Sleep Restriction Alters Physiological and Emotional Responses to Emotion Induction

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New Findings

The aim of the present study was to assess the effects of sleep restriction on self report and autonomic responses to neutral and sad film clips. Ratings of sadness and heart rate deceleration were greater while watching the sad clip, with no effect of sleep restriction; whereas heart rate variability and skin conductance were impacted by sleep restriction and to a lesser extent by film clips. Results suggest that autonomic function was adaptively altered by sleep restriction, so as to maintain a 'normal' response to emotional cues, despite mounting fatigue.

Abstract

Habitual insufficient sleep has long-term health consequences via its impact on the autonomic nervous system (ANS) function and on emotion regulation. To our knowledge, the effects of insufficient sleep on emotion-induced ANS function has not been tested. The present study aimed to address this lacuna. Using an emotion induction procedure, the effects of sleep restriction on physiological responses to validated neutral and sad film clips were assessed in a 2-by-2, pseudo-randomized, cross-over design. Thirty-one participants, ages 20-33, were assessed after sleeping either 5 hrs (sleep restricted, SR) or 8 hrs (well rested, WR) per night, three consecutive nights. Physiological measures included heart rate (HR), heart rate variability (HRV), skin conductance response (SCR), and participants' ratings of affect and fatigue. There was no effect of sleep condition on self-reported negative affect, however watching the sad clip reduced self-reported fatigue in the SR condition. There was greater HR deceleration while watching sad relative to neutral clips, independent of the sleep condition. SR increased HRV measures with no effect of emotion induction. There was an interaction of emotion induction by sleep condition for SCR, with more SCRs to sad relative to neutral clips in the WR condition, and the opposite effect in the SR condition. Combined the results suggest that ANS response to an emotional cue was altered by sleep restriction. The results suggest an adaptive ANS response to mild, but chronic, sleep restriction, resulting in constant HR response and self-reported experience across well-rested and sleep deprived conditions, despite mounting fatigue.

Introduction

According to a recent worldwide poll, more than 60% of adults attain less than 7 hours sleep per night, with 6 in 10 reporting difficulties with daytime sleepiness (© Koninklijke Philips N.V., 2019); data consistent with results from several other large scale studies (Fund et al., 2020; Luckhaupt, 2010; National Sleep Foundation, 2013). It is, therefore, safe to say that in today's society the vast majority of us habitually attain less than sufficient amounts of sleep for a variety of reasons ranging from work schedule, socioeconomic stress and health problems (Bixler, 2009). Chronic insufficient sleep has long-term health consequences, including increased risk for heart and metabolic diseases (Altevogt & Colten, 2006, pp 21). While mechanisms linking insufficient sleep with said diseases are not well understood, and may vary with the underlying cause of the sleep difficulty, insufficient sleep impacts the function of the autonomic nervous system (ANS, Kuetting et al., 2019; Mullington, Haack, Toth, Serrador, & Meier-Ewert, 2009; Zhong et al., 2005) and disrupts the capacity to regulate emotions , which – in turn - may further exacerbate ANS dysfunction (Smith & Blumenthal, 2011).

However, not all studies report changes in autonomic activity due to insufficient sleep, in fact several studies found little to no change after experimental sleep deprivation (Kato et al., 2000; Meerlo, Sgoifo, & Suchecki, 2008; Vaara, Kyröläinen, Koivu, Tulppo, & Finni, 2009). It has been argued that these inconsistencies are due to methodological differences in body position or degree of physical and cognitive effort (Meerlo et al., 2008). For example, modulation of cardiac activity under sleep deprived conditions was more pronounced while participants were sitting up compared to when they were lying supine (Zhong et al., 2005). Thus, some degree of physical and/or cognitive effort may be necessary to reveal the effects of insufficient sleep on ANS function.

A large body of evidence demonstrates the disruptive effects of insufficient or disturbed sleep on emotional processes. The majority of studies, both laboratory-based and in real-world settings, find that inadequate sleep is linked with more negative and fewer positive emotions (A. N. Goldstein & Walker, 2014; Pilcher & Huffcutt, 1996; Talbot, McGlinchey, Kaplan, Dahl, & Harvey, 2010; Yoo, Gujar, Hu, Jolesz, & Walker, 2007; Zohar, Tzischinsky, Epstein, & Lavie, 2005). These effects are

not restricted to self-report measures. For example, Minkel, et al. (2011) found decreased emotional facial expressiveness following sleep deprivation, despite no subjective effects on experienced emotion. Franzen, et al., (2009) found more robust and anticipatory pupillary dilation when viewing images with negative emotional content in sleep deprived relative to non-sleep deprived participants. In a similar vein, using computerized acoustic analysis, McGlinchey et al., (2011) found that sleep deprived participants' voices had a deeper pitch and were less intense.

While researchers do not necessarily agree on the role of ANS activation in specific emotions (Kreibig, 2010) there is clear evidence of ANS responses to variations in arousal and emotional states (Cacioppo, Berntson, Klein, & Poehlmann, 1997), albeit inconsistent. In her comprehensive review, Kreibig (2010) found both increases and decreases in heart rate (HR, number of heart beats per minute) and heart rate variability (HRV, fluctuation in the time intervals between adjacent heartbeats) to both negative and positive emotions. Broadly, the relationship between HRV and HR is determined by reciprocal sympathetic/parasympathetic drive, wherein greater parasympathetic drive is associated with slower HR and higher HRV and vice versa (Kreibig, 2010; Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996). However, this relationship may be complicated as HRV is also under regulatory control of a variety of other systems that operate on different time scales, e.g., gas exchange or gut muscle tone (Shaffer & Ginsberg, 2017). Unsurprisingly, paradoxical findings have been reported, for example decreases in HR (i.e., increased parasympathetic activity) coupled with increases in skin conductance response (which reflects sympathetic drive) when negative emotions were elicited (e.g., anger, fear, Kreibig, 2010).

