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Recruitment dynamics of cognitive control in insomnia

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ABSTRACT

Study Objectives: Individuals with insomnia disorder (ID) commonly report complaints of cognitive control functioning. Conversely, both behavioral and neurological evidence supporting subjective cognitive control impairments in insomnia remain remarkably scarce and inconclusive. To investigate this discrepancy, the present study used next to behavioral measures, event-related potentials (ERPs) to assess proactive control and reactive control in insomnia.

Methods: Individuals with insomnia disorder ($n = 18$) and good sleeper controls (GSC; $n = 15$) completed the AX-Continuous Performance Task, while electroencephalography (EEG) was recorded. This task required participants to maintain specific cue-information active in order to prepare an adequate response to a subsequent probe, which allowed us to measure participants' reliance on both proactive and reactive control.

Results: The results indicate that, although ID show a comparable level of performance as GSC, they show a reduced proactive engagement of cue-induced maintenance and response preparation processes (as reflected by the P3b and the contingent negative variation components). Moreover, in contrast to GSC, ID fail to engage reactive control (as indexed by the P3a component) in order to overcome invalid response tendencies.

Conclusions: This study provides neurological evidence for impairments in cognitive control functioning in insomnia. As such, our study contributes to a better understanding of the discrepancy between the commonly reported cognitive impairments in insomnia and the scarce objective evidence for these cognitive complaints.

Keywords: insomnia, sleep, cognitive control, EEG, AX-CPT

STATEMENT OF SIGNIFICANCE

Insomnia represents an enormous burden for society with high personal (e.g. risk for mental disorders) and economic/societal costs (e.g. health care). Individuals with insomnia disorder (ID) not only report sleep disturbances, but also significant alterations of daytime functioning, including fatigue and impaired cognitive control functioning. However, neurobehavioral research on these complaints remains remarkably scarce and inconclusive. Here, we provide evidence that ID show an altered cognitive control functioning compared with good sleepers controls (GSC). In particular, ID present with a significantly reduced proactive control engagement along with an absence of behavioral performance impairments. These findings provide neurological evidence for impairments in cognitive control in insomnia, which in turn provides objective evidence for the commonly reported subjective cognitive impairments in insomnia.

INTRODUCTION

Approximately 30 to 50% of the adult population reports occasional sleep disturbances and 6 to 10% meets the diagnostic criteria for an insomnia disorder [1-3]. According to the International Classification of Sleep Disorders (ICSD-3) [4] and the Diagnostic and Statistics Manual of Mental Disorders (DSM-V) [5], insomnia disorder is defined as an individual's report of recurrent difficulties with initiating or maintaining sleep, accompanied by clinically significant distress in daytime functioning. Interestingly, it is often these perceived functional daytime impairments rather than the sleep disturbances per se that have a seemingly profound impact on patients' daily life [6], forcing them to seek medical care [7].

Complaints with regards to altered wake-time functioning reported by individuals with insomnia disorder (ID) include not only fatigue and mood disturbances, but also several complaints about cognitive impairments [8-10], such as difficulties in suppressing repetitive thoughts [11-13]. For example, individuals with insomnia disorder tend to ruminate about the diurnal consequences of their chronic sleep difficulties. This extensive rumination and worry have also been proposed to contribute to the onset and maintenance of insomnia [11, 14-18]. Rumination and worry reflect impairments in the ability to effectively and efficiently inhibit irrelevant information, which is an important component of cognitive control [19-21]. Cognitive control comprises cognitive operations such as planning a new strategy, evaluating it, controlling its execution and correcting possible errors. It kicks in when routine activation of behavior is no longer sufficient for optimal performance [22]. The prefrontal cortex (PFC) plays a crucial role in cognitive control [23]. Interestingly, alterations of activation patterns have been reported in several cognitive control related brain areas in individuals with insomnia disorder [24-26]. Some studies revealed a hypoactivation in prefrontal and frontoparietal regions [24-25]. In contrast, a more recent study observed a hyperactivation in cognitive control brain circuits in insomnia [26]. Interestingly, in all these studies, the observed changes in brain activation of individuals with insomnia disorder were not accompanied by impairments in behavioral task performance. This finding is in line with the compensatory recruitment hypothesis [27]. This hypothesis assumes that unaltered task performance can be explained by an overrecruitment of cerebral resources in response to cognitive challenges. Individuals with insomnia disorder would thus mobilize compensatory cognitive effort

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(which would be indexed by overactivation in task-relevant brain areas) in order to maintain a comparable level of performance as good sleeper controls [27]. In turn, this reduction in cognitive efficiency might explain why individuals with insomnia disorder experience having to engage more effort to reach similar performance levels as good sleepers. Additionally, this could also contribute to their frequent feelings of increased mental fatigue.

Although individuals with insomnia disorder report deficits in cognitive control functioning, it becomes clear that objective neurobehavioral evidence for these complaints remains surprisingly scarce and inconclusive [24-32]. This discrepancy might stem from the fact that previous neuroimaging studies focused on the under- or overrecruitment of cognitive control brain areas, assuming a generic deficit in cognitive control functioning in insomnia. Instead, a more fruitful approach to study the altered neurological profile of individuals with insomnia disorder, is departing from a dynamic viewpoint. The Dual Mechanisms of Control (DMC) [33] theory offers a different approach to cognitive efficiency in which the temporal dynamics of cognitive control recruitment are pivotal. The DMC theory distinguishes two cognitive control modes which differ with regards to the time-scale on which they act. During proactive control, goal-relevant information is sustainably maintained to anticipate and prevent interference before it occurs. Reactive control is a late correction mechanism mobilized just in time, to detect and resolve interference after its onset. Although both control modes activate the same brain region, namely the lateral PFC (lPFC), proactive control does so in a sustained and/or anticipatory way, whereas reactive control only triggers transient activation [33]. It is postulated that both strategies have costs and benefits, and efficient cognition mainly relies on a mixture of both [33]. The DMC predicts that intrusive worry and rumination so often reported by individuals with insomnia disorder may hamper the active maintenance of task relevant information, which implies deficient proactive control [33]. To reveal this weaker proactive control in insomnia, a temporal analysis is needed. Methodologically, electroencephalography (EEG) is particularly suited to study the temporal recruitment of the different cognitive control modes in insomnia, since it has a superior temporal resolution and since a series of EEG components reflecting proactive and reactive control have already been identified [34, 35]. Although some previous studies have measured EEG differences between individuals with insomnia

disorder and good sleeper controls, these have focused on brain responses to auditory stimuli in or timed closely to transition phases between sleep and wakefulness [36, 37]. In contrast, the current study focused on event-related potentials (ERPs) associated with response preparation and cognitive control during wakefulness after potential effects of sleep inertia have dissipated.

In this study, we examine cognitive control mechanisms in individuals with insomnia disorder and good sleeper controls, using the AX-continuous performance task (AX-CPT) [38]. Performance on the AX-CPT depends on the successful execution of two cognitive control processes. More specifically, this task requires the participants to maintain and update task-relevant task information (i.e. proactive control) as well as to use additional task information to inhibit inappropriate response tendencies (i.e. reactive control). We will combine this task with EEG measurement. Due to its dynamic and temporal approach, this design will allow us to independently measure both proactive and reactive control mechanisms in individuals with insomnia disorder and good sleeper controls. This will provide us with a better understanding of how individuals with insomnia disorder recruit cognitive control and under which circumstances its efficiency fails and consequently offers the potential to improve current intervention strategies by targeting specific cognitive control impairments.

METHODS

Participants

   The participants included 20 individuals with insomnia disorder (ID) and 16 age- and sex-matched good sleeper controls (GSC). Individuals with insomnia complaints met the DSM-V criteria for Insomnia Disorder: a predominant complaint of a dissatisfaction with sleep quality or quantity associated with (1) difficulty initiating, maintaining sleep or early morning awakenings; (2) the sleep disturbance is occurring at least three nights per week for at least three months and (3) the sleep disturbance causes a clinically significant distress or impairment in social, occupational, behavioral or other important areas of functioning; (4) the sleep difficulty occurs despite adequate opportunities (e.g. enough time is allotted for sleep) and adequate circumstances (e.g. quiet and comfortable sleep environment) to sleep and is not better explained by another sleep-wake disorder, physiological effects of a substance (e.g. drug or medication) nor by a coexisting mental and/or medical disorder. GSC were satisfied with their sleep and did not meet the diagnostic criteria for Insomnia Disorder. Exclusion criteria for both groups were (1) presence of a medical or neurological disorder likely to interfere with sleep or cognitive functioning; (2) medication use altering sleep or cognitive functioning; (3) presence of a psychiatric disorder (i.e. current major depressive episode, generalized anxiety disorder, bipolar disorder or a history of a manic episode or a lifetime history of psychotic symptoms), assessed using the Mini International Neuropsychiatric Interview (M.I.N.I.) [39]; (4) substance abuse (including alcohol) in the previous year; (5) night or shift workers; individuals with irregular sleep-wake rhythms or abnormal habitual bedtime hours (< 09:00 PM or > 01:00 AM); (6) pregnant women or parents with newborns; (7) presence of other primary sleep disorders (e.g. Restless Legs Syndrome, Obstructive Sleep Apnea Syndrome); (9) a body mass index < 18 and > 30 kg/m^2. All participants had to be free of medication altering sleep or cognitive functioning at least four consecutive weeks before the start of the study as well as during the two weeks of the study (see flowchart of the recruitment procedure in Figure 1). All participants had normal or corrected-to normal vision, were right- or left-handed and received a financial compensation (€ 25) for the completion and received a detailed clinical report regarding their

sleep. The study protocol was approved by the ethical board of the Vrije Universiteit Brussel, Brussel, Belgium (reference: 2014, 234). All participants provided written informed consent.
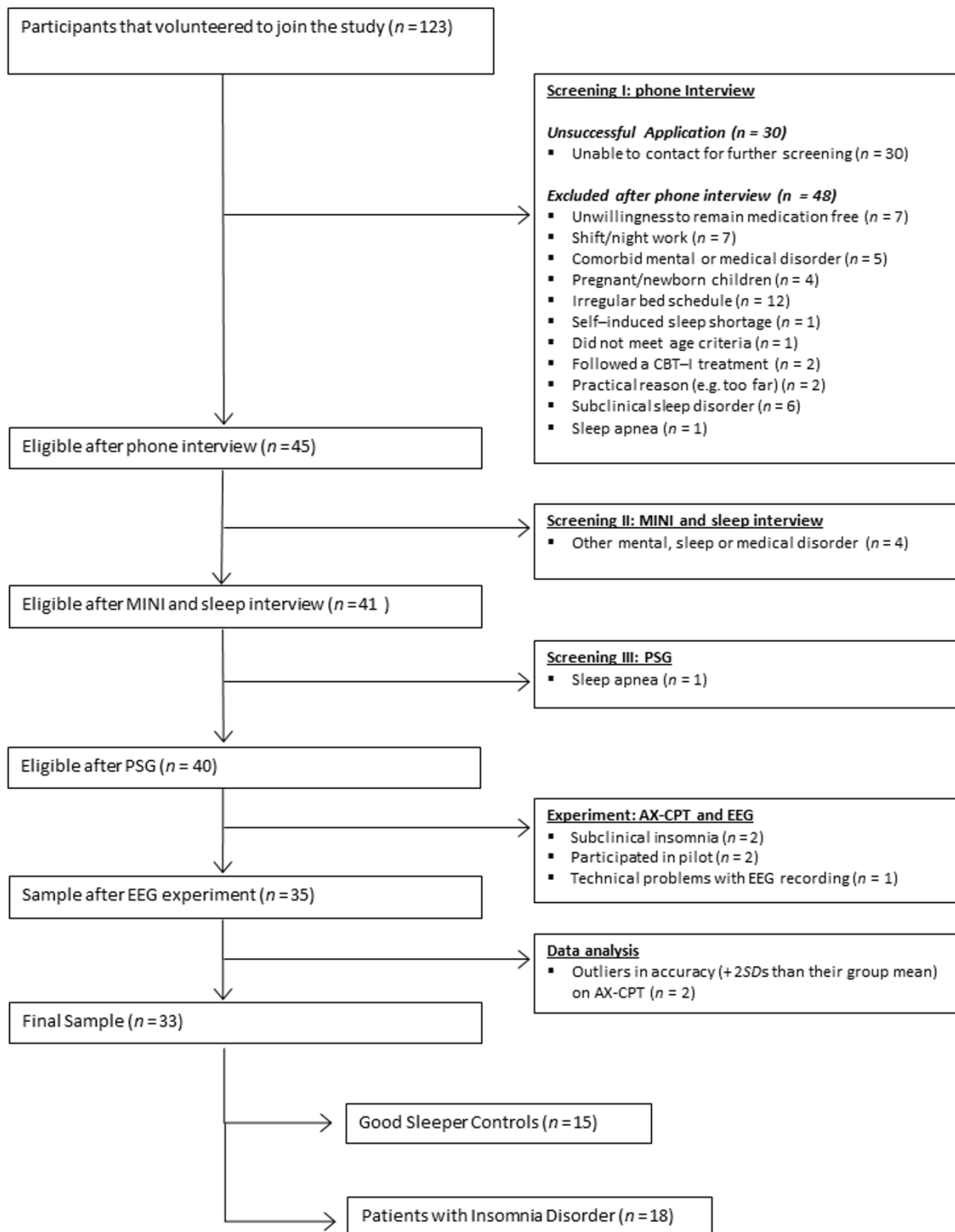

