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Sleep restriction alters physiological and emotional responses to emotion induction

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Abstract

Habitual insufficient sleep has long-term health consequences via its impact on autonomic nervous system (ANS) function and on regulation of emotion. To our knowledge, the effects of insufficient sleep on emotion-induced ANS function have 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 two-by-two, pseudo-randomized, cross-over design. Thirty-one participants, aged 20–33 years, were assessed after sleeping for either 5 h (sleep restricted, SR) or 8 h (well rested, WR) per night, for three consecutive nights. Physiological measures included heart rate, heart rate variability, skin conductance response (SCR) and participants' ratings of affect and fatigue. There was no effect of sleep conditions on self-reported negative affect, but watching the sad clip reduced self-reported fatigue in the SR condition. There was greater heart rate deceleration while watching sad relative to neutral clips, independent of the sleep condition. Sleep restriction increased heart rate variability measures, with no effect of emotion induction. There was an interaction of emotion induction with 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 the ANS response to an emotional cue was altered by sleep restriction. The results suggest an adaptive ANS response to mild, chronic sleep restriction, resulting in constant heart rate response and self-reported experience across WR and SR conditions, despite mounting fatigue.

KEYWORDS
autonomic system, sleep deprivation, vagal tone

1 | INTRODUCTION

According to a recent worldwide poll, >60% of adults attain less than 7 h of sleep per night, with six 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, including 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: p. 21). Although the mechanisms linking insufficient sleep with these diseases are not well understood and might 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, might further exacerbate ANS dysfunction (Smith & Blumenthal, 2011).

However, not all studies report changes in autonomic activity owing 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 attributable to methodological differences in body position or the degree of physical and cognitive effort (Meerlo et al., 2008). For example, modulation of cardiac activity in sleep-deprived conditions was more pronounced while participants were sitting up compared with when they were lying supine (Zhong et al., 2005). Thus, some degree of physical and/or cognitive effort might 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 (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, Htaik, Banks, and Dinges (2011) found decreased emotional facial expressiveness after sleep deprivation, despite a lack of subjective effects on experienced emotion. Franzen, Buysse, Dahl, Thompson, and Siegle (2009) found more robust and anticipatory pupillary dilatation 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.

Although 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, i.e. number of heart beats per minute) and heart rate variability (HRV, i.e. fluctuation in the time intervals between adjacent heart beats) to both negative and positive emotions. Broadly speaking, the relationship between HRV and HR is determined by reciprocal sympathetic and 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 can be complicated because HRV is also under regulatory control of a variety of other systems that operate on different time scales, such as 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 the generation and regulation of emotion and that emotional states are linked with autonomic activity. Additionally, insufficient sleep *per se* may alter autonomic function (Wright 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), but, to our knowledge, this possibility has not been tested before in human participants. Therefore, the aim of the present study was 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 used 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 h in bed or 5 h of sleep. Given that sleep disruption is associated with more negative and fewer positive emotions (Goldstein & Walker, 2014; Pilcher & Huffcutt, 1996; Talbot et al., 2010; Yoo et al., 2007; Zohar et al., 2005), we predicted increased negative and decreased positive affect to the sad film clip in the sleep-restricted condition, combined with more robust physiological responses.

New Findings

- **What is the central question of this study?**
  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.
- **What is the main finding and its importance?**
  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. The results suggest that autonomic function was adaptively altered by sleep restriction, in order to maintain a 'normal' response to emotional cues, despite mounting fatigue.

2 | METHODS

2.1 | 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, before beginning the study. The confidentiality and privacy rights of participants were observed at all times.

2.2 | Participants

Thirty-six participants (25 women and 11 men) took part in the study (ages 20–33 years, mean = 25.35 years, SD = 2.51 years). Of these, five were excluded because of 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%, and 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 who took medication for diabetes, and they were therefore included in the study.

2.3 | 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.

2.3.1 | The Pittsburgh Sleep Quality Index

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 whether 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 zero to three. Component scores are summed to yield a global score, ranging from zero to 21, with higher scores indicative of worse sleep quality. In validation studies with college students, a cut-off score of six 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 by Shochat, Tzischinsky, Oksenberg, and Peled (2007).

2.3.2 | State anxiety

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 α in the well-rested condition = 0.93, and in the sleep-restricted condition $\alpha = 0.91$.

