotype group variable, 'morning-leaning' or 'evening-leaning', was calculated based on participants' SMid IM during weekends. Weekend data was chosen with the assumption that participants would be more likely to have obligations that could alter their preferred sleep/wake pattern on weekdays, and because IIV in SMid has been found to be smaller than interindividual variability in SMid during free days (Lenneis et al., 2021). Participants in the morning-leaning group had their $SMid_{Weekend}$ IM before 05.00, while participants in the evening-leaning group had their $SMid_{Weekend}$ IM after 05.00. This classification was based on associations between chronotypes and SMid IM on free days found in previous research (Roenneberg et al., 2003; Roenneberg, Kuehnle, et al., 2007; Santisteban et al., 2018; Zavada et al., 2009). Furthermore, a continuous 'test-time' variable in

units of hrtm was created to account for the timepoint that participants completed the cognitive control tasks.

To provide more information about the participants' TST IIV, two sleep restriction variables and two 'over-sleep' variables were created. The first sleep restriction variable, called SR sum, counted how many days participants were sleep restricted, i.e., how many days they slept less than 7 hours. The second sleep restriction variable, called SR average, calculated the mean of how much less than 7 hours participants slept on sleep restriction days. The first over-sleep variable, called OS sum, counted how many days participants slept more than 9 hours. The second over-sleep variable, called OS average, calculated the mean of how much more than 9 hours participants slept on over-sleep days.

All data were checked for outliers prior to statistical analyses. Datapoints that were more than 3 SD units away from the mean were considered outliers, but they were only removed from the dataset if they were clearly erroneous or contradicted the validity of the variable. One participant had a negative shifting score (i.e., they responded faster in switching trials than in repeat trials on average) and this datapoint was thus removed from all analyses that included the shifting score variable.

2.6. Statistical analyses

All statistical analyses in the current study were performed in RStudio for MacOS (v2021.9.2.382), with the additional packages 'lubridate', 'psych', 'lm.beta', and 'ggplot2'. All tests were evaluated with alpha = .050 and 95% CI were calculated for point estimates. Effect sizes were calculated for significant statistics with 95% CI (except η^2, for which 90% CI were used due to the distribution being one-sided). The hypotheses of the current study were tested using t-tests and multiple regression models. Furthermore, Analyses of Variance (ANOVAs) were used for descriptive purposes. The assumptions for the t-tests were that the data were normally distributed and that the variance was homogenous (homoscedasticity). The assumptions for the multiple regression models were homoscedasticity of the residual variance, linear relationships in the data, normally distributed residuals, independent residual errors, and no presence of multicollinearity. The assumptions for the ANOVAs were homoscedasticity and normally distributed data. Linearity was assessed using Residuals vs Fitted plots, normality using Shapiro-Wilk tests and QQ plots, homoscedasticity using Scale-Location plots and Levene's tests, independence of errors using Cook's distance plots, and multicollinearity using Variance Inflation Factors (VIF). Unless otherwise specified, all data and models met the forementioned assumptions. In cases where single outlier datapoints

caused assumption violations for statistical tests, the outlier datapoint was assigned a new value of 3 SD away from the mean and included in the tests. In cases where assumption violations could not be attributed to single datapoints, non-parametric tests were chosen instead.

H1 was tested with a two-tailed independent sample t-tests and a two-tailed Wilcoxon rank-sum test. The t-test was used to test the difference in SQ IM between morning-leaning and evening-leaning groups, while the Wilcoxon rank-sum test was used to test the difference in TST IIV between morning-leaning and evening-leaning groups. The Wilcoxon rank-sum test was chosen because the distribution of TST IIV was non-normally distributed for the evening-leaning group.

H2 was tested using one multiple regression model. In this model, ADHD symptoms was the dependent variable, and the independent variables were TST IIV, SO IIV, SQ IM, and $SMid_{Weekends}$ IM. Furthermore, two multiple regression models were calculated post-hoc to test for potential differences between the ADHD symptoms subscales of inattention and hyperactivity-impulsivity. In these models, inattention and hyperactivity-impulsivity were the dependent variables and the previously stated sleep variables were the independent variables.

H3 was tested with three different multiple regression models. In these models, the three cognitive control performance measures (inhibiting, shifting, and updating scores) were the dependent variables. The independent variables were test-time and $SMid_{Weekends}$ IM, and an interaction term was included to test the interaction effect.

H4 and ***H5*** were tested with three different multiple regression models. In these models, the three cognitive control performance measures were the dependent variables. The independent variables were ADHD symptoms, TST IIV, SO IIV, and $SMid_{Weekends}$ IM. *H4* was tested by evaluating the associations between ADHD symptoms and the cognitive control performance measures. *H5* was tested by comparing the relative sum of the beta coefficients for the sleep variables that had a significant effect on the cognitive control performance measures. To further explore *H4*, two multiple regression models were calculated post-hoc to test associations between the ADHD symptoms subscales and the three cognitive control performance measures. In these models, inattention and hyperactivity-impulsivity were the dependent variables, and the three cognitive control performance measures were the independent variables.

3. Results

3.1. Descriptive statistics

Descriptive statistics for sleep measures, cognitive control performance measures, and ADHD symptoms measures can be found in Table 1.

Table 1

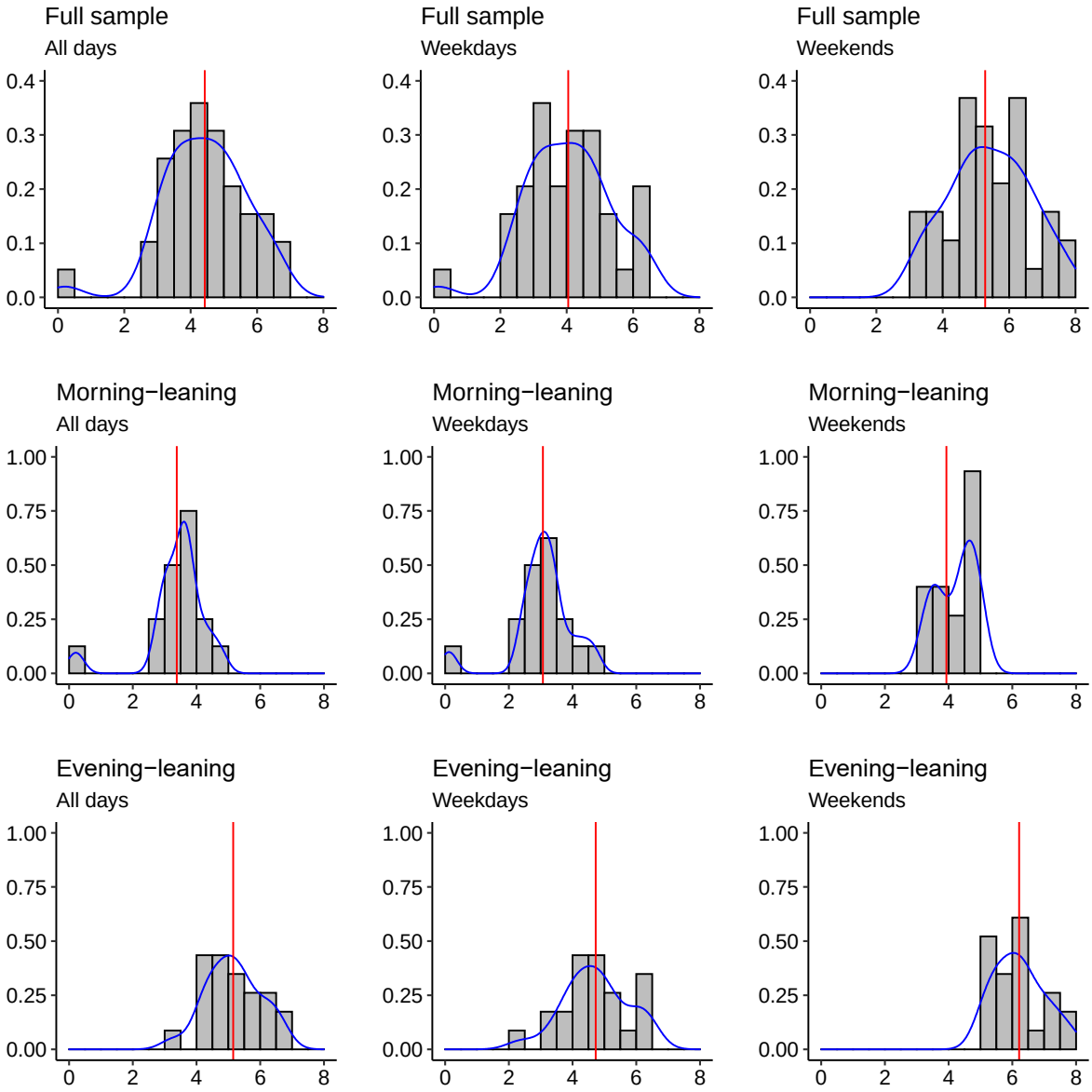
Descriptive statistics for continuous variables.