Figure 1. Flowchart of recruitment procedure

Procedures

The experimental procedure is summarized in Figure 2. The participants were recruited through advertisements distributed via clinical sleep centers, primary care physicians, and social media. Participants that volunteered to participate or showed interest in the study were contacted by phone or e-mail for a short information briefing and a screening interview, in which sleep complaints, sleep schedules, medication and/or substance consumption along with clinical history information were checked. Eligible candidates after this preliminary anamnesis participated in a first session (at home between 07:00 and 8:30 PM). This first session consisted of a complete briefing of the study's content and procedure. After giving informed consent, participants underwent a clinical assessment including the M.I.N.I. and a DSM-V-based semi-structured interview for sleep disorders, in order to assess mental and sleep disorders, respectively. A home-based polysomnography (PSG) was then performed with the sole purpose to further exclude comorbid or previously unknown sleep disorders. At preparation for PSG, the participants were able to become accustomed to the equipment before going to bed at their habitual bedtime. Between session 1 and session 2 (see Figure 2), participants were asked to complete questionnaires assessing sleep functioning, mental health, daytime and cognitive functioning and general medical history. The following clinical instruments were administered: Pittsburgh Sleep Quality Index (PSQI) [40], Insomnia Severity Index (ISI) [41], Ruminative Response Scale (RRS) [42], Beck Depression Inventory-II (BDI-II) [43], State-Trait Anxiety Inventory (STAI) [44], Multidimensional Fatigue Inventory (MFI) [45], Epworth Sleepiness Scale (ESS) [46], Dysfunctional Beliefs and Attitudes about Sleep (DBAS-16) [47], Cognitive Failures Questionnaire (CFQ) [48] and Multifactorial Memory Questionnaire (MMQ) [49]. During two consecutive weeks between session 1 and 2, participants were also required to complete a sleep diary and to wear an actigraph to check for the regularity of sleep-wake schedules. After this first session, participants were either assigned to the experimental (ID) or the control group (GSC) based upon DSM-V semi-structured interview for sleep disorders and the sleep diaries (see also recommendations [50]). Eligible candidates were invited for the second session, consisting of a cognitive continuous performance task (AX-CPT) while under continuous EEG recording. After the completion of the experimental AX-CPT task, participants also

completed the NASA Task Load Index (NASA-TLX) [51], in order to assess the subjective cognitive load they experienced during the experiment. The testing session lasted about 65 minutes. Participants were instructed to refrain from caffeine and alcohol intake for 72 h prior to both of the sessions, except for small habitual quantities of caffeine in the morning (i.e. maximum 2 units). Participants refrained from caffeine intake approximately 2 h before the start of the experimental AX-CPT task.
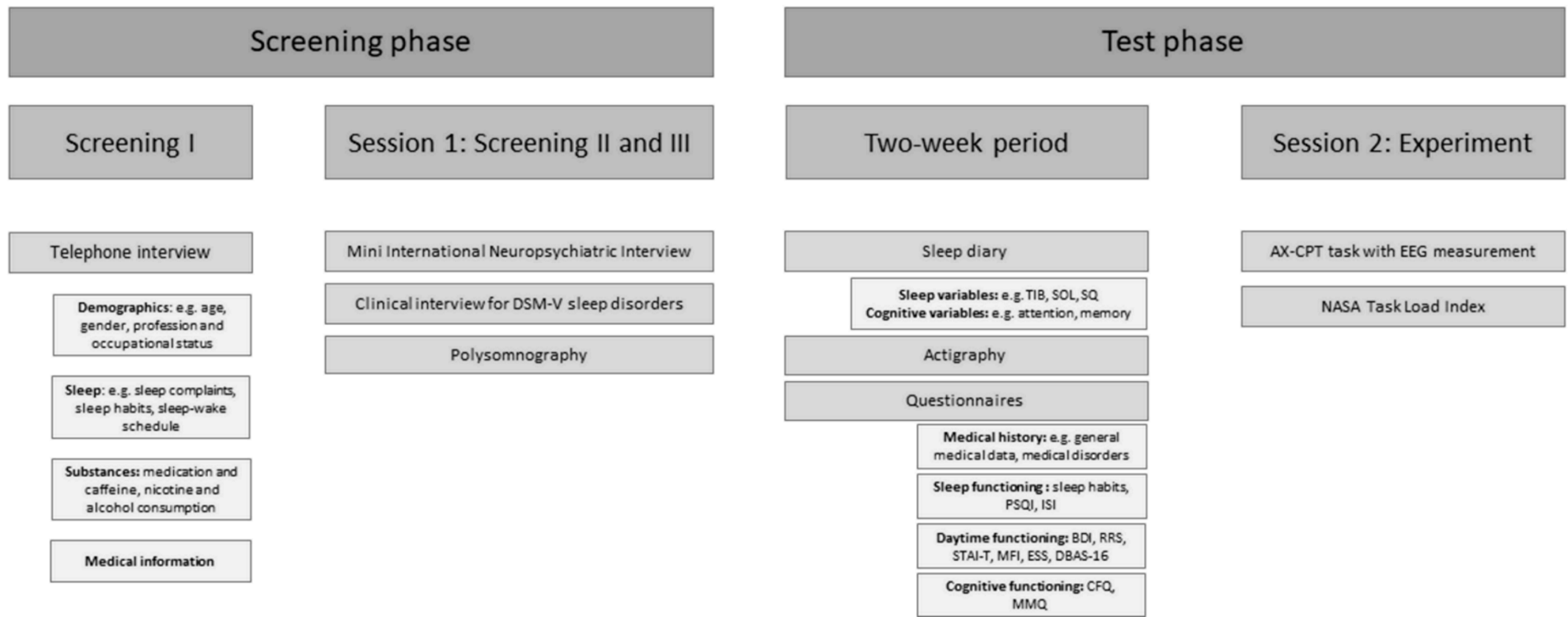
Screening phase		Test phase	
Screening I	**Session 1: Screening II and III**	**Two-week period**	**Session 2: Experiment**
Telephone interview	Mini International Neuropsychiatric Interview	Sleep diary	AX-CPT task with EEG measurement
Demographics: e.g. age, gender, profession and occupational status	Clinical interview for DSM-V sleep disorders	**Sleep variables:** e.g. TIB, SOL, SQ **Cognitive variables:** e.g. attention, memory	NASA Task Load Index
Sleep: e.g. sleep complaints, sleep habits, sleep-wake schedule	Polysomnography	Actigraphy	
Substances: medication and caffeine, nicotine and alcohol consumption		Questionnaires	
Medical information		**Medical history:** e.g. general medical data, medical disorders	
		Sleep functioning: sleep habits, PSQI, ISI	
		Daytime functioning: BDI, RRS, STAI-T, MFI, ESS, DBAS-16	
		Cognitive functioning: CFQ, MMQ	

Figure 2. Experimental procedure

Measures and material

Polysomnography

Participants underwent a full-night home-based PSG with the sole purpose of excluding (comorbid) sleep disorders. In order to minimize a reversed first night effect in individuals with insomnia disorder [52] or a first night effect in good sleeper controls [53], we performed the PSG in the participants' home environment. Signals were acquired by means of the Alice PDX G3 Software (Philips Respironics Inc™ Alice PDX®, Philips Healthcare™, Eindhoven, The Netherlands). Bedtimes were set in accordance with the participants' habitual bedtime and wake-up time. The recording montage consisted of two electroencephalograms (EEG) recorded from F4-A1, C4-A1 sites; two electrooculograms (EOG; LOC-A2, ROC-A2) and two submental and bilateral anterior tibial electromyograms. Oral and nasal airflow were measured using oro-nasal cannulae (Pro-Flow Plus™ Pro-Tech® Mukilteo, WA, USA). Respiration effort was measured using thoracic and abdominal respiratory belts (Pro-Tech® CT2™, Mukilteo, WA, USA). Capillary oxygen saturation was measured using photo-sensitive finger-oximetry (Nonin® Flexi-Form® II 7000A Nonin Medical Inc, Minneapolis, MN USA and LINOP® Adt Masimo corp. Irvine, CA, USA). In addition, a single electrocardiography lead was measured. The maximal impedance for the EEG and EOG electrodes remained under the cut-off of 5 kΩ and signals were recorded with a sampling rate of 500 Hz. PSG measurements were visually analyzed on 21-inch monitors, displaying 30-second epochs. EEG and EOG signals were filtered using a high pass filter of 0.5 Hz and a low pass filter of 20 Hz. The 30 s epochs were scored in accordance with the American Academy of Sleep Medicine criteria [4] by the primary author and were revised by a trained technician and the study supervisor. Classical variables of sleep structure included time in bed (TIB; i.e. the interval between lights off and lights on), sleep onset latency (SOL; i.e. the time between lights out and the first epoch of recorded sleep), wake after sleep onset (WASO; i.e. wake duration between the first sleep and the last recorded sleep epochs), early morning awakenings (EMA; i.e. wake duration between last recorded sleep epoch and lights on) and total sleep time (TST; TST = TIB - SOL - WASO - EMA). Sleep efficiency (SE) was defined as the percentage of TST divided by TIB. Other variables related to sleep architecture were the TST percentages of slow-wave sleep (SWS), rapid eye movement sleep

(REM), stage 1 (N1) and stage 2 (N2) sleep. The arousal index and the apnea-hypopnea episodes were defined according to American Academy of Sleep Medicine procedures [4]. The criteria for defining sleep disorders were an apnea-hypopnea index greater than 15 events per hour for sleep apnea and a periodic limb movement index greater than 15 events per hour for periodic limb movements in sleep [4, 5].

Actigraphy

Following the first session, participants were asked to wear an actigraph during two weeks, in order to check for the regularity (stability) of sleep-wake schedules. Data were recorded using the data analysis software ActiLife (ActiGraph®, Groningen, The Netherlands). No irregular sleep-wake schedules were observed among the participants.

Sleep Diary

During the two weeks between the first and the second session, participants were asked to complete a sleep diary evaluating their sleep (every morning) and daytime cognitive functioning (every evening) over a period of 14 consecutive days. Variables were added to sleep diaries to evaluate daytime cognitive functioning. Participants were asked to provide a subjective rating of their overall sleep quality on a 10-point scale (0 = very poor sleep quality, 10 = excellent sleep quality). Additionally, participants were asked to rate on a 10-point scale the perceived frequency of difficulty with attention/concentration (0 = very low level of attention and concentration, 10 = very high level of attention and concentration), memory (0 = significant memory difficulties, 10 = no significant memory difficulties at all) and planning/organization (0 = significant difficulties with planning and organization, 10 = no significant difficulties with planning and organization at all).

Pre-experimental task questionnaires

The Pittsburgh Sleep Quality Index. The Pittsburgh Sleep Quality Index (PSQI) [40] is a self-report questionnaire which assesses subjective sleep quality and sleep disturbances. The 19 items generate seven component scores (i.e. subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleep medication, and daytime dysfunction). Higher scores suggest a poor subjective sleep quality. A global PSQI score > 5 distinguishes poor sleepers from good sleepers [40].

Insomnia Severity Index. The Insomnia Severity Index (ISI) [41] is a 7-item self-report questionnaire that assesses insomnia symptoms (i.e. difficulties initiating sleep, maintaining sleep and early morning awakenings), (dis)satisfaction with sleep, interference of insomnia symptoms with daytime functioning, the noticeability of functional disabilities associated with insomnia for others, and level of experienced distress caused by insomnia. Higher scores suggest more severe insomnia. A global ISI score > 10 is suggested as a clinical threshold in determining clinically significant insomnia [41].

Ruminative Response Scale. The Ruminative Response Scale (RRS) [42] is a 22-item scale used to measure the tendency to ruminate as a reaction to feelings of sadness or depression. A higher score indicates higher levels of rumination.

Beck Depression Inventory-II. Depressive symptomatology was measured using the Beck Depression Inventory-II (BDI-II) [43]. The BDI-II is a 21 item inventory measuring depressive symptoms, as defined by the DSM-V. The BDI-II comprises two subscales: a cognitive subscale and a somatic-affective subscale [54]. Higher total scores indicate a higher severity of depressive symptoms.

State-Trait Anxiety Inventory. The State-Trait Anxiety Inventory (STAI) [44] is a 40-item self-report scale and measures state (A-state) and trait anxiety (A-trait). The first 20 items (A-state) ask about feelings of anxiety in a specific situation, the following 20 items (A-trait) ask about feelings of anxiety in general. Higher scores suggest a higher degree of anxiety. Only the STAI (A-trait) was used.

Multidimensional Fatigue Inventory. The multidimensional Fatigue Inventory (MFI) [45] is a 20-item self-report instrument designed to measure different dimensions of fatigue (i.e. General Fatigue,

Physical Fatigue, Mental Fatigue, Reduced Motivation and Reduced Activity). Higher scores suggest a higher degree of fatigue.

Epworth Sleepiness Scale. The Epworth Sleepiness Scale (ESS) [46] is a 8-item self-report questionnaire that measures the likelihood of dozing off or falling asleep in seven specific situations. A higher score indicates a higher degree of experienced sleepiness. A global ESS score (range: 0-24) > 10 distinguishes normal daytime sleepiness from excessive daytime sleepiness.

Dysfunctional Beliefs and Attitudes about Sleep. The brief version of the Dysfunctional Beliefs and Attitudes about Sleep (DBAS-16) [47] is a self-report scale assessing different types of unhelpful sleep-related cognitions playing an important role in the maintenance of insomnia. Higher scores indicate a higher level of unhelpful cognitions.

Cognitive Failures Questionnaire. The Cognitive Failures Questionnaire (CFQ) [48] is a self-report questionnaire that assesses the frequency of cognitive failures (e.g. failures in perception, memory and motor function) in daily life. A higher scores indicate more reported cognitive failures.

Multifactorial Memory Questionnaire. The Multifactorial Memory Questionnaire (MMQ) [49] is a self-report questionnaire that assesses subjective feelings (e.g. satisfaction, embarrassment) about memory function (i.e. Contentment subscale), the frequency of day-to-day life memory failures (i.e. Ability subscale) and the use of memory strategies (i.e. Strategy subscale). Higher scores indicating respectively greater contentment with memory, better memory ability and more frequent use of memory strategies. Only the Contentment and the Ability subscale were used.

Cognitive experimental EEG task

Participants were seated in an electrically shielded, dimly lit room for the duration of the whole experimental EEG session. A version of the AX-CPT [38] was performed on a 15-inch color CRT monitor connected to a computer running a Windows operating system. Stimulus delivery and the recording of behavioral data (reaction time and accuracy) were controlled by E-prime (www.pstnet.com; Psychology Software Tools). The AX-CPT was administered to measure the recruitment of cognitive

control. Participants were presented with a sequence of letters which appeared on a computer screen. These letter sequences consisted of cue-probe pairs. The task requires participants to make a target response when an A-cue is followed by an X-probe (i.e. AX-trials) and to respond with a non-target response for all other cue-probe combinations (i.e. AY-trials, BX-trials, BY-trials). Participants were instructed to respond by pressing one of two keys on a Serial Response Box (Cedrus RB-830; Cedrus Corporation, San Pedro, CA) with their right hand. Participants had to respond with their index finger on the left key when an A-cue was followed by an X-probe (i.e. target response). For all other cue-probe combinations participants had to respond with their middle finger on the right key (i.e. non-target response). Participants were asked to respond as fast and as accurately as possible, but speed was emphasized. "B" cues for the non-target BX- and BY-trials could be any letter of the alphabet, except "X", "K", and "Y" (to avoid perceptual similarity with "X"). "Y" probes for the non-target AY- and BY-trials could be any letter of the alphabet expect for "A" and "K". All stimuli (in 30-point Courier New bold) were presented in black on a white screen. Target trials (i.e. AX-trials) constituted 70% of the trials. Non-target trials (i.e. AY-trials, BX-trials, BY-trials) constituted 10% of the trials, each. This frequency distribution induces an expectancy bias for AX-trials, leading to the preparation of a target response whenever an A-cue is presented.

