Poorer Sleep Health is Associated with Altered Cognitive Control Processing in Healthy Adults

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Sleep Health and Cognitive Control

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Abstract

This study investigated how proactive and reactive cognitive control processing in the brain was associated with habitual sleep health. BOLD fMRI data was acquired from 81 healthy adults with normal sleep (41 females, age 20.96 - 39.58 years) during a test of cognitive control (Not-X CPT). Sleep health was assessed in the week before MRI scanning, using both objective (actigraphy) and self-report measures. Multiple measures indicating poorer sleep health - including later/more variable sleep timing, later chronotype preference, more insomnia symptoms and lower sleep efficiency - were associated with stronger and more widespread BOLD activations (adjusted for age, sex, education, and fMRI task performance). Poorer sleep health was predominantly associated with stronger *reactive* cognitive control activation, indicating an overall "hyper-reactive" brain state. Analysis of time-on-task effects showed that, with longer time on task, poorer sleep health was predominantly associated with increased *proactive* cognitive control activation, indicating recruitment of more neural resources over time. Finally, shorter objective sleep duration was associated with poorer task performance and lower BOLD activation with time on task. In conclusion, even in normal sleepers, relatively poorer sleep health is associated with altered cognitive control processing, possibly reflecting compensatory mechanisms and / or inefficient neural processing.

Keywords: *Continuous Performance Test, Executive Function, Magnetic Resonance Imaging, Neuropsychology, Sleep*

Cognitive control underlies the regulation of thoughts, actions, and emotions, and relies on rapid, dynamic communication between widespread brain regions (Badre 2008; Braver 2012; Diamond 2013; Cole et al. 2014; Menon et al. 2020). Sleep is vital for brain health, everyday functioning, and quality of life (Walker and Stickgold 2006; Mignot 2008; Palmer and Alfano 2017; Tahmasian et al. 2020). Both cognitive control dysfunction and sleep-wake disturbances are transdiagnostic risk factors for developing mental health problems (McTeague et al. 2016; Freeman et al. 2020; Wainberg et al. 2021), and associated with negative outcomes across neurological and psychiatric disorders (Goschke 2013; Snyder et al. 2015; McTeague et al. 2016). However, we still lack knowledge on potential links between cognitive control function and normal sleep in the general adult population, as most studies on this topic have focused on clinical, adolescent, or aging populations, and / or have included experimental manipulations of sleep or circadian rhythm (Lim and Dinges 2010; Scullin and Bliwise 2015; Krause et al. 2017; Lowe et al. 2017; Tahmasian et al. 2021).

Even in"normal sleepers" (persons without sleep or mental health complaints), there is considerable inter- and intraindividual variability in habitual sleep patterns and sleep need, i.e., sleep health (Buysse 2014; Beattie et al. 2015; Allen et al. 2018). Sleep health is inherently multidimensional - encompassing the duration, efficiency, timing, and subjective perception of sleep - and may be assessed via self-report (e.g., questionnaires) and objective (e.g., polysomnography, actigraphy) measures (Buysse 2014). Better sleep health is characterized by having a sleep duration, sleep consistency, and sleep timing in line with recommendations (Hirshkowitz et al. 2015; Ohayon et al. 2017), and reporting little or no problems with sleep or daytime alertness. Meanwhile, indicators of poorer sleep health, including short sleep duration, poor sleep quality, and later chronotype (a preference for later sleep timing), have been linked with poorer health outcomes (Itani et al. 2017; Knutson and von Schantz 2018; Dong et al. 2019; Freeman et al. 2020).

To date, most studies on sleep and cognitive performance have relied on retrospective self-report assessment of sleep duration and/or quality (Buysse 2014; Scullin and Bliwise 2015; Tahmasian et al.

2021; van de Langenberg et al. 2022). This limits the interpretation of results, as self-report measures are influenced by recall bias, individual response style, personality traits, and cognitive and mental health status (Manconi et al. 2010; Kay et al. 2013; Herbert et al. 2017; Rezaie et al. 2018; Robbins et al. 2021; Scarlett et al. 2021; Trimmel et al. 2021). Furthermore, self-report versus objective measures of sleep are often uncorrelated (Klumpp et al. 2017; Thurman et al. 2018; Bernstein et al. 2019; Stefansdottir et al. 2020; Scarlett et al. 2021). The focus on singular measures of sleep health also makes it difficult to compare results between studies. For example, several studies have found that both shorter and longer *self-reported* sleep durations, as compared to the recommended number of hours for different age groups (Hirshkowitz et al. 2015), are linked to poorer cognitive performance (Ferrie et al. 2011; Lo et al. 2016; Richards et al. 2017; van Oostrom et al. 2018; Wild et al. 2018; Cheng et al. 2020; Ding et al. 2020; Ma et al. 2020). However, studies using *objective* sleep assessment - which are far fewer in number - have not identified a clear-cut relationship between sleep duration and cognitive performance (Kato et al. 2018; Bernstein et al. 2019; Stefansdottir et al. 2020; Scarlett et al. 2021). In fact, other dimensions of sleep health, in particular, the timing and variability of sleep, appear to be at least as closely linked to cognitive and brain function as sleep duration itself (Fuligni and Hardway 2006; Mezick et al. 2009; Lemola et al. 2013; Bei et al. 2016; Owens et al. 2016; Molzof et al. 2018; Facer-Childs et al. 2019; Gomez Fonseca and Genzel 2020; Hershner 2020; Lunsford-Avery et al. 2020; Zhang et al. 2020; Bredeli et al. 2022; Stefansdottir et al. 2022). To further elucidate the links between sleep, cognitive performance, and brain function, there is a need to include multidimensional assessment of sleep health within the context of normal, habitual sleep (van de Langenberg et al. 2022; Schiel et al. 2022).

The interplay between sleep and cognitive function is likely mediated by brain structure and functioning (Avinun et al. 2017; Cheng et al. 2018; Liu et al. 2018; Alfini et al. 2020; Grumbach et al. 2020; Tahmasian et al. 2020). Measures of poorer sleep health have been associated with a less robust structural brain architecture (i.e., lower white matter integrity, lower gray matter volume, and increased cortical thinning) (Joo et al. 2013; Weber et al. 2013; Sexton et al. 2014; Khalsa et al. 2017; Fjell et al.

2020; Grumbach et al. 2020; Chen et al. 2021; Wang et al. 2021) as well as altered functional connectivity within and between widespread brain regions (Curtis et al. 2016; Khalsa et al. 2016; Cheng et al. 2018; Tashjian et al. 2018; Lunsford-Avery et al. 2020). In task-based functional magnetic resonance imaging (fMRI) studies, shorter habitual sleep has been associated with reduced frontal, occipital, and insular blood oxygen level-dependent (BOLD) activations during affective regulation (Dimitrov et al. 2021), threat perception (Tashjian and Galván 2020), and risky decision making (Uy and Galván 2017). Poorer self-reported sleep quality has been linked to lower activation within the insular and anterior cingulate cortices during emotion processing (Klumpp et al. 2017: 2017; Guadagni et al. 2018), and inconsistent sleep timing has been linked to less occipital activation and worse task performance during high working-memory loads (Zhang et al. 2020). In sum, the emerging pattern is that indicators of poorer sleep health are associated with altered functional and structural characteristics of specific brain regions important for cognitive control function (Dosenbach et al. 2008; Olsen et al. 2013; Cole et al. 2014). However, there is a lack of theoretical frameworks to aid interpretation of extant studies, which were generally not designed to target cognitive control processing and are based on heterogeneous study samples.

The *dual mechanisms framework of cognitive control* focuses on the temporal aspects of cognitive control processing (Braver 2012). In this framework, *proactive* cognitive control supports processing occurring over relatively longer time periods, such as the maintenance of goal-relevant information and continuous monitoring of incoming stimuli. *Reactive* cognitive control underlies more rapid processing occurring on a trial-to-trial basis, and typically engages when a conflicting or interfering stimulus is detected. Anatomically, these temporal modes of cognitive control rely on distinct, yet closely interacting brain networks (Seeley et al. 2007; Dosenbach et al. 2008; Braver 2012; Olsen et al. 2013; Cole et al. 2014; Cai et al. 2016), and converge on “core” control regions including fronto-parietal, insular, and subcortical areas (Dosenbach et al. 2008; Niendam et al. 2012; Olsen et al. 2013; Cole et al. 2014). Optimal cognitive functioning is believed to rely on the dynamic interplay between proactive and

reactive cognitive control processes, where one type of processing may be more beneficial depending on context (Burgess and Braver 2010; Braver 2012; Lesh et al. 2013; Vanderhasselt et al. 2014; Olsen et al. 2015; Kleerekooper et al. 2016; Huang et al. 2017; Olsen et al. 2018). A relative increase in proactive cognitive control processing is associated with healthy brain development (Staub et al. 2014; Chevalier et al. 2015; Kubota et al. 2020; Niebaum et al. 2020), and has been linked to compensatory mechanisms associated with better every day cognitive control function in people with moderate/severe traumatic brain injury (Olsen et al. 2015). On the other hand, a relative increase in reactive cognitive control processing has been linked to poorer white matter organization, lower fluid intelligence, as well as higher levels of anxiety and stress (Fales et al. 2008; Burgess and Braver 2010; Schmid et al. 2015; Olsen et al. 2018; Husa et al. 2022).

Another temporal aspect of cognitive control function is the effect of time on task, typically observed as decline in performance and vigilance with sustained task performance (Langner and Eickhoff 2013). Such effects have been linked to mental fatigue and depletion of cognitive resources over time (Lim et al. 2010). Proactive cognitive control processing seems to be more sensitive to time-on-task effects (Olsen et al. 2013; Olsen et al. 2015), possibly due to having a higher metabolic cost (engaging more wide-spread brain areas over longer time periods), and consequently being more taxing to uphold over time (Burgess and Braver 2010; Braver 2012). Importantly, time-on-task effects are exacerbated by sleep deprivation (Hudson et al. 2020), and the two have been linked to brain activity changes in overlapping areas, suggesting possibly shared underlying mechanisms (Asplund and Chee 2013; Satterfield et al. 2017). Investigating associations between time-on-task effects and habitual sleep health may therefore shed light on more subtle neural processes underlying the association between sleep and cognitive function in the healthy brain.

The main aim of this study was to investigate whether and how proactive and reactive cognitive control processing in the brain are associated with sleep health in adult, “normal sleepers”. To capture the multidimensionality of sleep health, we collected a comprehensive selection of objective (actigraphy-

based) and self-reported (questionnaire-based) measures, encompassing those measures which have been most often used in previous literature (e.g., Beattie et al. 2015; Klumpp et al. 2017; Grumbach et al. 2020; Zhang et al. 2020). To assess cognitive control processing, we used a well-validated, fMRI-adapted continuous performance test (Olsen et al. 2013; Olsen et al. 2015; Olsen et al. 2018) which was specifically developed to assess the temporal dynamics of cognitive control, including time-on-task effects. This is the first study to investigate how sleep health may affect proactive and reactive cognitive control processing in the brain, and our approach was therefore explorative. We first tested the associations between overall proactive and reactive cognitive control brain activations and the different aspects of sleep health. We then examined how cognitive control activations changed as an effect of time on task, and whether such changes were related to any of the sleep health measures. Age, sex, years of education, as well as fMRI task performance were selected as covariates a-priori, to control for possible confounding factors related to sleep health, cognitive control function, and BOLD activation (Price et al. 2006; Yarkoni et al. 2009; Olsen et al. 2015; Schiel et al. 2022; Wassing and D'Rozario 2022).