Variable	*n*	*M*	*SD*	Min	Max	Skew	Kurtosis
TST IM	39	7.55	0.57	6.17	8.70	0.05	-0.42
TST IIV	39	1.14	0.41	0.42	2.01	0.50	-0.65
SO IM	39	0.37	1.18	-3.30	2.45	-0.43	0.62
SO IIV	39	1.20	0.48	0.56	2.98	1.28	2.63
SMid IM	39	4.42	1.26	-0.21	6.69	-0.62	1.29
SMid IIV	39	1.10	0.41	0.50	2.29	0.68	0.21
SQ IM	32	3.51	0.54	2.36	4.86	0.08	-0.21
SQ IIV	32	0.70	0.25	0.00	1.17	-0.60	0.01
SR sum	39	4.41	2.51	1	9	0.27	-1.31
SR average	39	-0.75	0.41	-2.24	-0.04	-1.24	2.47
OS sum	39	1.77	1.84	0	7	1.00	0.18
OS average	27	0.76	0.59	0.00	2.17	0.77	-0.43
Test-time	39	14.34	2.63	10.12	19.10	0.36	-1.16
Inhibiting	39	201.23	21.37	167.28	251.60	0.44	-0.51
Switching	38	161.90	48.16	80.14	265.77	0.17	-0.78
Updating	39	163.04	80.95	27.51	415.11	0.36	0.78
ADHD symptoms	39	5.79	3.99	1	20	1.36	2.43
Inattention	39	3.56	2.50	0	11	0.89	0.40
Hyperactivity-impulsivity	39	2.23	1.93	0	9	1.30	1.91

Note. TST = total sleep time, SO = sleep onset, SMid = sleep midpoint, SQ = sleep quality, SR = sleep restriction, and OS = over-sleep. TST measures are in units of hours, while SO, SMid and test-time measures are in units of hrtm. SR sum and OS sum are in units of days, while SR average and OS average are in units of hours. The sample for OS average was 27 because only 27 participants had at least one day with TST > 9 hours. Table shows values calculated before potential outlier datapoints were replaced in statistical tests.

Figure 1

Histograms of SMid IM for different groups and days.



Note. X-axes denote hrtm, Y-axes denote density. The blue lines represent the smoothed distributions, and the red lines represent the mean values. The left column represents SMid IM for the different samples calculated across both weekdays and weekends, while the middle column represents only weekdays and the right column only weekends. The top row represents the full sample, while the middle row represents only the morning-leaning group and the bottom row only the evening-leaning group.

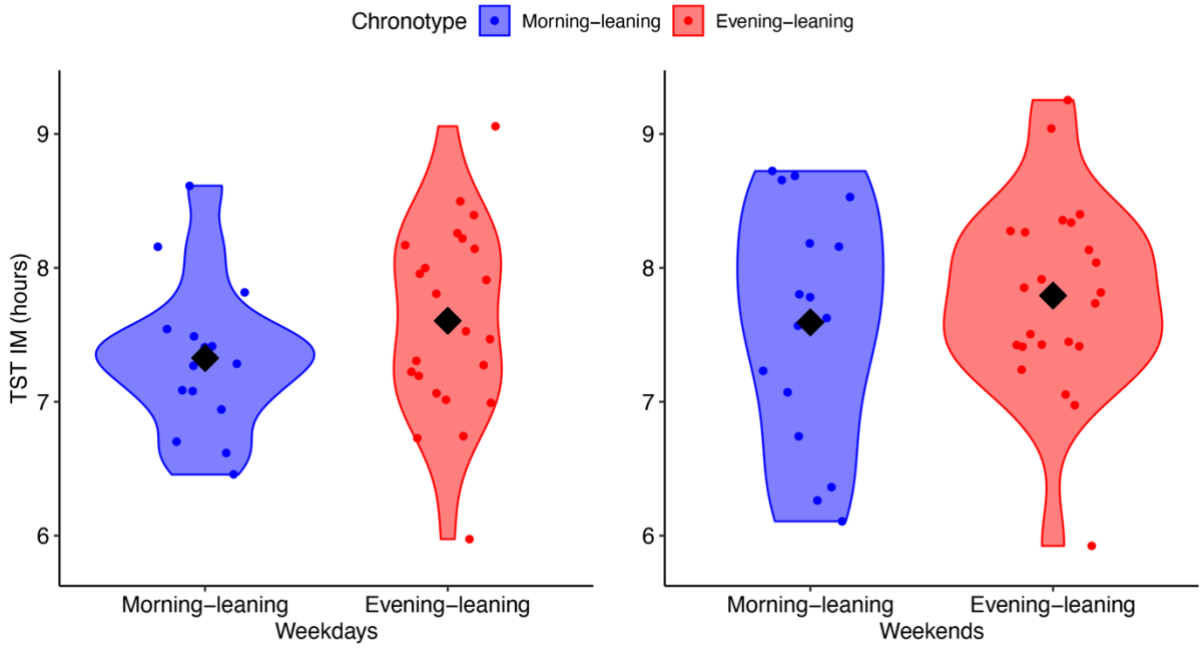
The distributions of SMid IM for the full two-week data collection period, as well as the distributions for only weekdays or weekends, can be seen in the first row of Figure 1.

Based on $SMid_{Weekends}$ IM, 16 participants were assigned to the morning-leaning chronotype group and 23 participants to the evening-leaning group. A two-way repeated measure ANOVA was conducted to explore whether the change in SMid IM between weekdays and weekends differed between morning-leaning and evening-leaning groups. There was a significant main effect of being in the morning-leaning versus evening-leaning group on SMid IM ($F(1, 38) = 24.83$, $p < .001$, $\eta^2 = .23$, 90% CI [.11, .38]), as well as a significant main effect of calculating SMid IM from weekdays versus weekends ($F(1, 38) = 5.93$, $p = .020$, $\eta^2 = .05$, 90% CI [.01, .18]). However, there was no significant interaction effect ($F(1,38) = 1.77$, $p = .187$). The SMid IM distributions for the morning-leaning and evening-leaning groups can be seen in the second and third row of Figure 1.

Furthermore, a two-way repeated measure ANOVA was calculated to explore whether TST IM differed between the morning-leaning and evening-leaning groups and between weekdays and weekends. There was no significant effect of morning-leaning versus evening-leaning group ($F(1,38)= 1.40$, $p = .241$), no significant effect of weekdays versus weekends ($F(1,38) = 0.78$, $p = .380$), and no significant interaction effect ($F(1,38) = 0.06$, $p = .815$). Figure 2 illustrates violin plots of TST IM, while Figure 3 illustrates violing plots of SR sum and OS sum.