A total of 400 experimental trials were administered and equally divided over five blocks (i.e. 80 trials per block). Each block included 56 target trials and 8 of each of the non-target trials types in a random order. After each block, the participants were allowed to take a short break. The experiment started with 10 practice trials (50% target and 50% non-target trials) where the participants received feedback on the accuracy of their responses (i.e. the message "correct" or "incorrect"). This feedback was then omitted during the experimental blocks. Each experimental trial started with the presentation of a fixation cross which was shown for 1000 ms. Subsequently, a cue-letter was presented for 300 ms again followed by a fixation cross for 4900 ms (i.e. delay between cue and probe). Then, a probe-letter was presented for 300 ms followed by a blank screen which was presented for 1000 ms. Participants were able to provide a response from probe-onset until 1000 ms after probe offset. If they failed to

respond within this time window, the trial was marked as "no response". The inter-trial interval (ITI) randomly varied from 1000 ms to 1500 ms.

Post-experimental task questionnaire

National Aeronautics Space Administration-Task Load Index. The National Aeronautics Space Administration-Task Load Index (NASA-TLX) [51] assesses subjective workload on six dimensions. The six visual analogue subscales include: mental demand (MD), physical demand (PD), temporal demand (TD), frustration level (FL), effort level (EL) and performance level (PL). Overall workload is estimated by averaging the scores of the six subscales. Higher scores indicate a greater level of experienced subjective workload. Participants had to complete the NASA Task Load Index, in order to evaluate the subjective cognitive load they experienced during the experimental AX-CPT.

EEG recording and data pre-processing

EEG data were recorded from 64 scalp locations (BioSemi ActiveTwo System, BioSemi, Amsterdam, The Netherlands) with a sample rate of 2048 Hz. Eye movements were recorded with electrode pairs placed 1 cm above and below the eye (vertical EOG) and from the outer canthi of each eye (horizontal EOG). After recording, the EEG was down-sampled offline to a 512 Hz sample rate. For the pre-processing of the P3b cue-locked activity, recordings were epoched from -0.5 s to +2.5 s relative to the onset of the cue. For the pre-processing of the CNV cue-locked activity, recordings were epoched from -0.5 s to +5 s relative to the onset of the cue. For the pre-processing of probe-locked activity (N2 and P3a), recordings were epoched from -0.5 s to +2 s relative to the onset of the probe. Baseline correction was performed on 200 ms prior to cue and probe onset. Artefact rejection was conducted by visual inspection. Artefacts not corresponding to eye blinks were manually removed. Next, independent component analysis (ICA) was performed, using MatLab (The Mathworks, Natick, MA, USA) EEGlab toolbox [55]. Subsequently, blink components and oculomotor artefacts were identified as a result of the ICA. Based on visual inspection, these artefactual blink components were removed from the EEG data. Noisy channels were replaced by an interpolated weighted average from surrounding electrodes using the MatLab EEGlab toolbox [55]. Data from 22 participants (cue-locked pre-processing) and 14 participants (probe-locked pre-processing) contained noisy channels. For the P3b cue-locked pre-processing, 3.05 channels on average were interpolated and for the CNV cue-locked pre-processing 6.44 channels on average were interpolated. For the probe-locked (N2 and P3a) pre-processing, 3.21 channels on average were interpolated. Subsequently, trials with artefacts (voltage exceeding ± 200 μV relative to baseline, at any electrode) were removed using extreme value rejection. Finally, segments containing further artefacts, identified by visual inspection, were removed prior to averaging. Before averaging ERPs, the signals were re-referenced to the average of all 64 electrodes. For plotting purposes, data were filtered using a 25 Hz low pass filter.

Statistical approach

All statistical analyses were conducted using IBM SPSS 25 (International Business Machines, Armonk, NY, USA). When the assumption of homogeneity of variances was violated in the analyses, we reported the corrected values for degrees of freedom, *t*-values and *p*-values. A Greenhouse-Geisser correction was applied to the *p*-values (*p*GG) when the assumption of sphericity was violated. We used the Bonferroni-corrected *α*-level (0.05 / number of comparisons) in order to determine significance of the comparisons in the pairwise samples *t*-tests in order to correct for multiple comparisons.

Sleep and self-report analyses

The data from the sleep diaries were averaged over the 14 days between session 1 and session 2. The self-report data (sleep-, daytime- and cognitive functioning), the sleep diary data and the PSG data were evaluated using a non-parametric Mann-Whitney *U* test, since normality was not observed in all these variables.

ERP components and analyses

In previous ERP studies using the AX-CPT, ERPs that are modulated by recruitment of proactive and reactive control have been reliably identified (e.g. [34, 35]). To examine whether Insomnia Disorder was related to impaired proactive control, we examined standard ERPs for cue-related components (P3b and CNV) and probe-related components (N2 and P3a). Only the experimental trials of the correct responses (on average 76-77% for the CNV and P3b cue-locked analyses and 84% for the N2 and P3a probe-locked analyses, respectively) were included in the ERP analyses. To define the spatial topography and time-windows, we first averaged the waveforms across groups for each cue (i.e. A-cue and B-cue) and each Trial Type (i.e. AX, AY, BX, BY). Based on the collapsed waveforms, we defined the spatial topography for the analysis of respectively cue components based on the grand-average difference plot between B-cues and A-cues, and probe components based on a grand-average difference plot of response conflict (BY-trials - AY-trials) again without taking group into account. In line with

previous research, these components were examined by averaging the relevant region of interest [e.g. 56-57]. Based on the collapsed waveforms and on prior research focusing on these ERP components, we defined the time-windows for each component. Subsequent EEG analysis, data averaging and data handling were conducted using MatLab and custom-built MatLab scripts.

Proactive control can be indexed by several ERP components during the cue-probe interval. First, the centro-parietal P3b component peaks around 300-600 ms after cue presentation and is believed to reflect target categorization, context updating and maintenance of task-relevant information [58-60]. Its amplitude increases with the presentation of novel task-relevant stimulus information [61]. For example, larger P3b amplitudes have been observed for B-cues compared to A-cues, since B-cues appear with a lower frequency than A-cues and can therefore be considered as more novel [34]. This cue-dependent modulation of the P3b component reflects that context-relevant novel information has been correctly updated and maintained, which characterizes enhanced proactive control mechanisms. Later in time, the contingent negative variation (CNV) emerges post-cue, which is thought to reflect expectation and general response preparation [62]. The CNV is a slow cortical potential that appears after a warning stimulus (e.g. a cue) and that announces the preparation of a motor action to a subsequent stimulus (e.g. a probe). Larger CNV amplitudes have been associated with increased response preparatory processes and thus an increased proactive control engagement (e.g. [35]). Since the interval between cue and probe is longer than one second in our design, the CNV can be separated into an initial CNV (iCNV) and a late CNV (lCNV) [63, 64]. The iCNV is supposed to reflect attention allocation to task-relevant information in order to prepare for an adequate response. The lCNV is assumed to reflect the readiness potential preceding actions [65].

P3b. For the P3b, the EEG over the centro-parietal electrodes CP1, CPz, CP2, P1, Pz, P2, PO3, POz, and PO4 was filtered at 0.01 Hz (high-pass filter) and 30 Hz (low-pass filter), slope 24 dB/octave. B-cues showed a later peak amplitude than the A-cues due to the fact that B-cues were presented with a lower frequency than A-cues. For that reason and in line with the approach used in prior research using the AX-CPT [34, 35], the P3b amplitude was calculated over two time-windows (i.e. 300 ms to 500 ms

after cue presentation and 400 ms to 700 ms after cue presentation), corresponding to the latencies which the grand averages exceeded a quarter of the peak amplitudes for respectively A-cues and B-cues.

CNV. For the CNV, the EEG was filtered over the fronto-central electrodes Fz, FCz, Cz, and CPz at 0.01 Hz (high-pass filter) and 30 Hz (low-pass filter), slope 24 dB/octave. As explained above [63, 64], we studied the CNV during two time-periods, more precisely, we examined the initial CNV (i.e. iCNV) in a time window between 1000 ms and 2500 ms post-cue and the late CNV (i.e. lCNV) in a time window between 3400 ms and 4900 ms post-cue.

Both the P3b and the iCNV and lCNV following the cue were analyzed by comparing amplitudes in a repeated-measures ANOVA with the factors Cue (2 levels: A-cue, B-cue) and Group (2 levels: ID or GSC), in line with previous research with this task [34, 35].

Reactive control can be indexed by several ERP components following the probe presentation. First, a fronto-central N2 component around 200-300 ms post-probe, which correlates with the activation of the anterior cingulate cortex (ACC), a structure that is associated with conflict detection [66]. The N2 is assumed to be involved in inhibiting an incorrect response tendency that is for example elicited by invalid cue information and conflicts with the probe information. For example, larger N2 amplitudes have been observed in AY-trials compared to the other types of trials, where participants prepared a target response after the presentation of an A-cue, which conflicted with the subsequent Y-probe information (e.g. [35]). Following the N2, a later P3a component emerges around 300-600 ms post-probe, indexing stimulus evaluation [59; 67, 68] and response inhibition [69, 70]. The evaluative P3a is supposed to reflect the need to monitor inhibitory processes required to overcome robust response tendencies. In AY-trials, for example, larger P3a amplitudes have been observed, which reflected inhibitory processes engaged to overcome a prepared target response elicited by the A-cue (e.g. [34]).

N2. In order to avoid masking of the N2 by the larger P3a amplitudes a 2 Hz (high-pass) and a 12 Hz (low-pass), slope 24 dB/octave filter was applied to filter out the P3a component [34, 35, 71]. The N2 was calculated over the centro-parietal electrodes FCz, Cz, CPz and Pz in the 40 ms period around the

peak of the component, which resulted in a time-window between 270 ms and 310 ms (for a similar method, see also [56, 57]).

P3a. For the P3a, the EEG over the frontal electrode Fz was filtered at 0.01 Hz (high-pass) and 30 Hz (low-pass), slope 24 dB/octave. The P3a was calculated over the electrode Fz in the 80 ms period around the peak of the component. This resulted in a time-window between 300 ms and 380 ms (for a similar method, see also [57]).

Both the N2 and the P3a following the probe were analyzed by comparing amplitudes in a repeated-measures ANOVA with the factors Trial Type (4 levels: AX, AY, BX, BY) and Group (2 levels: ID and GSC), in line with previous research with this task [35].

Cognitive performance analyses

We conducted a repeated-measures ANOVA with the factors Trial Type (4 levels: AX, AY, BX, BY) and Group (2 levels: ID and GSC) on the median RTs of correct trials (on average 96%) and the mean error rates on the AX-CPT.

Predictions

Figure 3 summarizes the underlying control mechanisms related to the four trial types and the involved ERP components associated with these control processes.

Cognitive performance hypothesis

An increased use of proactive control (i.e. maintenance of the task-relevant cue information to prepare a response in advance) will result in the preparation of a target response whenever an A-cue is encountered and the preparation of a non-target response whenever a B-cue is encountered. This cue-based preparation will lead to decreased performance (i.e. slower reaction times and higher error rates) on AY-trials, where the initial preparation of a target response based on the cue needs to be suppressed in order to make the correct (i.e. non-target) response. Impaired proactive control will result in the opposite pattern, that is, an enhanced or preserved performance (i.e. faster reaction times and lower error rates or no change in reaction times and error rates) on AY-trials. As the maintenance of the A-cue is impaired, it will not induce an incorrect response tendency towards a target response on these AY-trials.

In summary, we hypothesize behavioral AY-performance to be preserved or even better (i.e. faster reaction times and lower error rates) in ID compared to GSC, as ID are less hindered by the maintenance of the A-cue information on this type of trial. According to previous studies examining cognitive control functions in insomnia [24, 25], it may also be that no differences in behavioral performance between groups can be evidenced due to compensatory mechanisms resulting in preserved performance in insomnia.

ERP hypothesis

For the cue-related components, we generally expect that the P3b amplitude will be larger for B-cues compared to A-cues since the B-cues are presented with a lower frequency (20% compared to 80%) and thus a higher novelty. Based on our hypothesis that ID individuals present with a specific alteration of

proactive control, we predict the P3b to be reduced for both conditions in this group, reflecting their impaired engagement with this task relevant cue. Moreover, we expect an iCNV and lCNV after A-cues and B-cues, since both ask for a response preparation. However, we expect that the iCNV and lCNV amplitudes will be larger for B-cues compared to A-cues. Specifically, previous research has demonstrated that prior probability information (e.g. a cue stimulus) provides advance information regarding a subsequent stimulus requiring a response (e.g. a probe stimulus) and this probability information induces preparatory processes at a premotor stage. This premotor preparation, which is reflected by a modulation of the CNV component, leads to facilitation of the subsequent processing and results in faster reaction times [72]. In the AX-CPT task, participants should prepare a response to the upcoming probe after the presentation of an A-cue and B-cue. In case of B-cues, participants should prepare a non-target response regardless of the probe presented. In contrast, although A-cues will require a target response on the majority of trials, the final response can only be determined once the probe is presented. As such, participants should have greater response preparation in the cue period following B-cues. This response preparation should evoke a clear differential activation in CNV amplitudes for A-cues and B-cues, indicating more adequate response preparation processes. Crucially, we hypothesize that ID will be associated with impaired proactive control and thus impaired use of the cue information to adequately prepare for the upcoming likely response. Since, as described above, effective use of B-cues should result in greater response preparation (compared to A-cues), we expect the difference in CNV between A-cues and B-cues to be smaller in the ID group, reflecting a reduced ability to engage in proactive control.