Materials and Methods

Study Design and Procedure

Data were collected as part of a randomized controlled trial spanning three weeks. The current study uses data from the first week of this trial (baseline - before randomization or any intervention). The study had a prospective design, which included two on-site visits separated by seven days (questionnaires and cognitive testing at T1, fMRI at T2; see Figure 1a). Naturalistic sleep-wake data were recorded between visits using actigraphy and sleep diaries. Participants were asked to "sleep as usual" throughout the study period. At T1, participants performed a standardized test of cognitive control function and completed questionnaires assessing demographic information and sleep health. They received their actigraph and sleep diary and were instructed to keep the actigraph on 24/7 throughout the study period. At T2, participants completed a continuous performance test during fMRI (Not-X-CPT, Figure 1b) and were

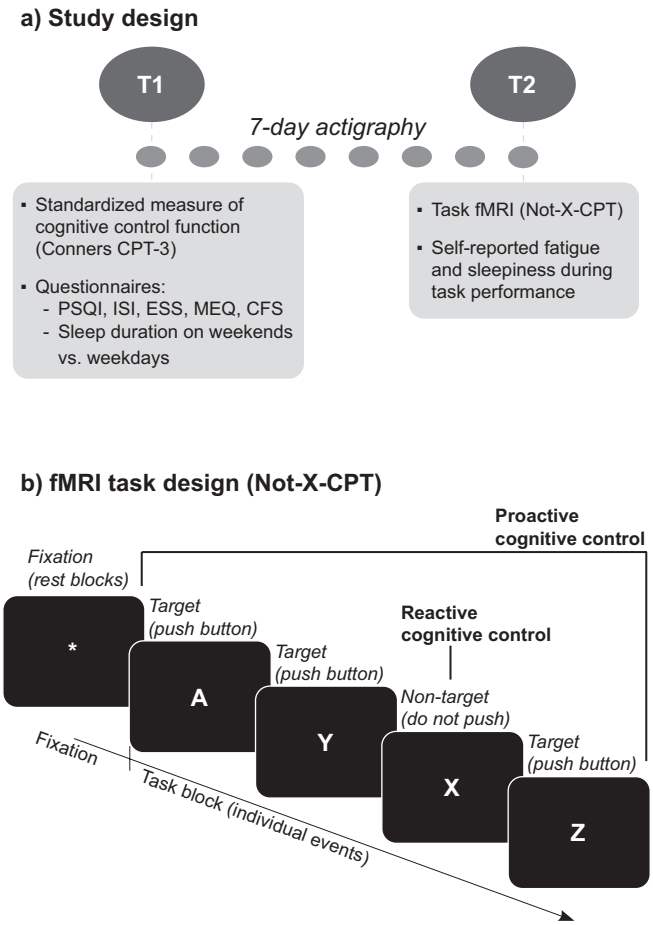

Figure 1. Overview of Study Design and Sleep Health Measures. a) Participants completed two study visits, and naturalistic, habitual sleep was measured during the 7 nights between visits using actigraphy. Visits were at the same time of day (between 8 AM and 3 PM) for each participant. At T1, participants completed a computerized, standardized test of cognitive control (Conners CPT-3) as well as a series of validated questionnaires on sleep health and fatigue. They also received actigraphs and sleep diaries (used for quality control of actigraphy data). At T2, participants completed a ~30 minute task fMRI session, and were asked to report on their current level of mental fatigue and sleepiness halfway through the task. b) To test cognitive control function, we used a 'Not-X-CPT' task adapted to a mixed block/event-related fMRI design (Olsen et al. 2013). Individual letters (A-Z) were presented consecutively, in pseudo-random order, on the screen. Participants were asked to respond as quickly and accurately as possible to every letter appearing (targets), except for the letter X (non-target). Task blocks vs. fixation stars were contrasted to study proactive cognitive control, and non-targets versus targets were contrasted to study reactive cognitive control. CPT = Continuous Performance Test; MEQ = Morningness-Eveningness Questionnaire; PSQI = Pittsburgh Sleep quality Index; ISI = Insomnia Severity Index; ESS = Epworth Sleepiness Scale; CFS = Chalder Fatigue Scale; fMRI: functional magnetic resonance imaging.

asked to report their levels of mental fatigue and sleepiness halfway through the task. All on-site testing was conducted between 8 AM and 3 PM. Participants were scheduled for testing at the same time of day on both visits to control for circadian effects in each individual. They were also asked to avoid caffeine and nicotine in the last two hours before testing.

Participants

Healthy volunteers without sleep complaints were recruited via online advertisements, public posters, and word-of-mouth in and around the city of Trondheim, Norway. Inclusion and exclusion criteria were assessed in a structured phone interview. Inclusion criteria were: 1) being between 20-40 years of age, 2) having normal or corrected-to-normal vision, and 3) fluency in the Norwegian language. Exclusion criteria were: 1) contraindications for MRI, 2) any chronic or ongoing medical, neurological or mental illness (including sleep disorders), and 3) obvious factor(s) that would likely influence results or adherence to the study protocol, such as night work or shift work, irregular sleep or perceived sleep problems, excessive alcohol use, previous habitual use of psychotropic drugs (e.g. marijuana, stimulants), or any use of such drugs in the last three months. Specific exclusion criteria concerning sleep were based on the Research Diagnostic Criteria for Normal Sleepers (Edinger et al. 2004) and operationalized at levels 1 & 2 according to suggestions by Beattie et al. (2015).

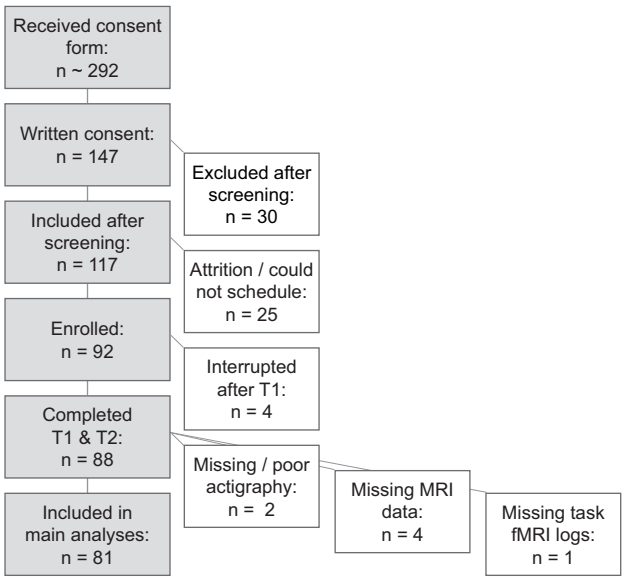

Figure 2. Overview of Inclusion Process. T1 = visit 1, T2 = visit 2, fMRI = functional magnetic resonance imaging.

A flowchart of the inclusion process is presented in Figure 2. Of the 92 participants enrolled in the study, four were excluded due to only having completed T1, four were excluded due to technical problems with the MRI scanner, one was excluded due to technical problems with logging task fMRI behavioral data, and two were excluded due to missing (n = 1) or poor (n = 1) actigraphy data. This left a final sample of 81 participants who completed both study visits and had usable fMRI, actigraphy and questionnaire data (41 women; mean age = 27.82 years, SD = 5.42; mean education = 16.28 years, SD = 2.34).

The study was approved by the Regional Committee for Medical and Health Research Ethics in Central Norway (REK number 2017/85) and conducted in accordance with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards.

Measures of Sleep Health, Fatigue, and Standardized Measure of Cognitive Control Function

Objective Measures of Sleep Health

Objective, prospective assessment of participants' habitual sleep was obtained using actigraphy (Actiwatch Spectrum Pro, Philips Respironics Inc., Murrysville, USA). The actigraphs recorded the participants' light exposure and daily activity via a light sensor and a piezoelectric accelerometer, respectively. Data were recorded using 15-second epoch lengths. Participants were asked to use an event marker on the actigraph to indicate when they lay down to sleep each night. Sleep-wake periods were automatically classified using the Actiware software (Philips Actiware 6.0.9) using a medium sensitivity setting, and thereafter systematically inspected by trained study staff (Boyne et al. 2013). For qualitative assessment of the actigraphy data, participants filled out sleep diaries based on the Consensus sleep diary each morning (Carney et al. 2012). In cases of obvious misclassifications of rest periods, or large discrepancies between actigraphy and sleep diary data, rest interval onsets and offsets were manually adjusted according to event markers, activity, light levels and / or sleep diaries using a standardized procedure (Follesø et al. 2021).

For each night, the following variables were extracted from the raw actigraphy data: sleep onset, sleep offset, sleep duration (total sleep time during the night, excluding brief wakes after sleep onset (WASO)), sleep efficiency (sleep duration divided by time in bed) and sleep onset latency (interval between going to bed and sleep onset (SOL)). Sleep midpoints were calculated by dividing the sleep interval (sleep offset - sleep onset) by 2 and subtracting the resulting duration from the sleep offset time point. Individual, 7-day averages of sleep duration, midpoint, efficiency and SOL were calculated for use in statistical analyses, and the standard deviation of sleep duration and midpoint were used as measures of the intra-individual variability of sleep. Of the 81 subjects included, 80 had seven nights of actigraphy data and one had six nights.

Self-Report Measures

Questionnaire-based, retrospective measures of sleep health were obtained at T1 using a selection of validated and commonly used inventories. The Pittsburgh Sleep Quality Index (PSQI) (Buysse et al. 1991) was used to assess sleep quality (global score) and self-reported habitual sleep duration (item 4), the Insomnia Severity Index (ISI) (Morin 1993) was used to assess insomnia symptoms, the Epworth Sleepiness Scale (ESS) was used to assess daytime sleepiness (Johns 1991), and the Horne-Östberg Morningness / Eveningness Questionnaire (MEQ) (Horne and Ostberg 1976) was used to assess chronotype preference. Participants were also asked to report their usual duration of sleep on weekdays (WD) versus weekends (WE), and the difference score was used to estimate self-reported sleep variability (WE-WD difference). Finally, the Chalder Fatigue Scale (CFS) was used to assess problems with fatigue in daily life (Chalder et al. 1993). For the PSQI, the global score was calculated where higher scores indicate poorer sleep quality. For the ISI and ESS, the sum score of all items was used, where higher scores indicate more problems with insomnia symptoms and daytime sleepiness. For the MEQ, the global score was calculated, where higher scores indicate a stronger preference for morningness. For alignment with actigraphy midpoint data, the MEQ global score was inverted in all correlation analyses such that higher scores indicate a preference for eveningness (later chronotype preference). For the CFS, the mean

of all items was calculated, where a mean of 1 indicates no current problems with fatigue and higher scores indicate more problems.

Standardized Measure of Cognitive Control Function

To obtain a standardized measure of cognitive control function, participants completed the widely used Conners Continuous Performance Test 3 (CCPT-3) at T1 (Conners 2014). In this computerized task, a series of letters (A-Z) appear on the screen in random order, and participants are asked to press the spacebar everytime they see a letter, except for the letter X ("Not-X-CPT"). Participants were told to respond as quickly and accurately as possible. Using norms from the test provider (MHS Scoring Software, version 5.6.0, Multi-Health Systems Inc., Canada), T-scores for hit reaction time (hit RT), hit reaction time standard deviation (hit RT SD), commission errors (non-targets responded to), omission errors (targets missed), as well as the derivative measure detectability (d'), were extracted for a descriptive characterization of the participants.

Neuroimaging Protocol and Data Acquisition

fMRI task

To assess the neural correlates of cognitive control processing, we used a well-validated Not-X-CPT task which was specifically developed for a mixed block/event-related fMRI design and allows for the study of proactive and reactive cognitive control processing, including time-on-task effects (Olsen et al. 2013; Olsen et al. 2015; Olsen et al. 2018). The paradigm and stimulus presentation are described in greater detail elsewhere (Olsen et al. 2013). Briefly, letters from A-Z were presented for 250 ms each, with an interstimulus interval varying between 1, 2 and 4 seconds. Participants were asked to respond whenever they saw a letter appear (targets), but to withhold their response when the letter X appeared (non-targets) (Figure 1b). They were told to respond as quickly and accurately as possible. Response speed and accuracy were recorded using fiber-optic response grips (Nordic Neurolabs, Bergen, Norway) held in the participants' dominant hand (determined using the Edinburgh Handedness Inventory (Oldfield 1971)). In total, 480 stimuli were presented, and non-target (X) frequency was 10%. The task paradigm consisted of

two task runs (duration ~14.5 minutes each), which were counterbalanced with regard to stimulus presentation to allow for investigation of time-on-task effects. The task was presented on an HDMI monitor (Nordic Neurolabs, Bergen, Norway) via the EPrime 3.0 software (Psychology Software Tools, Pittsburgh, PA). Participants viewed the HDMI screen via a coil mounted mirror (Siemens, Erlangen, Germany). The following behavioral measures were extracted from the behavioral logs and were collapsed across the two runs: average hit RT and hit RT SD in milliseconds (target stimuli), number of omissions (targets missed), number of commission errors (non-targets responded to). Additionally, to investigate time-on-task effects, individual change-scores (Δ) were calculated for each response measure by dividing each task run into four time epochs and subtracting responses during epoch 1 from responses made during task epoch 4.