Combined, there is strong evidence that disrupted sleep interferes with emotion generation and regulation, while emotional states are linked with autonomic activity. Additionally, insufficient sleep per se may alter autonomic function (Wright Jr et al., 2015; Zhong et al., 2005). Taken together, it is likely that sleep deprivation will impact affect-driven autonomic responses (Meerlo et al., 2008), yet, to our knowledge, this possibility has not been previously tested directly in human participants. Therefore, the present study aimed to assess the effects of sleep restriction on autonomic responses to

emotional stimuli in an emotion induction procedure, using validated film clips, the most common and effective method for inducing emotional arousal (Kreibig, 2010; Westermann, Spies, Stahl, & Hesse, 1996).

To minimize between-subject variability of ANS measures, we employed a cross-over, within-subject, design such that all participants were assessed in all conditions, viewing previously validated neutral and sad film clips after three nights of either 8 hours in bed or 5 hours of sleep. As sleep disruption is associated with more negative and fewer positive emotions (A. N. Goldstein & Walker, 2014; Pilcher & Huffcutt, 1996; Talbot et al., 2010; Yoo et al., 2007; Zohar et al., 2005) we similarly predicted increased negative and decreased positive affect to the sad film clip in the sleep restricted condition, combined with more robust physiological responses.

Methods

Ethical Approval

Approval for the study was granted by the Institutional Review Board of the Academic College of Tel Aviv Yafo (2013181/40). The study conformed to the standards set by the World Medical Association Declaration of Helsinki (Brazil, 2013), except for registration in a database. All participants received a brief verbal explanation of the procedures of the study and its purposes, and signed informed consent forms, in person, prior to beginning the study. The confidentiality and privacy rights of participants were observed at all times.

Participants

Thirty-six participants (25 women and 11 men) took part in the study, ages 20-33 (M=25.35, SD=2.51). Of these, five were excluded due to electrical noise and poor physiological signal. Thus, the final sample included 31 participants with usable physiological data. Participants were recruited from the student body through advertisements published at the College. Hebrew was the native tongue of 94%, 83% reported being married or co-habitating with a significant other. Sixty-one per cent reported exercising regularly. Only six reported taking

medications regularly, five of whom took contraceptives and one took medication for diabetes, and were therefore included in the study.

Instruments

Demographic and general health information was obtained using a brief questionnaire regarding gender identification, age, education, marital status, primary language, employment, education, and health information.

The Pittsburgh Sleep Quality Index (PSQI), a 19-item, self-report questionnaire designed to measure sleep quality and disturbance over the past month (Buysse, Reynolds, Monk, Berman, & Kupfer, 1989), was used to determine if participants had an underlying sleep disorder. Items of the PSQI are grouped into seven components: (sleep duration, sleep disturbance, sleep latency, daytime dysfunction, sleep efficiency, overall sleep quality and medication use) with each component yielding a score from 0 to 3. Component scores are summed to yield a global score, ranging from 0 to 21, with higher scores indicative of worse sleep quality. In validation studies with college students a cut-off score of 6 demonstrated improved specificity and sensitivity to clinically significant sleep disturbance (Aloba, Adewuya, Ola, & Mapayi, 2007; Dietch et al., 2016; Manzar et al., 2015). Internal reliability in this study was acceptable (Cronbach's $\alpha = 0.72$), similar to the values reported in the validation study in Shochat et al., (2007).

State anxiety was measured using the Hebrew version of the 'state' sub-scale (STAI-S) of the State-Trait Anxiety Inventory (STAI) (Spielberger, Gorscuch, & Lushene, 1970; Teichman. & Melineck, 1979). The STAI-S consists of 20 items measuring situational anxiety. Internal reliability in this study was high with Cronbach's alpha in the well-rested condition: $\alpha = 0.93$, and in the sleep restricted condition $\alpha = 0.91$.

Positive (PA) and negative (NA) affect Schedule was used to assess Positive (PA) and Negative Affect (NA, Ben-Zur, 2002; Watson, Clark, & Tellegen, 1988) after viewing the film clips. The PANAS is a 20-item self-report measure that includes a list of adjectives depicting emotional state (e.g., “ashamed”, “excited”) to which participants are instructed to indicate "to what extent do you feel this way now?" on a 5-point Likert scale (1 = very slightly or not at all; 5 = extremely). To avoid repetition, sets of ten items (five positive, five negative) were used after each clip, with random selection of the items. Due to this procedure internal consistency is not reported.

Visual Analogue Scales (VAS, Aitken, 1969) were used to obtain sadness and arousal levels. VAS is a standard psychometric scale used for various contents. A line is shown to participants with each end representing the extreme. There was a VAS for sadness (“how sad did you feel while watching the film clip?”), “not at all” <----------> “extremely sad”; VAS for fatigue/arousal (“how tired did you feel while watching the film clip?”) “Fully awake” <----------> “extremely tired”. Values ranged 0 to 100.

Sleep monitoring: Daily sleep diaries and actigraphy were used to confirm that participants adhered to the prescribed sleep schedules. Sleep schedules were either three consecutive nights of 5 hrs in bed (sleep restriction, SR), or three consecutive nights of at least 8 hrs in bed (well rested, WR). During these days, participants were required to complete sleep diaries every morning, regarding bedtime, wake-up time, if and when they removed the actigraphy device during the previous day, and how refreshed they felt in the morning on a scale of 1 ‘very refreshed’ to 3 ‘very tired’. Actigraphy devices were Mini-Mitter Actiwatch-2 devices (Philips Respironics, Bend, Oregon), worn on the non-dominant hand throughout the three days and nights of each phase of the study. Data were downloaded and analyzed using Actiware 5.0 software (Philips Respironics). Output included bedtime, wake-up time, time in bed (TIB), total sleep time (TST), sleep onset latency (SOL), wake

after sleep onset (WASO), number of awakenings, and sleep efficiency (SE; [TST/TIB]*100).