Figure 2

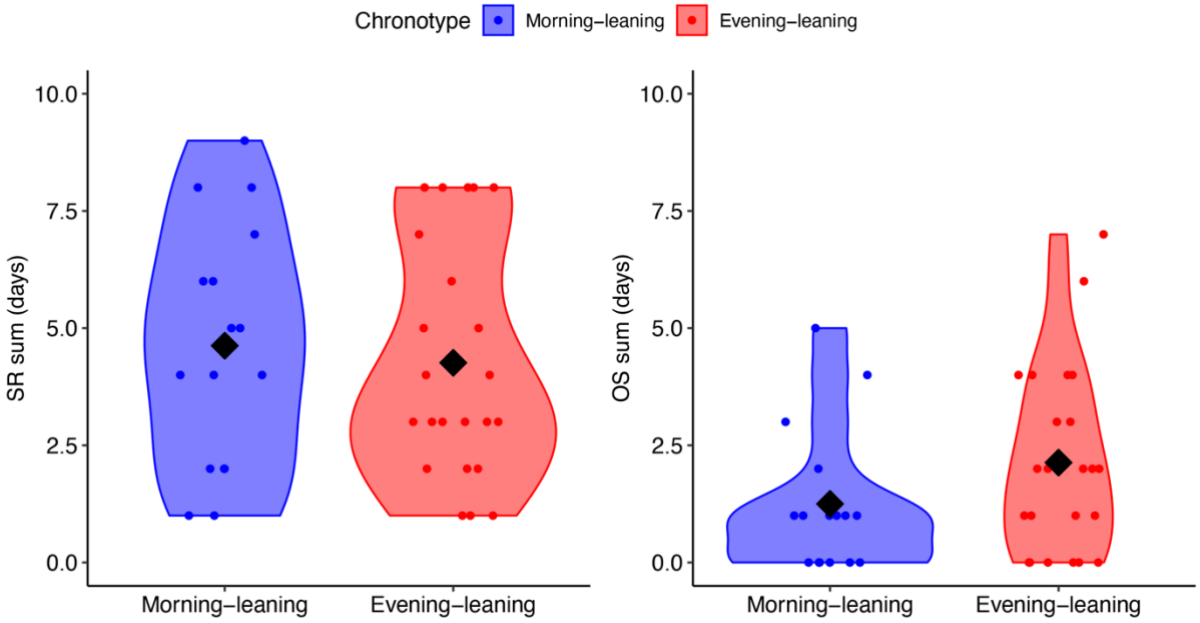
Violin plots of TST IM for chronotype groups and weekdays versus weekends.



Note. Black squares denote the means. Points were jittered on the x-axis to limit overlap.

Figure 3

Violin plots of the number of days participants slept less than 7 hours and more than 9 hours by chronotype group.



Note. Black squares denote the means. Points were jittered on the x-axis to limit overlap.

Correlation matrices were calculated for the sleep measures, the cognitive control performance measures, and the ADHD symptoms measures prior to hypothesis testing with multiple regression models. The sleep variables included were both IM and IIV for TST, SO, and SQ (6 variables, 15 correlations total). There were significant correlations between TST IIV and SO IIV ($r(37) = .46$, $p = .003$), and SQ IIV and SQ IM ($r(37) = -.42$, $p = .016$). There were no other significant correlations between the other sleep measures. Furthermore, there were no significant correlations between the cognitive control performance measures (3 variables, 3 correlations total). As the two subscales add up to the ADHD symptoms scale, there were significant correlations between ADHD symptoms and inattention ($r(39) = .93$, $p < .001$), ADHD symptoms and hyperactivity-impulsivity ($r(39) = .87$, $p < .001$), and inattention and hyperactivity-impulsivity ($r(39) = .62$, $p < .001$).

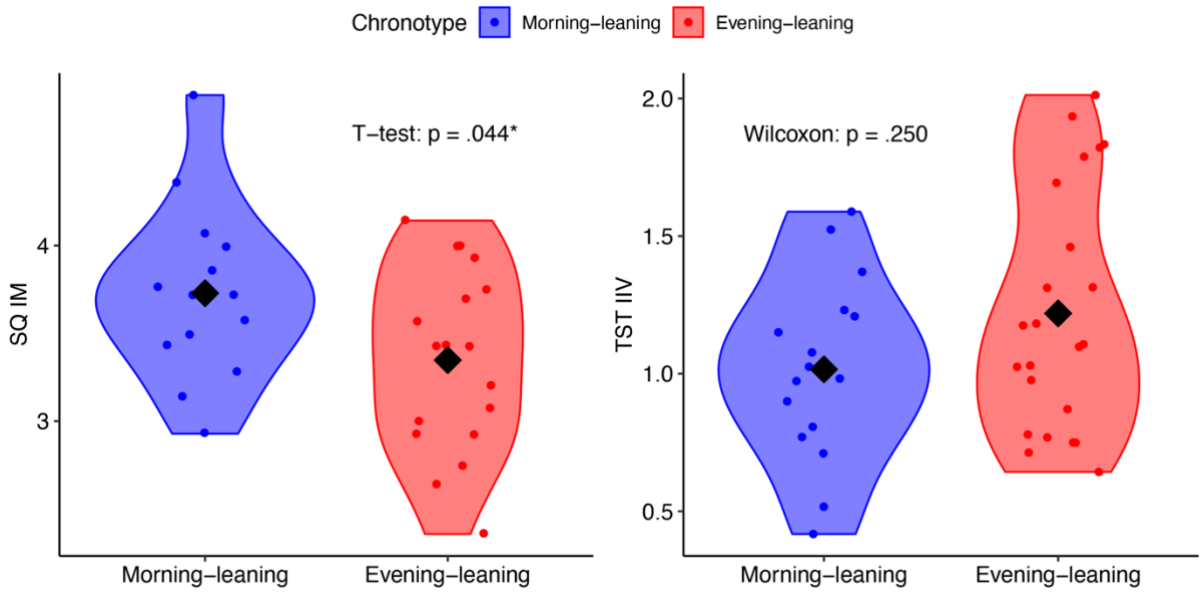
3.2. Hypotheses and post-hoc tests

H1. A two-tailed independent sample t-test found that there was a significant difference in SQ IM between morning-leaning ($M = 3.73$, 95% CI [3.44, 4.02]) and evening-leaning ($M = 3.35$, 95% CI [3.09, 3.61]) chronotype groups ($t(30) = 2.10$, $p = .044$), and the

effect size was moderate (d = 0.75, 95% CI [0.04, 1.51]). There was no significant difference between the groups in TST IIV ($Mdn_{\text{Morning-leaning}}$ = 1.00 [IQR = 0.42], $Mdn_{\text{Evening-leaning}}$ = 1.11 [IQR = 0.75]) when tested with a two-tailed Wilcoxon rank-sum test (W(38) = 143, p = .251). Figure 4 illustrates violin plots of the two tested variables grouped by chronotype.

Figure 4

Violin plots of SQ IM and TST IIV by chronotype group.



Note. * denotes significance at p < .050. Black squares denote the means. Points were jittered on the x-axis to limit overlap.

H2. A multiple regression model was calculated to test associations between ADHD symptoms and sleep variables. One outlier value of 20 (3.56 SD) on the ADHD symptoms measure was replaced with a value of 3 SD away from the mean to comply with model assumptions. The regression model explained 9% of the variance in ADHD symptoms (adjusted R^2 = .09) and the results can be seen in Table 2. There was a significant negative association between SQ IM and ADHD symptoms (see Table 2), but the overall model was not statistically significant (F(4, 27) = 1.80, p = .159). There were no significant associations between ADHD symptoms and TST IIV, SO IIV, and $SMid_{\text{Weekends}}$ IM.