2.3.3 | Positive and negative affect schedule

Positive and negative affect schedule (PANAS) 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 five-point Likert scale (1 = very slightly or not at all; 5 = extremely). To avoid repetition, sets of 10 items (five positive, five negative) were used after each clip, with random selection of the items. Owing to this procedure, internal consistency is not reported.

2.3.4 | Visual analog scales

Visual analog scales (VAS) (Aitken, 1969) were used to obtain sadness and arousal levels. The 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' and a VAS for fatigue/arousal ('how tired did you feel while watching the film clip?') 'Fully awake' ←→ 'extremely tired'. Values ranged from zero to 100.

2.3.5 | 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 h in bed (sleep restriction, SR) or three consecutive nights of ≥8 h in bed (well rested, WR). During these days, participants were required to complete sleep diaries every morning with regard to 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 from one 'very refreshed' to three 'very tired'. Actigraphy devices were Mini-Mitter Actiwatch-2 devices (Philips Respironics, Bend, OR, USA), worn on the non-dominant hand throughout the 3 days and nights of each phase of

the study. Data were downloaded and analysed using Actiware v.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).

2.3.6 | 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 who died. The 'neutral' clips were different segments of a scenic drive from the film '*Gerry*' (2002). Each film clip ran for 1.5–2 min 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 mean = 11.58, SD = 4.55, and to the sad clips mean = 9.35, SD = 3.44; Student's paired *t* test $t = 3.86$, $P < .001$. The NA response to the neutral clips was mean = 9.50, SD = 4.77, and to the sad clips mean = 12.72, SD = 4.50; Student's paired *t* test $t = -4.68$, $P < 0.001$.

2.3.7 | Acquisition of physiological data

Physiological data were acquired at 1 kHz, using a BIOPAC base unit (MP150-BIOPAC Systems Inc., Goleta, CA, USA) 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), and skin conductance was acquired with Ag/AgCl electrodes place on the second and fourth digits of the left hand. Data were acquired and analysed using AcqKnowledge v.4.0 software, and a transistor–transistor logic (TTL) digital signal, presented via Inquisit v.3.0, marked the onset and offset of baseline and film presentation periods. After manual removal of artefacts and application of a 1 Hz lowpass filter, three measures were derived: HR (in beats per minute), derived from the R–R interval, HRV with a combination of time-domain and frequency-domain measurements. Time-domain indices of HRV quantify the variability in interbeat 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 (HF) band (0.15–0.4 Hz), which reflects parasympathetic nerve activity, and a low-frequency (LF) band (0.04–0.15 Hz), which relates to both sympathetic and parasympathetic tone. Given that the time frame for detecting change in the LF component is longer than the 1–2 min 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 v 4.0 built-in algorithms: (i) the 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); and (ii) the frequency-domain method, where the power spectrum density is estimated for the R–R interval series, using fast Fourier transformation. The power in the HF band, and the ratio of LF to HF are reported.

Skin conductance measures fluctuations in electrical properties of the skin attributable to sweat secretion and reflects sympathetic drive, because 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 50 Hz bandpass filter. Measures were the average SCL and the number of SCR events (# SCRs), identified as transient increases in SCL of >0.05 μS within the period measured (baseline or film clip). After quality control, data extraction was conducted by research assistants blinded to the study protocol. All measurements were normalized to the baseline period before the presentation of each clip (see Statistics section).

2.4 | Procedure

After signing informed consent, participants completed the demographic and PSQI questionnaires. Participants were then given actigraphy devices and sleep diaries and were instructed which schedule to maintain before their subsequent visits, three consecutive nights of 5 h in bed (sleep restriction, SR), and three consecutive nights of ≥8 h 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 in the laboratory. To allow physiological signals to stabilize before beginning data 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 film clip condition was identical: an initial 30 s when participants were asked to focus on a plus symbol ('+') to obtain baseline measurements; presentation of the video clip; and responses to each film clip assessed with the VASs and 10 items from the PANAS. Between film clip periods, participants played computer games for ~7 min.