Emotion induction stimuli: 'Sad' clips included a scene from the movie 'The Champ' (1979) depicting a boy crying over the death of his father, and 'In Memory of Gabby Anderson' (http://tiny.cc/gbxxez), a memorial clip for a child that died. The 'neutral' clips were different segments of a scenic drive from the film 'Gerry' (2002). Each film clip ran 1.5 – 2 minutes and was previously validated on a separate sample of 20 participants in accordance with standard protocols (Gross & Levenson, 1995) using the PANAS. In the validation sample, PA response to the neutral clips was M=11.58, SD=4.55, and to the sad clips M=9.35, SD=3.44, paired t-test T=3.86, p<.001. The NA response to the neutral clips was M=9.50, SD=4.77, and to the sad clips M=12.72, SD=4.50, paired t-test T=-4.68, p<.001.

Physiological data were acquired at 1kHz, using a BIOPAC base unit (MP150-BIOPAC Systems Inc., Goleta, CA) with the electrocardiogram (ECG100C) and galvanic skin conductance (GSR100C) modules. For cardiac signals, Ag/AgCl electrodes were placed in a bipolar precordial lead (lead II), while skin conductance was acquired with Ag/AgCl electrodes place on the 2nd and 4th digits of the left hand. Data was acquired and analyzed using AcqKnowledge v4.0 software, and a transistor-transistor Logic (TTL) digital signal, presented via Inquisit 3.0, marked onset and off-set of baseline and film presentation periods. After manual removal of artefacts, and application of 1Hz low-pass filter, three measures were derived: [1] Heart rate (HR) was expressed in units of beats-per-minute (BPM), derived from R-R moment. [2] Heart rate variability with a combination of time-domain and frequency-domain measurements. Time-domain indices of HRV quantify the variability in inter-beat interval, whereas frequency-domain measurements estimate the distribution of absolute or relative power in several frequency bands (Shaffer & Ginsberg, 2017). The two main components derived from frequency analysis are a high-frequency band (HF, 0.15 to 0.4 Hz) that reflects parasympathetic nerve activity, and a low-frequency (LF, 0.04 to 0.15 Hz) that relates to both sympathetic and parasympathetic tone. As the time frame for detecting change in the LF component is greater than 1-2

minutes timeframe of the film clip typically used in emotion-induction studies, we report HF and the ratio of LF/HF, the latter reflecting the balance between sympathetic and parasympathetic systems. Here, HRV was derived using AcqKnowledge 4.0 built-in algorithms. (1) Time domain method, calculated as the standard deviation of successive differences (SDSD) of the R-R interval, appropriate for short-term variability (Shaffer & Ginsberg, 2017). (2) Frequency domain method, where the power spectrum density is estimated for the R-R interval series, using Fast Fourier Transformation. The power in the high frequency (HF, 0.15-0.4 Hz) band, and the ratio of LF to HF are reported.

Skin Conductance measures fluctuations in electrical properties of the skin due to sweat secretion, and reflects sympathetic drive as sweat glands are innervated by sympathetic nerves (Dawson, Schell, & Filion, 2007; Kreibig, 2010). Changes in skin conductance typically include skin conductance level (SCL, the overall conductivity of the skin) and skin conductance response rate ([SCR] the number of skin conductance responses per minute, Kreibig, 2010). Here skin conductance measures were derived from the signal recorded on the GSR100 amplifier, after applying a 50Hz band pass filter. Measures were the average skin conductance level (SCL), and the number of SCR (# SCRs) events, identified as transient increases in skin conductance level that exceed 0.05 microSiemens.

After quality control, data extraction was conducted by research assistants blinded to study protocol. All measurements were normalized to baseline period acquired prior to the presentation of each clip. For HR, HF, LF/HF and SCL values were normalized by division by baseline. For # SCRs, the value obtained during the baseline was subtracted from the value during the film clip, to avoid dividing by zero.

Procedure

After signing informed consent, participants completed the demographic and PSQI questionnaires. Participants were then given actiwatches and sleep diaries and were instructed which schedule to maintain prior to their subsequent visits, three consecutive nights of 5 hrs in bed (sleep restriction, SR), and three consecutive nights of at least 8 hrs in bed (well rested, WR), in counterbalanced order. If participants were assigned first to the SR

condition, their WR condition started only after a 3-day "washout" period. On each subsequent laboratory visit, participants were hooked up to the physiological set-up upon arrival to the lab. To allow physiological signals to stabilize before beginning acquisition, participants completed the STAI-S. The experimental procedure included the two emotion induction conditions - neutral and sad, in counterbalanced order across visits and participants. The course of events for each clip condition was identical: [1] an initial 30 sec where participants were asked to focus on a plus symbol ('+') to obtain baseline measurements; [2] presentation of the video clip; [3] responses to each film clip assessed with the VASs and to 10 items from the PANAS. Between film clip periods, participants played computer games for approximately 7 minutes.

Statistics

Statistical analyses were performed in SPSS V24.0. Change values were calculated for each of the physiological measures. For HR, SDSD, HF, LF/HF, and SCL change values were the quotient of division by baseline, for # SCRs, change was calculated by subtracting the number of events during baseline. To determine whether the film clips induced statistically significant changes in physiological measures, one sample T-tests were run on change values, with hypothesized means of 1 for HR, SDSD, HF, LF/HF, and SCL, and 0 for SCRs. For testing the effects of sleep restriction and emotion induction on the different dependent variables, repeated measures ANOVAs were used.

RESULTS

Descriptive statistics and actigraphy results are reported in Table 1. As can be seen, the majority of the sample (87%) were below the PSQI cut off of 6.0, reflecting normal sleep. Of the remainder (n=4), the average score was 7.5. Overall, participants adhered to the sleep restriction schedule, with significant reductions in time in bed (TIB) and total sleep time (TST), mainly by delaying their bedtime. The intervention also resulted in less wake time after sleep onset (WASO) and fewer nocturnal awakenings. According to their sleep diaries, participants felt significantly less refreshed after sleep restriction nights. Additionally, participants reported greater state anxiety when sleep restricted compared to the well-rested condition.