For the probe-related components, we generally expect that the N2 and P3a amplitude will be larger for AY-trials compared to the other types of trials. More specifically, behavioral performance on AY-trials will generally be impaired compared to other types of trials, due to the fact that the vast majority of A-cues (on 7 out of 8 trials) are followed by an X-probe, and thus require a target response. As such, on AY-trials, the A-cue information (that activates a target response) will conflict with the Y-probe information (that activates non-target response). Hence, participants will detect a response conflict (as reflected by larger N2 amplitudes) and will need to inhibit a pre-potent tendency to make a target

response (as reflected by larger P3b amplitudes). Due to an impaired proactive maintenance of cue-information, we expect that the N2 and P3a amplitudes during AY-trials will be smaller for ID compared to GSC. More precisely, the A-cue information will then not be maintained. Consequently, this A-cue information will conflict to a lesser extent with the Y-probe information leading to smaller N2 amplitudes (due to a smaller response conflict) and smaller P3b amplitudes (due to a lesser need for inhibition of an incorrect response).

Cue	Probe	% Trials	Required response	Response preparation	Underlying mechanisms for correct response	Preferred control mode
A	X	70%	Target	Cue and probe activate target response		
B	X	10%	Non-target	**Bias:** probe induces target response	Active maintenance of the cue	Proactive control
A	Y	10%	Non-target	**Bias:** cue prepares target response	Suppression of cue activation	Reactive control
B	Y	10%	Non-target	Cue and probe activate non-target response		
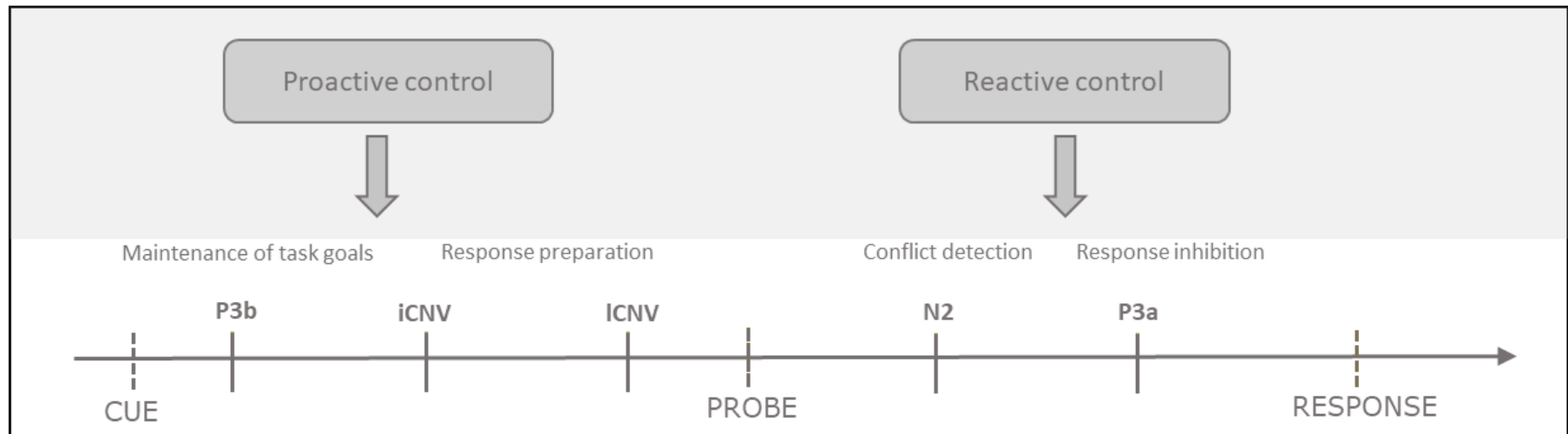
Figure 3. Schematic overview of the underlying control mechanisms related to the four trial types (upper table) and the involved ERP components associated with these control processes (lower figure)

RESULTS

Sample description

Thirty-six volunteers participated in the experiment. The ID group consisted of 20 participants and the good sleeper controls consisted of 16 participants. Two participants made significantly more errors (+ 2.5 SDs) than their group mean (i.e. ID or GSC) and were therefore excluded from further analysis. The data of one additional participant was excluded because of technical problems with the EEG recording. Thus, the final ID group consisted of 18 participants with a mean age of 32 years ($SD = 11.36$, age range 20 - 53 years) and the GSC group consisted of 15 participants with a mean age of 32 years ($SD = 10.94$, age range 21 - 53 years). Participants of each group were statistically equivalent in age ($t = 0.082$, $p = 0.94$) and in gender distribution ($\chi^2 = 0.071$, $p = 0.79$). There were no significant differences on the apnea/hypopnea index ($z = -1.84$, $p = 0.067$) and the periodic limb movements during sleep index ($z = -0.13$, $p = 0.90$).

Sleep parameters

Medians, interquartile ranges and between-group comparisons for the sleep variables are presented in Table 1.

Polysomnography. Due to technical problems with the PSG recording equipment, the data of five participants were not included in between-group comparisons for recorded sleep variables. The ID group and the GSC group did not differ with respect to any of the recorded sleep architecture and sleep continuity PSG parameters. Significant differences in sleep architecture and sleep continuity between ID and GSC based on one-night PSG are rarely found (for similar results see for example [25, 29]).

Sleep Diary and Self-Report. The ID group reported significantly more insomnia complaints (ISI; $z = -4.85$, $p < 0.001$, $r = 0.84$) and a worse sleep quality over the past month (PSQI; $z = -4.26$, $p < 0.001$, $r =$

0.74) compared to the GSC group. With regards to the sleep diary variables, all group differences are in line with our expectations. Finally, the ID group reported a lower sleep quality the night before session 2 compared to the GSC group ($z = -3.07$, $p = 0.002$, $r = -0.53$).

Table 1. Between-group comparisons on subjective and objective sleep variables.

	ID (n = 18)	GSC (n = 15)	ID vs GSC
	Median (± *IQR*)	Median (± *IQR*)	Effect size (*r*)
ISI	15.00 (7.25)	3.00 (4.00)	-0.84***
PSQI	10.00 (6.00)	4.00 (3.00)	-0.74***
Sleep Diaries (average 14 nights)			
SOL (min)	33.38 (15.00)	8.57 (22.50)	-0.60**
WASO (min)	35.90 (57.83)	7.50 (16.07)	-0.62***
EMA (min)	27.51 (20.76)	12.86 (15.00)	-0.35*
TIB (min)	483.25 (55.21)	492.86 (43.93)	-0.24
TST (min)	385.09 (91.63)	456.43 (70.71)	-0.60***
% SE	80.83 (15.81)	93.11 (6.67)	-0.67***
SQ	5.68 (1.33)	8.07 (0.93)	-0.80***
Sleep Diaries (night before session 2)			
SQ	7.00 (1.75)	8.00 (2.00)	-0.53**
PSG (1 night)			
AHI (events/hour)	1.25 (3.30)	0.30 (1.70)	-0.32
PLMSi (events/hour)	0.00 (1.73)	0.00 (2.20)	-0.022
	ID (n = 16)	GSC (n = 12)	ID vs GSC
	Median (± *IQR*)	Median (± *IQR*)	Effect size (*r*)
SOL (min)	20.50 (19.38)	9.25 (17.00)	-0.12
WASO (min)	24.00 (40.13)	39.50 (45.50)	-0.070
EMA (min)	2.25 (19.10)	5.54 (16.99)	-0.045
TIB (min)	485.20 (103.93)	504.40 (112.05)	-0.11
TST (min)	408.75 (73.00)	440.75 (125.88)	-0.19
% SE	87.50 (15.68)	87.30 (12.68)	-0.12
N1 (%)	10.80 (9.85)	12.65 (8.05)	-0.08
N2 (%)	44.55 (14.83)	39.70 (7.98)	-0.18
SWS (%)	25.40 (10.45)	22.00 (15.43)	-0.070
REM (%)	19.50 (9.95)	22.90 (7.85)	-0.28
ArI (arousals/hour)	10.91 (6.15)	9.72 (13.61)	-0.001

ID (Individuals with Insomnia Disorder); GSC (Good Sleeper Controls); ISI (Insomnia Severity Index); PSQI (Pittsburgh Sleep Quality Index); SOL (Sleep Onset Latency), WASO (Wake After Sleep Onset), TWT (Total Wake Time), TIB (Time In Bed) and TST (Total Sleep Time) in minutes (min); SE (Sleep Efficiency = (TST/TIB)x100) in percent (%); SQ (Perceived Sleep Quality); N1 (Sleep Stage 1), N2 (Sleep Stage 2), SWS (Slow Wave Sleep) and REM (Rapid Eye Movement Sleep) in percent (%) of TST; ArI (Arousal Index) in arousals per hour of sleep; AHI (Apnea-hypopnea Index) per hour of sleep; PLMSi (Periodic Limb Movements during Sleep index) per hour of sleep; IQR (Interquartile range). *$p < 0.05$, **$p < 0.01$, ***$p < 0.001$.

Daytime Symptoms.

Medians, interquartile ranges and between-group comparisons for the daytime variables are presented in Table 2. With respect to daytime symptoms, significant group differences were found in the expected direction. With regard to the cognitive parameters, ID reported more everyday cognitive failures compared to GSC (CFQ; z = -2.53, p = 0.011, r = -0.44), while no difference was observed between groups regarding reported memory contentment and memory ability. All daytime cognitive variables (sleep diaries) showed a significant difference between groups. Finally, the ID group and the GSC group did not differ with regard to any of the experienced cognitive task-workload dimensions (NASA-TLX).

Table 2. Between-group comparisons on daytime variables and subjective cognitive variables.

	ID (*n* = 18)	GSC (*n* = 15)	ID vs GSC
	Median (± *IQR*)	Median (± *IQR*)	Effect size (*r*)
Daytime variables			
BDI	12.00 (12.75)	1.00 (6.00)	-0.57**
Cognitive	4.00 (3.25)	0.00 (2.00)	-0.46**
Somatic-Affective	6.50 (6.50)	1.00 (3.00)	-0.50**
RRS	41.50 (11.75)	28.00 (10.00)	-0.50**
STAI (Trait)	45.50 (16.50)	33.00 (6.00)	-0.56**
MFI			
General Fatigue	15.00 (4.50)	8.00 (5.00)	-0.76***
Physical Fatigue	12.50 (5.50)	7.00 (4.00)	-0.57**
Decreased Activity	11.50 (7.25)	6.00 (4.00)	-0.63***
Decreased Motivation	10.00 (5.00)	6.00 (3.00)	-0.46**
Mental Fatigue	13.50 (7.50)	8.00 (4.00)	-0.46**
ESS	6.50 (9.25)	6.00 (4.00)	-0.23
Cognitive variables			
CFQ	41.00 (18.75)	25.00 (19.00)	-0.44*
MMQ			
Contentment	54.50 (23.50)	58.00 (11.00)	-0.30
Ability	52.00 (22.50)	64.00 (19.00)	-0.26
Cognitive function (Sleep diary, average14 days)			
Attention and concentration	6.18 (1.41)	7.86 (0.86)	-0.74***
Memory	6.42 (1.44)	8.21 (1.32)	-0.67***
Planning and organization	6.74 (1.82)	8.21 (0.72)	-0.61***
NASA-TLX			
Mental demands	12.50 (6.25)	10.00 (11.00)	-0.20
Physical demands	6.50 (13.25)	3.00 (2.00)	-0.31
Temporal demands	5.00 (9.25)	7.00 (4.00)	-0.05
Performance	6.00 (5.75)	6.00 (2.00)	-0.10
Effort	13.00 (2.25)	13.00 (8.00)	-0.04
Frustration	11.00 (10.25)	4.00 (8.00)	-0.30

ID (Individuals with Insomnia Disorder); GSC (Good Sleeper Controls); BDI (Beck Depression Inventory); RRS (Ruminative Response Scale); STAI (State-Trait Anxiety Scale); MFI (Multidimensional Fatigue Scale); ESS (Epworth Sleepiness Scale); CFQ (Cognitive Failures Questionnaire); MMQ (Multifactorial Memory Questionnaire); NASA-TLX (National Aeronautics Space Administration-Task Load Index); IQR (Interquartile range). *$p < 0.05$, **$p < 0.01$, ***$p < 0.001$

EEG results

Cue locked ERPs[a]

P3b. Figure 4 presents the grand average cue-locked ERPs for the centro-parietal electrodes elicited by the different cues (i.e. A and B) for the different groups (i.e. ID and GSC) and the topographic distribution plots representing the average voltage measured for the P3b effect (B - A cues) over the first P3b time-window (300 ms - 500 ms).

A repeated measures ANOVA with the factors Cue (2 levels: A and B) and Group (2 levels: ID and GSC) on the mean average voltage of correct trials during the *first P3b time-window* (300 ms - 500 ms), across the centro-parietal electrodes showed a main effect of Cue ($F(1,31) = 4.22$, $p = 0.049$, $\eta_p^2 = 0.12$). The P3b amplitude was larger for B-cues ($M_{B\text{-cue}} = 2.66$ µV) compared to that for A-cues ($M_{A\text{-cue}} = 2.09$ µV). Crucially, there was a main effect of Group ($F(1,31) = 4.62$, $p = 0.040$, $\eta_p^2 = 0.13$). The P3b amplitude across cues was larger for GSC ($M_{GSC} = 3.03$ µV) compared to that for ID ($M_{ID} = 1.82$ µV), which indicates that the maintenance of task-related information is decreased in ID compared to GSC. No interaction between Cue and Group was found.

