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Relationships between affect, vigilance, and sleepiness following sleep deprivation

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SUMMARY

This pilot study examined the relationships between the effects of sleep deprivation on subjective and objective measures of sleepiness and affect, and psychomotor vigilance performance. Following an adaptation night in the laboratory, healthy young adults were randomly assigned to either a night of total sleep deprivation (SD group; $n = 15$) or to a night of normal sleep (non-SD group; $n = 14$) under controlled laboratory conditions. The following day, subjective reports of mood and sleepiness, objective sleepiness (Multiple Sleep Latency Test and spontaneous oscillations in pupil diameter, PUI), affective reactivity/regulation (pupil dilation responses to emotional pictures), and psychomotor vigilance performance (PVT) were measured. Sleep deprivation had a significant impact on all three domains (affect, sleepiness, and vigilance), with significant group differences for eight of the nine outcome measures. Exploratory factor analyses performed across the entire sample and within the SD group alone revealed that the outcomes clustered on three orthogonal dimensions reflecting the method of measurement: physiological measures of sleepiness and affective reactivity/regulation, subjective measures of sleepiness and mood, and vigilance performance. Sleepiness and affective responses to sleep deprivation were associated (although separately for objective and subjective measures). PVT performance was also independent of the sleepiness and affect outcomes. These findings suggest that objective and subjective measures represent distinct entities that should not be assumed to be equivalent. By including affective outcomes in experimental sleep deprivation research, the impact of sleep loss on affective function and their relationship to other neurobehavioral domains can be assessed.

Keywords

affect; emotion; neurobehavioral outcomes; sleep deprivation; sleepiness; vigilance

INTRODUCTION

Sleep loss is consistently associated with impairments in a range of neurobehavioral functions, including cognition, affect, and sleepiness. Such neurobehavioral impairments have been documented to be stable (Leproult *et al.*, 2003; Van Dongen *et al.*, 2004) but unrelated (Frey *et al.*, 2004; Van Dongen *et al.*, 2003, 2004), suggesting that sleep deprivation differentially affects brain functions controlling different dimensions of neurobehavioral impairment. The most well documented of these sleep deprivation effects are those on neurobehavioral deficits in vigilance and executive function. A potential mechanism for these deficits involves the effects of sleep deprivation on regions of the prefrontal cortex (PFC). Sleep loss strongly affects prefrontal regions (Chee and Choo, 2004; Thomas *et al.*, 2000, 2003), and leads to impairments observed in PFC-associated

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executive functions (e.g., Dinges *et al.*, 1997; Drummond *et al.*, 1999; e.g., Harrison *et al.*, 2000; Nilsson *et al.*, 2005).

Adverse effects of sleep deprivation on affect (used here as a superordinate term referring to discrete emotions, mood states, and other affective reactivity and regulation processes) have been less thoroughly explored in the experimental literature. Prefrontal regions are also critically involved in regulating mood, particularly by inhibiting brain structures such as the amygdala that are important to the generation and recognition of affect (Davidson *et al.*, 2000; Hariri *et al.*, 2000; Ochsner and Gross, 2005; Phillips *et al.*, 2003; Urry *et al.*, 2006). Thus, sleep deprivation-related cognitive and affective impairments may share similar pathophysiologic bases and could plausibly be related.

Sleep loss has been linked to emotional and mood dysregulation (e.g., Dinges *et al.*, 1997; Haack and Mullington, 2005), which may lead to serious consequences in real-world settings (Baldwin and Daugherty, 2004; Lingenfelser *et al.*, 1994). For example, sleeping less than 8 h is associated with increased risk for adolescent suicidal behavior (Liu, 2004). Experimental studies of chronic sleep restriction and acute sleep deprivation in healthy individuals have documented larger mood effects than either cognitive or motor responses (Pilcher and Huffcutt, 1996). These studies, however, have almost exclusively used self-report outcomes. The reliability of such self-reported data is uncertain due to contextual factors (e.g., intentional or unintentional report bias, scale interpretation, and demand characteristics and self-presentational concerns). Quantifying physiological responses to emotional information is one method to objectively measure affective responses to sleep deprivation. To the extent that sleep deprivation-related effects on mood are similar to other mood disruptions such as dysphoria or anxiety (Challis and Krane, 1988; Deldin *et al.*, 2001; Siegle *et al.*, 2002), sleep deprivation may be associated with disruptions in the dynamic time-course of physiological responses to emotional information, which occurs on the time scale of milliseconds or seconds. Such physiological measures might therefore complement subjective assessments of mood by providing evidence for other aspects of affect that are altered during sleep deprivation. It is unclear to what extent such objective affective measures are associated with other objective measures of sleep deprivation-related neurobehavioral impairment (e.g., sleepiness and performance).