Repeated measures ANOVAs were run to assess the effects of sleep restriction and emotion induction on ratings on the VAS scales and PANAS (Table 2). For the fatigue VAS, there was a main effect of sleep condition ($F_{1,30}$=45,33, p<.001, partial η^2=0.602), a main effect of emotion condition ($F_{1,30}$=21.20, p<.001, partial η^2=0.414) and a significant interaction ($F_{1,30}$=10.11, p=.004, partial η^2=0.250). Post hoc analysis revealed significantly lower fatigue ratings after viewing the sad clip in the sleep restricted condition ($F_{1,30}$=30.95, p<.001, partial η^2=0.508), but no difference in the well-rested condition ($F_{1,30}$<1.0). For the sadness VAS, there was a main effect of emotion induction ($F_{1,30}$=155.51, p<.001, partial η^2=0.916), but no effect of sleep condition nor an interaction (Fs<1.0). There was a main effect of emotion induction condition for PANAS-NA ($F_{1,30}$=9.40, p=.004, partial η^2=0.227), with no effect of sleep restriction or interaction (Fs<1.0). There were no main or interaction effects for the PANAS-PA (Fs<1.0).

To assess whether sleep restriction impacted physiological measures, independent of emotional state, we first analyzed the averaged 30-sec baselines of the two film clips and performed paired T-tests on the different physiological measures to compare the sleep

restricted to well-rested conditions. Heart rate was higher in the well-rested (M = 81.08, SD = 15.78) compared with the sleep restricted condition (M = 77.25, SD = 11.89, p=0.042), and SCR rate was marginally higher in the well-rested (M = 3.01, SD = 1.21) compared with the sleep restricted condition (M = 2.53, SD = 1.01, p=0.053). All other baseline measurements did not differ between the sleep schedule conditions (p's>.050, data not shown).

To determine whether presentation of clips induced significantly changed physiological measures, change values for each physiological measure were calculated and single sample T-tests were run. For the majority of measurements there was a significant change relative to baseline (Table 3). Exceptions were HR during the neutral clip under sleep restriction condition, and SDSD during the neutral clips, in both sleep-restricted and well-rested condition, as well as SDSD during the sad clip in the well-rested condition.

The combined effects of sleep restriction and emotion induction on physiological measures were assessed using repeated measures ANOVAs. For HR (Figure 1a), there was a main effect of emotion induction ($F_{1,29}$=7.31, p=.011, partial η^2=.201) with greater attenuation in heart rate, relative to baseline, while watching the sad film clip compared with the neutral clip, with no main effect of sleep schedule nor an interaction (Fs<1.0). For SDSD (Figure 1b), there was a main effect of sleep schedule ($F_{1,29}$=5.298, p=.029, partial η^2=.154) due to greater increase from baseline in the sleep restriction condition, with no effect of emotion induction nor an interaction (Fs<1.0). For HF (Figure 1c), there a significant interaction of emotion induction and sleep condition ($F_{1,30}$=5.285, p=.029, partial η^2=.150), with no main effect of sleep condition ($F_{1,30}$=2.284, p=.141) nor emotion induction (F<1.0). Post-hoc analysis revealed significantly lower HF during sad vs. neutral clip in the well-rested condition (p=.046), while significantly higher HF to the neutral clip in the well-rested vs. sleep restricted condition (p=.020). For the LF/HF ratio (Figure 1d), there was a main effect of sleep condition ($F_{1,30}$=5.016, p=.033, partial η^2=.147) with a greater increase from

baseline in the sleep restricted condition. There was no main effect of emotion induction ($F_{1,30}$=2.575, p=.119) nor an interaction ($F_{1,30}$=2.253, p=.144). For SCL (Figure 1e), there were no main effects nor interactions (Fs<1.0). For SCR rate (Figure 1f), there was a significant interaction of sleep condition by emotion induction ($F_{1,28}$=4.843, p=.036, partial η^2=.139), and no significant main effects (Fs<=1.0). These were due to more SCRs in the sleep restricted neutral movie clip condition compared with well-rested neutral condition (p=.043).

Discussion

The aim of the present study was to assess the effects of sleep restriction on emotion-induced autonomic responses and self-reported affective experience using a sleep restriction protocol of three consecutive nights, to simulate real-world chronic insufficient sleep. Our predictions, that sleep restriction would enhance self-reported and physiological responses to a sad film clip were not supported, however our results provide a different perspective to the effects of sleep restriction on emotion. In a nutshell, we found a dissociation between emotion induction effects on heart rate and sleep restriction effects on heart rate variability and skin conductance, which we interpret to represent an adaptation of the ANS to sleep restriction.

In contrast to other studies (Dinges et al., 1997; Franzen et al., 2009; e.g., Talbot et al., 2010) sleep deprivation did not amplify self-reported negative affect ratings to the sad film clip. This unexpected finding is likely due to the fact that in the plurality of other studies negative affect was measured generally and not in response to a discrete cue. Consistently, in this study, state anxiety significantly increased in the sleep restricted condition commensurate with a more general effect of sleep restriction on mood. That said, Zohar et al, (2005) (2005), who used an experience-sampling methodology in a sample of medical residents, found that sleep loss amplified negative emotive responses to disruptive events, highlighting the advantage of ecologically-valid paradigms.

As lower power in the HF band and a higher LF/HF ratio have been linked with stress and anxious states (Thayer, Ahs, Fredrikson, Sollers, & Wager, 2012), it has been postulated that sleep deprivation may have similar effects, i.e., increased sympathetic and diminished parasympathetic drive (Meerlo et al., 2008; Tobaldini, Pecis, & Montano, 2014). Thus, we expected that sleep restriction would increase sympathetic and decrease parasympathetic measures at baseline. Yet in contrast to several previous reports (Prado, Bevilacqua, Trabattoni, Porta, & Montano, 2013; Tobaldini et al., 2014; Zhong et al., 2005) but consistent with others (Kato et al., 2000; Vaara et al., 2009), baseline heart rate decreased in the sleep restriction condition, with only a marginal increase in SCR rate, while other baseline measures of sympathetic activity (e.g., LF/HF and SDSD) were not impacted by sleep restriction. Again, a direct comparison with other studies is problematic as baseline measurements in this study constituted a total of one minute, which may not be sufficient to detect state-level changes. Further, this baseline was obtained while participants were instructed to focus on a single point on the computer monitor, which was meant to be emotionally neutral, but may not be a true baseline.