2.5 | Statistics

Statistical analyses were performed in IBM SPSS Statistics for Macintosh, Version 24.0. 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, Student's one-sample *t* tests were

TABLE 1 Descriptive statistics of actigraphy and sleep diary data

Parameter	Well rested (mean [SD])	Sleep restriction (mean [SD])	Paired *t*
Bedtime (h:min)	10:00 [11:04]	3:56 [7:01]	2.293*
Wake up (h:min)	8:37 [1:04]	7:09 [0:57]	7.241**
TIB (h:min)	8:23 [1:02]	5:52 [0:53]	9.014**
TST (h: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**
No. of 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**

Abbreviations: PSQI, Pittsburgh Sleep Quality Index; SE, sleep efficiency; SOL, sleep onset latency; STAI-S, State and Trait Anxiety Index, State subscale; TIB, time in bed; TST, total sleep time; and WASO, wake after sleep onset. Feeling refreshed from sleep was obtained from diary reports. Student's paired *t* test:
*$P < 0.05$;
**$P < 0.001$.

TABLE 2 Responses on the visual analog scales (VAS) and to positive and negative affect schedule (PANAS)

Condition	Film clip	VAS fatigue	VAS sadness
Well rested	Neutral	27.74 [25.78]*	15.48 [22.19]†
	Sad	24.19 [24.60]*	71.94 [25.49]
Sleep restricted	Neutral	75.48 [22.78]*†	12.94 [16.55]†
	Sad	48.06 [31.67]	74.19 [26.05]
		Positive affect	**Negative affect**
Well rested	Neutral	11.58 [3.95]	9.12 [4.08]†
	Sad	10.48 [3.51]	11.48 [3.00]
Sleep restricted	Neutral	10.82 [4.39]	9.21 [4.07]†
	Sad	10.00 [3.06]	11.45 [3.99]

Values represent means [SD] of the participant's response collected immediately after watching each film clip.
*Significant difference between well-rested and sleep-restricted condition.
†Significant difference between neutral and sad clips.

conducted on change values, with hypothesized means of one for HR, SDSD, HF, LF/HF and SCL, and zero for SCRs. To test the effects of sleep restriction and emotion induction on the different dependent variables, repeated-measures ANOVAs were used.

3 | RESULTS

Descriptive statistics and actigraphy results are reported in Table 1. As can be seen, the majority of the sample (87%) was 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 TIB and 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 with the WR 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 ($F_{1,30} = 45{,}33$, $P < 0.001$, partial $\eta^2 = 0.602$), a main effect of emotion condition ($F_{1,30} = 21.20$, $P < 0.001$, partial $\eta^2 = 0.414$) and a significant interaction ($F_{1,30} = 10.11$, $P = 0.004$, partial $\eta^2 = 0.250$). *Post hoc* analysis revealed significantly lower fatigue ratings after viewing the sad clip in the SR condition ($F_{1,30} = 30.95$, $P < 0.001$, partial $\eta^2 = 0.508$), but no difference in the WR condition ($F_{1,30} < 1.0$). For the sadness VAS, there was a main effect of emotion induction ($F_{1,30} = 155.51$, $P < 0.001$, partial $\eta^2 = 0.916$), but no effect of sleep condition nor any interaction ($Fs < 1.0$). There was a main effect of emotion induction condition for PANAS-NA ($F_{1,30} = 9.40$, $P = 0.004$, partial $\eta^2 = 0.227$), with no effect of SR 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 initially analysed the averaged 30 s baselines of the two film clips and performed Student's paired *t* tests on the different physiological measures to compare the SR and WR conditions. Heart rate was higher in the WR (mean = 81.08 beats min^{-1}, SD = 15.78 beats min^{-1}) compared with the SR condition (mean = 77.25 beats min^{-1}, SD = 11.89 beats min^{-1}, $P = 0.042$), and SCR rate was marginally higher in the WR (mean = 3.01, SD = 1.21) compared with the SR condition (mean = 2.53, SD = 1.01, $P = 0.053$). All other baseline measurements did not differ between the sleep schedule conditions (*P*-values > 0.050, data not shown).

To determine whether presentation of film clips induced significant changes in physiological measures, change values for each physiological measure were calculated, and Student's 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 in the SR condition, SDSD during the neutral clips in both SR and WR conditions, and SDSD during the sad clip in the WR 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 ($F_{1,29} = 7.31$, $P = 0.011$, partial $\eta^2 = 0.201$), with greater attenuation in HR, relative to baseline, while watching the sad film clip compared with the neutral clip, with no main effect of sleep schedule and no interaction ($Fs < 1.0$). For SDSD (Figure 1b), there was a main effect of sleep schedule ($F_{1,29} = 5.298$, $P = 0.029$, partial $\eta^2 = 0.154$) owing to a greater increase from baseline in the SR condition, with no effect of emotion induction and no interaction ($Fs < 1.0$). For HF (Figure 1c), there was a significant interaction of emotion induction and sleep condition ($F_{1,30} = 5.285$, $P = 0.029$, partial $\eta^2 = 0.150$), with no main effect of sleep condition ($F_{1,30} = 2.284$, $P = .141$) or