In this pilot study, we examined neurobehavioral outcomes of sleepiness, affect, and vigilance performance in healthy young adults randomly assigned to a night of total sleep deprivation (SD) or to a normal sleep (non-SD) control group. Sleepiness was measured by self-report and objectively with the Multiple Sleep Latency Test (MSLT) and the Pupil Sleepiness Test (Wilhelm *et al.*, 1998). In the latter task, sleepiness-associated spontaneous oscillations of pupil size (e.g., Lowenstein *et al.*, 1963; Newman and Broughton, 1991; Yoss *et al.*, 1970) were quantified. Affect was likewise measured by self-report (i.e., positive and negative mood) and objectively by quantifying physiological responses to emotional stimuli. Vigilance was measured during the Psychomotor Vigilance Test (PVT), a sustained attention task known to be quite sensitive to sleep loss (Doran *et al.*, 2001; Graw *et al.*, 2004).

We addressed the following aims: (1) To examine the impact of sleep deprivation on three domains of neurobehavioral function (affect, vigilance, and sleepiness); (2) to examine relationships among the neurobehavioral domains; and (3) to examine how sleep deprivation changes the relationships among the neurobehavioral domains. Effect size estimates formed the basis for comparing the relative sensitivity of the neurobehavioral outcomes to sleep deprivation, and exploratory factor analyses were used to examine possible relationships/independencies among the neurobehavioral outcomes.

METHODS

Following an adaptation night of sleep in the laboratory, healthy young adult participants were randomly assigned to either one night of total SD or to a non-SD control condition. Neurobehavioral testing occurred the following day. The specific tasks and neurobehavioral outcomes are summarized in Table 1 and described below.

Subjects

Potential participants were recruited with poster advertisements. Participants included 29 healthy adult volunteers ages 21–30: 15 females/14 males (8/7 in SD group, respectively), ages 21–30, mean (standard deviation) = 24.4 (2.76). One out of the 14 control participants had incomplete data and was excluded from all analyses; in addition, one participant's data on the Pupillary Sleepiness Test was missing due to technical error, and thus this participant was excluded in a pairwise fashion from analyses involving this measure. After providing informed consent and signing a University of Pittsburgh Institutional Review Board-approved consent form, individuals were first screened with an in-person structured psychiatric and sleep disorders interview. Potential participants were excluded if they had any of the following: current or past psychiatric or sleep disorders; presence of significant sleep disordered breathing or leg movements (10 or more events per hour of sleep) detected during the adaptation night in the laboratory; significant hearing or vision problems; health problems thought to interfere with performance or medications other than contraception; psychoactive drug abuse within the past 6 months; maintained very early or late bedtimes (earlier than 22:00 h. or later than 02:00 h), or an irregular sleep/wake schedule (i.e., 2 or more hours of variability in sleep/wake times based on participants' 1 week sleep diary data; Monk *et al.*, 1994); used tobacco; or used more than 400 mg of caffeine daily. Alcohol and caffeine were avoided 1 day to prior to and throughout the experimental protocol.

Experimental protocol

At 20:00 h, participants arrived to the Clinical Neuroscience Research Center (CNRC), a satellite laboratory of the University of Pittsburgh General Clinical Research Center, for an adaptation night. Polysomnographic (PSG) electrodes and sensors were applied, as well as inductance plethesmography, nasal pressure, oximetry, and electrodes on the tibial muscle to screen for sleep apnea (defined as an apnea–hypopnea index of ≥10) and periodic limb movement (PLM) disorder (defined as a PLM index of ≥10). Good night time was between 23:00 and 24:00 hours, based on participants' average bedtime for the past 7 days as recorded on a daily sleep diary. Following sleep onset, participants had 8 h of time in bed. The following evening, participants returned to the CNRC and PSG electrodes and sensors were applied. Participants in the non-SD group slept according to their habitual schedule. Participants in the SD group spent the night in the CNRC lounge under continual PSG and frequent behavioral monitoring by CNRC sleep technicians. During the SD night, participants had access to food and non-caffeinated beverages, TV and VCR, and internet access. Participants were able to freely eat and move about the CNRC lounge and laboratory hallways.