A reduction in heart rate was observed while participants watched both sad and neutral film clips, a response more robust during the sad clip but unaffected by sleep restriction. In the well-rested condition, this sad clip-related attenuation in heart rate was coupled with increased HF, a small increase in LF/HF ratio, and an increase in SCR rate. This pattern is in line with several studies by Porges and colleagues (Cheung & Porges, 1977; Porges, 2007; Porges & Coles, 1982; Porges & Raskin, 1969; e.g., Walter & Porges, 1976) who demonstrated that brief attentional demand causes heart rate deceleration, while others have reported that higher HRV was associated with improved attention allocation (Park, Vasey, Van Bavel, & Thayer, 2013). Potentially, the sad movie clips were more engaging than the neutral clips.

An alternate explanation is that the pattern of physiological responses in the well-rested condition represents a 'deactivating sadness response', observed when participants are exposed to cues (e.g., film clips/music) that elicit sadness without crying. This deactivating pattern has been found to occur in response to film clips that depict scenes related to loss that has already occurred (as in this study) as opposed to anticipatory loss (e.g., a loved one dying from cancer) (Kreibig, 2010). This evoked despondency associated with increases in both sympathetic (i.e., SCR rate) and parasympathetic (i.e., HF and LF/HF ratio) tone may indicate a greater increase in parasympathetic activity to counter sympathetic arousal.

Unlike HR, HRV measures were impacted by sleep restriction but not - for the most part - by the emotion induction. Both SDSD and LF/HF were higher, across emotion clips, in the sleep restriction condition compared to the well-rested condition. HF, on the other hand, was higher in the sleep-restricted condition only when watching the neutral clip. This pattern suggests that under sleep restriction, the sad emotion induction resulted in a smaller response in *both* the low frequency and the high frequency components of the HRV, leading to similar LF/HF values for the neutral and sad clips. While LF is no longer believed to represent sympathetic activity per se (D. S. Goldstein, Bentho, Park, & Sharabi, 2011; Hayano & Yuda, 2019), involvement of the sympathetic system is a potential explanation of the finding and in line with the reduction in SCRs observed in the sleep restriction, sad clip condition. Typically, it is assumed that the sympathetic and parasympathetic systems are complementary, and thus it would be expected that as HF declines LF (and SCRs) would increase; yet it has been found that fluctuations in the two bands may be on different time scales (Shaffer & Ginsberg, 2017), thereby temporally uncoupling the responses of the two measures. However, this intriguing pattern has also been reported after steady-state exercise in healthy participant (Grant & Ker, 2008; Maud & Foster, 2006 pp., 52; Povea et al., 2005), suggesting that the combination of sleep restriction and an emotional challenge induce an

adaptive response to maintain constant heart rate response across well-rested and sleep deprived conditions. This conjecture is also consistent also with the decreased self-reported fatigue when watching the sad clip in the sleep restriction condition.

Clearly, these findings require further investigation, and several limitations of this study should be highlighted. First, the sample was relatively small and uniform with respect to age and health status. Specifically, participants were screened not to have significant sleep disturbances, thereby limiting the generalizability to young, healthy, urban populations. Second, we used only one emotion and limited physiological sampling of baseline ANS activity. The effects of sleep restriction should be studied using a broader range of emotions, and allow for longer changes in the physiological measurements. Third, further controls should be established to assess sensitivity to stressors which could perhaps explain some of the variance in physiological measures. Fourth, while we attempted to minimize the effects of anticipation of a negative stimulus by randomization, the potential biasing effects of anticipation cannot be denied. Finally, as noted above, the laboratory environment, while providing a controlled setting, diminishes the ecological validity of the results.

The abovementioned limitations notwithstanding, to our knowledge this is the first study to assess the effects of sleep restriction on physiological correlates of emotional responses. Combined these finding are in line with the notion that ANS functioning is altered by sleep restriction, but may not be in line with the hypothesized increased sympathetic / decreased parasympathetic activity (Meerlo et al., 2008; Tobaldini et al., 2014). The picture that can be drawn from our results points to the recruitment of the autonomic system in an adaptive manner, so as to adjust heart rate and perhaps the cognitive-emotional experience, to attain ‘normal’ responding to emotional cues, despite increased fatigue. Additional research is required to confirm this theory and examine the potential cost of this adaptation over time.

Bibliography

© Koninklijke Philips N.V. (2019). *The global pursuit of better sleep health*. Retrieved from https://www.usa.philips.com/c-dam/b2c/master/experience/smartsleep/world-sleep-day/2019/2019-philips-world-sleep-day-survey-results.pdf

Aitken, R. C. (1969). A growing edge of measurement of feelings: Measurement of feelings using visual analogue scales. *Proceedings of the Royal Society of Medicine*, *62*, 989–993.

Aloba, O. O., Adewuya, A. O., Ola, B. A., & Mapayi, B. M. (2007). Validity of the Pittsburgh sleep quality index (PSQI) among Nigerian university students. *Sleep Medicine*, *8*(3), 266–270.

Altevogt, B. M., & Colten, H. R. (2006). *Sleep disorders and sleep deprivation: An unmet public health problem*. National Academies Press.

Ben-Zur, H. (2002). Coping, affect and aging: The roles of mastery and self-esteem. *Personality and Individual Differences*, *32*(2), 357–372.

Bixler, E. (2009). Sleep and society: An epidemiological perspective. *Sleep Medicine*, *10*, S3–S6.