TABLE 3 Physiological responses to neutral and sad film clips

	Well rested		Sleep restricted	
Parameter	Neutral film clip	Sad film clip	Neutral film clip	Sad film clip
HR	0.981 [0.04]‡	0.960 [0.06]‡	0.988 [0.04]	0.963 [0.05]‡
SDSD	0.965 [0.05]	0.972 [0.06]	1.163 [0.09]‡	1.054 [1.00]
HF	1.386 [0.71]*†‡	1.859 [1.44]‡	2.238 [2.24]‡	1.730 [1.25]‡
LF/HF	2.979 [2.39]†‡	1.873 [1.19]*‡	3.165 [1.87]‡	3.235 [2.64]‡
SCL	0.896 [0.14]‡	0.954 [0.16]‡	0.927 [0.14]‡	0.939 [0.15]‡
No. of SCRs	2.063 [1.05]*†‡	2.251 [1.23]‡	2.637 [1.48]‡	2.305 [1.22]‡

Values represent means [SD] of change from baseline. Abbreviations: HF, high frequency; HR, heart rate (in beats per minute); LF, low frequency; SCL, skin conductance level; SCR, skin conductance response rate (per minute analysed); SDSD, standard deviation of successive R–R interval differences.

*Significant difference between well-rested and sleep-restricted condition.

†Significant difference between neutral and sad clips.

‡Significant change from baseline.