Daytime testing commenced at 10:00 with a Multiple Sleep Latency Test (MSLT) consisting of five naps at 2-h intervals. Naps lasted up to 20 min and were terminated upon three consecutive epochs of stage 1 or one epoch of stage 2 non-rapid eye movement sleep. Prior to each nap, participants completed two self-report questionnaires measuring mood and sleepiness, followed by a 10-min Psychomotor Vigilance Task (PVT) to assess sustained attention. A task involving presentation of emotional pictures and the Pupil Sleepiness Test (Wilhelm *et al.*, 1998) were also administered during an afternoon test session between MSLT naps 3–4 or 4–5.

Subjective measures of sleepiness and mood

Subjective sleepiness and mood were assessed with 14 visual analog scale (VAS) items, in which responses are indicated along 100-mm lines. Eight of these items included the Global Vigor and Affect (GVA) visual analog scales validated by Monk (1989), which are summarized by a Vigor (sleepiness/alertness) scale and an Affect scale (with opposite weightings for positive and negative mood items). The GVA has been shown to be sensitive to sleep loss (Brendel *et al.*, 1990) and circadian variation (Monk *et al.*, 1997). We supplemented the GVA items with additional visual analog mood and sleepiness items described by our group as being sensitive to sleep disruption (Buysse *et al.*, 2007). Principal components analysis of the final 14 VAS items supported two orthogonal factors (mood and sleepiness; all factor loadings were >0.72). The responses to VAS items sleepy, fatigued, weary, exhausted, effort, and alert (reverse scored) were therefore averaged together to index subjective sleepiness on a range of 0–100. VAS items sad, anxious, tense, stressed, irritable, and happy, calm, relaxed (reverse scored) were averaged together to index negative mood (0–100 range). Subjective mood was also measured with the Positive and Negative Affect Schedule (PANAS; Watson *et al.*, 1988). The PANAS is a Likert-style questionnaire that consists of two 10-item mood scales consisting of words (e.g., interested, excited, distressed, upset). Participants are asked to rate how each of the words describe them right now on a 5-item scale from ‘very slightly or not at all’ to ‘extremely’. Positive affect and negative affect mood scores range between 10 and 50; both scales are internally consistent (coefficient alphas ≥0.85; Watson *et al.*, 1988) and largely orthogonal.

Objective, physiological measures of sleepiness

Objective sleepiness outcomes included mean sleep latency on the MSLT and the Pupillary Unrest Index (PUI) during the Pupil Sleepiness Test (Wilhelm *et al.*, 1998). The Pupil Sleepiness Test quantifies slow pupillary oscillations that are characteristic of sleepy but not alert individuals, due to unstable fluctuations in central sympathetic activity under conditions of sleepiness (Wilhelm *et al.*, 2001). Participants sat for 11 min in a completely dark and quiet room, with their head affixed by a chin and forehead rest, staring at a dim red dot centered on an otherwise black computer screen. To prevent participants from falling asleep, they were alerted by sending a soft ‘click’ over an intercom if their eyes were observed to close for 3 s (using a protocol developed by the measures’ authors; Wilhelm, personal communication); stronger interventions to maintain wakefulness were not necessary. Pupil diameter was continuously recorded at 60 Hz and later down-sampled to 25 Hz. Data were cleaned following methodology derived from Granholm *et al.* (1996) which included identifying blinks as large changes in pupil dilation occurring too rapidly to signify actual dilation or contraction. Linear interpolations replaced blinks throughout the dataset. Data were smoothed by applying a 3-point flat filter twice. Sleepiness-related pupillary instability was quantified with the PUI – the integrated sum of slow pupillary oscillations over time (Ludtke *et al.*, 1998). Higher numbers on the PUI indicate greater sleepiness.