The same repeated measures ANOVA on the *second P3b time-window* (400 ms - 700 ms) across centro-parietal electrodes, similarly showed a main effect of Cue ($F(1,31) = 31.41$, $p < 0.001$, $\eta_p^2 = 0.50$). The P3b amplitude was larger for B-cues ($M_{B\text{-cue}} = 3.24$ µV) compared to that for A-cues ($M_{A\text{-cue}} = 1.36$ µV). None of the other main or interaction effects reached significance.
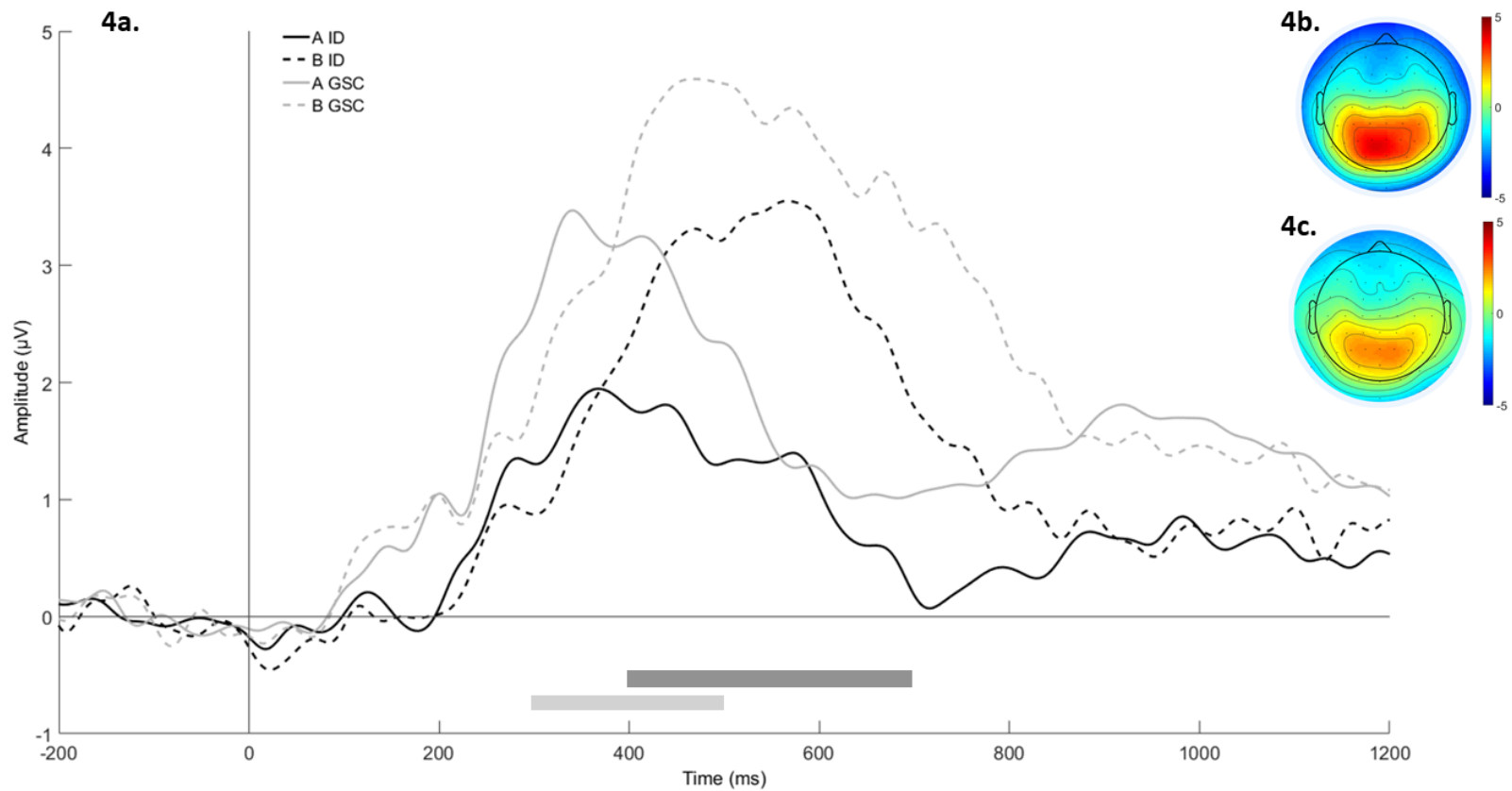
Figure 4. 4a. Grand averages of the cue-locked ERPs evoked at the centro-parietal electrodes (CP1, P1, PO3, POz, Pz, CPz, CP2, P2 and PO4) for GSC and for ID. Light gray and dark gray horizontal bars indicate the two P3b time-windows of analysis, respectively 300 ms to 500 ms and 400 ms to 700 ms. 4bc. Topographic distribution plots for the P3b effect (B - A cues) over the 300 ms to 500 ms time-window for GSC (4b) and for ID (4c). ID (Individuals with Insomnia Disorder); GSC (Good Sleeper Controls); A (A cue); B (B cue).

CNV. Figure 5 presents the grand average cue-locked ERPs for the fronto-central electrodes elicited by the different cues (i.e. A and B) for the different groups (i.e. ID and GSC) and the topographic distribution plots representing the average voltage measured for the iCNV effect (B - A cues) over the iCNV time-window (1000 ms - 2500 ms)

A repeated measures ANOVA with the factors Cue (2 levels: A and B) and Group (2 levels: ID and GSC) on the mean average voltage of correct trials during the *iCNV time-window* (1000 ms - 2500 ms), across the fronto-central electrodes showed a main effect of Cue ($F(1,31) = 20.86$, $p < 0.001$, $\eta_p^2 = 0.40$). The iCNV amplitude for B-cues ($M_{\text{B-cue}} = -0.72$ µV) was larger compared to that for A-cues ($M_{\text{A-cue}} = 0.69$ µV). Crucially, there was an interaction between Cue and Group ($F(1,31) = 4.54$, $p = 0.041$, $\eta_p^2 = 0.13$), indicating that the difference between the iCNV amplitudes for B-cues and A-cues was smaller for ID ($M_{\text{ID-B-A-cue}} = -0.79$ µV), compared to for GSC ($M_{\text{ID-B-A-cue}} = -2.16$ µV). This result indicates that response preparation after cue presentation is hampered in ID compared to GSC. No main effect of Group was observed.

The same repeated measures ANOVA on the *lCNV time-window* (3400 ms - 4900 ms), across the fronto-central electrodes similarly showed a main effect of Cue ($F(1,31) = 4.59$, $p = 0.040$, $\eta_p^2 = 0.13$). The lCNV amplitude was larger for B-cues ($M_{\text{B-cue}} = -1.60$ µV) compared to that for A-cues ($M_{\text{A-cue}} = -0.66$ µV). None of the other main or interaction effects reached significance.
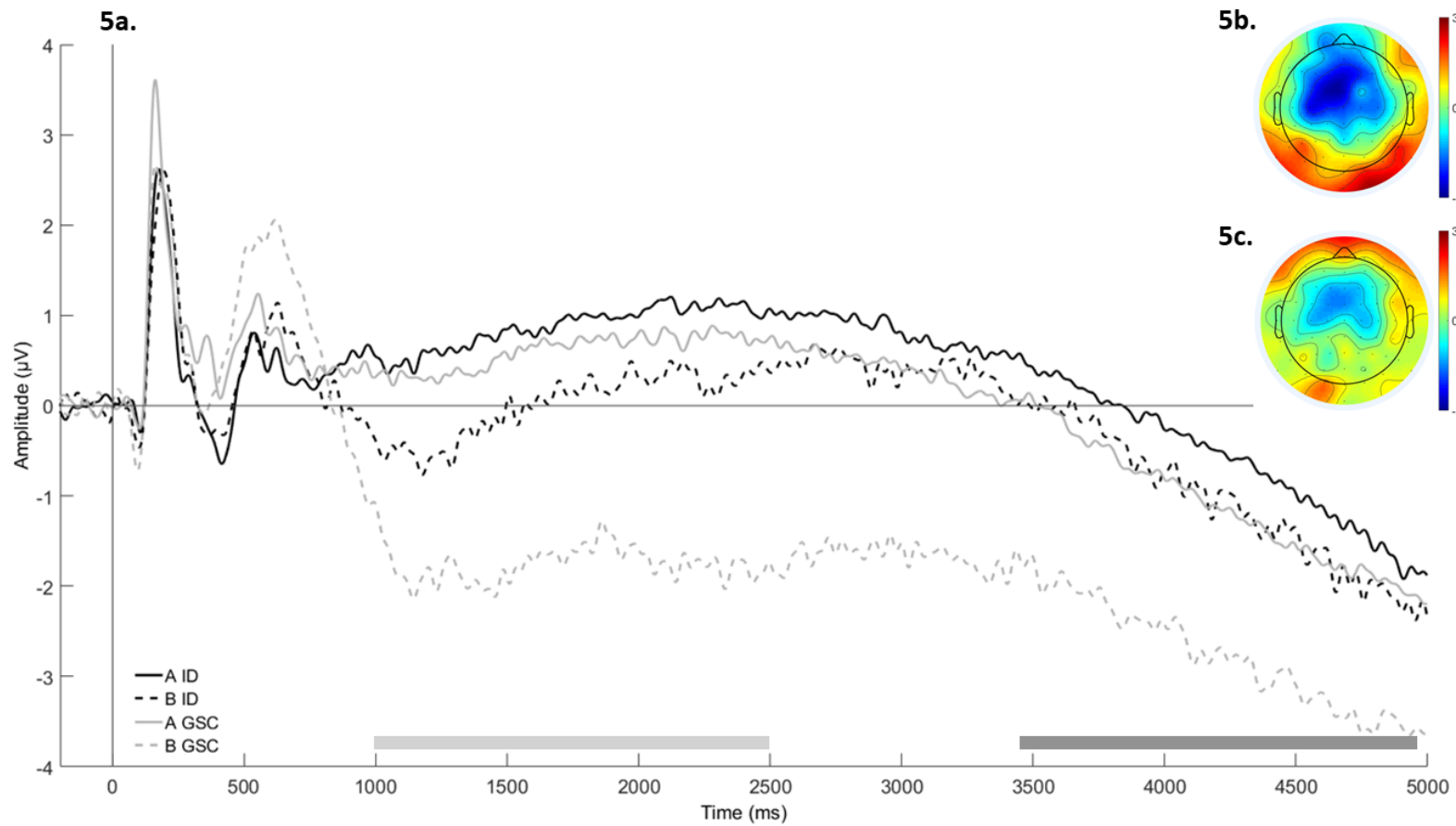
Figure 5. 5a. Grand averages of the cue-locked ERPs evoked at the fronto-central electrodes (Fz, FCz, Cz and CPz) for GSC and for ID . The light grey bar indicates the iCNV window of

analysis (1000 ms to 2500 ms) and the dark-grey bar indicated the lCNV window of analysis (3400 ms to 4900 ms). 5bc. Topographic distribution plots for the iCNV effect (B - A cues) over the

1000 ms to 2500 ms time-window for GSC (5b) and for ID (5c). ID (Individuals with Insomnia Disorder); GSC (Good Sleeper Controls); A (A cue); B (B cue).

Probe locked ERPs

N2. Figure 6 presents the grand average probe-locked ERPs for the centro-parietal electrodes elicited by the different trial types (i.e. AX, AY, BX, BY) for the different groups (i.e. ID and GSC). Figure 7 presents the grand average probe-locked ERPs for the centro-parietal electrodes elicited by the different trial types (i.e. AY and BY) for the different groups (i.e. ID and GSC) and the topographic distribution plots representing the average voltage measured for the N2 effect (BY - AY trials) over the N2 time-window (270 ms - 310 ms).