Buysse, D. J., Reynolds, C. F., Monk, T. H., Berman, S. R., & Kupfer, D. J. (1989). The Pittsburgh Sleep Quality Index: A new instrument for psychiatric practice and research. *Psychiatry Research*, *28*(2), 193–213.

Cacioppo, J. T., Berntson, G. G., Klein, D. J., & Poehlmann, K. M. (1997). Psychophysiology of emotion across the life span. *Annual Review of Gerontology and Geriatrics*, *17*, 27–74.

Cheung, M. N., & Porges, S. W. (1977). Respiratory influences on cardiac responses during attention. *Physiological Psychology*, *5*(1), 53–57.

Dawson, M. E., Schell, A. M., & Filion, D. L. (2007). The electrodermal system. *Handbook of Psychophysiology*, *2*, 200–223.

Dietch, J. R., Taylor, D. J., Sethi, K., Kelly, K., Bramoweth, A. D., & Roane, B. M. (2016). Psychometric evaluation of the PSQI in US college students. *Journal of Clinical Sleep Medicine*, *12*(08), 1121–1129.

Dinges, D. F., Pack, F., Williams, K., Gillen, K. A., Powell, J. W., Ott, G. E., … Pack, A. I. (1997). Cumulative sleepiness, mood disturbance, and psychomotor vigilance performance decrements during a week of sleep restricted to 4–5 hours per night. *Sleep*, *20*(4), 267–277.

Franzen, P. L., Buysse, D. J., Dahl, R. E., Thompson, W., & Siegle, G. J. (2009). Sleep deprivation alters pupillary reactivity to emotional stimuli in healthy young adults. *Biological Psychology*, *80*(3), 300–305. doi: 10.1016/j.biopsycho.2008.10.010

Fund, N., Green, A., Chodick, G., Orin, M., Koren, G., Shalev, V., & Dagan, Y. (2020). The epidemiology of sleep disorders in Israel: Results from a population-wide study. *Sleep Medicine*, *67*, 120–127.

Goldstein, A. N., & Walker, M. P. (2014). The Role of Sleep in Emotional Brain Function. *Annual Review of Clinical Psychology*, *10*, 679–708. doi: 10.1146/annurev-clinpsy-032813-153716

Goldstein, D. S., Bentho, O., Park, M.-Y., & Sharabi, Y. (2011). Low-frequency power of heart rate variability is not a measure of cardiac sympathetic tone but may be a measure of modulation of cardiac autonomic outflows by baroreflexes. *Experimental Physiology*, *96*(12), 1255–1261.

Grant, C. C., & Ker, J. A. (2008). Autonomic response to exercise as measured by cardiovascular variability. *South African Journal of Sports Medicine*, *20*(4), 102–108.

Gross, J. J., & Levenson, R. W. (1995). Emotion elicitation using films. *Cognition & Emotion*, *9*(1), 87–108.

Hayano, J., & Yuda, E. (2019). Pitfalls of assessment of autonomic function by heart rate variability. *Journal of Physiological Anthropology*, *38*(1), 1–8.

Kato, M., Phillips, B. G., Sigurdsson, G., Narkiewicz, K., Pesek, C. A., & Somers, V. K. (2000). Effects of sleep deprivation on neural circulatory control. *Hypertension*, *35*(5), 1173–1175.

Kreibig, S. D. (2010). Autonomic nervous system activity in emotion: A review. *Biological Psychology*, *84*(3), 394–421. (20371374). doi: 10.1016/j.biopsycho.2010.03.010

Kuetting, D. L., Feisst, A., Sprinkart, A. M., Homsi, R., Luetkens, J., Thomas, D., … Dabir, D. (2019). Effects of a 24-hr-shift-related short-term sleep deprivation on cardiac function: A cardiac magnetic resonance-based study. *Journal of Sleep Research*, *28*(3), e12665.

Luckhaupt, S. E. (2010). *Short sleep duration among workers–United States, 2010.* (No. 513-841–4123). Centers for Disease Control and Prevention.

Manzar, M. D., Moiz, J. A., Zannat, W., Spence, D. W., Pandi-Perumal, S. R., BaHammam, A. S., & Hussain, M. E. (2015). Validity of the Pittsburgh sleep quality index in Indian university students. *Oman Medical Journal*, *30*(3), 193.

Maud, P. J., & Foster, C. (2006). *Physiological assessment of human fitness*. Human Kinetics.

McGlinchey, E. L., Talbot, L. S., Chang, K., Kaplan, K. A., Dahl, R. E., & Harvey, A. G. (2011). The effect of sleep deprivation on vocal expression of emotion in adolescents and adults. *Sleep*, *34*(9), 1233–1241.

Meerlo, P., Sgoifo, A., & Suchecki, D. (2008). Restricted and disrupted sleep: Effects on autonomic function, neuroendocrine stress systems and stress responsivity. *Sleep Medicine Reviews*, *12*(3), 197–210.

Minkel, J., Htaik, O., Banks, S., & Dinges, D. (2011). Emotional expressiveness in sleep-deprived healthy adults. *Behavioral Sleep Medicine*, *9*(1), 5–14.

Mullington, J. M., Haack, M., Toth, M., Serrador, J. M., & Meier-Ewert, H. K. (2009). Cardiovascular, inflammatory, and metabolic consequences of sleep deprivation. *Progress in Cardiovascular Diseases*, *51*(4), 294–302. (19110131). doi: 10.1016/j.pcad.2008.10.003

National Sleep Foundation. (2013). *2013 International Bedroom Poll*.

Park, G., Vasey, M. W., Van Bavel, J. J., & Thayer, J. F. (2013). Cardiac vagal tone is correlated with selective attention to neutral distractors under load. *Psychophysiology*, *50*(4), 398–406. (23418911). doi: 10.1111/psyp.12029

Pilcher, J. J., & Huffcutt, A. I. (1996). Effects of sleep deprivation on performance: A meta-analysis. *Sleep*, *19*(4), 318–326.

Porges, S. W. (2007). The polyvagal perspective. *Biological Psychology*, *74*(2), 116–143.