Mental Fatigue and Sleepiness During fMRI Task Performance

To assess subjective levels of mental fatigue and sleepiness during fMRI task performance, participants were asked the following questions via the MRI speaker system halfway through the task: 1) "On a scale from 1 to 10, how mentally fatigued do you feel right now, where 1 equals 'not at all' and 10 equals 'severely'?", and 2) "On a scale from 1 to 10, how sleepy do you feel right now, where 1 equals 'extremely alert' and 10 equals 'can't keep awake'?". The question about sleepiness was adapted from the 10-point version of the Karolinska Sleepiness Scale (KSS) (Akerstedt and Gillberg 1990; Shahid et al. 2011).

MRI Data Acquisition and Preprocessing

MRI data were acquired on a 3T Skyra scanner using a 32-channel head-coil (Siemens, Erlangen, Germany). For the task fMRI, two series of multiband T2*-weighted echo planar images (EPI) with whole-brain coverage were acquired (947 volumes; phase encoding direction = A-P; SMS = 6; TR = 0.970 s; TE = 34.2 ms; FA = 60°; voxel size = 2.5 mm isotropic, FOV = 260 mm). For correction of susceptibility induced distortions, two series of spin-echo planar images in opposite phase encoding directions (AP-PA) were acquired after each fMRI run (3 volumes; TR = 7.33 s; TE = 60.8 ms; FA = 90°;

voxel size = 2.5 mm isotropic, FOV = 260 mm). For anatomical co-registration, a high-resolution, 3D T1-weighted MPRAGE volume was acquired (TR = 2.3 s; TE = 29.2 ms; FA = 9°, voxel size = 1 x 1 x 1.2 mm; FOV = 256 mm).

Anatomical T1-weighted (T1w) images were preprocessed using fMRIPrep version 20.2.3 (Esteban et al. 2018; Esteban et al. 2019) (RRID:SCR_016216), which is based on Nipype 1.6.1 (Gorgolewski et al. 2011; Gorgolewski et al. 2018) (RRID:SCR_002502). The T1w images were corrected for intensity non-uniformity with N4BiasFieldCorrection (Tustison et al. 2010), distributed with ANTs 2.3.3 (Avants et al. 2008) (RRID:SCR_004757) and thereafter skull-stripped with a Nipype implementation of the antsBrainExtraction.sh workflow (from ANTs), using OASIS30ANTs as a target template. Brain tissue segmentation of cerebrospinal fluid (CSF), white-matter (WM) and gray-matter (GM) was performed on the brain-extracted T1w image using FAST (FSL 5.0.9, RRID:SCR_002823) (Zhang et al. 2001). Brain surfaces were reconstructed using recon-all (Dale et al. 1999) (FreeSurfer 6.0.1, RRID:SCR_001847), and the T1 brain mask was refined with a custom variation of the method to reconcile ANTs-derived and FreeSurfer-derived segmentations of the cortical gray-matter of Mindboggle (Klein et al. 2017) (RRID:SCR_002438). The resulting brain mask was used to run brain extraction on the T1w image using BET (Smith 2002), after mean dilation of non-zero voxels to ensure full brain coverage in the resulting brain-extracted image.

FMRI data pre-processing was carried out using FEAT (FMRI Expert Analysis Tool) Version 6.00, part of FSL (FMRIB's Software Library, Oxford, UK) (Jenkinson et al. 2012). First, to improve registrations from functional to structural space, single-band reference images from the multiband EPI sequences were used, and susceptibility distortion correction was applied by running TOPUP (Andersson et al. 2003) on the spin-echo planar images, followed by b0 unwarping as implemented in FEAT. Registration of functional images to the preprocessed T1w image/the 2-mm MNI standard space template was carried out using FLIRT with boundary-based registration/12 degrees of freedom (Jenkinson and Smith 2001; Jenkinson et al. 2002) and further refined using FNIRT nonlinear registration with a 10-mm

warp resolution (Andersson et al. 2007). The following pre-statistical processing steps were applied: motion correction using MCFLIRT (Jenkinson et al. 2002); non-brain removal using BET (Smith 2002); spatial smoothing using a Gaussian kernel of FWHM 6 mm and grand-mean intensity normalization of the entire 4D dataset by a single multiplicative factor. High-pass temporal filtering (Gaussian-weighted least-squares straight line fitting) was applied using sigmas of 50.0 s for block-related analyses, and 25.0 s for event-related analyses.

Statistical Analysis

IBM SPSS Statistics version 27 was used to calculate the daily sleep midpoints, as well as 7-day averages and standard deviations for all actigraphy outcome measures. Actigraphy, fMRI performance and questionnaire data were then further analyzed and visualized in RStudio (version 1.4.1103; R version 4.0.3) using the *tidyverse* (Wickham et al. 2019)*, lubridate* (Grolemund and Wickham 2011), *psycho* (Makowski 2018)*, psych* (Revelle 2022) and *corrplot* (Wei and Simko) packages. Functional MRI data were analyzed using FSL FEAT (FMRI Expert Analysis Tool) Version 6.00 (FMRIB's Software Library, Oxford, UK).

fMRI Task Performance

Individual mean hit RT, hit RT SD, number of omissions, commissions, and detectability were calculated for use in further analyses. Detectability was calculated using the *psycho* package in R (Makowski 2018). Both overall task performance (collapsed across the two task runs) and time-on-task changes (Δ) were calculated for all measures. To investigate associations between fMRI performance and the different measures of sleep health, partial correlation analyses (adjusted for age, sex, and education, determined a-priori) were performed (n = 78, alpha level = 0.05). Spearman's correlation coefficient was used to account for non-normally distributed data.

Whole-brain fMRI Analyses

Prior to statistical analysis, all individual runs were checked for excessive motion. The values for relative root mean square displacement were very low (mean: 0.1 mm: max: 0.25 mm), and all subjects were

therefore included in subsequent analyses. First, for each task run (run 1 and run 2), a single-subject general-linear model (GLM) was applied using FSL's FILM with local autocorrelation correction (Woolrich et al. 2001). The following contrasts were computed for the task as a whole: task blocks > fixation blocks (Proactive Cognitive Control), and non-targets (X) > targets (A-Z) (Reactive Cognitive Control). The contrasts from the two task runs were combined using a fixed effects model for each participant, to generate mean task activation across both task runs. To investigate time-on-task effects, each task run was divided into four time epochs in which Proactive / Reactive Cognitive Control BOLD activations were estimated. Time-on-task effects across runs (ΔProactive Cognitive Control and Δ Reactive Cognitive Control) were then calculated for each participant using a fixed effects model by contrasting time epoch 1 with time epoch 4.

Group average, whole-brain analyses were carried out using a mixed-effects model (FLAME 1 and 2) (Woolrich et al. 2004). Group Z statistical images were obtained using a cluster threshold of Z > 3.1 and a (corrected) cluster significance threshold of P=0.05 (Worsley 2011). Separate GLMs were run to test group mean activations for (1) Proactive Cognitive Control and Reactive Cognitive Control, (2) Δ Proactive Cognitive Control and ΔReactive Cognitive Control (time-on-task change), as well as (3) associations with sleep health measures. All models were adjusted for age, sex, education, and fMRI task performance (hit RT, omissions, and commissions), to control for confounding factors related to sleep health, cognitive control function, and BOLD signal (Price et al. 2006; Yarkoni et al. 2009; Shanmugan and Satterthwaite 2016; Gurvich et al. 2018). Models of Proactive and Reactive Cognitive Control activations were adjusted for overall (whole-task) performance, and models of ΔProactive Cognitive Control and ΔReactive Cognitive Control (time-on-task change) were adjusted using performance Δ scores. The six motion parameters from FEAT were also added to each model as separate regressors. For associations with sleep health, the following measures were demeaned and included as covariates in separate analyses: actigraphy-derived mean sleep duration, sleep duration SD, mean midpoint, midpoint SD, sleep efficiency, and SOL; self-reported sleep duration (PSQI), chronotype (MEQ inverse global

score), sleep variability (WE-WD difference), sleep quality (PSQI), insomnia symptoms (ISI), problems with fatigue in daily life (CFS), daytime sleepiness (ESS), mental fatigue during the task, and sleepiness during the task (KSS).

For statistically significant results, maximum Z-values with up to five local maxima and the size of clusters (number of voxels) in standard 2x2x2mm MNI space were extracted for descriptive purposes. Anatomical locations were determined using the FSLeyes software, version 1.3.0, with the incorporated Harvard Oxford cortical and subcortical structural brain atlases and visual inspection.

Results

Demographics and Standardized Cognitive Control Function

An overview of demographic variables and scores on the standardized assessment of cognitive control function (CCPT-3) can be found in Table 1. For the CCPT-3, lower scores reflect better performance. On the group level, participants had relatively faster reaction times while maintaining an expected number of errors as compared to the norm group (hit RT: mean T-score = 43.01, SD = 6.09; commission errors: mean T-score = 50.99, SD = 9.53).

Table 1 *Demographics and Standardized Cognitive Control Function*

Variable	n	Mean (SD)	Min	Max
Demographics				
Female / Male	41 / 40	n/a	n/a	n/a
Age (years)	81	27.82 (5.42)	20.96	39.58
Years of education[a]	81	16.28 (2.39)	12	21
Standardized Cognitive Control Function (Conners CPT-3, T-scores)				
Hit RT, mean	81	43.01 (6.09)	33	63
Hit RT, SD	81	41.49 (5.84)	32	66
Omission errors	81	45.60 (2.72)	43	60
Commission errors	81	50.99 (9.53)	36	78
Detectability (d')	81	47.49 (7.39)	32	66

The standardized scores on the CPT-3 have a mean of 50 and a standard deviation of 10. Higher scores indicate poorer performance, and lower scores indicate better performance. [a]Years of education = the number of years corresponding to the highest completed level *or* ongoing level of education (educational attainment). SD = standard deviation; RT = reaction time.

Measures of Sleep Health

Table 2 provides an overview of measures of sleep health. The mean objective sleep duration (averaged over 7 days) was 7.21 hours (SD = 0.66 hours), which is within the range of recommended sleep duration for adults (7 - 8 hours) (Hirshkowitz et al. 2015). The mean sleep efficiency and SOL also indicated overall good quality sleep (≥85% and <30 minutes, respectively) (Ohayon et al. 2017). For sleep timing, the mean midpoint was at 03:52 AM, and mean of scores on the MEQ was 54.29 (SD = 8.86). The distribution of scores on the MEQ showed that a majority of participants fell within the "intermediate" type, with a slight lean toward "moderately morning" (Horne and Ostberg 1976). Finally, the mean scores on the PSQI and ISI suggest a low prevalence of sleep problems (using a cut-off for "normal sleep" of 5 on the PSQI, and 7 for the ISI (Buysse et al. 2008; Morin et al. 2011)). Taken together, the results show that participants had good sleep overall, but that there was inter-individual variability as expected within the normal range.