A repeated measures ANOVA with the factors Trial Type (4 levels: AX, AY, BX, BY) and Group (2 levels: ID and GSC) on the mean average voltage of correct trials during the *N2 time-window* (270 ms - 310 ms) across the centro-parietal electrodes showed a main effect of Trial Type ($F(3,29) = 21.38$, $pGG < 0.001$, $\eta_p^2 = 0.69$). Post-hoc Bonferroni-corrected paired samples *t*-tests indicated that the N2 amplitude for AY-trials ($M_{AY} = -0.92$ µV) was larger than that for AX-trials ($M_{AX} = -0.23$ µV, $t = 4.49$, $p < 0.001$), BX-trials ($M_{BX} = 0.10$ µV, $t = -7.64$, $p < 0.001$) and BY-trials ($M_{BY} = -0.32$ µV, $t = -4.25$, $p < 0.001$). The N2 amplitude for AX-trials was also larger than that for BX-trials ($t = -3.41$, $p = 0.002$) and the N2 amplitude for BY-trials was larger than that for BX-trials ($t = 4.73$, $p < 0.001$). No difference was found between the N2 amplitude for AX-trials and BY-trials. An interaction between Trial Type and Group was present ($F(3,29) = 2.64$, $pGG = 0.047$, $\eta_p^2 = 0.21$). Planned comparisons between AY-trials and the other types of trials showed that only the difference in N2 amplitude between AY- and BY-trials was significantly larger for GSC ($M_{GSC\_BY-AY} = 0.98$ µV) than for ID ($M_{ID\_BY-AY} = 0.28$ µV, $F(1,31) = 7.31$, $p = 0.011$, $\eta_p^2 = 0.19$) which indicates that ID experience less conflict to AY trials compared to GSC. No difference in N2 amplitude between groups was found between AY-trials and AX-trials and between AY-trials and BX-trials. Means, standard deviations, standard errors of the mean of the Trial Types contrasts (i.e. AY trials vs the other types of trials) on the N2 amplitudes for each group separately and between-group comparisons are presented in Table 3.
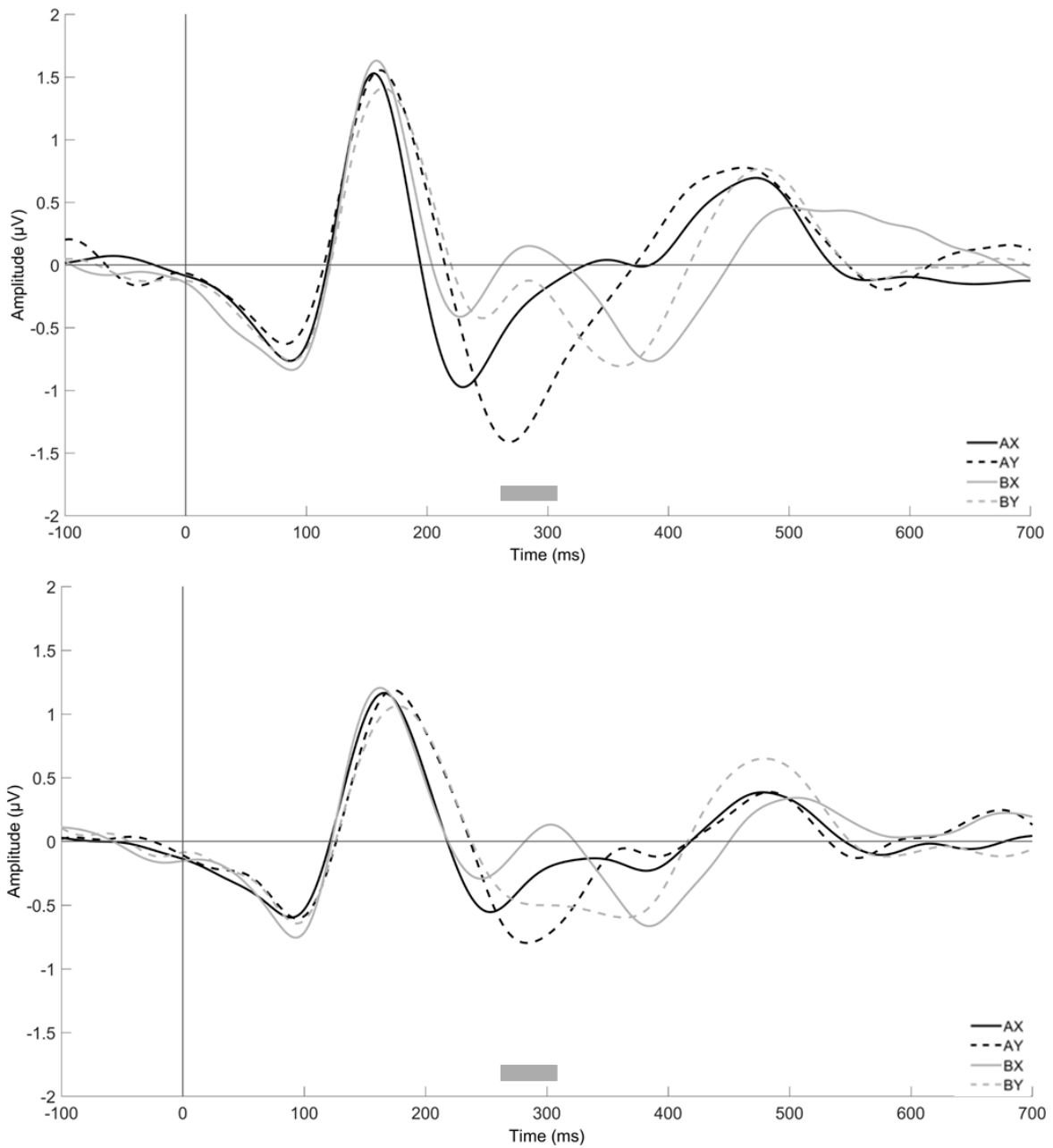
Figure 6. Grand averages of the probe-locked ERPs evoked over the centro-parietal electrodes (FCz, Cz, CPz and Pz) for GSC (upper plot) and for ID (lower plot). The grey bar indicates the N2 window of analysis (270 ms to 310 ms). ID (Individuals with Insomnia Disorder); GSC (Good Sleeper Controls); AX (AX-trials); AY (AY-trials); BX (BX-trials); BY (BY-trials).
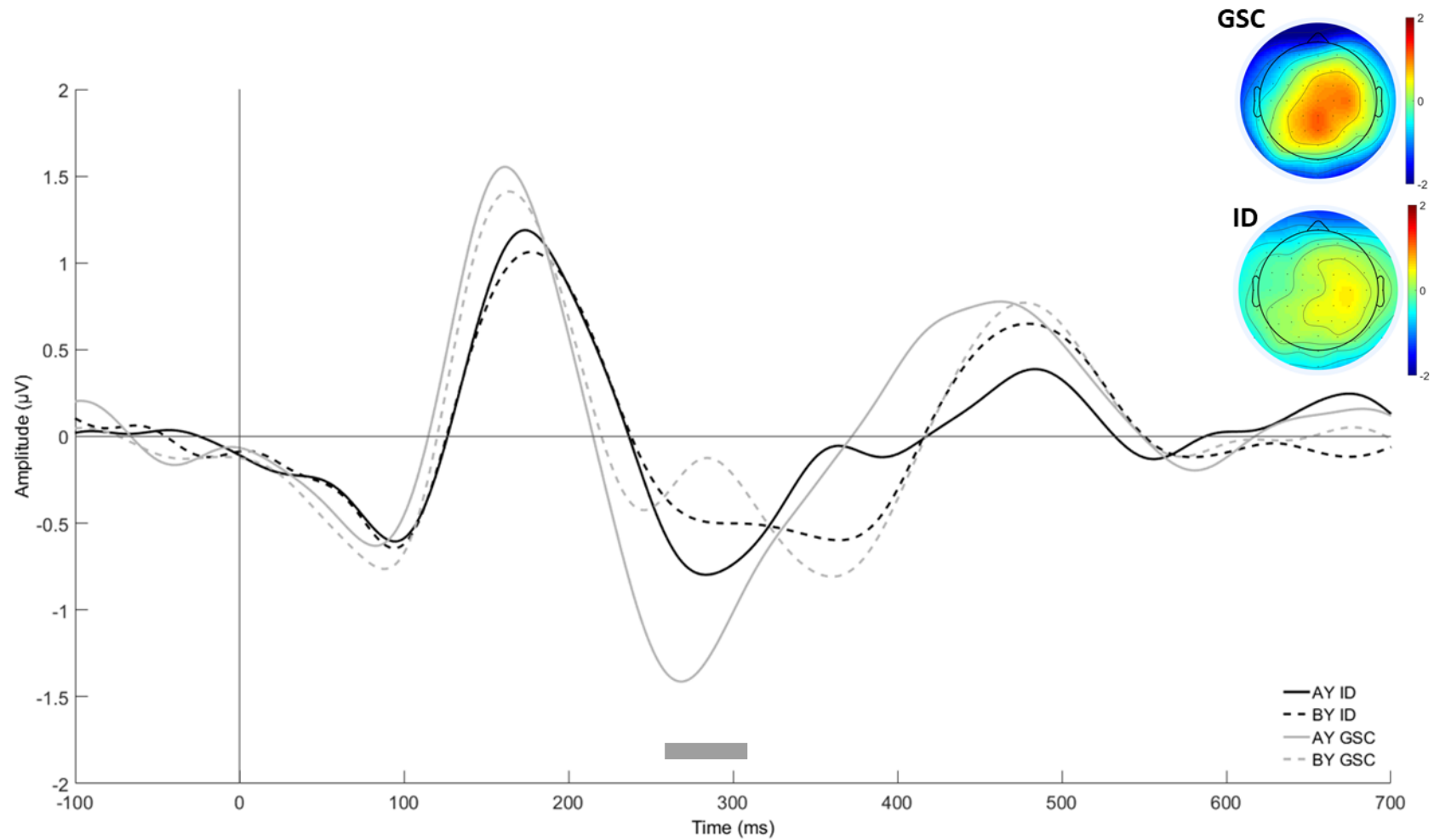
Figure 7. 7a. Grand averages of the probe-locked ERPs evoked over the centro-parietal electrodes (FCz, Cz, CPz and Pz) for GSC and for ID. The grey bar indicates the N2 window of analysis (270 ms to 310 ms). 7bc. Topographic distribution plots for the N2 effect (BY - AY trials) over the 270 ms to 310 ms time-window for GSC (7b) and for ID (7c). ID (Individuals with Insomnia Disorder); GSC (Good Sleeper Controls); AY (AY trials); BY (BY trials).

Table 3. Contrasts comparing the N2 amplitude per Trial Type for each group separately and between groups.

Trial Types		ID	GSC	ID vs GSC
		Mean ± *SD*	Mean ± *SD*	Mean ± *SE*
AY	AX	0.48 (0.73)*	0.91 (0.98)**	0.43 (0.30)
	BX	-0.82 (0.76)***	-1.27 (0.73)***	-0.45 (0.26)
	BY	-0.28 (0.62)	-0.98 (0.86)**	-0.70 (0.26)*

ID (Individuals with Insomnia Disorder); GSC (Good Sleeper Controls); AX (AX-trials); AY (AY-trials); BX (BX-trials); BY (BY-trials) *$p < 0.05$, **$p < 0.01$, ***$p < 0.001$.

P3a. Figure 8. presents the grand average probe-locked ERPs for the frontal electrode elicited by the different trial types (i.e. AX, AY, BX, BY) for the different groups (i.e. ID and GSC).

A repeated measures ANOVA with the factors Trial Type (4 levels: AX, AY, BX, BY) and Group (2 levels: ID and GSC) on the mean average voltage of correct trials during the P3a time-window (300 ms - 380 ms) across the frontal electrode showed a main effect of Trial Type ($F(3,29) = 6.51$, $pGG < 0.001$, $\eta_p^2 = 0.40$). Post-hoc Bonferroni-corrected paired samples t-tests indicated that the P3a amplitude for AY-trials ($M_{AY} = -1.63$ µV) was larger than that for AX-trials ($M_{AX} = -2.94$ µV, $t = -3.38$, $p = 0.002$), BX-trials ($M_{BX} = -3.60$ µV, $t = 3.92$, $p < 0.001$) and BY-trials ($M_{BY} = -4.06$ µV, $t = 4.59$, $p < 0.001$). The P3a amplitude for AX-trials was also larger than that for BY-trials ($t = 3.51$, $p = 0.001$). No difference in P3a amplitude was found between BX-trials and BY-trials and between AX-trials and BX-trials. None of the other main or interaction effects reached significance.
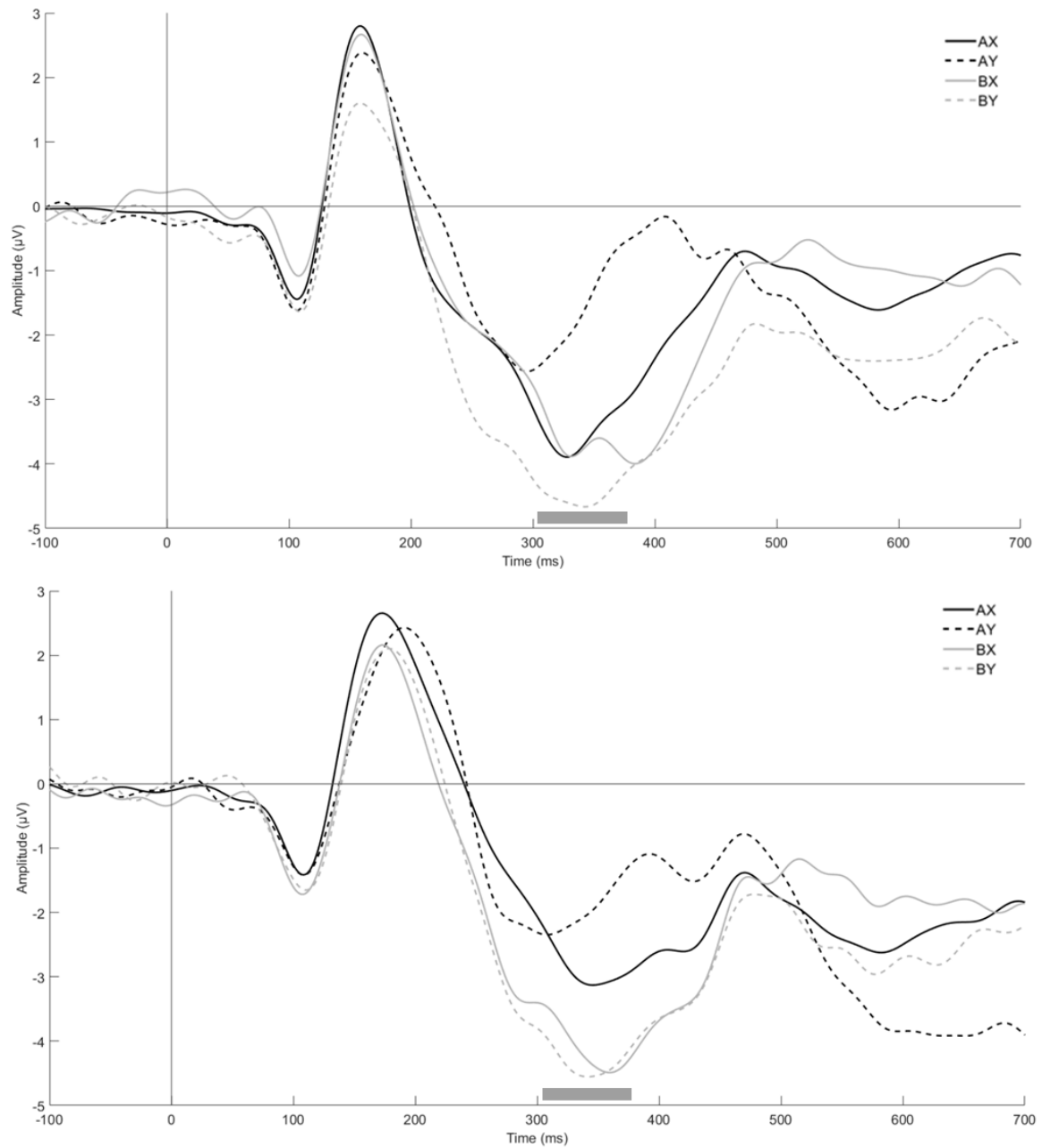
Figure 8. Grand averages of the probe-locked ERPs evoked at the frontal electrode (Fz) for GSC (upper plot) and for ID (lower

plot). The grey bar indicates the P3a window of analysis (300 ms to 380 ms). ID (Individuals with Insomnia Disorder); GSC

(Good Sleeper Controls); AX (AX-trials); AY (AY-trials); BX (BX-trials); BY (BY-trials).

Cognitive performance results

The median RTs of correct responses and mean error rates as a function of Trial Type (i.e. AX, AY, BX, BY) and Group (i.e. ID and GSC) are summarized in Table 4.

Reaction Times

Inaccurate responses (on average 4.00 %) were discarded for the RT analyses. A repeated measures ANOVA with the factors Trial Type (4 levels: AX, AY, BX, BY) and Group (2 levels: ID and GSC) on the median RTs showed a main effect of Trial Type ($F(3,29) = 132.50$, pGG < 0.001, $\eta_p^2 = 0.93$). Post-hoc Bonferroni-corrected paired samples t-tests indicated that participants were slower on AY-trials ($M_{AY} = 627$ ms) compared to AX-trials ($M_{AX} = 502$ ms, $t = 13.36$, $p < 0.001$), BX-trials ($M_{BX} = 433$ ms, $t = 15.69$, $p < 0.001$) and BY-trials ($M_{BY} = 433$ ms, $t = 17.95$, $p < 0.001$). Participants were also slower on AX-trials compared to BX-trials ($t = 5.38$, $p < 0.001$) and BY-trials ($t = 5.51$, $p < 0.001$). No difference in reaction times was found between BX-trials and BY-trials. None of the other main or interaction effects reached significance.