Two similar multiple regression models were calculated post-hoc to test whether the inattention and hyperactivity-impulsivity subscales were differentially associated with the sleep variables. One outlier value of 9 (3.51 SD) on the hyperactivity-impulsivity measure was replaced with a value of 3 SD away from the mean to comply with model assumptions.

The regression models' results can be seen in Table 2. The models explained 0% of the variance in inattention (adjusted R^2 = -.01) and 15% of the variance in hyperactivity-impulsivity (adjusted R^2 = .15). Neither the inattention model (F(4, 27) = 0.94, p = .458), nor the hyperactivity-impulsivity model (F(4,27) = 2.31, p = .084) were statistically significant. SQ IM was a significantly negatively associated with hyperactivity-impulsivity, but not with inattention (see Table 2). There were no significant associations between inattention or hyperactivity-impulsivity and TST IIV, SO IIV, and $SMid_{Weekends}$ IM. Figure 5 illustrates violin plots of ADHD symptoms, inattention, and hyperactivity-impulsivity by chronotype group.

Table 2

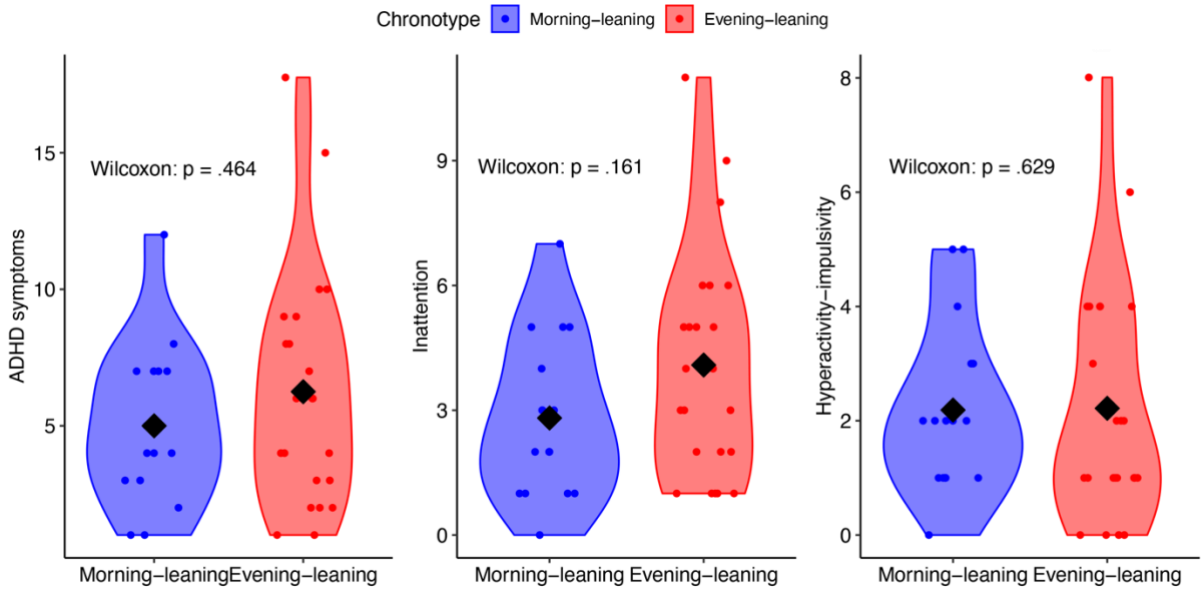
Regression model results for ADHD symptoms and inattention and hyperactivity-impulsivity.

Models & variables	*b*	95% CI	SE	*β*	*t*	*p*
ADHD symptoms (intercept)	17.43	[4.19, 30.67]	6.45			
TST IIV	0.77	[-3.10, 4.64]	1.89	0.08	0.41	.688
SO IIV	0.83	[-3.03, 4.69]	1.88	0.08	0.44	.663
SQ IM	-3.53	[-6.35, -0.71]	1.37	-0.48	-2.57	.016*
$SMid_{Weekends}$ IM	-0.25	[-1.27, 0.77]	0.50	-0.10	-0.51	.615
Post-hoc models & variables						
Inattention (intercept)	7.99	[-1.35, 17.34]	4.55			
TST IIV	0.53	[-2.20, 3.26]	1.33	0.08	0.40	.693
SO IIV	0.66	[-2.06, 3.38]	1.32	0.10	0.50	.624
SQ IM	-1.64	[-3.36, 0.34]	0.97	-0.34	-1.70	.101
$SMid_{Weekends}$ IM	-0.03	[-0.75, 0.69]	0.35	-0.02	-0.08	.940
Hyperactivity-impulsivity (intercept)	9.63	[3.39, 15.86]	3.04			
TST IIV	0.16	[-1.67, 1.98]	0.89	0.03	0.18	.862
SO IIV	0.27	[-1.55, 2.08]	0.89	0.06	0.30	.765
SQ IM	-1.96	[-3.28, -0.63]	0.65	-0.55	-3.03	.005*
$SMid_{Weekends}$ IM	-0.21	[-0.69, 0.27]	0.23	-0.18	-0.91	.372

Note. TST = total sleep time, SO = sleep onset, SQ = sleep quality, and SMid = sleep midpoint. TST is in units of hours, while SO and SMid are in units of hrtm. Minimum possible SQ IM was 1, maximum possible was 5. * denotes significance at $p < .050$.

Figure 5

Violin plots of ADHD symptoms, inattention, and hyperactivity-impulsivity by chronotype group.



Note. Black squares denote the means. Points were jittered on the x-axis to limit overlap. The minimum and maximum possible values for the measures were 0 and 26 for ADHD symptoms, 0 and 14 for inattention, and 0 and 12 for hyperactivity-impulsivity. The distributions of both ADHD symptoms, inattention, and hyperactivity-impulsivity were non-normal in the evening-leaning chronotype group. For descriptive purposes, the significance levels of the differences between chronotype groups were calculated using Wilcoxon rank-sum tests.

H3. Three multiple regression models were calculated to test whether there was an interaction effect between chronotype and test-time on cognitive control performance. The results of these model can be seen in Table 3. The model predicting inhibiting score explained 1% of the variance (adjusted R^2 = .01) and was not statistically significant (F(3, 35) = 1.12, p = .356). The model predicting shifting score explained 1% of the variance (adjusted R^2 = .01) and was not statistically significant (F(3, 34) = 1.13, p = .353). One outlier value of 415.11 (3.11 SD) on the updating measure was replaced with a value of 3 SD away from the mean to comply with model assumptions. The model predicting updating score explained 0% of the variance (adjusted R^2 = -.02) and was not statistically significant (F(3, 35) = 0.80, p = .502). There were no significant main effects or interactions effects in any of the three models (see Table 3). Figure 6 illustrates scatterplots of test-time and cognitive control performance

measures. The plot lines illustrate the trends for the morning-leaning and evening-leaning groups, and the shape of the dots indicate which chronotype group participants belonged to. Furthermore, the colour of the dots indicates the degree to which a participant's chronotype was more morning-leaning or evening-leaning. Neither of the three plots have any distinguishable patterns in the distributions of shapes and colours, which indicates the lack of a relationship between test-time and chronotype. The chronotype group trend lines also lack indications of relationships between test-time and performance.