Affective reactivity/regulation

Physiological responses to emotional stimuli were objectively measured during a picture viewing task in which positive, negative, and neutral International Affective Picture System (IAPS; Lang *et al.*, 2005) stimuli were presented (15 of each valence for a total of 45 trials). Each trial consisted of a 2-s cue and a 6-s stimulus presentation period, followed by a 6–8 s interstimulus interval. Pupil diameter was continuously recorded as an objective index of brain activity in response to the pictures. The pupil dilates in response to emotional information (Bitsios *et al.*, 2004; Janisse, 1973; Siegle *et al.*, 2001, 2003; Urry *et al.*, 2006) as well as cognitive load (e.g., Beatty, 1982), and is innervated by multiple brain structures involved in emotional reactivity, emotion regulation, and cognitive information processing. Thus, pupil diameter can be considered a summative index of brain activity associated with

emotional information processing. For the present analysis, we calculated the average millimeter change in pupil diameter during the stimulus presentation period for negative pictures to index brain responses to the emotional stimuli including initial affective reactivity and regulation; higher numbers indicate higher levels of brain activity in response to negative emotional information. Though there were group differences in pupillary responses for all three valences, pupillary responses were significantly elevated to negative compared with neutral or positive pictures in the SD group. As such, we examined pupillary responses to negative pictures as probably the most reliable and ecologically valid (e.g., Pilcher and Huffcutt, 1996) index of sleep deprivation-related phenomena on this task, as well as to simplify the analyses given the small sample size. Nuances of the relationships between pupillary reactivity to positive, negative, and neutral pictures will be more fully explored in a future report on just the pupil data.

Objective measure of performance/alertness

Sustained attention was assessed with a PVT task presented on a computer with the Eprime software suite (Psychology Software Tools; Pittsburgh, PA). Targets were presented randomly every 3–10 s for 10 min, and reaction time (RT) feedback on each trial was provided. Participants were instructed to respond as quickly as possible to targets. PVT outcomes include lapses (RTs >500 ms) and speed (harmonic mean RT in milliseconds).

Data analysis

Data were analyzed with SPSS version 13.0 (SPSS, Inc., Chicago, IL, USA) software. Outcome variables that were measured multiple times were first examined with repeated measures analysis of variance (ANOVA). Main effects of time of day were not significant, and therefore mean scores were calculated. Distributions and outliers were examined graphically. Positively skewed variables were natural log transformed. Data from an individual outlier in the control group on the PUI were rescaled to the median value plus two times the interquartile range. Data are summarized as mean ± standard deviation, and are reported in Table 1 in the original units. The overall difference across the nine neurobehavioral outcome variables between the SD and non-SD control groups was examined with a multivariate analysis of variance (MANOVA), which protects against Type 1 errors that might occur if comparisons for each outcome were conducted separately. The significant multivariate analysis was followed up with univariate analyses. We considered differences significant at $P < 0.05$ for this exploratory study. To examine associations among the neurobehavioral outcomes, we then conducted an exploratory principal components analysis (PCA) using Varimax rotation with Kaiser normalization to obtain orthogonal factors. Factors were extracted using two criteria: (1) examination of scree plot and eigenvalues >1 and (2) accounting for a total of >70% of the variance for all of the factors. Items with factor loadings >0.5 were considered to load on a given factor. To examine extent to which factor structures were similar across groups, we then conducted separated PCA's per group.

RESULTS

First specific aim: To examine the impact of sleep deprivation on the three domains of neurobehavioral function (affect, vigilance, and sleepiness)

The MANOVA revealed a significant effect of group across neurobehavioral outcomes, $F(9,17) = 6.37$, $P = 0.001$, accounting for 77% of the overall variance (partial eta-squared effect size; $\eta^2 = 0.771$). Follow-up univariate analyses, presented in Table 1, revealed significant differences for all outcomes except for PANAS-negative mood. The effect sizes ranged medium to very large. Subjective sleepiness and mean sleep latency on the MSLT had largest effect sizes ($\eta^2 = 0.631$ and 0.501, respectively); the objective measures affective

reactivity/regulation and PUI had similar effect sizes (η^2 = 0.265 and 0.317, respectively); and subjective mood (VAS-negative mood and PANAS-positive affect) and PVT outcomes had similar effect sizes, ranging from η^2 = 0.161 to 0.224. The effect size for PANAS-negative mood was medium (η^2 = 0.065), although the SD and non-SD groups did not differ significantly on this variable. Of note, however, this variable also had a floor effect with one-third of SD group participants and one-half of non-SD group participants responding with the minimum value for each negative mood item. PANAS-negative mood was therefore excluded from the subsequent principal components analysis (PCA), so that relationships were examined only among the variables that were affected by the SD manipulation.