Error rates

A repeated-measures ANOVA with the factors Trial Type (4 levels: AX, AY, BX, BY) and Group (2 levels: ID and GSC) on the mean error rates showed a main effect of Trial Type ($F(3,29) = 9.34$, pGG < 0.001, $\eta_p^2 = 0.49$). Post-hoc Bonferroni-corrected paired samples t-tests indicated that participants made more errors on AY-trials ($M_{AY} = 10.64$ %) compared to AX-trials ($M_{AX} = 3.18$ %, $t = -4.13$, $p < 0.001$), BX-trials ($M_{BX} = 4.58$ %, $t = 2.90$, $p = 0.007$) and BY-trials ($M_{BY} = 2.09$ %, $t = 4.87$, $p < 0.001$). No differences in error rates were found between BX-trials and AX-trials and between BX-trials and BY-trials. None of the other main or interaction effects reached significance.

Link between experienced cognitive task-load and electrophysiological results

Based on the results of the cue-locked data, we can conclude that ID show a reduced ability to engage in proactive control as reflected by an ineffective use of the cue-information in order to prepare a

response (i.e. smaller difference in iCNV between A-cues and B-cues in the ID group compared to the GSC group). Interestingly, we can investigate whether this impaired engagement of proactive control is crucially linked to the often reported cognitive control complaints in insomnia. If this is the case, a relation between self-reported task performance (as indexed by the performance scale of the NASA-TLX) and the magnitude of the interaction in the iCNV component (reflecting the difference in iCNV activation to A-cues and B-cues) should be expected. A correlational analysis confirmed this relation in individuals with insomnia disorder, $R^2 = 0.35$, $\beta = -0.59$, $t(17) = -2.92$, $p = 0.010$. The worse ID indicated they performed on the AX-CPT task, the larger the difference in iCNV amplitudes between A-cues and B-cues. However, an absence of this relation was observed in the GSC group.

Table 4. Means (*SD*) of the median RTs (in ms) of the correct responses and mean error rates (in %) as a function of Trial Type (i.e. AX, AY, BX, BY) and Group (i.e. ID and GSC) in the AX-CPT.

Group	RTs (ms)			Error rates (%)		
	ID	GSC	ID vs GSC	ID	GSC	ID vs GSC
	Mean (± *SD*)	Mean (± *SD*)	Effect size (*r*)	Mean (± *SD*)	Mean (± *SD*)	Effect size (*r*)
AX	527 (123)	473 (158)	0.19	4.72 (6.77)	1.33 (1.76)	0.42
AY	645 (120)	607 (136)	0.15	9.17 (8.18)	12.40 (13.90)	0.15
BX	461 (176)	402 (143)	0.18	6.56 (8.38)	2.20 (2.81)	0.41
BY	461 (152)	399 (135)	0.22	2.44 (3.68)	1.67 (1.95)	0.13

ID (Individuals with Insomnia Disorder); GSC (Good Sleeper Controls); AX (AX-trials); AY (AY-trials); BX (BX-trials); BY (BY-trials)

*$p < .05$, **$p < .01$, ***$p < .001$

DISCUSSION

This study is the first to address the temporal dynamics of cognitive control in insomnia. Using a continuous performance task (AX-CPT), we examined how individuals with insomnia disorder recruit different cognitive control mechanisms and whether insomnia was specifically associated with a decreased proactive control. As hypothesized, our results indicate that in general, compared to good sleeper controls, individuals with insomnia disorder showed indeed a significantly reduced proactive control engagement along with an absence of clear behavioral performance impairments.

Consistent with previous findings [24, 25], our behavioral data indicate that individuals with insomnia disorder exhibit similar cognitive performance compared with good sleepers. Individuals with insomnia disorder however, show a decreased proactive control recruitment as reflected in the ERP cue and probe-related components compared to GSCs. First, individuals with insomnia disorder exhibit less positive P3b amplitudes to A-cues and B-cues, potentially reflecting a weaker engagement and maintenance of task-relevant cue-information [35, 58]. Second, the amplitude modulation of the iCNV in response to different cues (i.e. larger iCNV amplitudes for B-cues compared to A-cues) in good sleeper controls, is attenuated for individuals with insomnia disorder, suggesting that they do not proactively prepare an adequate response when possible (i.e. for B-cues). The decreased P3b combined with an absence of the modulation of iCNV amplitude suggest that the engagement and maintenance of task-relevant cue information is hampered in insomnia, which may subsequently interfere with the selection of the appropriate response. Third, the observed reduced proactive control engagement in the cue-locked data is further confirmed by smaller N2 amplitudes to AY-trials (i.e. smaller N2 amplitudes for the BY-AY contrast) in individuals with insomnia disorder. The reduced N2 amplitudes to AY-trials indicate that individuals with insomnia disorder experience less conflict in these trials, which can result from an impaired maintenance of the A-cue information (i.e. a reduced proactive control). Taken together, these observations reliably demonstrate that the recruitment of proactive control processes are significantly impaired in individuals with insomnia disorder.

Decreased P3b and CNV amplitudes have previously been related to impairments in proactive control [35], assumingly reflecting a weakened ability to engage attention to relevant task goals, which is often

the case during rumination [19, 20]. As by Braver [33], proactive control is a cognitive control strategy that is strongly resource consuming, as task-goals are continuously maintained active over a certain period. Given that individuals with insomnia disorder in our study report significantly higher levels of trait rumination compared to the good sleeper control group, these task-irrelevant thoughts might occur during task execution and might consequently substantially reduce available cognitive capacity for maintenance of task-relevant information. Subsequently, this might limit efficient goal maintenance, and therefore reduce proactive control. One way to confirm this hypothesis, is to examine whether the P3b component (reflecting the capacity to engage with and maintain task-relevant information) was associated with higher levels of rumination. We observed that $P3b_{B-A}$ amplitudes were indeed significantly related to higher levels of rumination in the ID group ($R^2 = 0.27$, $\beta = -0.52$, $t = -2.35$, $p = 0.026$). The more individuals with insomnia disorder reported to ruminate, the larger the difference in P3b amplitudes between A-cues and B-cues. A closer inspection reveals that this effect was driven by a relation between lower P3b amplitudes to B-cues and higher levels of rumination in insomnia ($R^2 = 0.25$, $\beta = -0.50$, $t = -2.29$, $p = 0.036$), suggesting that the more individuals with insomnia disorder reported to ruminate, the smaller the P3b amplitudes to B-cues were, reflecting an impaired engagement with the task-relevant cue-information. For the GSC group on the other hand, where low levels of rumination were reported, this relation between the $P3b_{B-A}$ amplitudes and rumination was absent ($R^2 = 0.011$, $\beta = 0.10$, $t = 0.38$, $p = 0.71$). These findings may partly support the hypothesis that rumination interferes with the effective engagement and maintenance of task-relevant information and that a high tendency to ruminate might account for the impaired cognitive engagement and maintenance of relevant information in insomnia. However, since in this study we only assessed trait rumination, we cannot make strong claims regarding state rumination processes affecting proactive control engagement during task execution. For that reason, further research is needed to examine whether state rumination is contributing to the relationship between trait rumination and proactive control that we observed in our study or whether impaired proactive control mechanisms account for increased levels of trait rumination.

Interestingly, decreased CNV amplitudes have also been found in combination with increased activity in different regions of the Default Mode Network [73], a set of brain areas thought to be active

at rest and when the subject is not engaging in task-relevant behaviors [74]. The activation in this network is hypothesized to be associated with off-task internal thoughts often present during distraction, mind wandering and rumination for example. Disengagement from the Default Mode Network is crucial in order for cognitive resources to become available, to allow focus on task-relevant information and to optimize goal-oriented behavior. A decreased ability to disengage will consequently result in distracting subjects from anticipating the upcoming stimulus, thus implying a deficient proactive control. Interestingly, a recent study showed that individuals with insomnia disorder demonstrate reduced deactivation of default mode regions with increasing task difficulty [25] compared to good sleepers. This impaired disengagement of default mode in insomnia was accompanied by a decreased ability to concentrate during the task. Our data seem to support this finding. That is, we found that decreased subjective performance during the task correlated with increased iCNV modulation and this was only observed in individuals with insomnia disorder. These results therefore corroborate the hypothesis that individuals with insomnia disorder experience more difficulty to focus on the task at hand and are therefore forced to exert more cognitive effort, which may in turn explain their subjective complaints with regard to cognitive functioning. The previous explanation assumes that subjective performance is primarily predicted by proactive control mechanisms. However, other alternative explanations (e.g. reactive control mechanisms driving subjective performance) can be envisaged as well. For that reason, future studies should include more sensitive measures such as a trial-by-trial investigation of subjective performance and cognitive effort, to reliably map these subjective processes related to proactive and reactive control mechanisms.

Although our data suggest that individuals with insomnia disorder only show decrements in the recruitment of proactive control, we cannot explain the overall pattern of our results solely based on the reliance of one single mechanism. Our data suggest that individuals with insomnia disorder show deficits in the recruitment of reactive control as well. We observe that individuals with insomnia disorder fail to differentiate between the A-cue and the B-cue information (see iCNV). According to the compensatory recruitment hypothesis [27], we can expect that individuals with insomnia disorder would mobilize extra effort (i.e. reactive control) in order to maintain a comparable level of performance. Specifically, when

the B-cue information is not adequately processed and used to prepare a non-target response, reactive control can eventually be engaged in order to compensate for the decreased proactive control and to maintain performance (i.e. make a non-target response to BX-trials). This should be reflected in increased P3a amplitudes to BX-trials (reflecting an increased response inhibition in order to suppress a target response to BX-trials). However, our results indicate that individuals with insomnia disorder did not differ with regards to the P3a amplitudes on BX-trials compared to good sleepers controls ($t = 0.43$, $p = .67$). This finding demonstrates that individuals with insomnia disorder do not compensate for their decreased proactive control. This deficient recruitment of reactive control mechanisms in turn affects BX-performance in individuals with insomnia disorder. More specifically, individuals with insomnia disorder showed a higher proportion of errors on BX-trials compared to BY-trials ($t = 2.18$, $p = 0.044$). In contrast, good sleepers did not show a performance decrement on BX-trials ($t = 1.05$, $p = 0.31$). Although, individuals with insomnia disorder made more errors on BX-trials (see larger BX-BY contrast) compared to good sleeper controls, this difference in BX-BY contrast was not statistically significant between good sleeper controls and individuals with insomnia disorder ($t = 1.83$, $p = 0.082$).

A possible explanation for the lack of clear behavioral differences between good sleeper controls and individuals with insomnia disorder might arise from the heterogeneity within our sample of individuals with insomnia disorder. Indeed, a recent study [29] has showed that performance differences exist between individuals with insomnia disorder with cognitive complaints and without cognitive complaints. More specifically, individuals with insomnia disorder who report more severe cognitive complaints exhibited a poorer neurobehavioral performance compared to individuals with insomnia disorder without cognitive complaints and good sleeper controls. Additionally, by not taking into account the severity of subjective cognitive impairment among individuals with insomnia disorder, we might have failed to uncover clear differences in cognitive performance between individuals with insomnia disorder and good sleeper controls. Possibly, the heterogeneity in cognitive complaints among individuals with insomnia disorder might also account for the fact that we did not find differences in memory contentment and ability between individuals with insomnia disorder and good sleeper controls. Indeed, when examining our ID sample more closely and focusing on the reported cognitive complaints,

we observe that almost half of the sample of individuals with insomnia disorder exhibited a similar cognitive profile as good sleeper controls. The absence of severe cognitive complaints in a subset of individuals with insomnia disorder might cancel out subjective and objective cognitive impairments in insomnia and might in turn also account for the contradictory previous findings in the literature [29].

Some limitations to our study must be taken into account. First of all, our individuals with insomnia disorder were, according to the ISI, varying in insomnia severity (i.e. ranging from subthreshold insomnia to severe clinical insomnia). This may be misleading as well and certain existing deficits characterizing certain subgroups of insomnia severity may consequently be overlooked. Future research may thus take into account different phenotypes in insomnia severity and intensity of cognitive complaints. Furthermore, future research should take into account the different phenotypes related to cognitive complaints in individuals with insomnia disorder, in order to achieve more reliable findings regarding cognitive impairment in insomnia, which may be underestimated in this current study. Note that, we cannot make any absolutely pure inferences regarding proactive control engagement, based on post-response behavioral data, since from the moment a response is required also reactive processes come into play. However, the EEG method we used in the current paper allows us to isolate brain activity related to proactive control from reactive control.

While previous approaches have examined the neurocognitive control impairments in insomnia focusing on a general deficit in cognitive control, the current study is the first study to investigate how individuals with insomnia disorder recruit different cognitive control mechanisms and when this recruitment becomes inefficient from a more dynamic, temporal perspective. In summary, our results showed a reduced engagement of proactive control mechanisms in insomnia compared to good sleepers. This inefficient proactive control recruitment might actually contribute to the common cognitive complaints in individuals with insomnia disorder. Specifically, it is possible that individuals with insomnia disorder might become aware of their impaired proactive control engagement and experience it as cognitively challenging, resulting in the report of decreased cognitive performance. These findings are especially relevant given that recent reports [25, 26] highlight the importance to consider the altered neurological profile of individuals with insomnia disorder instead of only focusing on behavioral

measures. In conclusion, our study provides reliable and consistent neurological evidence for impairments in cognitive control functioning in insomnia and contributes to an improved understanding of the discrepancy between the commonly reported cognitive impairments in insomnia and the scarce objective findings for these cognitive complaints.

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DISCLOSURE STATEMENT

REFERENCES

1. Ohayon MM. Epidemiology of insomnia: what we know and what we still need to learn. Sleep Med Rev. 2002; 6 (2): 97–111.

2. Morin CM, LeBlanc M, Daley M, Gregoire JP, Mérette C. Epidemiology of insomnia: prevalence, self-help treatments, consultations, and determinants of help-seeking behaviors. Sleep Med. 2006; 7 (2): 123–30.