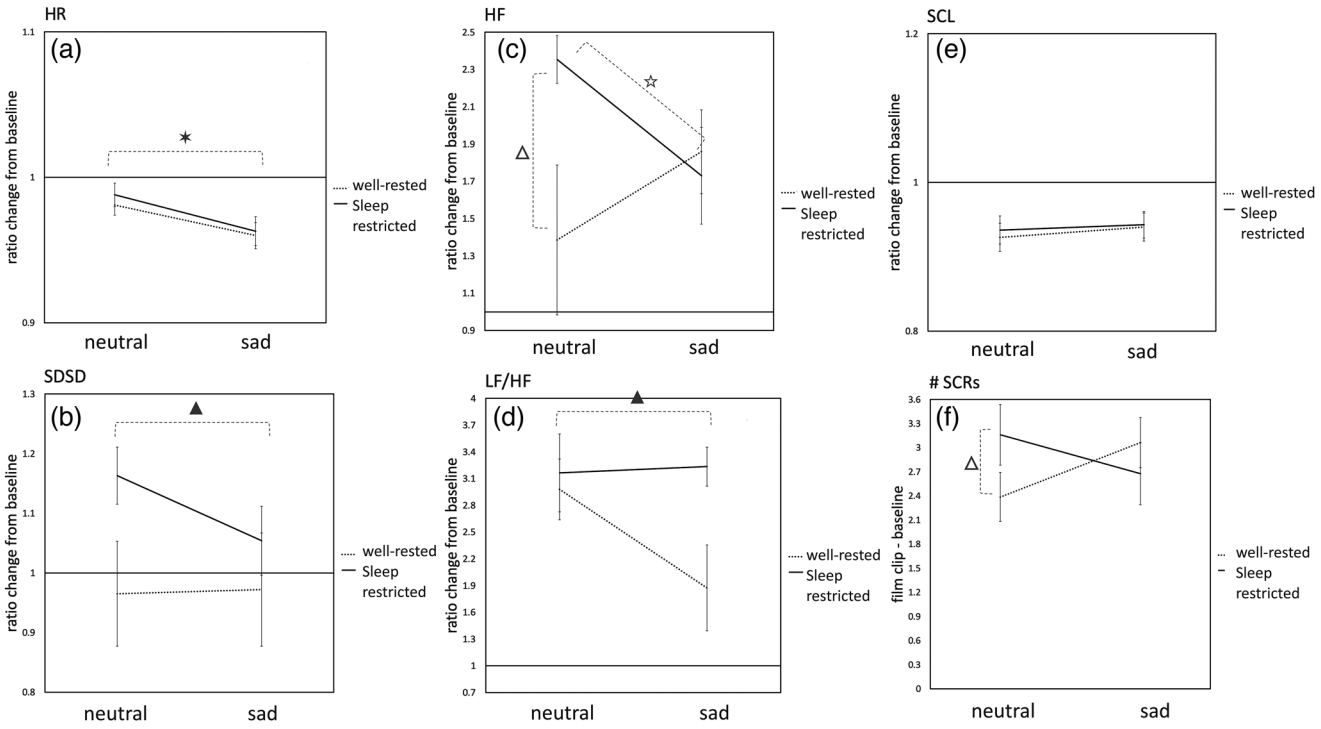

FIGURE 1 Effects of sleep schedule and mood induction on physiological measures. (a) Heart rate (HR). (b) Standard deviation of successive R–R interval differences (SDSD). (c) High frequency (HF). (d) Low frequency/high frequency (LF/HF) ratio. (e) Skin conductance level (SCL). (f) Skin conductance response rate (per minute analysed; # SCRs). Except for SCR, all values represent the quotient of baseline, obtained before 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; continuous lines are measurements obtained in the sleep-restricted condition. Filled star, main effect of emotion; open star, *post hoc* effect of emotion; filled triangle, main effect of sleep; and open triangle, *post hoc* effect of sleep

emotion induction ($F < 1.0$). *Post hoc* analysis revealed significantly lower HF during the sad *versus* neutral film clip in the WR condition ($P = 0.046$), but significantly higher HF in response to the neutral clip in the WR *versus* SR condition ($P = 0.020$). For the LF/HF ratio (Figure 1d), there was a main effect of sleep condition ($F_{1,30} = 5.016$, $P = 0.033$, partial $\eta^2 = 0.147$), with a greater increase from baseline in the SR condition. There was no main effect of emotion induction ($F_{1,30} = 2.575$, $P = 0.119$) and no interaction ($F_{1,30} = 2.253$, $P = 0.144$). For SCL (Figure 1e), there were no main effects or interactions ($Fs < 1.0$). For SCR rate (Figure 1f), there was a significant interaction of sleep condition with emotion induction ($F_{1,28} = 4.843$, $P = 0.036$, partial $\eta^2 = 0.139$), and no significant main effects ($Fs <= 1.0$). These were attributable to more SCRs in the SR neutral movie clip condition compared with the WR neutral condition ($P = 0.043$).

4 | 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 prediction that sleep restriction would enhance self-reported and physiological responses to a sad film clip was not supported, but our results provide a different perspective on the effects of sleep restriction on emotion. In a nutshell, we found a dissociation between emotion induction effects on HR and SR effects on HRV and skin conductance, which we interpret to represent an adaptation of the ANS to sleep restriction.

In contrast to other studies (e.g. Dinges et al., 1997; Franzen et al., 2009; Talbot et al., 2010), sleep deprivation did not amplify self-reported negative affect ratings to the sad film clip. This unexpected finding is probably because in most other studies negative affect was measured generally and not in response to a discrete cue. Consistently, in the present study, state anxiety increased significantly in the SR condition commensurate with a more general effect of insufficient sleep on mood. That being said, Zohar et al. (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.

Given that 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 might have similar effects, i.e. increased sympathetic and diminished parasympathetic drive (Meerlo et al., 2008; Tobaldini, Pecis, & Montano, 2014). Thus, we expected that SR 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 HR decreased in the SR condition, with only a marginal increase in SCR rate, and other baseline measures of sympathetic activity (e.g. LF/HF and SDSD) were not impacted by SR. Again, a direct comparison with other studies is problematic because baseline measurements in the present study constituted a total of 1 min, which might not be sufficient to detect state-level changes. Furthermore, this baseline was obtained while participants were instructed to focus on a single point on the computer monitor, which was intended to be emotionally neutral, but might not be a true baseline.