Second specific aim: To examine relationships among the neurobehavioral domains

PCA performed on the entire sample (SD and non-SD groups) revealed three factors explaining 77% of the variance (see Table 2). Objective/physiological outcomes of sleepiness and affective reactivity/regulation loaded on the first factor, self-reported mood and sleepiness on the second, and lapses and speed on the PVT on the third. Factor scores were calculated and group differences on factor scores were examined with independent samples *t*-tests. The groups differed significantly on the first and second factors (i.e., objective/physiological outcomes and subjective outcomes, respectively). The groups did not differ on the third factor (PVT outcomes), although the Cohen's *d* effect size was medium.

Third specific aim: To examine how sleep deprivation changes the relationships among the neurobehavioral domains

PCA was conducted in each group separately. The factor structure in the SD group (Table 3) was very similar to the structure in the entire sample (Table 2). A distinction between the objective and subjective outcomes was less clear in the non-SD control group (Table 4), in which subjective outcomes of mood and sleepiness loaded with decreased pupillary affective reactivity/regulation on the first factor, MSLT and PVT outcomes on the second, and PUI on the third.

DISCUSSION

This study examined the impact of one night of total sleep deprivation on affect, vigilance, and sleepiness-related outcomes using both objective and subjective measures. Sleep deprivation had strong effects on all the domains assessed in this study, resulting in greater physiological and subjective sleepiness, a reduction in positive mood and increase in negative mood self-reports, greater levels of physiological reactivity/regulation in response to emotional stimuli, and slower over all reaction times and greater lapses on the PVT. The variables with the largest magnitude effects were mean sleep latency and subjective sleepiness. Objective, physiological outcomes measuring sleepiness (PUI) and affective reactivity/regulation had similar magnitude effects, as did subjective mood and PVT outcomes. Thus, the impact of sleep deprivation on the affective outcomes (subjective mood and a physiological measure of affective reactivity/regulation) was as large as or larger than the PVT, a gold-standard measure of sleep deprivation-related performance impairment.

Exploratory factor analyses performed across the entire sample and within the SD group alone revealed that the outcomes clustered on three orthogonal dimensions reflecting the method of measurement: physiological measures of sleepiness and affective reactivity/regulation, subjective measures of sleepiness and mood, and performance measures of sustained attention. Given the exploratory nature of these analyses, in particular the sub-group analyses, the present findings must be considered preliminary and will need to be replicated. These preliminary results, however, suggest that although sleep deprivation has a

strong effect across multiple neurobehavioral domains, sleep deprivation-related impairments were more homogenous across the type of measurement (objective/physiological versus subjective versus performance) than across specific neurobehavioral domains (affect, sleepiness, and vigilance).

Vigilance performance and objective measures of sleepiness

Lapses and speed on the PVT were orthogonal to the other outcomes examined in this study. Two other sleep deprivation studies have reported similar findings that the PVT loaded independently of other performance and sleepiness outcomes. Frey *et al.* (2004) reported that objective sleepiness (Maintenance of Wakefulness Test), subjective sleepiness, and outcomes on the PVT each loaded independently on three of five factors. A similar factor structure to the present study was reported by Van Dongen *et al.* (2004), finding three distinct dimensions of inter-individual vulnerability to sleep deprivation that were stable across repeated sleep deprivation probes: subjective sleepiness, fatigue, and mood; cognitive processing capability; and lapses on the PVT. In a study of non-deprived individuals (Kraemer *et al.*, 2000), pupillographic measures of sleepiness (e.g., PUI) and MSLT outcomes factored together and independent of other performance and subjective sleepiness measures, suggesting that the MSLT and PUI outcomes reflect a similar physiological sleepiness trait that is independent of what is measured by the PVT. These findings highlight the multi-dimensional nature of sleepiness, and have implications for assessing sleepiness (e.g., clinically, or in the work place). For example, a sleep-restricted individual who does not feel sleepy while working or driving may nonetheless have increased attentional lapsing or physiological sleepiness and therefore increased risk of having an accident.

Relationships of sleepiness to subjective and objective measures of affect

The present study extends previous research findings by including an objective measure of affective information processing. Pupillary responses to affective stimuli were associated with the two physiological sleepiness outcomes, suggesting that these three measures may share a similar neurobiological substrate. Subjective mood (increased negative mood and decreased positive mood) was also associated with subjective sleepiness, suggesting again that these two domains share a similar pathway, although sleep deprivation affected the objective and subjective pathways differentially. On a subjective level, sleepiness may be intertwined with mood (i.e., they may be related to the same latent construct). Being sleep restricted or sleep deprived is typically an unpleasant experience. It may therefore be difficult to differentiate perceptions of sleepiness from those of mood. Constructs such as sleepiness and mood have fluctuating time courses, further complicating their subjective assessment.