Table 2 *Sleep Health Measures (N = 81)*

Variable	Mean (SD)	Min	Max
Actigraphy (7-Day Averages)			
Sleep duration, mean (hours)	7.21 (0.66)	4.97	9.01
Sleep duration SD (hours)	0.95 (0.43)	0.28	2.68
Sleep midpoint, mean (time)	03:52 am (53 min)	02:25 am	06:20 am
Sleep midpoint SD (hours)	0.94 (0.42)	0.26	2.23
Sleep efficiency (%)	86.73 (4.04)	73.44	94.78
Sleep onset latency (SOL) (minutes)	20.44 (12.93)	4.18	68.82
Self-Reported Sleep and Fatigue			
Sleep duration (hours)[a]	7.46 (0.61)	6.50	9.50
Sleep quality (PSQI global score)	3.41 (1.79)	0	10
WE-WD sleep duration difference (hours)	1.04 (0.87)	-1	3
Insomnia symptoms (ISI global score)	3.63 (2.84)	0	13
Daytime sleepiness (ESS sum score)	5.12 (2.84)	0	13
Chronotype (MEQ global score)	54.29 (8.86)	73	31
Fatigue (CFS global score)	1.00 (0.31)	0.27	2.09

[a]Assessed using PSQI item 4. SD = standard deviation; PSQI = Pittsburgh Sleep Quality Index; WE-WD = weekend-weekday; ISI = Insomnia Severity Index; ESS = Epworth Sleepiness Scale; MEQ = Horne-Östberg Morning/Eveningness Questionnaire; CFS = Chalder Fatigue Scale.

fMRI Task Performance and Associations with Sleep Health

Behavioral data from the fMRI task are presented in Figure 3 and Supplementary Table 1. Data are presented both for the task as a whole and for time-on-task changes (Δ).

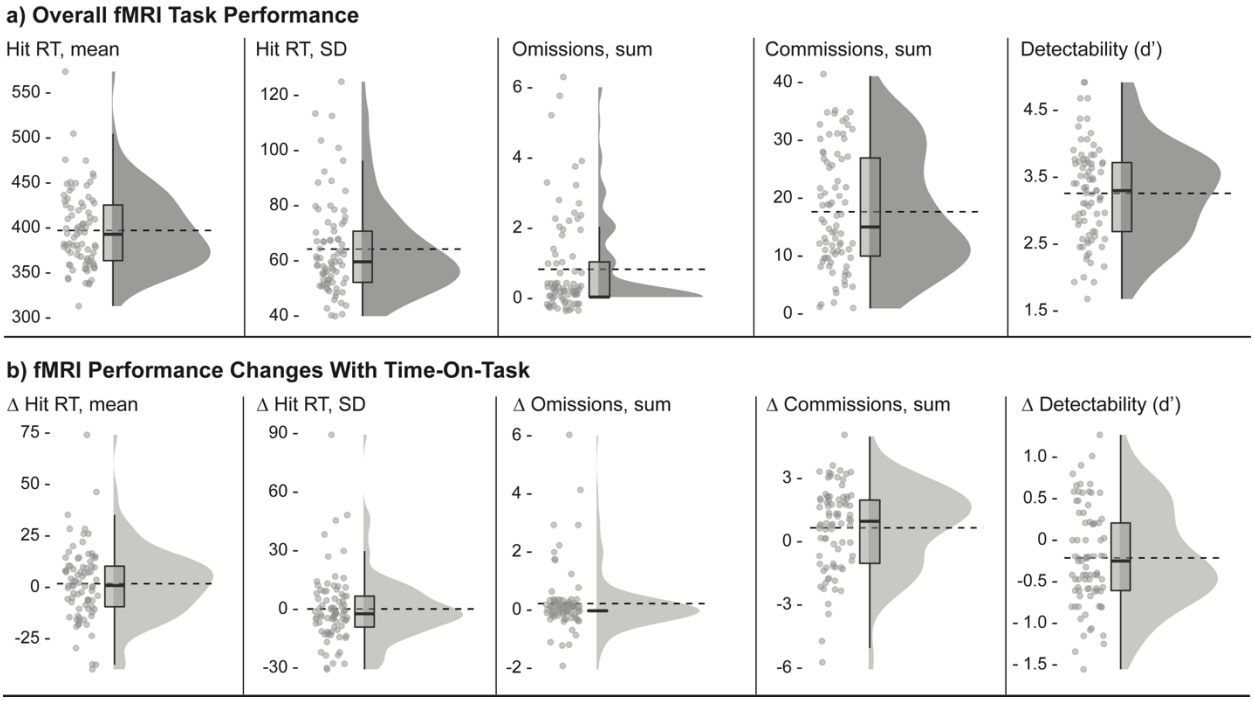

Figure 3. fMRI Task Performance. Individual means for each outcome variable are plotted as raincloud plots (Allen et al. 2021) with overlaid boxplots, as well as dashed horizontal lines indicating group means. Figure a) shows overall task performance and b) shows performance change scores with time on task (time epoch 4 - time epoch 1). Hit RT and hit RT SD refer to target responses (letters A - Z). Omissions refer to missed target letters (A-Z), and commissions refer to pushed non-targets (X). Detectability (d') refers to the ability to discriminate targets from non-targets. fMRI = functional magnetic resonance imaging, RT = reaction time, SD = standard deviation.

Associations between fMRI task performance, measures of sleep health as well as mental fatigue and sleepiness during the task are presented in Figure 4. There were statistically significant associations between actigraphy-derived sleep duration and overall task performance: shorter habitual sleep duration was associated with poorer performance (higher hit RT SD, more errors, and lower detectability). More variable habitual sleep duration (higher SD) was associated with more commission errors and lower detectability. Shorter habitual sleep durations were also associated with poorer performance with time on

task (longer hit RT, higher hit RT SD, and lower detectability). Furthermore, shorter *self-reported* sleep duration was associated with poorer performance with time on task (more omission errors and lower detectability). Finally, relatively later chronotype preference was associated with a lower hit RT SD with time on task, and higher levels of daytime sleepiness were associated with more omissions and longer hit RTs with time on task. There were no other statistically significant associations between fMRI task performance and sleep health measures. The full correlogram - including correlations between the different sleep health measures - can be found in Supplementary Figure 1.

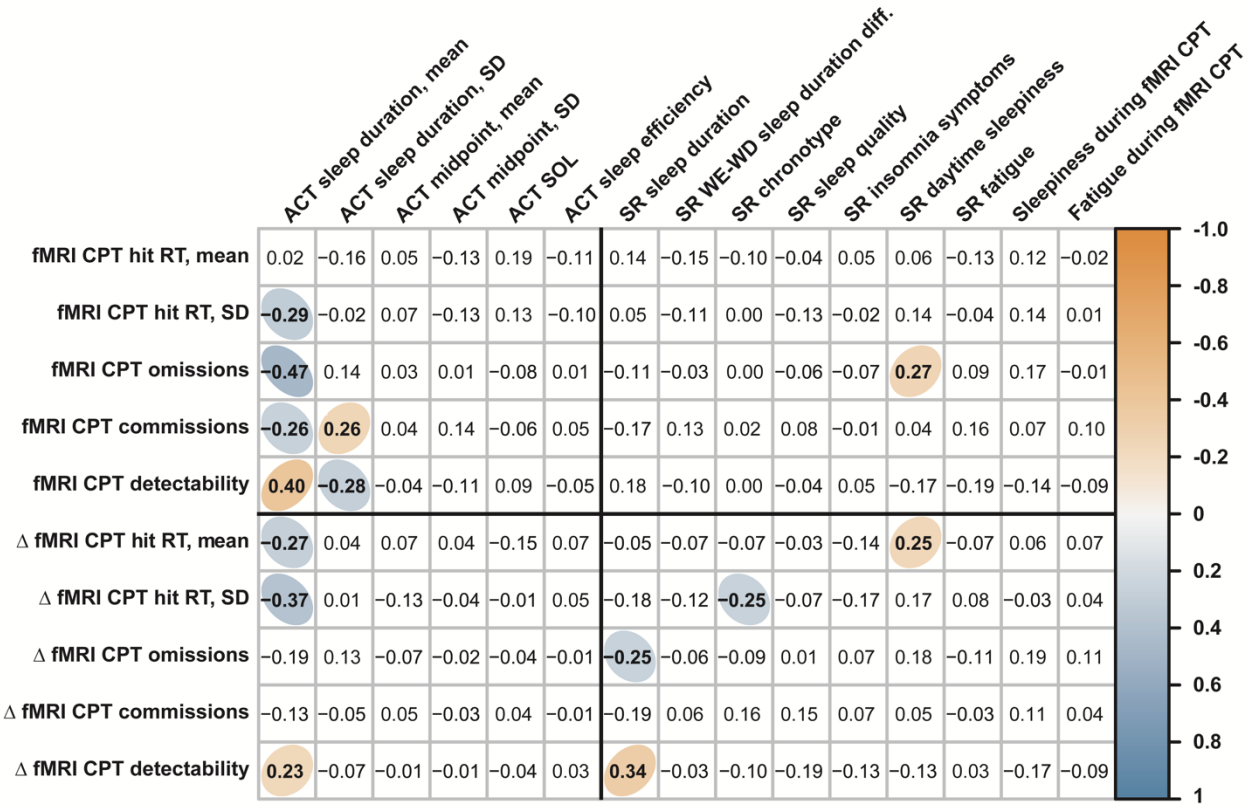

Figure 4. Partial Correlations between Task fMRI Performance and Sleep Health Measures. The correlogram depicts correlation coefficients (Spearman's rho) between fMRI task performance measures (Y axis) and the different sleep health measures (X axis). Measures of overall task performance are listed above the horizontal black line and measures of time-on-task changes (Δ) are listed below the line. Objective measures of sleep (actigraphy-derived) are listed to the left of the vertical black line and self-report measures are listed to the right of the line. Statistically significant correlations ($p < .05$, not corrected for multiple comparisons) are marked with colored ellipses. Positive correlations in orange and negative correlations in blue. ACT = actigraphy; SR = self-reported; fMRI = functional magnetic resonance imaging; CPT = continuous performance test; RT = reaction time, SD = standard deviation.

Whole-Brain fMRI Analyses

Group Mean Proactive and Reactive Cognitive Control Activations

The group average whole-brain analyses revealed widespread Proactive and Reactive Cognitive Control activations, with overlapping activation in fronto-parietal regions, the insular cortex, dorsal striatum, thalamus, and the cerebellum - indicating core regions for cognitive control processing as observed in previous studies (Dosenbach et al. 2008; Niendam et al. 2012; Olsen et al. 2013). With longer time on task, there was a general *decrease* in Proactive Cognitive Control activation within core control regions (which are typically "task-positive" regions), and *increased* Proactive Cognitive Control activations in the precuneus, medial prefrontal cortex, and middle temporal gyrus (typically "task-negative" regions). For Reactive Cognitive Control, there was *increased* activation in core cognitive control areas with time on task, largely overlapping with regions displaying decreased Proactive Cognitive Control activation. This change in activity patterns suggests a relative shift from Proactive to Reactive Cognitive Control processing with increasing time on task. Group mean activations are presented in Supplementary Tables 2, 3 and visualized in Supplementary Figure 2.

Associations between Cognitive Control Processing and Sleep Health

Statistically significant associations between cognitive control activations and sleep health measures are presented in Figures 5, 6 and in Table 3. Most were found for Reactive Cognitive Control activation.

For Proactive Cognitive Control, a more variable sleep midpoint (higher midpoint SD) was associated with higher BOLD activation in the left postcentral gyrus (Figure 5). None of the other sleep health variables was associated with overall Proactive Cognitive Control activation.

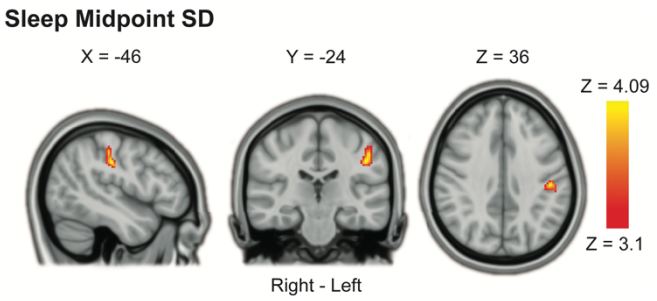

Figure 5. **Positive Association between Proactive Cognitive Control Processing and Sleep Midpoint SD.** More variable sleep midpoint (midpoint SD) was associated with stronger proactive cognitive control activation in the left postcentral gyrus. Results were obtained using a cluster threshold of Z>3.1, p = .05, corrected for multiple comparisons, and are presented on a 1-mm MNI standard space template. SD = standard deviation; MNI = Montreal Neurological Institute.