A reduction in HR was observed while participants watched both sad and neutral film clips, a response more robust during the sad clip but unaffected by SR. In the WR condition, this sad clip-related attenuation in HR 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 (e.g. Cheung & Porges, 1977; Porges, 2007; Porges & Coles, 1982; Porges & Raskin, 1969; Walter & Porges, 1976), who demonstrated that brief attentional demand causes HR deceleration, whereas 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 alternative explanation is that the pattern of physiological responses in the WR 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 the present 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 might 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 SR condition compared with the WR condition. In contrast, HF was higher in the SR condition only when watching the neutral clip. This pattern suggests that during sleep restriction, the sad emotion induction resulted in a smaller response in both the LF and the HF components of the HRV, leading to similar LF/HF values for the neutral and sad clips. Although LF is no longer believed to represent sympathetic activity *per se* (Goldstein, Bentho, Park, & Sharabi, 2011; Hayano & Yuda, 2019; Kreibig, 2010), involvement of the sympathetic system is a potential explanation of the finding and is in line with the reduction in SCRs observed in the SR, 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; however, it has been found that fluctuations in the two bands might be on different time scales (Shaffer & Ginsberg, 2017), thereby temporally uncoupling the responses of the two measures. This intriguing pattern has also been reported after steady-state exercise in healthy participants (Grant & Ker, 2008; Maud & Foster, 2006: p. 52; Povea et al., 2005), suggesting that the combination of sleep restriction and an emotional challenge induces an adaptive response to maintain a constant HR response across WR and SR conditions. This conjecture is also consistent with the decreased self-reported fatigue when watching the sad film clip in the SR condition.

Clearly, these findings require further investigation, and several limitations of the present 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 should 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 should be

acknowledged. Finally, as noted above, the laboratory environment, although providing a controlled setting, diminishes the real-life 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 findings are in line with the notion that ANS functioning is altered by sleep restriction, but might not be in line with the hypothesized increase in sympathetic and decrease in 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 ANS in an adaptive manner, in order to adjust heart rate and perhaps the cognitive–emotional experience, to attain 'normal' responses to emotional cues, despite increased fatigue. Additional research is required to confirm this theory and examine the potential cost of this adaptation over time.

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COMPETING INTERESTS

None declared.

AUTHOR CONTRIBUTIONS

Both authors were involved in conception and design of the project. I.S.H. was the primary contributor to data acquisition, analysis and interpretation and to drafting and revising the manuscript. Both authors approved the final version of the manuscript and agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. Both persons designated as authors qualify for authorship, and all those who qualify for authorship are listed.

DATA AVAILABILITY STATEMENT

Given that data were collected and analysed in Hebrew, right-to-left systems, the data that support the findings of this study are available from the corresponding author upon reasonable request.

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REFERENCES

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*, 266-270.

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

Ben-Zur, H. (2002). Coping, affect and aging: The roles of mastery and self-esteem. *Personality and Individual Differences*, *32*, 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*, 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*, 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*, 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*, 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*, 300-305.

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, 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*, 1255-1261.

Goldstein, A. N., & Walker, M. P. (2014). The role of sleep in emotional brain function. *Annual Review of Clinical Psychology*, *10*, 679-708.

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

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

Hayano, J., & Yuda, E. (2019). Pitfalls of assessment of autonomic function by heart rate variability. *Journal of Physiological Anthropology*, *38*, 3.

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*, 1173-1175.

Koninklijke Philips N.V. (2019). *PHILIPS global sleep survey. 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

Kreibig, S. D. (2010). Autonomic nervous system activity in emotion: A review. *Biological Psychology*, *84*, 394-421.

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*, e12665.

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

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*, 193.

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

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*, 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*, 197-210.

Minkel, J., Htaik, O., Banks, S., & Dinges, D. (2011). Emotional expressiveness in sleep-deprived healthy adults. *Behavioral Sleep Medicine*, *9*, 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*, 294-302.

National Sleep Foundation. (2013). 2013 International bedroom poll. https://www.sleepfoundation.org/professionals/sleep-americar-polls/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*, 398-406.

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

Porges, S. W. (2007). The polyvagal perspective. *Biological Psychology*, *74*, 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*, 215-220.

Porges, S. W., & Raskin, D. C. (1969). Respiratory and heart rate components of attention. *Journal of Experimental Psychology*, *81*, 497-503.

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*, 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. *European Journal of Internal Medicine*, *24*, 664–670.

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*, 853–856.

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*, 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*, 831-841.

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.

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*, 747-756.

Tobaldini, E., Pecis, M., & Montano, N. (2014). Effects of acute and chronic sleep deprivation on cardiovascular regulation. *Archives Italiennes de Biologie*, *152*, 103–110.

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*, 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*, 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*, 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*, 557-580.

Wright, K. P. Jr, 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*, 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*, 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*, 47-54.

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