Relationships between subjective and objective assessments may be affected by measurement sensitivity. Physiological measures can be more precisely measured, and may therefore be more sensitive than self-report measures. This would be consistent with the effect sizes which, with the exception of subjective sleepiness, were larger for the physiologic measures (sleepiness and affective reactivity/regulation) than subjective mood and PVT outcomes.

Finally, our objective measure of affective information processing may represent multiple related constructs leading to a more complicated picture. Pupil dilation in response to emotional information was negatively associated with a factor including subjective sad mood and sleepiness, but only in the non-SD group. This could reflect the pupil's dual reflection of brain processes underlying emotional reactivity and regulation. In non-SD individuals who have adequate prefrontal resources to devote to emotion regulation, increased sad mood and feelings of sleepiness may reflect protective pre-stimulus

engagement of regulatory mechanisms, and thus, systematically less stimulus-related reactivity/regulation, yielding the observed negative relationship. Decreased prefrontal control in the SD group could thus have ameliorated this association. If the SD group reacted more strongly to emotional pictures because of their sleep deprivation, objective measures of sleep deprivation would be expected to cluster with the objective measure of emotional reactivity afforded by pupil dilation. Alternatively, that the factor loadings were different for the SD and non-SD groups may be related to small sample sizes, which are problematic for determining stable factor solutions.

Limitations

Although the study design had a number of strengths, including multiple measures of both objective and subjective outcomes measured under rigorous experimental control conditions, there are also some limitations. Foremost is the small sample size – future replications with larger samples would help to examine the robustness of the current results and would allow examination of individual differences in response to sleep deprivation (Van Dongen *et al.*, 2004). Similarly, individual differences in factors such as motivation, trait-predisposition to nap quickly ('somnotypes'; Lavie, 1991) as well as demand characteristics and self-presentational concerns (i.e., social desirability to minimize deficits or unwillingness to report negative affect) could also have confounded both self-report and objective measures. Individual differences in subjective awareness of emotions may also be an important confound. Logistical limitations precluded us from conducting the pupillary assessments of objective sleepiness and affective reactivity/regulation with the same frequency as the other outcomes that were repeatedly assessed. Perhaps the afternoon testing time confounded their association with the MSLT, although this confound is less likely for PUI as previous studies have reported similar time-of-day variability in the MSLT and PUI in non-deprived individuals (Danker-Hopfe *et al.*, 2001).

In conclusion, one night of sleep deprivation had strong effects on psychomotor vigilance performance, sleepiness, and on affect. The association of sleepiness and affective responses to sleep deprivation, although separately for objective and subjective measures, should be explored in greater attention in experimental research. As there is extensive overlap between cognitive and affective processes (e.g., Davidson, 2003), the impact of sleep loss on affective function may have critical implications for cognitive function (and vice versa). PVT performance was also independent of the sleepiness and affect outcomes. These findings suggest that objective and subjective measures represent distinct entities that should not be assumed to be equivalent. Different answers may emerge depending upon the specific outcomes selected, and the methods by which these outcomes are measured. For understanding the effects of sleep deprivation and their impact on functioning, future studies may benefit from including a full compliment of dimensions and measurement-methods (i.e., multitrait, multimethod assessments). Carefully examining the effects of sleep deprivation on sleepiness, and cognitive and affective outcomes under controlled conditions may reveal predictors of trait vulnerability for a variety of negative repercussions due to sleep loss. The identification of such sleep-related risk factors may suggest not only therapeutic but also preventative strategies to reduce the neurobehavioral consequences of sleep loss, such as accidents, psychopathology, and cognitive impairment.