For Reactive Cognitive Control, later sleep midpoint was associated with greater BOLD activation in the cerebellum, lingual gyrus, thalamus, right precuneus cortex, posterior cingulate gyrus, and right middle frontal gyrus (Figure 6a). A more variable sleep midpoint (higher midpoint SD) was associated with greater Reactive Cognitive Control in the right precentral / postcentral gyrus, as well as the juxtapositional lobule (supplementary motor area) (Figure 6b). Later chronotype preference was associated with greater Reactive Cognitive Control activation in widespread regions encompassing frontal, parietal, and temporal cortex as well as the cerebellum, caudate and thalamus (Figure 6c). Finally, a higher level of insomnia symptoms was associated with greater Reactive Cognitive Control activation in the right precuneus and ventromedial frontal cortex (Figure 6d).

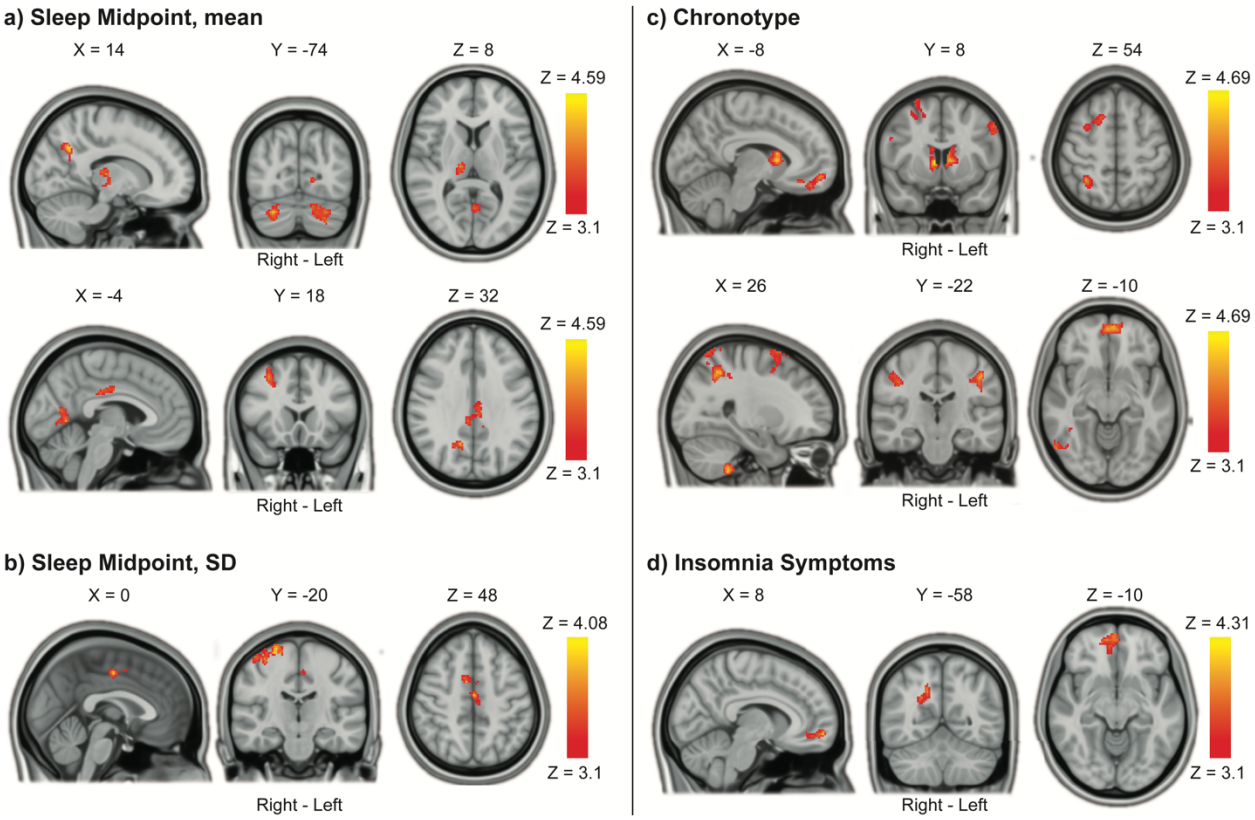

Figure 6. Positive Associations between Reactive Cognitive Control Processing and Sleep Health. a) Later sleep midpoint was associated with higher reactive cognitive control activations in the cerebellum, lingual gyrus, thalamus, right precuneus cortex, posterior cingulate gyrus, and right middle frontal gyrus. b) More variable sleep midpoint (midpoint SD) was associated with higher reactive cognitive control activation in the precentral / postcentral gyrus, and in the juxtapositional lobule (supplementary motor area). c) Later chronotype preference was associated with widespread higher reactive cognitive control activation, including frontal, parietal and temporal cortex as well as the cerebellum, caudate and thalamus. d) Higher levels of insomnia symptoms were associated with higher reactive cognitive control processing in the right precuneus and ventromedial frontal cortex. SD = standard deviation. Results were obtained using a cluster threshold of Z>3.1, p = .05, corrected for multiple comparisons, and are presented on a 1-mm MNI standard space template. SD = standard deviation; MNI = Montreal Neurological Institute.

Table 3 *Positive Associations Between Cognitive Control Activations and Sleep Health*

Contrast / Region	Right / Left	Size (#voxels)	P Value	Peak Z Value	Peak Coordinates (MNI)		
					X	Y	Z
Proactive Cognitive Control: Higher Sleep Midpoint SD							
Postcentral Gyrus	L	198	.0366	4.09	-46	-24	36
Reactive Cognitive Control: Later Sleep Midpoint							
Cerebellum: Right Crus I	R	332	.0020	4.22	28	-74	-32
Cerebellum: Left Crus I & II	L	306	.0032	4.08	-18	-70	-36
Cingulate Gyrus, posterior division	R	266	.0068	4.37	4	-36	26
Lingual Gyrus / Intracalcarine Cortex	L	248	.0096	4.37	-6	-62	2
Precuneus Cortex	R	235	.0123	4.38	14	-60	36
Thalamus	R	226	.0148	3.98	14	-20	8
Middle Frontal Gyrus	R	210	.0204	4.59	32	12	52
Reactive Cognitive Control: Later Chronotype Preference							
Superior Parietal Lobule	R	760	< .0001	-4.69	38	-58	68
Cerebellum: Right VIIIb	R	709	< .0001	-4.40	26	-42	-54
Caudate	R / L	495	.0001	-4.68	8	8	4
Middle Temporal Gyrus, temporo-occipital part	R	367	.0010	-4.38	48	-52	2
Superior Frontal Gyrus	R	311	.0028	-4.41	24	14	72
Inferior Frontal Gyrus, *pars opercularis*	R	282	.0047	-4.03	40	10	22
Frontal Pole / Frontal Medial Cortex	R / L	273	.0056	-4.22	-4	58	-10
Postcentral Gyrus	L	252	.0085	-4.38	-46	-18	30
Postcentral Gyrus	R	242	.0103	-3.95	40	-24	38
Precentral Gyrus	L	228	.0136	-4.08	-52	6	42
Reactive Cognitive Control: Higher Sleep Midpoint SD							
Precentral & Postcentral Gyri	R	592	< .0001	4.08	24	-18	74
Juxtapositional Lobule	R	169	.0488	3.85	0	-12	48

Table 3 Continued

Contrast / Region	Right / Left	Size (#voxels)	P Value	Peak Z Value	Peak Coordinates (MNI)		
					X	Y	Z
Reactive Cognitive Control More Insomnia Symptoms							
Precuneus Cortex	R	247	.0097	3.81	16	-58	36
Frontal Pole / Frontal Medial Cortex	R	201	.0245	4.31	4	58	-8

Results were obtained using a mixed-effects model corrected for multiple comparisons using a cluster threshold of $Z > 3.1$ and a corrected cluster significance threshold of $p < 0.05$. Main peaks within each cluster are reported, and only statistically significant results are included in the table. Anatomical regions for each peak Z value were labeled using visual inspection and the Harvard Oxford cortical / subcortical structural atlases and the Cerebellar atlas (normalized with FLIRT) within the FSL software. MNI = Montreal Neurological Institute, R = Right, L = Left.

Associations between Time-On-Task Effects and Sleep Health

Statistically significant associations between time-on-task changes in cognitive control activation and sleep health measures are presented in Figures 7, 8 and in Table 4. Most associations with time-on-task effects were found for Δ Proactive Cognitive Control activation.

For Δ Proactive Cognitive Control, later sleep midpoint was associated with increased BOLD activation with time on task in the cerebellum, precuneus cortex, and right middle frontal gyrus (Figure 7a). Lower sleep efficiency was associated with increased Proactive Cognitive Control activation in the left paracingulate gyrus / frontal pole with time on task (Figure 7b). More problems with fatigue in daily life were associated with increased Proactive Cognitive Control activation with time on task in widespread areas, including bilateral cerebellum, occipital cortex, precuneus cortex, and frontal pole (Figure 7c). More self-reported sleepiness during task performance was associated with increased Proactive Cognitive Control activation with time on task in the left precentral / postcentral gyrus (Figure 7d). Finally, shorter self-reported sleep duration was associated with *decreased* Proactive Cognitive Control activation with time on task in the left middle temporal gyrus (Figure 8a).

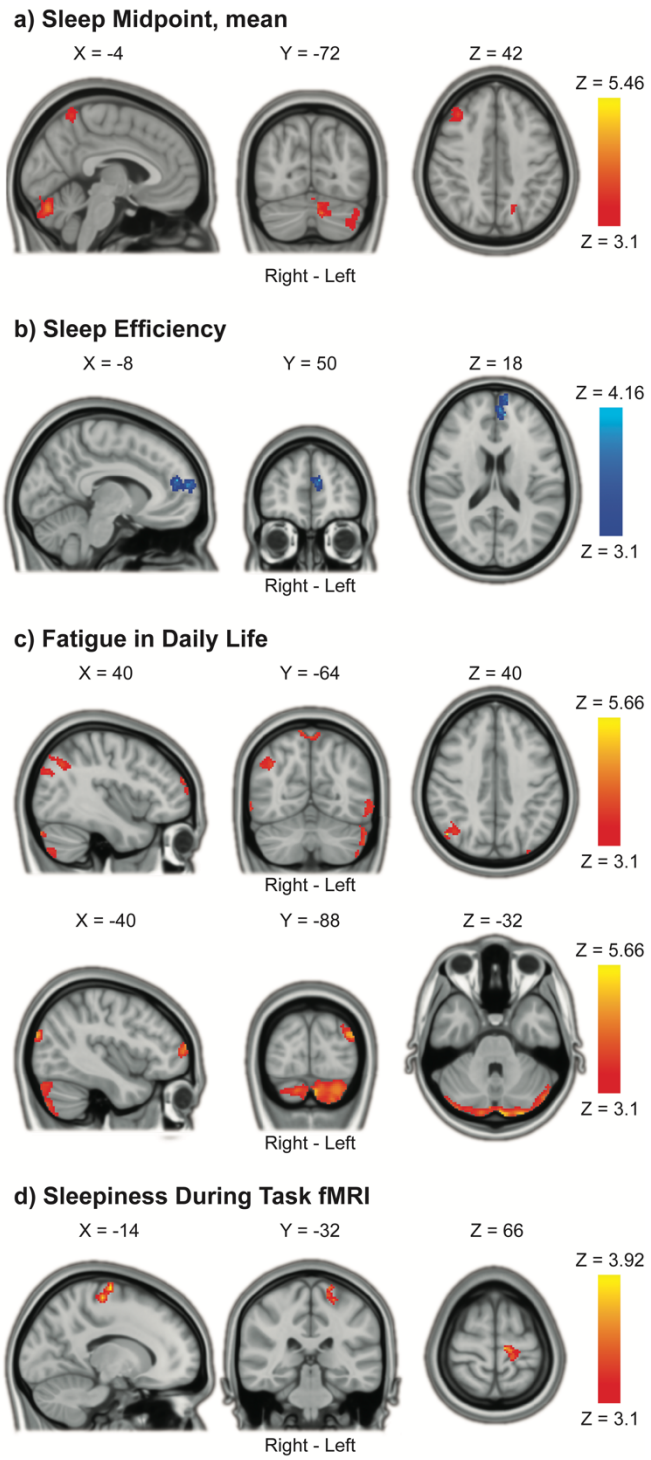

Figure 7. Time-On-Task (TOT) Increases in Proactive Cognitive Control Associated with Sleep Health. a) Later sleep midpoint was associated with increased Proactive Cognitive Control activations in the cerebellum, precuneus cortex and right middle frontal gyrus with TOT. b) Lower sleep efficiency was associated with increased Proactive Cognitive Control activations in the paracingulate gyrus / left frontal pole with TOT. c) More problems with fatigue in daily life were associated with increased Proactive Cognitive Control activations in widespread areas of the brain, including the cerebellum, occipital cortex, precuneus and frontal pole with TOT. d) Higher levels of sleepiness during fMRI task performance were associated with increased Proactive Cognitive Control activations in the left precentral / postcentral gyrus with TOT. Results were obtained using a cluster threshold of Z>3.1, p = .05, corrected for multiple comparisons, and are presented on a 1-mm MNI standard space template. TOT = time-on-task; fMRI = functional magnetic resonance imaging; MNI = Montreal Neurological Institute.