3. Morin CM, Jarrin DC. Insomnia and healthcare-seeking behaviors: impact of case definitions, comorbidity, sociodemographic, and cultural factors. Sleep Med. 2013; 14 (9): 808–809.

4. American Academy of Sleep Medicine. International classification of Sleep Disorders, 3rd edn. American Academy of Sleep Medicine, Darien, IL, 2014.

5. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders. 5th ed. Arlington, VA: American Psychiatric Publishing, 2013.

6. Moul DE, Nofzinger EA, Pilkonis PA, Houck PR, Miewald JM, Buysse DJ. Symptom reports in severe chronic insomnia. Sleep. 2002; 25 (5): 553–63.

7. Stepanski E, Koshorek G, Zorick F, Glinn M, Roehrs T, Roth T. Characteristics of individuals who do or do not seek treatment for chronic insomnia. Psychosomatics. 1989; 30 (4): 421–7.

8. Roth T, Ancoli-Israel S. Daytime consequences and correlates of insomnia in the United States: results of the 1991 National Sleep Foundation Survey. II. Sleep. 1999; 1; 22(Suppl.2): S354e8.

9. Riedel BW, Lichstein KL. Insomnia and daytime functioning. Sleep Med Rev. 2000; 4 (3): 277–298.

10. Buysse DJ, Thompson W, Scott J, et al. Daytime symptoms in primary insomnia: a prospective analysis using ecological momentary assessment. Sleep Med. 2007; 8 (3): 198–208.

11. Harvey AG. A cognitive model of insomnia. Behav Res Ther. 2002; 40 (8): 869–893

12. Carney CE, Harris AL, Falco A, Edinger JD. The relation between insomnia symptoms, mood, and rumination about insomnia symptoms. J Clin Sleep Med. 2013; 9 (6): 567–575.

13. Ballesio A, Ottaviani C, Lombardo C. Poor Cognitive Inhibition Predicts Rumination About Insomnia in a Clinical Sample. Behav Sleep Med. 2018; 20: 1-20.

14. Morin CM. Insomnia: Psychological Assessment and Management. New York, USA: Guilford Publications, 1996.

15. Harvey AG, Tang NK, Browning L. Cognitive approaches to insomnia. Clin Psychol Rev. 2005; 25 (5): 593–611.

16. Carney CE, Edinger JD, Meyer B, Lindman L, Istre T. Symptom-focused rumination and sleep disturbance. Behav Sleep Med. 2006; 4 (4): 228–41.

17. Carney CE, Harris AL, Moss TG, Edinger JD. Distinguishing rumination from worry in clinical insomnia. Behav Res Ther. 2010; 48 (6): 540–6

18. Carney CE, Harris AL, Falco A, Edinger JD. The relation between insomnia symptoms, mood, and rumination about insomnia symptoms. J Clin Sleep Med. 2013; 9 (6): 567–75.

19. Phillippot P, Brutoux F. Induced rumination dampens executive processes in dysphoric young adults. J Behav Ther Exp Psychiatry, 2008; 39 (9): 219–27.

20. Krompinger JW, Simons RF. Cognitive inefficiency in depressive undergraduates: stroop processing and ERPs. Biol Psychol, 2011; 86 (3): 239–46.

21. Owens M, Derakshan N, Richards A. Trait susceptibility to worry modulates the effects of cognitive load on cognitive control: an ERP study. Emotion, 2015; 15 (5): 544–549.

22. Norman DA, Shallice T. Attention to action: willed and automatic control of behaviour. In: Davidson RJ, Schwartz GE, Shapiro D, ed. Consciousness and Self-Regulation: Advances in Research and Theory. New York, USA: Plenum Press, 1986.

23. Miller EK, Cohen JD. An integrative theory of prefrontal cortex function. Annu Rev Neurosci. 2001; 24: 167–202.

24. Altena E, Van Der Werf YD, Sanz-Arigita EJ, et al. Prefrontal hypoactivation and recovery in insomnia. Sleep. 2008; 31 (9): 1271–6.

25. Drummond SP, Walker M, Almklov E, Campos M, Anderson DE, Straus LD. Neural correlates of working memory performance in primary insomnia. Sleep. 2013; 36 (9): 1307–16.

26. Son YD, Kang JM, Cho SJ, Lee JS, Hwang HY, Kang SG. fMRI brain activation in patients with insomnia disorder during a working memory task. Sleep Breath. 2018; 22 (2): 487–493.

27. Orff HJ, Drummond SP, Nowakowski S, Perils ML. Discrepancy between subjective symptomatology and objective neuropsychological performance in insomnia. Sleep. 2007; 30 (9): 1205–11.

28. Fortier-Brochu E, Beaulieu-Bonneau S, Ivers H, Morin CM. Insomnia and daytime cognitive performance: a meta-analysis. Sleep Med Rev. 2012; 16 (1): 83–94.

29. Fortier-Brochu E, Morin CM. Cognitive impairment in individuals with insomnia: clinical significance and correlates. Sleep. 2014; 37 (11): 1787–98.

30. Shekleton JA, Rogers NL, Rajaratnam SM. Searching for the daytime impairments of primary insomnia. Sleep Med Rev. 2010; 14 (1): 47–60.

31. Perrier J, Chavoix C, Bocca ML. Functioning of the three attentional networks and vigilance in primary insomnia. Sleep Med. 2015; 16 (12): 1569–75.

32. Li Y, Liu L, Wang E, et al. Abnormal neural network of primary insomnia: evidence from spatial working memory task fMRI. Eur Neurol. 2016; 75 (1-2); 48–57.

33. Braver TS. The variable nature of cognitive control: A dual-mechanisms framework. Trends Cogn Sci. 2012; 16 (2): 106–13.

34. Morales J, Yudes C, Gómez-Ariza CJ, Bajo MT. Bilingualism modulates dual mechanisms of cognitive control: Evidence from ERPs. Neuropsychologia. 2015; 66: 157–69

35. van Wouwe NC, Band GP, Ridderinkhof KR. Positive affect modulates flexibility and evaluative control. J Cognitive Neurosci. 2010; 23 (3): 524–39.

36. Bastien CH, , St-Jean G, Morin CM, Turcotte I, Carrier J. Chronic Psychophysiological Insomnia: Hyperarousal and/or Inhibition Deficits? An ERPs Investigation. Sleep. 2008; 31 (6): 887–898.

37. Bastien CH, Turcotte I, St-Jean G, Morin CM, Carrier J. Information Processing Varies Between Insomnia Types: Measures of N1 and P2 During the Night. Behav Sleep Med. 2013; 11 (1): 56 –72.

38. Servan-Schreiber D, Cohen JD, Steingard S. Schizophrenic deficits in the processing of context: A test of a theoretical model. Arch Gen Psych. 1996; 53 (12): 1105–12.

39. Sheehan DV, Lecrubier Y, Sheehan KH, et al. The Mini-International Neuropsychiatric Interview (MINI): the development and validation of a structured diagnostic psychiatric interview for DSM-IV and ICD-10. J Clin Psychiatry; 1998, 59, 22–33.

40. Buysse DJ, Reynolds CF, Monk TH, Berman SR, Kupfer DJ. The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research. Psychiatry Res. 1989; 28 (2): 193–213.

41. Bastien CH, Vallières A, Morin CM. Validation of the Insomnia Severity Index as an outcome measure for insomnia research. 2001; 2 (4): 297–307.

42. Treynor W, Gonzalez R, Nolen-Hoeksema S. Rumination reconsidered: a psychometric analysis. Cognit Ther and Res. 2003; 27 (3), 247–259.

43. Beck AT, Steer RA, Brown GK. Manual for the Beck Depression Inventory-II. San Antonio, TX: Psychological Corporation; 1967.

44. Spielberger CD. Manual for the State-Trait Anxiety Inventory. Palo Alto, CA: Consulting Psychologists Press, 1983.

45. Smets EM, Garssen B, Bonke B, De Haes JC. The Multidimensional Fatigue Inventory (MFI) psychometric qualities of an instrument to assess fatigue. J Psychosom Res. 1995; 39 (3): 315–25.

46. Johns MW. A new method for measuring daytime sleepiness: the Epworth sleepiness scale. Sleep. 1991; 14 (6): 540–5

47. Morin CM, Vallières A, Ivers H. Dysfunctional beliefs and attitudes about sleep (DBAS): validation of a brief version. Sleep. 2007; 30 (11): 1547–54.

48. Broadbent DE, Cooper PF, Fitzgerald P, Parkes KR. The Cognitive Failures Questionnaire (CFQ) and its correlates. Br J Clin Psychol. 1982; 21: 1–16.

49. Troyer AK, Rich JB. Psychometric properties of a new metamemory questionnaire for older adults. J Gerontol B Psychol Sci Soc Sci. 2002; 57 (1): 19–27.

50. Chesson AJr, Hartse K, Anderson WM, et al. Practice parameters for the evaluation of chronic insomnia. An American Academy of Sleep Medicine report. Standards of Practice Committee of the American Academy of Sleep Medicine. Sleep. 2000; 23 (2): 237–41.

51. Hart SG, Staveland LE. Development of NASA-TLX (Task Load Index): results of empirical and theoretical research. Advances in Psychology. 1988; 52: 139–183.

52. Hauri PJ, Olmstead EM. Reverse first night effect in insomnia. Sleep. 1989; 12 (2): 97–105.

53. Agnew HW, Webb WB, Williams RL. The first night effect: an EEG study of sleep. Psychophysiology, 1966; 2 (3): 263–6.

54. Steer RA, Ball R, Ranieri WF, Beck AT. Dimensions of the Beck Depression Inventory-II in clinically depressed outpatients. J Clin Psychol. 1999; 55 (1): 117–28.

55. Delorme A, Makeig S. EEGLAB: an open source toolbox for analysis of single-trial EEG dynamics including independent component analysis. J Neurosci Methods. 2004; 134 (1): 9–21.

56. Clayson PE, Larson MJ. Conflict adaptation and sequential trial effects: support for the conflict monitoring theory. Neuropsychologia. 2011; 49 (7): 1953–1961.

57. Desender K, Van Opstal F, Hughes G, Van den Bussche E. The temporal dynamics of metacognition: Dissociating task-related activity from later metacognitive processes. Neuropsychologia. 2016; 82: 54–64.

58. Polich J, Comerchero MD. P3a from visual stimuli: typicality, task, and topography. Brain Topogr. 2003; 15 (3): 141–52.

59. Polich J. Updating P300: an integrative theory of P3a and P3b. Clin Neurophysiol. 2007; 118 (10): 2128–48.

60. Donchin E, Coles MGH. Is the P300 component a manifestation of context updating? Behav Brain Sci. 1988; 11 (3) :357–74.

61. Knight RT. Decreased response to novel stimuli after prefrontal lesions in man. Electroencephalogr Clin Neurophysiol. 1984; 59 (1): 9–20.

62. Walter WG, Cooper R, Aldridge VJ, Mccallum WC, Winter AL. Contingent Negative Variation: an electric sign of sensorimotor association and expectancy in the human brain. Nature. 1964; 203: 380–384.

63. Verleger R, Wauschkuhn B, van der Lubbe R, Jaskowski P, Trillenberg P. Posterior and anterior contribution of hand-movement preparation to late CNV. J Psychophysiol. 2000; 14 (2): 69–86.

64. Frömer R, Stürmer B, Sommer W. (Don't) Mind the effort: Effects of contextual interference on ERP indicators of motor preparation. Psychophysiology. 2016; 53: 1577–1586.

65. Prescott J. The effects of response parameters on CNV amplitude. Biological Psychology. 1986; 22 (2); 107–135.

66. Van Veen V, Carter CS. The timing of action-monitoring processes in the anterior cingulate cortex. J. Cognit. Neurosci. 2002; 14 (4): 593–602.

67. Coles MGH, Gratton G, Bashore TR, Eriksen CW, Donchin E. A psychophysiological investigation of the continuous flow model of human information processing. J Exp Psychol Hum Percept Perform. 1985; 11 (5): 529–561.

68. Purmann S, Badde S, Luna-Rodriguez A, Wendt M. Adaptation to frequent conflict in the eriksen flanker task. J. Psychophysiol. 2011; 25 (2): 50–59.

69. Schupp HT, Lutzenberger W, Rau H, Birbaumer N. Positive shifts of event related potentials: a state of cortical disfacilitation as reflected by the startle reflex probe. Electroencephalogr. Clin. Neurophysiol. 1994; 90 (2): 135–144.

70. Smith JL, Johnstone SJ, Barry RJ. Movement-related potentials in the Go/NoGo task: the P3 reflects both cognitive and motor inhibition. Clin. Neurophysiol. 2008; 119 (3), 704–714.

71. Donkers FCL, Nieuwenhuis S, van Boxtel GJ. Mediofrontal negativities in the absence of responding. Cognit. Brain Res. 2005; 25 (3): 777–787.

72. Scheibe C, Schubert R, Sommer W, Heekeren HR. Electrophysiological evidence for the effect of prior probability on response preparation. Psychophysiology. 2009; 46: 758–770.

73. Vanderhasselt MA, De Raedt R, De Paepe A et al. Abnormal proactive and reactive cognitive control during conflict processing in major depression. J Abnorm Psychol. 2014; 123 (1): 68–80.

74. Raichle ME, MacLeod AM, Snyder AZ, Powers WJ, Gusnard DA, Shulman GL. A default mode of brain function. PNAS. 2001; 98 (2): 676–682.

FOOTNOTES

[a] We investigated whether the specific differences observed between groups regarding proactive control engagement were not simply due to the fact that individuals with insomnia disorder were less engaged in the task compared to good sleeper controls. To examine this, we investigated early visual ERP components, i.e. the P1. We did not observe a difference in P1 amplitudes between groups, which strengthens our claim that both groups were equally engaged in the task. A repeated measures ANOVA with the factors Cue (2 levels: A and B) and Group (2 levels: ID and GSC) on the mean average voltage of correct trials during the first P1 time-window (80 ms - 120 ms), across the occipital electrodes (O1, Oz, O2) did not show a main effect of Cue ($F(1, 31) = 1.074$, $p = 0.31$, $\eta_p^2 = 0.033$). There was no main effect of Group ($F(1, 31) = 0.29$, $p = 0.60$, $\eta_p^2 = 0.009$). No interaction between Cue and Group was found ($F(1, 31) = 1.10$, $p = 0.30$, $\eta_p^2 = 0.034$).