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Table 1

Means and standard deviation of outcome measures for the SD and non-SD control groups, and univariate group comparisons and partial eta-squared (η^2) effect sizes

Domain	Task/scale	Outcome measure	Mean ± standard deviation		*F*	*P*-value	Effect size (η^2)
			SD group	non-SD group			
Sleepiness	MSLT	Mean sleep latency (minutes, less sleepy)*	3.0 ± 2.9	9.9 ± 4.7	25.08	<0.001	0.501
	Pupil sleepiness test	Pupillary unrest index (PUI; mm/min, more sleepy)	3.9 ± 1.83	1.91 ± 0.77	11.58	0.002	0.317
	VAS: sleepiness	Sleepiness (0–100, more sleepy)	57.0 ± 14.4	23.6 ± 11.2	42.80	<0.001	0.631
Affect	VAS: mood	Negative mood (0–100, more negative)	35.5 ± 14.9	22.6 ± 14.1	4.76	0.039	0.160
	PANAS	Positive affect (10–50, more positive)	20.2 ± 7.2	25.6 ± 6.0	4.78	0.038	0.161
		Negative affect (10–50, more negative)	13.1 ± 2.7	11.8 ± 1.8	1.74	0.199	0.065
	Pupillary affective reactivity/regulation	Task-related pupil dilation (mm, more reactive)	0.015 ± 0.133	−0.150 ± 0.189	9.01	0.006	0.265
Vigilance	Psychomotor	Lapses (RTs >500 ms, more impaired)*	10.6 ± 7.8	4.7 ± 3.9	7.23	0.013	0.224
	Vigilance task	Speed (harmonic mean RT)	346.4 ± 53.4	309.7 ± 26.2	5.04	0.034	0.168

MSLT, Multiple Sleep Latency Test; VAS, visual analog scale; PANAS, Positive and Negative Affect Schedule; RT, reaction time.

*Positively skewed variables that were natural log transformed prior to parametric analyses. Means and standard deviations are reported in the original units.

Table 2

Factor loadings Varimax rotation (Kaiser normalized) across the entire sample and group comparisons on the factor scores

	Factor 1	Factor 2	Factor 3
MSLT	**−0.812**	−0.286	−0.228
PST PUI	**0.818**	0.188	0.172
Pupillary affective reactivity	**0.817**	−0.188	0.059
PANAS: positive affect	−0.084	**−0.823**	−0.070
VAS: negative mood	−0.094	**0.817**	0.250
VAS: sleepiness	0.426	**0.757**	0.281
PVT lapses	0.246	0.130	**0.870**
PVT speed (RT)	0.112	0.273	**0.852**
% Variance explained/cumulative variance	28.34	27.02/55.36	21.42/76.78
SD group (mean ± standard deviation)	0.561 ± 0.967	.434 ± 0.878	0.256 ± 1.08
non-SD group (mean ± standard deviation)	−0.706 ± 0.501	−0.487 ± 0.958	−0.311 ± 0.884
Group comparison (*t*-test)	$t = 4.110$, $P < 0.001$ Cohen's $d = 1.65$	$t = 2.302$, $P = 0.015$ Cohen's $d = 1.00$	$t = 1.469$, $P = 0.154$ Cohen's $d = 0.57$

MSLT, Multiple Sleep Latency Test; VAS, visual analog scale; PANAS, Positive and Negative Affect Schedule; PST, Pupil Sleepiness Test; PUI, Pupillary Unrest Index; PVT, psychomotor vigilance performance; RT, reaction time.

Table 3

Factor loadings Varimax rotation (Kaiser normalized) in the sleep-deprived group

	Factor 1	Factor 2	Factor 3
MSLT	**−0.882**	−0.123	−0.018
PST PUI	**0.885**	−0.102	0.179
Pupillary affective reactivity	**0.746**	−0.178	−0.375
PANAS: positive affect	0.048	**−0.674**	−0.331
VAS: negative mood	−0.259	**0.761**	−0.034
VAS: sleepiness	0.188	**0.892**	0.039
PVT lapses	−0.003	−0.003	**0.882**
PVT speed (RT)	−0.003	0.305	**0.826**
% Variance explained/cumulative variance	27.78	24.82/52.60	23.58/76.18

See details of abbreviations in Table 2.

Table 4

Factor loadings Varimax rotation (Kaiser normalized) in the non-sleep deprived control group

	Factor 1	Factor 2	Factor 3
MSLT	0.145	**−0.702**	0.314
PST PUI	−0.035	0.098	**0.957**
Pupillary affective reactivity	**−0.699**	0.153	−0.104
PANAS: positive affect	**−0.506**	0.186	−0.497
VAS: negative mood	**0.916**	0.098	−0.010
VAS: sleepiness	**0.895**	0.062	−0.007
PVT lapses	0.091	**0.870**	0.104
PVT speed (RT)	−0.023	**0.882**	0.106
% Variance explained/cumulative variance	30.25	26.34/56.59	16.19/72.78

See details of abbreviations in Table 2.