For Δ Reactive Cognitive Control, shorter objective (actigraphy-derived) sleep duration was associated with decreased BOLD activation with time on task in the right paracingulate / anterior cingulate gyrus (Figure 8b). None of the other sleep health measures was significantly associated with Δ Reactive Cognitive Control.

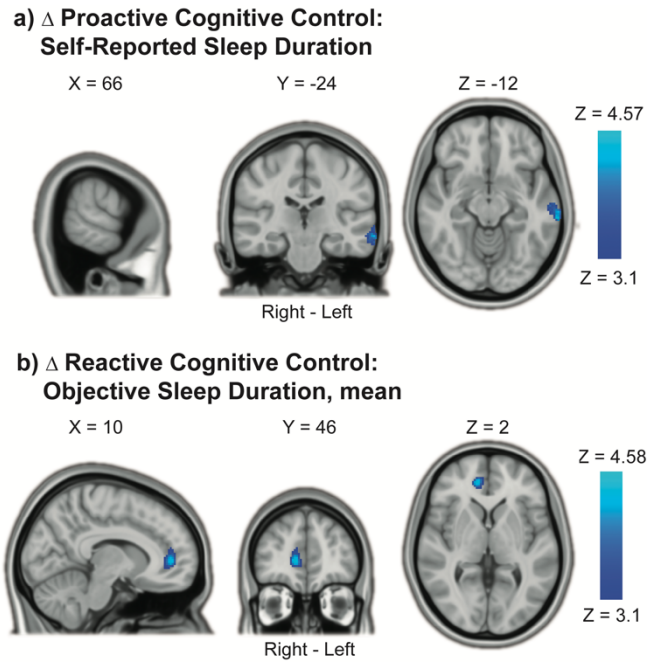

Figure 8. Time-On-Task (TOT) Decreases in Cognitive Control Processing with Shorter Sleep Duration. a) Shorter self-reported sleep duration was associated with decreased Proactive Cognitive Control activations in the left middle temporal gyrus with TOT. b) Shorter objective sleep duration (7-day mean) was associated with decreased Reactive Cognitive Control activation in the right paracingulate / anterior cingulate gyrus with TOT. Results were obtained using a cluster threshold of $Z>3.1$, $p = .05$, corrected for multiple comparisons, and are presented on a 1-mm MNI standard space template. TOT = time-on-task; MNI = Montreal Neurological Institute.

Table 4 *Associations Between Time-On-Task Change in Cognitive Control Activations and Sleep Health*

Contrast / Region	Right / Left	Size (#voxels)	P Value	Peak Z Value	Peak Coordinates (MNI)		
					X	Y	Z
Proactive Cognitive Control TOT increase: Later Sleep Midpoint							
Cerebellum: Left Crus I & II	L	810	< .0001	4.76	-6	-78	-28
Cerebellum: Left Crus I	L	355	.0025	4.13	-40	-66	-36
Precuneus Cortex	R / L	248	.0153	5.46	12	-62	68
Middle Frontal Gyrus	R	233	.0200	4.52	34	38	46
Lateral Occipital Cortex, superior division	L	193	.0423	4.04	-24	-58	34
Proactive Cognitive Control TOT increase: Lower Sleep Efficiency							
Paracingulate Gyrus / Frontal Pole	L	301	.0063	4.16	-10	48	18
Proactive Cognitive Control TOT increase: Fatigue in Daily Life							
Cerebellum: Left Crus II	L	1,877	< .0001	4.99	-4	-90	-32
Lateral Occipital Cortex, superior division	R	460	.0005	5.21	48	-68	40
Lateral Occipital Cortex, superior division / Occipital Pole	L	449	.0006	5.02	-34	-92	26
Lateral Occipital Cortex, inferior division	L	391	.0015	4.66	-56	-68	-6
Cerebellum: Right Crus II	R	371	.0020	4.61	18	-80	-54
Frontal Pole	R	288	.0077	5.66	16	70	26
Precuneus Cortex	R / L	283	.0083	4.30	10	-62	70
Frontal Pole	L	280	.0088	4.78	-42	60	12
Lateral Occipital Cortex, inferior division	R	188	.0467	4.15	62	-64	-6
Proactive Cognitive Control TOT increase: More Self-Reported Sleepiness On Task							
Precentral / Postcentral Gyrus	L	284	.0083	3.92	-14	-16	74
Proactive Cognitive Control TOT decrease: Shorter Self-Reported Sleep Duration							
Middle Temporal Gyrus, posterior division	L	193	.0436	4.57	-68	-30	-12

Table 4 Continued

Contrast / Region	Right / Left	Size (#voxels)	P Value	Peak Z Value	Peak Coordinates (MNI)		
					X	Y	Z
Reactive Cognitive Control Δ: Sleep Duration							
Paracingulate Gyrus / Cingulate Gyrus, anterior division	R	173	.0429	4.58	12	46	4

Results pertain to changes in brain activation with increased time-on-task (Δ). Results were obtained using a mixed-effects model corrected for multiple comparisons using a cluster threshold of $Z > 3.1$ and a corrected cluster significance threshold of $p < 0.05$. Main peaks within each cluster are reported, and only statistically significant results are included in the table. Anatomical regions for each peak Z value were labeled using visual inspection and the Harvard Oxford cortical / subcortical structural atlases and the Cerebellar atlas (normalized with FLIRT) within the FSL software. MNI = Montreal Neurological Institute, R = Right, L = Left.

Discussion

In this prospective study of adult, normal sleepers, we observed multiple associations between cognitive control processing and habitual sleep health. Using whole-brain fMRI analyses, we found that measures indicating poorer sleep health were predominantly associated with stronger and more widespread BOLD activations during cognitive control processing (adjusted for age, sex, years of education and Not-X-CPT performance). Analyses focused on different temporal aspects of cognitive control processing yielded unique and complementary results. Later and more variable sleep midpoints, a relatively later chronotype preference, and higher levels of insomnia symptoms were associated with stronger *reactive* cognitive control activations. Furthermore, later sleep midpoints, lower sleep efficiency, more problems with fatigue in daily life, and more sleepiness during task performance were associated with increased *proactive* cognitive control activations with increasing time on task, reflecting more neural recruitment with time. As an exception to the general pattern, shorter sleep duration was associated with *decreased* cognitive control activations with time on task, as well as poorer task performance. Taken together, our results show that cognitive control function is linked to different aspects of habitual sleep health even in high-functioning, normal sleepers. The dominating pattern of increased BOLD activations, when adjusted

for performance, indicates that individuals with relatively poorer sleep health need to recruit more neuronal resources to support cognitive control function, possibly reflecting compensatory mechanisms and / or less efficient neural processing (Drummond et al. 2005; Chee and Tan 2010; Schmidt et al. 2015; Maire et al. 2018; Olsen et al. 2020; Alsameen et al. 2021). Meanwhile, shorter sleep duration seems to be associated with a lower recruitment of neuronal resources necessary to sustain task performance over longer periods of time, in line with previous research indicating an overlap between general time-on-task effects and effects of sleep loss (Hudson et al. 2020).

Later and more variable sleep timing, later chronotype preference, and more insomnia symptoms were associated with stronger *reactive* cognitive control activations. This indicates that poorer sleep health is predominantly associated with a "hyper-reactive" brain state, possibly reflecting increased recruitment of cognitive control resources as a response to conflicting (non-target) stimuli. Increased reactive cognitive control has previously been associated with higher levels of stress (Husa et al. 2022) and anxiety (Fales et al. 2008; Schmid et al. 2015; Yang et al. 2018) in healthy individuals, and with poorer white matter organization, lower fluid intelligence, and more anxiety problems in adults born preterm (Olsen et al. 2018). The reactive hyperactivations observed here therefore suggest sub-optimal cognitive control processing in those with relatively poorer sleep health, and may point to a potential mechanism linking poorer sleep health to poorer mental health outcomes.

Beyond stronger activations in core cognitive control regions, later chronotype preference and more insomnia symptoms were also associated with higher activations in the ventromedial prefrontal cortex. The ventromedial prefrontal cortex has previously been implicated in self-referential cognition (Gusnard et al. 2001; Jenkins and Mitchell 2011; Abraham 2013) and processing of predictive value and reward (O'Doherty 2004; Etkin et al. 2011; Byrne et al. 2019). Heightened activations here may therefore indicate experiencing the conflicting stimuli (non-targets) as more salient, and/or being more self-aware during conflict processing. The association with insomnia symptoms is also interesting in light of the "hyperarousal" hypothesis, which postulates that insomnia disorder is linked to higher interoceptive

awareness (Wei and Van Someren 2020), paired with an increased tendency to ruminate about sleep problems (Riemann et al. 2010; Fasiello et al. 2022). Although our findings pertain to healthy participants with normal sleep, these patterns of altered brain activity may shed light on neural substrates underlying the development and maintenance of sleep problems (Nicolazzo et al. 2021; Faaland et al. 2022).

Analyses of time-on-task effects showed that measures of poorer sleep health were predominantly associated with increased *proactive* cognitive control activations in fronto-parietal and cerebellar regions. At the group-level, participants generally demonstrated a shift toward more *reactive* cognitive control processing with time on task, with decreasing proactive- and increasing reactive cognitive control activations in core control regions. Proactive cognitive control processing is believed to be more resource-demanding than reactive cognitive control (having a higher metabolic cost) (Burgess and Braver 2010; Braver 2012). The group-level shift toward more reactive cognitive control processing with time on task may therefore reflect an adaptive or more cost-efficient response - freeing up neural resources (lowering metabolic cost) and allowing participants to engage in other thought processes while still maintaining a satisfactory performance (Petersen et al. 1998; Neubauer and Fink 2009). Hence, the time-on-task increases in proactive cognitive control activations observed with poorer sleep health may indicate compensatory mechanisms, i.e., recruitment of relatively more neural resources in order to maintain cognitive control function over time (Olsen et al. 2015; Olsen et al. 2020).

The strongest association for time-on-task effects was linked to problems with fatigue in daily life, for which there were widespread BOLD increases in cerebellar, occipital, and prefrontal areas with longer time on task. Heightened task-related BOLD activity has previously been observed in patient groups suffering from chronic fatigue, as compared to healthy controls (Cook et al. 2007; DeLuca et al. 2009; Almutairi et al. 2020). Our findings therefore imply that analyses of time-on-task effects are sensitive to temporal changes in neural activity associated with subtle symptoms also in healthy individuals. We did not observe statistically significant associations between time-on-task changes and self-reported fatigue during task performance. There was, however, an association with self-reported

sleepiness during the task. Fatigue research has repeatedly demonstrated a distinction between fatigue and sleepiness, as well as "trait" (stable over time) versus "state" (momentary) measures (Kluger et al. 2013; Wylie et al. 2022). Our findings mirror this phenomenon, showing a widespread increase in brain activity related to "trait" fatigue (as measured by the CFS), no evidence for differences in brain activity related to "state" fatigue (mental fatigue during task performance), and unique activity changes related to task-related sleepiness centered in motor areas. One possible interpretation of these findings is that, while the time-on-task increases in BOLD activity may be compensatory in the moment, they might come with a longer-term cost (higher levels of trait fatigue) (Kohl et al. 2009; Olsen et al. 2015).