Table 3

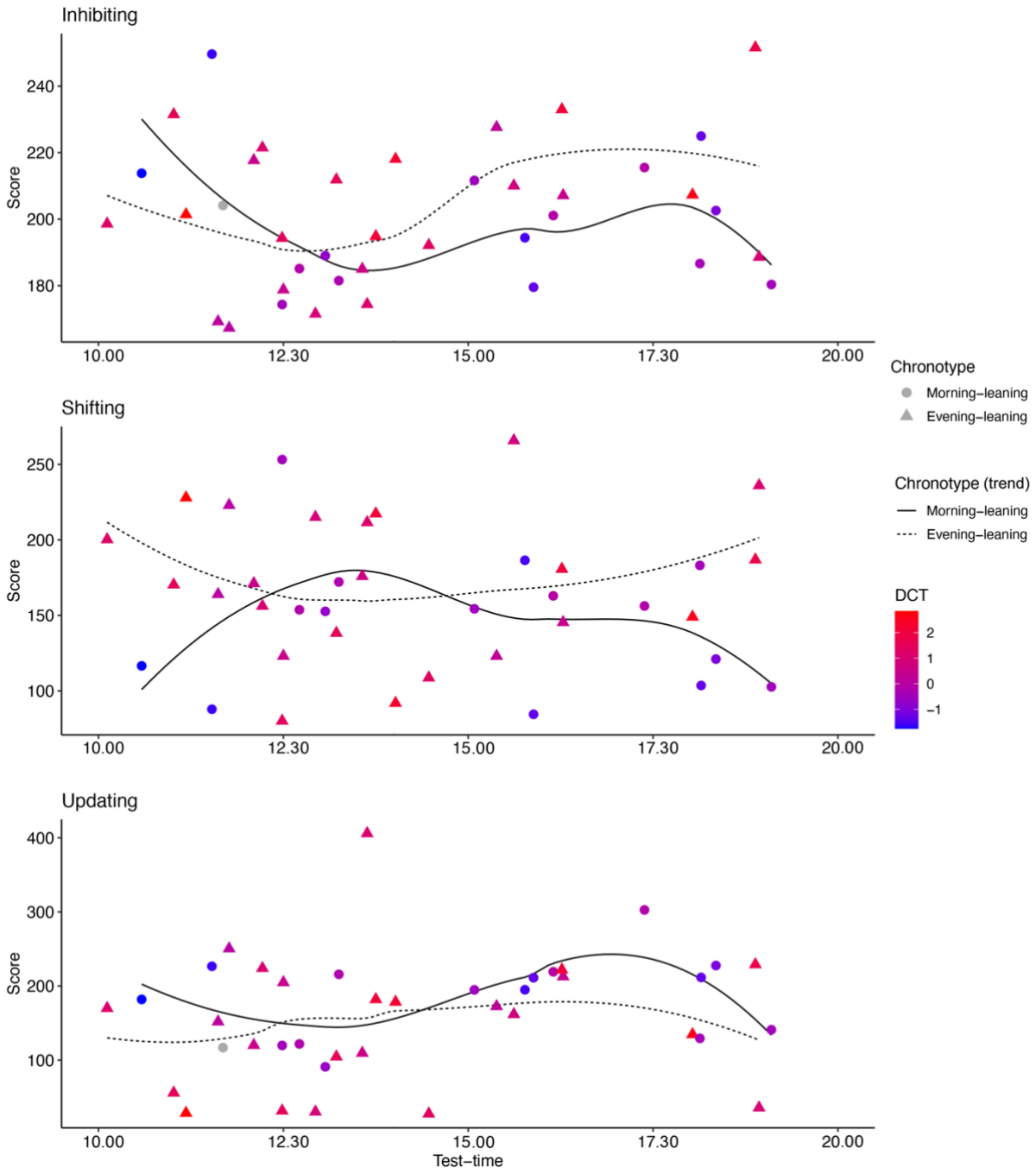
Regression model results for cognitive control performance by chronotype and test-time.

Models & variables	*b*	95% CI	SE	*β*	*t*	*p*
Inhibiting (intercept)	272.23	[141.14 403.31]	64.57			
$SMid_{Weekends}$ IM	-16.66	[-40.75, 7.43]	11.87	-1.19	-1.40	.169
Test-time	-5.87	[-15.46, 3.73]	4.73	-0.72	-1.24	.223
Interaction	1.33	[-0.43, 3.10]	0.87	1.58	1.53	.135
Shifting (intercept)	134.24	[-244.45, 512.93]	186.34			
$SMid_{Weekends}$ IM	8.35	[-59.76, 76.47]	33.52	0.21	0.25	.805
Test-time	-2.35	[-27.91, 23.21]	12.58	-0.13	-0.19	.853
Interaction	0.21	[-4.42, 4.84]	2.28	0.09	0.09	.928
Updating (intercept)	111.60	[-386.64, 609.85]	245.43			
$SMid_{Weekends}$ IM	-6.79	[-98.37, 84.79]	45.11	-0.13	-0.15	.881
Test-time	6.79	[-29.69, 43.28]	17.97	0.22	0.38	.708
Interaction	-0.14	[-6.85, 6.58]	3.31	-0.04	-0.04	.967

Note. SMid = sleep midpoint. SMid and test-time are in units of hrtm.

Figure 6

Scatterplots of cognitive control performance by test-time and chronotype group.



Note. DTC stands for 'Distance from Chronotype Threshold' and it represents the degree to which a participant's chronotype was leaning from the group cut-off. 0 DTC equals the cut-off of 05.00 on $SMid_{Weekends}$ IM, -1 DTC equals 04.00, and +1 DTC means 06.00. The grey dots in the inhibiting and updating plots are the datapoints for the participant that had their shifting score excluded from all analyses. This participant also had the lowest DTC (-5.46) of all participants, so to allow for ease of colour comparison between all three plots this participants' datapoints were greyed out.

H4* & *H5. Three multiple regression models were calculated to test associations between cognitive control performance measures, TST IIV, SO IIV, $SMid_{Weekends}$ IM, and ADHD symptoms. The results of these models can be seen in Table 4. The model predicting inhibiting score explained 4% of the variance (adjusted R^2 = .04), and was not statistically significant (F(4, 34) = 1.36, p = .267). The model predicting shifting score explained 17% of the variance (adjusted R^2 = .17), and was statistically significant (F(4, 33) = 2.91, p = .036). The model predicting updating score explained 0% of the variance (adjusted R^2 = -.02), and was not statistically significant (F(4, 34) = 0.82, p = .519). There were no statistically significant associations between the dependent and independent variables in the three models (see Table 4).

Table 4

Regression model results for cognitive control performance measures.

Models & variables	*b*	95% CI	SE	*β*	*t*	*p*
Inhibiting (intercept)	176.10	[144.31, 207.89]	15.64			
TST IIV	15.37	[-4.65, 35.38]	9.85	0.29	1.56	.128
SO IIV	5.82	[-10.36, 22.00]	7.96	0.13	0.73	.987
$SMid_{Weekends}$ IM	0.04	[-4.69, 4.77]	2.33	0.00	0.02	.923
ADHD symptoms	0.09	[-1.74, 1.92]	0.90	0.02	0.10	.470
Shifting (intercept)	122.90	[48.05, 197.74]	36.79			
TST IIV	-9.46	[-52.80, 33.87]	21.30	-0.08	-0.44	.660
SO IIV	-30.15	[-64.02, 3.73]	16.65	-0.31	-1.81	.079
$SMid_{Weekends}$ IM	12.94	[-0.22, 26.10]	6.47	0.33	2.00	.054
ADHD symptoms	2.71	[-1.15, 6.58]	1.90	0.22	1.43	.163
Updating (intercept)	204.67	[81.93, 327.42]	60.40			
TST IIV	-54.95	[-132.22, 22.32]	38.02	-0.28	-1.45	.158
SO IIV	32.01	[-30.45, 94.48]	30.74	0.19	1.04	.305
$SMid_{Weekends}$ IM	-4.93	[-23.20, 13.33]	8.99	-0.09	-0.55	.587
ADHD symptoms	1.43	[-5.65, 8.50]	3.48	0.07	0.41	.684

Note. TST = total sleep time, SO = sleep onset, and SMid = sleep midpoint. TST is in units of hours, while SO and SMid are in units of hrtm. Minimum possible SQ IM was 1, maximum possible was 5.