Porges, S. W., & Coles, M. G. H. (1982). Individual differences in respiratory-heart period coupling and heart period responses during two attention-demanding tasks. *Physiological Psychology*, *10*(2), 215–220.

Porges, S. W., & Raskin, D. C. (1969). Respiratory and heart rate components of attention. *Journal of Experimental Psychology*, *81*(3), 497.

Povea, C., Schmitt, L., Brugniaux, J., Nicolet, G., Richalet, J.-P., & Fouillot, J.-P. (2005). Effects of intermittent hypoxia on heart rate variability during rest and exercise. *High Altitude Medicine & Biology*, *6*(3), 215–225.

Prado, M., Bevilacqua, M., Trabattoni, D., Porta, A., & Montano, N. (2013). *One night on-call: Sleep deprivation affects cardiac autonomic control and inflammation in physicians ☆*.

Shaffer, F., & Ginsberg, J. P. (2017). An overview of heart rate variability metrics and norms. *Frontiers in Public Health*, *5*, 258.

Shochat, T., Tzischinsky, O., Oksenberg, A., & Peled, R. (2007). Validation of the Pittsburgh Sleep Quality Index Hebrew translation (PSQI-H) in a sleep clinic sample. *The Israel Medical Association Journal*, *9*(12), 853.

Smith, P. J., & Blumenthal, J. A. (2011). Psychiatric and behavioral aspects of cardiovascular disease: Epidemiology, mechanisms, and treatment. *Revista Española de Cardiología (English Edition)*, *64*(10), 924–933.

Spielberger, C. D., Gorscuch, R. L., & Lushene, R. E. (1970). *STAI manual for the state-trait anxiety inventory: ("Self-evaluation questionnaire").* Palo Alto, CA: Consulting Psychologists Press.

Talbot, L. S., McGlinchey, E. L., Kaplan, K. A., Dahl, R. E., & Harvey, A. G. (2010). Sleep deprivation in adolescents and adults: Changes in affect. *Emotion*, *10*(6), 831.

Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology. (1996). Heart rate variability. Standards of measurement, physiological interpretation, and clinical use. *Circulation*, *93*, 1043–1065. (8737210).

Teichman., Y., & Melineck, C. (1979). *Manual of the Hebrew state – Trait Anxiety Inventory*. Tel-Aviv, Israel: Tel Aviv University.

Thayer, J. F., Ahs, F., Fredrikson, M., Sollers, J. J., 3rd, & Wager, T. D. (2012). A meta-analysis of heart rate variability and neuroimaging studies: Implications for heart rate variability as a marker of stress and health. *Neuroscience and Biobehavioral Reviews*, *36*(2), 747–756. (22178086). doi: 10.1016/j.neubiorev.2011.11.009

Tobaldini, E., Pecis, M., & Montano, N. (2014). Effects of acute and chronic sleep deprivation on cardiovascular regulation. *Arch Ital Biol*, *152*(2–3), 103–10.

Vaara, J., Kyröläinen, H., Koivu, M., Tulppo, M., & Finni, T. (2009). The effect of 60-h sleep deprivation on cardiovascular regulation and body temperature. *European Journal of Applied Physiology*, *105*(3), 439–444.

Walter, G. F., & Porges, S. W. (1976). Heart rate and respiratory responses as a function of task difficulty: The use of discriminant analysis in the selection of psychologically sensitive physiological responses. *Psychophysiology*, *13*(6), 563–571.

Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: The PANAS scales. *Journal of Personality and Social Psychology*, *54*(6), 1063–1070.

Westermann, R., Spies, K., Stahl, G., & Hesse, F. W. (1996). Relative effectiveness and validity of mood induction procedures: A meta-analysis. *European Journal of Social Psychology*, *26*(4), 557–580.

Wright Jr, K. P., Drake, A. L., Frey, D. J., Fleshner, M., Desouza, C. A., Gronfier, C., & Czeisler, C. A. (2015). Influence of sleep deprivation and circadian misalignment on cortisol, inflammatory markers, and cytokine balance. *Brain, Behavior, and Immunity*, *47*, 24–34.

Yoo, S. S., Gujar, N., Hu, P., Jolesz, F. A., & Walker, M. P. (2007). The human emotional brain without sleep—A prefrontal amygdala disconnect. *Current Biology*, *17*(20), R877-R878.

Zhong, X., Hilton, H. J., Gates, G. J., Jelic, S., Stern, Y., Bartels, M. N., … Basner, R. C. (2005). Increased sympathetic and decreased parasympathetic cardiovascular modulation in normal humans with acute sleep deprivation. *Journal of Applied Physiology*, *98*(6), 2024–2032.

Zohar, D., Tzischinsky, O., Epstein, R., & Lavie, P. (2005). The effects of sleep loss on medical residents' emotional reactions to work events: A cognitive-energy model. *Sleep*, *28*(1), 47–54. (15700720).