Self-reported and objective measures of sleep duration were differently associated with cognitive control processing, task performance, and other sleep health measures in our study. This provides additional evidence that self-reported versus objective measures of sleep, even within the same dimension, capture different phenomena (Klumpp et al. 2017: 2017; Bernstein et al. 2019; Stefansdottir et al. 2020; Scarlett et al. 2021; Tahmasian et al. 2021). In the brain, shorter self-reported sleep duration was associated with *proactive* cognitive control decreases, and shorter objectively measured sleep duration was associated with *reactive* cognitive control decreases in BOLD activation with time on task (Figure 8). Relatively shorter sleep may therefore be linked to a lessened ability to recruit task-relevant brain areas with sustained time on task, perhaps reflecting a heightened sensitivity to general time-on-task effects due to insufficient sleep (Asplund and Chee 2013; Hudson et al. 2020). These findings are also in line with prior research showing that shorter sleep and sleep loss are associated with lower task-related BOLD activations (Krause et al. 2017; Uy and Galván 2017; Tashjian and Galván 2020; Dimitrov et al. 2021). Considering that the participants in our study largely had sleep durations within the recommended range for adults (7-8 hours), these results further suggest that time-on-task analyses are sensitive to subtle changes in neural activity within a homogenous, healthy sample.

Behaviorally, a shorter objective sleep duration was significantly associated with more variable RTs, more errors, and worse detectability, as well as slower and more variable RTs with time on task.

This supports extant findings linking shorter sleep to a more inattentive response style (Kuula et al. 2018; Saksvik-Lehouillier et al. 2020). Moreover, the objective sleep duration was associated with BOLD signal changes in the anterior cingulate cortex - a core region for cognitive control - whereas self-reported sleep was associated with BOLD signal change in temporal areas, which is typically implicated in memory function or semantic processing. Taken together, our results indicate that a shorter objectively measured sleep duration is associated with poorer cognitive control functioning in normal sleepers, whereas the association between self-reported sleep duration and cognitive control function was less clear. Moreover, the associations between task performance and sleep measures indicated that, while the objective sleep duration was the strongest predictor of task performance, shorter self-reported sleep duration was more associated with other self-reported measures of sleep health (in particular, sleep quality, insomnia symptoms, and fatigue problems) (see Supplementary Figure 1). We therefore speculate that shorter self-reported sleep duration might be linked to general symptom load, which may contribute to detrimental effects observed with shorter self-reported sleep previously. Future studies should investigate how objective versus self-report measures of sleep relate to objective versus self-report measures of cognitive function as well as mental health (Bernstein et al. 2019).

Through assessing the multidimensionality of habitual sleep health, some interesting patterns emerged. Overall, most associations with cognitive control processing were related to later and more variable sleep timings, providing additional evidence that the timing and variability of sleep are uniquely related to brain function (Byrne et al. 2019; Facer-Childs et al. 2019; Tian et al. 2020). Further, actigraphy-derived habitual sleep duration was the measure most closely associated with task performance, indicating that objective sleep duration is linked to cognitive performance even in healthy sleepers. Regarding sleep efficiency and quality, cognitive control activations were significantly associated with levels of insomnia symptoms and actigraphy-derived sleep efficiency, but not with self-reported sleep quality (as measured using the PSQI). Whereas the ISI is specific to problems with going to sleep and maintaining sleep, the PSQI is a broader measure of perceived sleep quality. Our findings

therefore suggest that the efficiency and consistency of sleep, rather than perceived sleep quality, are more closely related to cognitive control function in normal sleepers. Notably, the lack of a significant association with the PSQI is in contrast to several previous studies which have identified a link between poorer sleep quality and brain function (Elvemo et al. 2015; Curtis et al. 2016; Avinun et al. 2017; Cheng et al. 2018; Guadagni et al. 2018). However, most of these studies included patients or individuals with depressive symptoms, and / or were focused on emotion processing. One interpretation of our findings in this context may therefore be that poorer self-reported sleep quality is more related to affective regulation ("hot cognitive control function") as compared to "cold" cognitive processes (as was measured in our study) (Salehinejad, Ghanavati, et al. 2021). We also note that the participants in our study were considered eligible partly based on the *absence* of self-reported sleep and mental health problems, and hence, the overall prevalence of sleep problems was relatively low in our sample. These null-findings should thus be interpreted with caution.

Due to the cross-sectional design of our study, we cannot conclude on the directionality of our results. Our findings may suggest that poorer sleep health leads to altered cognitive control function, or they may reflect inherent differences in cognitive control and/or brain function which give rise to real-life differences in sleep behavior. For example, those with a more reactive cognitive control style (i.e., a more "hyper-reactive" brain signature) may be more impulsive in nature, leading to later and more variable bed/rise times. In line with this, previous studies have found a link between later chronotype preference and lower ability for self-regulation (Digdon and Howell 2008; Kroese et al. 2014; Owens et al. 2016; Kuula et al. 2018). Also, circadian phenotype is known to be partially dependent on genetic factors (Katzenberg et al. 1998), which could in turn affect the development of cognitive control function and sleep behavior. On the other hand, it seems unlikely that cognitive control function would affect actigraphy-derived sleep efficiency, which was associated with increased neural recruitment in frontal areas with time on task. This finding in particular might reflect an increased need for cognitive compensation, or increased efforts to maintain alertness, as a result of insufficient sleep. Furthermore, we

underline that our participants had good sleep health overall, and as such, our results cannot be generalized to clinical populations.

This is the largest study to date using neuroimaging combined with objective sleep assessment (actigraphy) data to investigate the relationship between sleep and brain function in healthy adults (Khalsa et al. 2016; Lunsford-Avery et al. 2020; Zhang et al. 2020). Our use of objective and prospective sleep assessment avoids retrospective biases, and more accurately accounts for intra-individual variability in normal sleep as compared to self-report measures. The restriction of inclusion to healthy, "normal sleepers" between the ages of 20-40 ensured a homogenous age sample, and reduces the likelihood of confounding factors affecting sleep health (e.g., brain aging and health problems) (Scullin and Bliwise 2015; Bei et al. 2016; Alfini et al. 2020). Although there was some variability in sleep health within our group, the use of strict inclusion criteria limits the generalizability of our findings to other populations (e.g., extreme chronotypes, clinical populations, and other age cohorts). Furthermore, our broad approach to sleep health generated a range of variables of interest, which necessitated the use of multiple comparisons with an increasing chance of Type I errors. This approach was used since we specifically sought to investigate the many different dimensions of sleep based on previously heterogeneous findings. To account for this limitation, we used a conservative statistical threshold in our whole-brain fMRI analyses, which were also adjusted for age, sex, education, fMRI task performance, and head movement. Finally, prior studies have shown that BOLD activations and task performance can vary throughout the day, and that people perform better when test times are aligned with their natural alertness peak ("the synchrony effect") (Goldstein et al. 2007; Schmidt et al. 2012; Song et al. 2018; Salehinejad, Wischnewski, et al. 2021). One concern could therefore be that our results - in particular, those pertaining to sleep timing and chronotype - were driven by inter-individual circadian effects. However, there was no systematic relationship between chronotype preference and test times in our study, and all testing was performed during standard working hours within a relatively large timespan (between 08:00 AM and 03:00 PM). There were also little-to-no associations between chronotype preference and task

performance, except that later chronotype preference was associated with lower hit RT variability with increased time on task (Figure 4). As this study was not specifically designed to test circadian effects, conclusions regarding such effects are beyond the scope of the current study, and results should be interpreted with this in mind.

In conclusion, in this study of adult, normal sleepers, we found that poorer sleep health was associated with a hyper-reactive brain state during a test of cognitive control function, as well as increased proactive cognitive control processing with longer time on task. Across the different dimensions of sleep health, later and more variable sleep timing was most closely associated with higher cognitive control BOLD activations, whereas shorter objective sleep duration was associated with poorer task performance and lower BOLD activations with time on task. Given that our fMRI analyses were adjusted for performance, we suggest that the altered brain activity observed with poorer sleep health may reflect compensatory neural recruitment and / or inefficient neural processing. Future studies should continue to focus on naturalistic measurement of normal sleep to elucidate the complex relationships between sleep and brain function.

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Competing Interest Statements