As there were no significant associations between ADHD symptoms and the cognitive control performance measures, two multiple regression models were calculated post-hoc to explore associations with the inattention and hyperactivity-impulsivity subscales. Inattention and hyperactivity-impulsivity were the dependent variables in these models, while the three

cognitive control performance measures were the independent variables. The results of these models can be seen in Table 5. The model predicting inattention explained 4% of the variance (adjusted R^2 = .04) and was not statistically significant ($F(3, 34) = 1.55$, $p = .220$). However, there was a significant positive association between inattention and shifting score in the model (see Table 5). The model predicting hyperactivity-impulsivity explained 0% of the variance (adjusted R^2 = -.04) and was not statistically significant ($F(3, 34) = 0.48$, $p = .696$). There were no significant associations between hyperactivity-impulsivity and the cognitive control performance measures.

No formal comparison of the beta coefficients for the different cognitive control performance measures was attempted since none of the models had any significant coefficients (see Table 4). However, since the model predicting shifting score was significant and explained the most variance out of the three, a stepwise regression model was calculated post hoc to explore the explained variance. Both-direction stepwise selection was used as the method of variable selection and the Akaike Information Criterion (AIC) was used to limit the variables that were included in the final model. The final stepwise regression model included shifting score as the dependent variable, and SO IIV, $SMid_{Weekends}$ IM, and ADHD symptoms as independent variables. The stepwise model explained 19% of the variance (adjusted R^2 = .19) and was statistically significant ($F(3, 34) = 3.91$, $p = .017$). In this model, shifting performance had a significant association with SO IIV (b = -33.50, 95% CI [-63.30, 3.69], β = -0.34, $p = .029$), but not with $SMid_{Weekends}$ (b = 11.870, 95% CI [-0.18, 23.92], β = 0.30, p = .053) nor with ADHD symptoms (b = 2.72, 95% CI [-1.10, 6.53], β = 0.22, $p = .157$).

Table 5

Regression model results for inattention and hyperactivity-impulsivity.

Post-hoc models & variables	*b*	95% CI	SE	*β*	*t*	*p*
Inattention (intercept)	-3.39	[-12.57, 5.79]	4.52			
Inhibiting	0.02	[-0.02, 0.06]	0.02	0.17	1.01	.321
Shifting	0.02	[0.00, 0.04]	0.01	0.35	2.10	.043*
Updating	-0.00	[-0.01, 0.01]	0.01	-0.00	0.02	.988
Hyperactivity-impulsivity (intercept)	1.87	[-5.21, 8.94]	3.48			
Inhibiting	-0.01	[-0.04, 0.03]	0.02	-0.06	-0.34	.736
Shifting	0.01	[-0.01, 0.02]	0.01	0.17	0.95	.349
Updating	0.00	[-0.01, 0.01]	0.00	0.09	0.53	.600

Note. * denotes significance at $p < .050$.

4. Discussion

With day-to-day variability and noticeable individual preferences for timing, sleep is an unavoidable subject when it comes to explaining the intricacies of human cognition. The aim of the current study was to investigate the interplay between intraindividual variability in sleep, chronotype, cognitive control, and ADHD symptoms in a sample of healthy adults. Based on previous findings, five hypotheses were formulated to explain the relationship between these concepts. The current study had mixed results in finding evidence to support *H1* and *H2*; individuals with an evening-leaning chronotype had significantly lower subjective SQ on average than individuals with a morning-leaning chronotype and ADHD symptoms was associated with lower SQ on average. On the other hand, the current study did not find statistically significant evidence to support *H3*, *H4*, and *H5*; neither chronotype test-time interactions, ADHD symptoms, nor IIV in sleep were associated with cognitive control performance. Nevertheless, descriptive statistics and post-hoc analyses indicate that there are trends in the data that are worthy of consideration. Finally, there are limitations worth noting which could explain the lack of significant findings in the current study.

4.1. Sleep

The current study used actigraphy to estimate participants' sleep-wake patterns in a naturalistic setting for two weeks, while also collecting subjective SQ data via sleep diaries. The sleep variables calculated from this data give a good insight into the habitual sleep of a sample of young healthy adults. At a first glance of TST IM, participants in the current study appeared to get more than the 7 hours of recommended sleep on average. However, when also considering TST IIV and the sleep restriction variables, the image is less homogenous. Not only was the mean TST IIV higher than the interindividual variability in TST IM, but all participants had at least one sleep restricted day throughout the two-week data collection period. In fact, the mean number of sleep restriction days was 4.41 (SD = 2.51), while the mean number of days with prolonged TST (> 9 hours) was only 1.17 (SD = 1.84). Interestingly, the current study found no significant differences between TST IM for weekdays and weekends, which suggests that participants did not catch up on sleep during weekends as is frequently found in sleep research (Monk, 2012; Wittmann et al., 2006).

On the other hand, there was a significant difference in participants' timing of sleep between weekdays and weekends; on average, participants' SMid IM was shifted to be later during weekends than during weekdays. As expected, participants in the morning-leaning group had earlier SMid IM than the evening-leaning group on average, but there was no

interaction effect between the groups with weekdays vs weekends, meaning that the observed shift in sleep timing was similar for both groups (this can also be seen in Figure 1). Thus, the current study did not replicate the previous finding that later/evening chronotypes have larger differences in sleep timing between workdays and free days than earlier/morning chronotypes (McGowan et al., 2016; Wittmann et al., 2006). One potential explanation for this failed replication is the influence of the COVID-19 pandemic on sleep/wake patterns, which is discussed further with other limitations in section 4.6.

The first hypothesis of the study concerned the differences in sleep between individuals with earlier/morning chronotypes and individuals with later/evening chronotypes. In line with previous findings, the current study found that individuals with an evening-leaning chronotype had significantly lower subjective SQ than individuals with a morning-leaning chronotype. Although the average rating fell between 'fair' and 'good' for both groups, the morning-leaning group were closer to rating their sleep as 'good', while the evening-leaning group were closer to rating their sleep as 'fair'. This difference might not appear large, but the effect size was moderate and since the current study was conducted with healthy participants with no sleep disorders it remains an important finding. One would not expect to see major differences in SQ between healthy adults, so the finding that later/evening chronotypes had lower SQ on average should not be taken lightly despite the causality being unknown.

Nevertheless, the current study did not find evidence to support the hypothesis that individuals with later/evening chronotypes had higher IIV in sleep than earlier/morning chronotypes. Participants in the morning-leaning chronotype group did not significantly differ in TST IIV from participants in the evening-leaning group. However, a different method of sleep estimation and a different variable tested could potentially explain why this result differs from previous findings. The previous investigations into chronotype differences for IIV in sleep have estimated sleep measured from sleep diaries, not actigraphy, and they only found significant differences in bedtime, sleep timing, and time in bed (Bei et al., 2016). Therefore, the lack of a difference TST IIV between the chronotype groups does not necessarily constitute a failed replication of previous findings, but rather an addition of new results pertaining to different sleep estimation parameters.

4.2. Sleep and symptoms of attention-deficit/hyperactivity disorder

The second hypothesis of the current study concerned the relationship between sleep and ADHD symptoms in a healthy population sample. To investigate this, a measure of

ADHD symptoms, as well as two subscale measures for inattention and hyperactivity-impulsivity, were derived from the participants' self-reported behaviours, thoughts, and feelings in the ASEBA-ASR. The hypothesised relationships were that ADHD symptoms would be associated with higher IIV in sleep, later/evening chronotype, and lower SQ IM. Of these three sleep aspects, the current study only found evidence for lowered SQ IM.