Legends

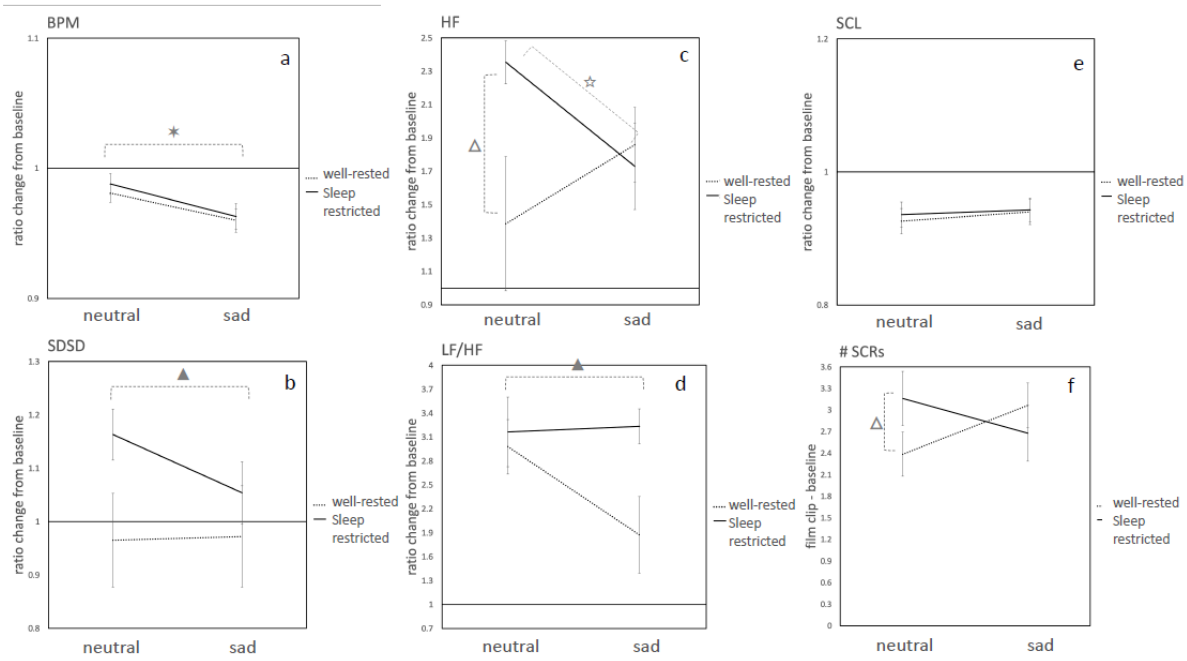

Figure 1 Effects of sleep schedule and mood induction on physiological measures. [a] HR – heart rate; [b] SDSD - standard deviation of successive R-R interval differences; [c] HF – high frequency; [d] LF/HF ratio; [e] SCL – skin conductance level; [f] SCR – skin conductance response rate (per minute analyzed). Except for SCR, all values represent the quotient of baseline, obtained prior to each film clip. For SCR, baseline was subtracted from the values obtained while watching the film clip. Dashed lines are measurements obtained in the well-rested condition, full lines are measurements obtained in the sleep restricted condition; ✯ = main effect of emotion; ☆ = post-hoc effect of emotion; ▲ = main effect of sleep; ∆ = post-hoc effect of sleep.

Table 1: Descriptive Statistics of Actigraphy and Sleep Diary Data

PSQI (M [range])	4.04 [0 – 8.0]		
PSQI % >6	13 [*n*=4]		
	Well Rested (M [SD])	Sleep Restriction (M [SD])	Paired *t*
Bedtime (hrs:min)	10:00 [11:04]	3:56 [7:01]	2.293*
Wake up (hrs:min)	8:37 [1:04]	7:09 [0:57]	7.241**
TIB (hrs:min)	8:23 [1:02]	5:52 [0:53]	9.014**
TST (hrs:min)	7:05 [0:40]	5:00 [0:35]	16.369*
SOL (min)	9.68 [12.00]	7.15 [8.69]	0.989
WASO (min)	52.38 [20.08]	32.43 [12.78]	4.586**
# awakenings	27.11 [10.30]	17.74 [6.29]	5.134**
SE (% of TIB)	85.95 [5.04]	86.28 [4.90]	-0.326
Feeling refreshed	1.04 [0.56]	2.22 [0.50]	-9.843*
STAI-S	33.34 [9.09]	44.01 [5.47]	-10.68**

Note: PSQI - Pittsburgh Sleep Quality Index; TIB - time in bed; TST - total sleep time; SOL - sleep onset latency; WASO - wake after sleep onset; SE - sleep efficiency; Feeling refreshed from sleep diary reports, STAI-S – State and Trait Anxiety Index, State subscale. '*' – $p<.05$; '**' - $p<.001$.

Table 2: Responses on the Visual Analogue Scales (VAS) and to PANAS

		VAS Fatigue	VAS Sadness
Well Rested	Neutral	27.74 [25.78][1]	15.48 [22.19][2]
	Sad	24.19 [24.60][1]	71.94 [25.49]
Sleep Restricted	Neutral	75.48 [22.78][1,2]	12.94 [16.55][2]
	Sad	48.06 [31.67]	74.19 [26.05]
		PA	NA
Well Rested	Neutral	11.58 [3.95]	9.12 [4.08][2]
	Sad	10.48 [3.51]	11.48 [3.00]
Sleep Restricted	Neutral	10.82 [4.39]	9.21 [4.07][2]
	Sad	10.00 [3.06]	11.45 [3.99]

Note: Values represent means and SD of participants response collected immediately after watching each clip. 1=significant difference between well-rested and sleep restricted condition, 2=significant difference between neutral and sad clips.

Table 3: Physiological Responses to Neutral and Sad Film Clips

	Well Rested		Sleep Restricted	
	Neutral	Sad	Neutral	Sad
HR_{BPM}	0.981 [0.04][3]	0.960 [0.06][3]	0.988 [0.04]	0.963 [0.05][3]
SDSD	0.965 [0.05]	0.972 [0.06]	1.163 [0.09][3]	1.054 [1.00]
HF	1.386 [0.71][1,2,3]	1.859 [1.44][3]	2.238 [2.24][3]	1.730 [1.25][3]
LF/HF	2.979 [2.39][2,3]	1.873 [1.19][1,3]	3.165 [1.87][3]	3.235 [2.64][3]
SCL	0.896 [0.14][3]	0.954 [0.16][3]	0.927 [0.14][3]	0.939 [0.15][3]
# SCRs	2.063 [1.05][1,2,3]	2.251 [1.23][3]	2.637 [1.48][3]	2.305 [1.22][3]

Note: Values represent means and SD of change from baseline. HR – heart rate; BPM - beats per minute; HF – high frequency; LF – low frequency; SDSD - standard deviation of successive R-R interval differences; SCL – skin conductance level; SCR – skin conductance response rate (per minute analyzed). '1' - significant difference between well-rested and sleep restricted condition; '2'– significant difference between neutral and sad clips; '3' – significant change from baseline.