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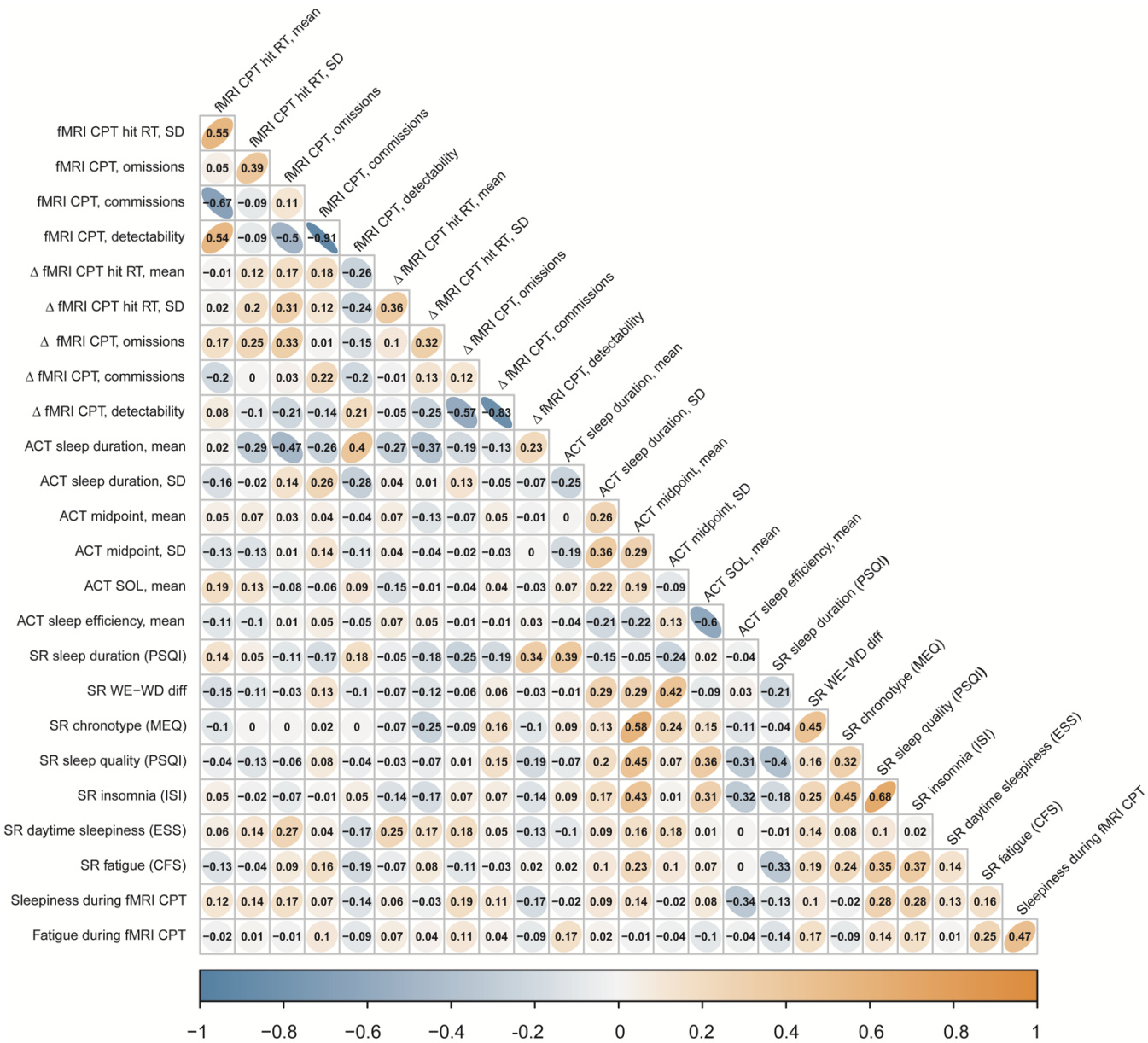
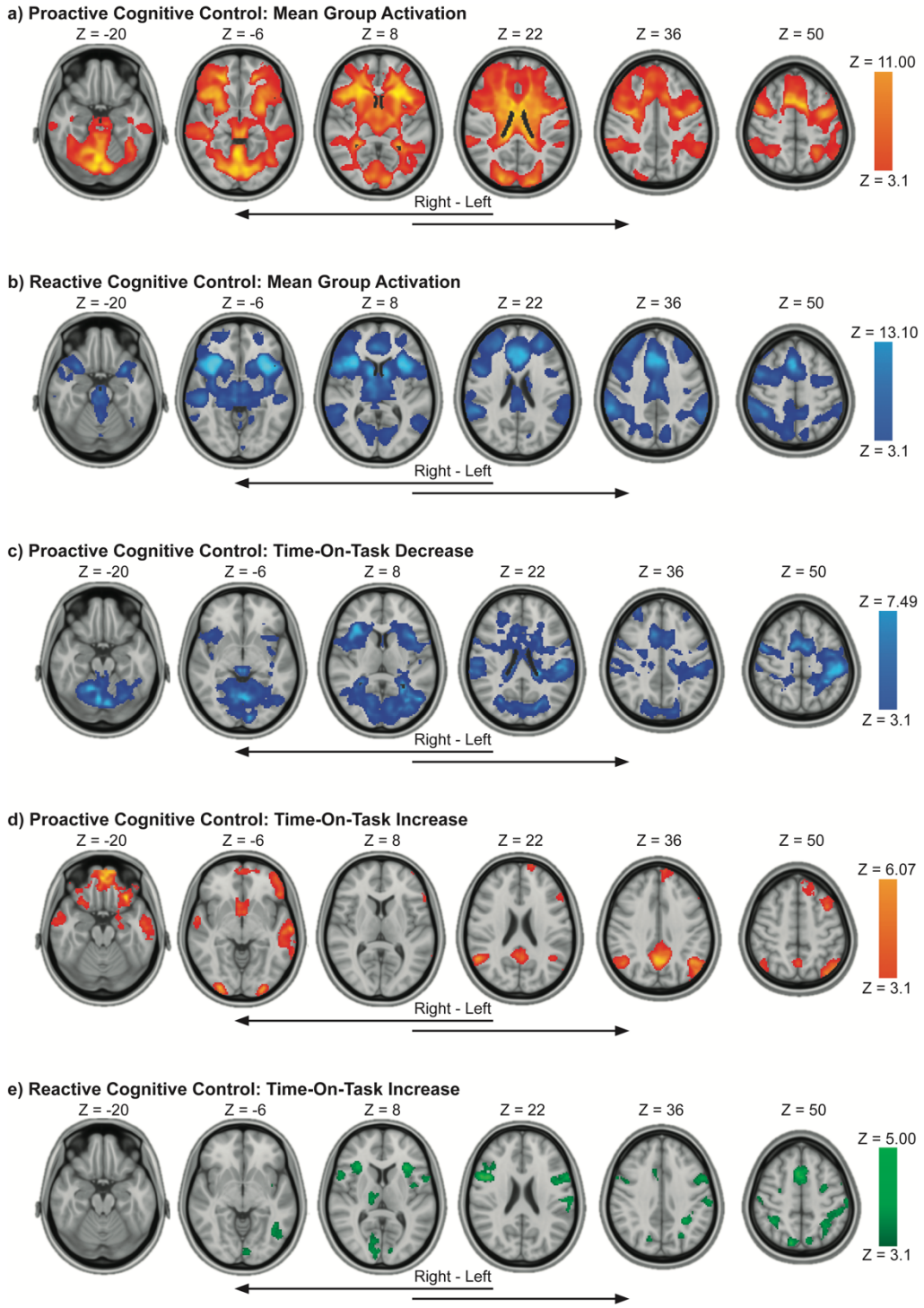


Supplementary Figure 1. Associations Between fMRI Task Performance and Sleep Health Measures. Full correlation matrix, depicted as a correlogram. The correlogram shows correlation coefficients (Spearman's rho) between fMRI task performance measures and all sleep health measures. The colors and shapes of the circles indicate size of the correlation coefficient (higher correlations = stronger color and more elliptical in shape). Blue color indicates negative correlations and red color indicates positive correlations. ACT = actigraphy; SR = self-reported; fMRI = functional magnetic resonance imaging; CPT = continuous performance test; RT = reaction time, SD = standard deviation.



Supplementary Figure 2. Group Average Cognitive Control Activations. a) Proactive Cognitive Control, b) Reactive Cognitive Control, c) Proactive Cognitive Control Time-on-Task Decrease, d) Proactive Cognitive Control Time-On-Task Increase, e) Reactive Cognitive Control Time-on-Task Increase. Results were obtained using a cluster threshold of Z>3.1, p = .05, corrected for multiple comparisons, and are presented on a 1- mm MNI standard space template. MNI = Montreal Neurological Institute.

Supplementary Table 1 *fMRI Task Performance and Self-Report Measures (N = 81)*

Variable	Mean / SD	Min	Max
Hit RT, mean (ms)	396.48 (43.36)	312.40	573.43
Δ Hit RT, mean (ms)	1.66 (17.95)	-39.98	74.15
Hit RT SD, mean (ms)	64.08 (17.36)	39.76	129.94
Δ Hit RT SD, mean (ms)	0.54 (17.86)	-30.18	89.30
Omission errors, sum (n)	0.79 (1.41)	0	6
Δ Omission errors, sum (n)	0.25 (1.08)	-2	6
Commission errors, sum (n)	17.62 (10.14)	1	41
Δ Commission errors, sum (n)	0.69 (2.00)	-6	5
Detectability (d')	3.27 (0.71)	1.68	4.92
Δ Detectability (d')	-0.22 (0.60)	-1.56	1.27
Fatigue during task (1-10)[a]	4.63 (1.65)	1	9
Sleepiness during task (1-10)[a]	4.81 (1.55)	1	8

Δ indicates time-on-task changes (time epoch 4 - time epoch 1). [a]Self-report measures during task performance were obtained by asking the participants to rate their level of mental fatigue and sleepiness via the MRI speaker system between task runs. Levels of sleepiness were rated according to the Karolinska Sleepiness Scale (Akerstedt and Gillberg 1990). RT = reaction time, ms = milliseconds, SD = standard deviation.

Supplementary Table 2 *Group Mean Activations for Proactive and Reactive Cognitive Control Processing*

Contrast / Region	Right / Left	Size (#voxels)	P Value	Peak Z Value	Peak Coordinates (MNI)		
					X	Y	Z
Proactive Cognitive Control							
Insular Cortex / Frontal Operculum / Frontal Orbital Cortex	R / L	93,854	< .0001	11.00	-24	20	12
Frontal Operculum	L	lm	-	11.00	-38	16	14
Insular Cortex / Frontal Operculum	R	lm	-	10.90	26	22	12
Lingual Gyrus	R	lm	-	10.80	8	-76	-16
Cerebellum – Right V	R	lm	-	10.80	4	-58	-8
Lingual Gyrus / Cerebellum – Left VI	L	lm	-	10.70	-6	-76	-16
Reactive Cognitive Control							
Insular Cortex	R / L	77,594	< .0001	13.10	-36	16	-6
Insular Cortex	R	lm	-	13.10	38	16	-4
Insular Cortex / Frontal Operculum	R	lm	-	12.90	34	22	2
Insular Cortex	L	lm	-	12.80	-30	22	-2
Insular Cortex / Frontal Orbital Cortex	R	lm	-	12.60	32	20	-8
Paracingulate Gyrus	R	lm	-	11.50	4	22	42
Cerebellum – Right Crus I	R	367	.0012	5.73	38	-52	-32

Main peaks and local maxima (lm) within each cluster are reported. Results were obtained using a mixed-effects model corrected for multiple comparisons using a cluster threshold of $Z > 3.1$ and a corrected cluster significance threshold of $p < 0.05$. Anatomical regions for each peak Z value were labeled using visual inspection and the Harvard Oxford cortical / subcortical structural atlases and the Cerebellar atlas (normalized with FLIRT) within the FSL software. Note that clusters are large and span over several brain regions. MNI = Montreal Neurological Institute, lm = local maxima.

Supplementary Table 3 *Group Mean Time-On-Task Changes for Proactive and Reactive Cognitive Control Processing*

Contrast / Region	Right / Left	Size (#voxels)	P Value	Peak Z Value	Peak Coordinates (MNI)		
					X	Y	Z
Proactive Cognitive Control: Time-On-Task Decrease							
Superior Parietal Lobule / Postcentral Gyrus	R / L	61,822	< .0001	-7.49	-32	-46	58
Juxtapositional Lobule	R	lm	-	-7.33	12	2	56
Cerebellum – Right V / VI	R	lm	-	-7.24	6	-64	-14
Left Lateral Ventricle	L	lm	-	-7.20	-16	-30	18
Lingual Gyrus / Cerebellum	R	lm	-	-7.17	8	-72	-14
Superior Parietal Lobule / Postcentral Gyrus	L	lm	-	-7.09	-42	-40	60
Proactive Cognitive Control: Time-On-Task Increase							
Frontal Orbital Cortex	L	4,938	< .0001	6.07	-30	26	-18
Middle Temporal Gyrus	L	2,654	< .0001	5.52	-58	-14	-14
Lateral Occipital Cortex	L	2,166	< .0001	5.63	-44	-66	56
Precuneus Cortex	R	2,138	< .0001	5.51	4	-58	34
Suprior Temporal Gyrus	R	1,516	< .0001	4.71	60	-4	-8
Angular Gyrus	R	1,336	< .0001	5.59	52	-56	26
Cerebellum: Right Crus I	R	737	< .0001	4.86	22	-82	-28
Cerebellum – Left Crus II	L	634	< .0001	5.09	-18	-84	-38
Occipital Pole	R	338	.0036	5.27	28	-96	-4
Occipital Pole	L	300	.0066	5.80	-26	-96	-10
Reactive Cognitive Control: Time-On-Task Increase							
Superior Parietal Lobule	L	1,791	< .0001	4.23	-26	-48	40
Inferior Frontal Gyrus, pars opercularis	L	1,658	< .0001	4.55	-58	12	26
Inferior Frontal Gyrus, pars opercularis	R	1,271	< .0001	5.00	54	12	18
Paracingulate Gyrus	R	984	< .0001	4.59	6	10	52
Occipital Pole	R	434	.0003	4.05	8	-90	4

Supplementary Table 3 Continued

Contrast / Region	Right / Left	Size (#voxels)	P Value	Peak Z Value	Peak Coordinates (MNI)		
					X	Y	Z
Reactive Cognitive Control: Time-On-Task Increase							
Inferior Temporal Gyrus	L	433	.0003	4.62	-42	-50	-10
Precentral Gyrus	R	407	.0005	4.00	46	-8	60
Superior Parietal Lobule	R	404	.0006	4.03	38	-48	50
Lateral Occipital Cortex, superior division	R	282	.0049	4.56	14	-78	48
Postcentral Gyrus	L	205	.0223	3.88	-46	-16	58
Precuneus Cortex	R	197	.0263	4.18	-8	-72	46
Precentral Gyrus	L	186	.0332	3.82	-26	-8	54
Thalamus	R	186	.0332	4.40	12	-16	6
Postcentral Gyrus	R	185	.0339	3.73	54	-20	42

Main peaks within each cluster are reported. For Proactive Cognitive Control Time-On-Task Decrease, local maxima (lm) are included due to the cluster's size and span. Results were obtained using a mixed-effects model corrected for multiple comparisons using a cluster threshold of $Z > 3.1$ and a corrected cluster significance threshold of $p < 0.05$. Anatomical regions for each peak Z value were labeled using visual inspection and the Harvard Oxford cortical / subcortical structural atlases and the Cerebellar atlas (normalized with FLIRT) within the FSL software. Note that many clusters span over several brain regions. MNI = Montreal Neurological Institute, lm = local maxima.