In a multiple regression model (controlling for TST IIV, SO IIV, and SMidWeekends IIM), though the overall model was not significant, SQ IM had a significant negative association. A 1 SD unit increase in SQ IM was associated with a 0.48 SD unit decrease in ADHD symptoms. This result is in line with previous associations found between ADHD and SQ in clinical groups (Snitselaar et al., 2013; van Veen et al., 2010; Ziegler et al., 2021), and importantly, it replicates the finding that SQ is related to ADHD symptoms in healthy adults (McGowan et al., 2016). Furthermore, post-hoc tests of the ADHD symptoms subscales indicated that the SQ association could be attributed to hyperactivity-impulsivity and not inattention. This finding is similar to the negative association between SQ and trait impulsivity in healthy adults found by McGowan et al. (2016), as well as similar to the negative association between SQ and hyperactivity-impulsivity symptoms in individuals with ADHD found by Mahajan et al. (2010). Without further research, however, interpreting the causal nature of this finding is difficult; a lowered SQ could cause heightened symptoms of hyperactivity-impulsivity or vice versa, and bidirectional effects cannot be ruled out.

In contrast to the previous finding that later/evening chronotype is associated with ADHD symptoms in healthy young adults (McGowan et al., 2016), the current study found no significant associations between chronotype (estimated with $SMid_{Weekends}$ IM) and ADHD symptoms. Moreover, post-hoc tests found no significant associations between chronotype and the inattention and hyperactivity-impulsivity subscales. Hence, the current study also failed to find evidence for a connection between impulsivity and later/evening chronotypes as has been reported previously (Muro et al., 2012; Prat & Adan, 2013). Interestingly, the lack of an association between ADHD symptoms and chronotype suggests that chronotype and ADHD may be associated with SQ differently.

Lastly, results from the current study do not support the hypothesis that IIV in sleep is associated with more ADHD symptoms. Neither TST IIV nor SO IIV were found to be significantly associated with ADHD symptoms, nor with the inattention and hyperactivity-impulsivity subscales. Previous research on IIV in sleep and ADHD has found that individuals diagnosed with ADHD have significantly higher IIV in sleep than control groups (Langberg et al., 2019; van Veen et al., 2010; Ziegler et al., 2021), but the results of the

current study suggest that such an association is either negligible or non-existent in a small sample of healthy adults.

4.3. Chronotype and cognitive control performance

The third hypothesis of the current study concerned the interaction between chronotype and test-time for cognitive control performance. Participants' performance in the three core components of cognitive control outlined by the unity and diversity framework (Friedman & Miyake, 2017; Miyake et al., 2000) were estimated with 3 different cognitive control tasks. Based on previous research, the hypothesis was that performance would be poorer for participants with a later/evening chronotype tested early in the day and poorer for earlier/morning chronotype tested late in the day. The performance measures were modelled in three multiple regression models that included test-time and chronotype (estimated with $SMid_{Weekends}$ IM) as continuous predictors and an interaction term. All three of these models explained little to no variance and were not statistically significant. There were neither significant main effects nor significant interaction effects on the cognitive control performance measures, meaning that the current study did not find evidence to support this hypothesis.

However, there is little reason to think that an individual's chronotype does not influence their cognitive control performance at all; there are design flaws in the current study that should be accounted for before the hypothesis is rejected. In contrast to previous studies showing that cognitive performance varies systematically depending on test-time and chronotype (Chellappa et al., 2018; Facer-Childs et al., 2018; Martínez-Pérez et al., 2020; Salehinejad et al., 2021), participants in the current study were only tested at one timepoint of the day and they were not assigned test-times with potential chronotype effects in mind. The average test-time across participants was 14.20 and the SD was 2 hours and 38 minutes, indicating that most participants were tested in the afternoon when alertness and vigilance is generally high and potential effects of chronotype would be small (Schmidt et al., 2007; Valdez et al., 2012).

4.4. Symptoms of attention-deficit/hyperactivity disorder and cognitive control performance

The fourth hypothesis of the current study concerned the relationship between ADHD and cognitive control. ADHD symptoms in healthy adults have been linked with deficits in

multiple aspects of cognitive control (Das et al., 2015; Silverstein et al., 2020), and thus the current study hypothesised that there would be a negative association between ADHD symptoms and performance related to the three cognitive control components. However, this hypothesis was not supported by the results. Three multiple regression models were calculated for the three cognitive control performance measures, and when controlling for TST IIV, SO IIV, and $SMid_{Weekends}$ IM, there were no significant associations between any of the three cognitive control performance measures and ADHD symptoms. Nevertheless, there was one significant association in the post-hoc regression models calculated for the two ADHD symptoms subscales; inattention was negatively associated with shifting performance. This association is in line with previous research showing that cognitive control performance in clinical ADHD groups is largely explained by inattention symptomology rather than hyperactivity-impulsivity (Brocki et al., 2010; Chhabildas et al., 2001; Martel et al., 2011; Nigg et al., 2005; Sabhlok et al., 2022). Although the association effect was small, it is an interesting finding, as one might expect that higher levels of inattention would affect performance on the three cognitive control tasks in a uniform manner. Previous research in clinical ADHD groups indicate that inattention symptoms are related to poor performance in many aspects of cognitive control, not just shifting (Chhabildas et al., 2001; Nigg, 2001). However, the level of symptoms among the healthy participants in the current study was much lower than what one would find in clinical groups.

Inattention traits have previously been found to be negatively associated with shifting performance for healthy adults in another task-switching paradigm (Arabacı & Parris, 2020). In this study, the researchers were able to show that the inattention shifting deficit was related to a neglect of preparatory processes rather than an inability to prepare. Furthermore, the observed inattention shifting deficit was also related to poor performance in a goal neglect task. Goal neglect is a type of cognitive performance failure described as not adhering to a rule that one is able to state verbally (Duncan et al., 1996), and it has previously been found to be associated with inattention symptoms and not hyperactivity-symptoms in a sample of healthy adults (Elisa et al., 2016). Taken together, these findings indicate that inattention symptomology in healthy adults has the potential to explain performance deficits in tasks where adhering to rules is a central aspect. Of the three cognitive control tasks used in the current study, the NLTST that estimated shifting was the task that required participants to follow rules most closely. In the SST and NBT, participants adhered to one rule at a time, whereas in the NLTST they were required to flexibly shift between two rulesets.

In summary, one potential explanation for why there was a significant association between inattention and shifting but not inhibiting and updating could be that the level of inattention symptoms in the current sample of healthy adults only made the inattention deficit apparent in the task that was the most demanding in terms of flexibly adhering to rules.

4.5. Sleep and cognitive control performance

The fifth and final hypothesis of the current study concerned the relationship between sleep and cognitive control. In research on sleep deprivation and sleep restriction, cognitive control performance has consistently been found to be affected by sleep, but not in a uniform manner (Gevers et al., 2015; Killgore, 2010; Kusztor et al., 2019; Satterfield & Killgore, 2019). Furthermore, to the author's knowledge no studies have investigated the relationship between IIV in sleep and cognitive control although both concepts are implicated in ADHD (Boonstra et al., 2005; Langberg et al., 2019; Nigg, 2001; van Veen et al., 2010; Ziegler et al., 2021). The current study hypothesised that potential associations with sleep would be lower for inhibiting performance than for shifting and updating performance, as previous studies had shown that more low-level cognitive control processes were less affected by sleep (Gevers et al., 2015; Kusztor et al., 2019). The same three multiple regression models calculated for hypothesis four were used to evaluate this hypothesis.

Of the three models, only the model predicting shifting performance was statistically significant. Although none had significant coefficients, TST IIV, SO IIV, $SMid_{Weekends}$ IM, and ADHD symptoms together explained 17% of the variance in shifting performance in the current study's sample of healthy adults. Since neither the inhibiting nor the updating performance measures were significant, the current study made no attempt to compare sleep associations between the three cognitive control components and thus could not find evidence to support the fifth hypothesis. Nevertheless, a stepwise regression model was calculated to explore the explained variance in shifting performance. The stepwise model included SO IIV, $SMid_{Weekends}$ IM, and ADHD symptoms as independent variables, but discarded TST IIV, and it explained 19% of the variance. In contrast to the original model, the stepwise model indicated that there was a significant negative association between SO IIV and shifting performance and the association with $SMid_{Weekends}$ IM was similar in effect size. At best these results indicate that there is a trend towards chronotype and IIV in timing of sleep being related to shifting performance, but the effect sizes and interpretations of significance limit the conclusions that can be drawn.

The lack of findings regarding IIV in sleep and cognitive control performance in the current study does not completely rule out a connection between the two concepts. In comparison to previous findings for healthy adults (Bartlett et al., 2008; Knutson et al., 2007; Mezick et al., 2009), the variability in both TST IIV and TST IM in the current study was small (around 30 minutes for both measures). Additionally, there was similar variability within and between participants in SO. As such, it might have been a poor choice to only estimate IIV in sleep with the TST and SO, and different results might have been found if IIV in sleep variables such as SQ or sleep efficiency were included as well. Furthermore, the detrimental effects of poor sleep on cognitive control have largely been found in controlled experiments with sleep deprivation and sleep restriction (Gosselin et al., 2005; Lowe et al., 2017; Martella et al., 2011; Nilsson et al., 2005; Qi et al., 2010; Tsai et al., 2005), and one would not expect to see effects of similar size associated with normal IIV in sleep for healthy adults. Additionally, the current study did not control for the sleep that took place the night before cognitive control performance testing. Given the small sample size, there is a high chance that the current study did not have enough statistical power to detect small-magnitude associations between cognitive control performance and day-to-day sleep variability.

4.6. Limitations and future research

As noted in previous sections, the current study's conclusive power is weakened by several limitations. The most over-arching of these limitations is the sample size. The current study aimed to explore many different concepts and variables but only had a sample of 39 participants (of which only 32 had SQ data). Additionally, the sample was primarily students and had a narrow age-range. Not only did this small sample limit the study's ability to detect small-magnitude effects, it also limited the number of variables of interest that could be included in the statistical analyses. The six multiple regression analyses that were calculated for hypotheses 3, 4, and 5 would ideally have been merged down to three (one for each cognitive control performance measure) so that potential interaction effects could be accounted for. Furthermore, it would have been beneficial to control for and test associations with sleep variables such as TST IM, SQ IM, and SQ IIV, which could include valuable variance (Bei, Manber, et al., 2017; Wiley et al., 2014). In the current study, however, merging the regression models and adding more predictors would have made the number of predictors unreasonably high in comparison to the small sample size. To be able to detect small-magnitude effects and to explore the interplay between IIV in sleep, chronotype,

cognitive control, and ADHD symptoms in more depth, future research should aim to analyse larger samples.

Other limitations of the current study concern sleep estimation and data collection during the COVID-19 pandemic. Research has shown that sleep/wake patterns were significantly altered during the pandemic compared to pre-pandemic (Benedict et al., 2021; Bottary et al., 2022; Brandão et al., 2021; Cellini et al., 2020; Storari et al., 2021), and approximately one third of the participants in the current study were recruited and tested during the pandemic. Rezaei & Grandner (2021) found that mean sleep duration was increased during the pandemic, whereas IIV in both sleep duration and bedtime decreased. Additionally, a large proportion of the participants were students, whom might have more flexible schedules compared to adult with other occupations. Thus, alterations to sleep-wake patterns in the form of being able to sleep more freely could potentially have reduced both the IIV in sleep and the differences between chronotype groups that have been found in previous research. Moreover, the use of actigraphy to estimate sleep could also have introduced measurement error to the current study, as actigraphy tends to overestimate TST in sleepers who lie motionless while still being awake (Martin & Hakim, 2011). The lack of validation for the chronotype estimates from $SMid_{Weekends}$ IM also introduces uncertainty. To address these limitations in sleep measurement, future research should aim to include a validated chronotype measure (such as the MEQ or the MCTQ), explore how measures of IIV differ between methods, and collect more information about participants' schedules.

Next, there are also aspects of the current study's experimental design that could have been changed to optimise hypothesis testing. Because participants completed both their test-sessions at the same time of day and were free to choose this test-time themselves, the current study was unable to compare the cognitive control performance of individual participants across different test-times. Additionally, no sleep data was collected prior to the first test-session, so potential effects of prior sleep could not be controlled for. To increase the likelihood of observing interactions between chronotype and test-time on cognitive control performance, future research should test participants at more than one timepoint as well as at timepoints that are more 'extreme' than the test-time range in the current study.

Finally, there are also limitations regarding the conceptualisation and measurement of cognitive control performance in the current study. The current study only used three tasks to measure inhibiting, shifting, and updating performance, and these tasks do not map onto the three components of the unity and diversity framework perfectly. In the framework, several other tasks were used to establish the three-component structure and the tasks within each

component had low correlations with each other (Friedman & Miyake, 2017). Furthermore, the variance associated with the inhibiting component is largely explained by a general cognitive control component. As a result, Friedman & Miyake (2017) argue that using multiple tasks for each component necessary to get accurate and meaningful measures for the components. Moreover, the current study only analysed one reaction-time outcome measure for each of the three tasks and did not account for other aspects of performance such as response accuracy. To get a better picture of the differential effects of sleep on cognitive control components, future research should include performance measures from multiple tasks for each component and address potential differences between different types of performance measures.

4.7. Concluding remarks

The current study sought to explore the role of sleep in cognition by investigating the interplay between IIV in sleep, chronotype, ADHD symptoms, and cognitive control performance in healthy adults. By measuring sleep in the participants' naturalistic sleep environments, the study was the first to test associations between IIV in sleep and performance related to three components of cognitive control: inhibiting, shifting, and updating. In line with previous research, the study found that individuals with earlier/morning chronotypes reported lower SQ on average than individuals with later/evening chronotypes. Furthermore, individuals with more severe ADHD symptoms also reported having lower SQ on average. As there were no significant associations between ADHD symptoms and chronotype in the current study, these findings could suggest that the associations between SQ and ADHD symptoms and SQ and chronotype were independent of each other.

In contrast to previous research, the current study found no interaction effect between chronotypes and time of testing for cognitive control performance. This null finding could potentially be explained by flaws in the experimental design, and thus the interaction effect should be investigated in a more systematic fashion in future research. Although the current study could not directly replicate the finding that ADHD symptoms are associated with poorer cognitive control performance in healthy adults, the results showed a differential effect for the two ADHD symptomology dimensions of inattention and hyperactivity-impulsivity. In line with previous findings, inattention was associated with poor performance in the task that demanded flexible allocation of attention based on two rulesets.

Finally, the current study could not draw any conclusions about the differential effects of sleep on cognitive control components and could not find strong associations between IIV

in sleep and cognitive control. Nevertheless, the role of sleep IIV in cognition should not be overlooked, as variability is a key aspect of sleep in both healthy and clinical populations. The current study serves as an example of how IIV in sleep can be investigated with high ecological validity, and it highlights the need to consider both variability, quality, and timing in investigations of sleep and cognition.

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