Psychoneuroendocrinology 81 (2017) 151–156

Contents lists available at ScienceDirect

Psychoneuroendocrinology

journal homepage: www.elsevier.com/locate/psyneuen

Poor habitual sleep efficiency is associated with increased cardiovascular and cortisol stress reactivity in men☆

Stijn A.A. Massar[a], Jean C.J. Liu[a,b], Nabilah B. Mohammad[a], Michael W.L. Chee[a,*]

[a] Centre for Cognitive Neuroscience, Duke-NUS Medical School, Singapore
[b] Division of Psychology, Yale-NUS College, Singapore

ARTICLE INFO

Keywords:
Habitual sleep
Actigraphy
Sleep efficiency
Psychosocial stress
Cortisol
Cardiovascular reactivity

ABSTRACT

Inadequate sleep and psychological stress can both elevate physiological stress markers, such as cortisol. Prior studies that have applied induced psychosocial stress after a night of experimental sleep deprivation have found these effects to be compounded. We examined whether the relationship between stress reactivity and poor sleep also extends to habitual sleep patterns. Fifty-nine adult male participants were recruited. Habitual sleep patterns were monitored with actigraphy for a week. Participants subsequently underwent the Trier Social Stress Test. Cardiovascular responses and salivary cortisol were measured at baseline, during stress, and during recovery. Subjects who showed poor habitual sleep efficiency during the week before stress induction responded with higher stress-related elevations of blood pressure and cortisol levels as compared to subjects with high sleep efficiency. This relationship between poor sleep efficiency and elevated blood pressure persisted during the post-stress recovery period. Similar associations between total sleep time in the week prior to the stress induction and physiological reactivity did not reach significance. Our findings indicate that habitual low sleep efficiency exaggerates cardiovascular and neuroendocrine effects of psychosocial stress, in a male population.

1. Introduction

Inadequate sleep and stress both have negative impact on health, increasing the risk for negative outcomes such as cardiovascular disease, diabetes and depression (Cappuccio et al., 2011; Cappuccio et al., 2010; Hamer et al., 2010; Hamer and Steptoe, 2012; Maglione et al., 2014; Susman et al., 1997). Moreover, the effects of inadequate sleep are known to potentiate the physiological effects of stress. Laboratory studies on the acute effects of total sleep deprivation (TSD) have found that TSD affects physiological markers of stress, elevating blood pressure (BP; Kato et al., 2000), bringing about immune suppression, and altered neuroendocrine function (Dinges et al., 1995; Spiegel et al., 1999; Wright et al., 2015). TSD also elevates evening cortisol levels, reflecting altered regulation of the hypothalamus-pituitary-adrenal (HPA) axis (Spiegel et al., 1999; Vgontzas et al., 1999). Over and above these effects on baseline physiology, sleep deprivation also exaggerates physiological responses to particular stressors. When exposed to stress induction, sleep deprived participants show greater physiological reactivity (i.e. increased BP, increased skin conductance levels and elevated cortisol responses) than those who had a night of normal sleep (Franzen et al., 2011; Liu et al., 2015; Minkel et al., 2014).

While experiments involving a night of total sleep deprivation on stress reactivity are important in uncovering the causal relationship of these effects, most persons are not regularly exposed to this type of sleep loss. Moreover, the impact of poor sleep on stress physiology may be particularly detrimental when patterns of inadequate sleep are experienced for longer periods of time. Long-term laboratory studies on the effects of short sleep are logistically difficult and expensive to undertake but studies examining the consequences of adverse habitual sleep patterns can be informative.

Several studies have investigated the association between habitual sleep patterns and stress reactivity in developmental contexts. In these studies, children and adolescents who report poor habitual sleep or have actigraphically verified low sleep efficiency, show higher cortisol elevations following stress induction (Mrug et al., 2016; Pesonen et al., 2012; Räikkönen et al., 2010), with some exceptions (Capaldi et al., 2005). As developmental changes occur both in sleep patterns and stress physiology during adolescence (Carskadon et al., 1998; Gunnar et al., 2009), it is important to investigate if these findings generalize to adults. The few studies of this type performed in adults have yielded inconclusive findings (Bassett et al., 2015; Mezick et al., 2014; Wright

☆ This work was performed at Duke-NUS Medical School Singapore.

* Corresponding author: Duke-NUS Medical School, 8 College Road, 169857, Singapore.
E-mail address: michael.chee@duke-nus.edu.sg (M.W.L. Chee).

http://dx.doi.org/10.1016/j.psyneuen.2017.04.013
Received 20 October 2016; Received in revised form 16 March 2017; Accepted 21 April 2017

et al., 2007). Possible reasons for this include the use of non-optimal stress induction procedures (Wright et al., 2007), the use of different physiological outcome measures (BP, cortisol, etc.; Mezick et al., 2014) or not objectively measuring habitual sleep (Bassett et al., 2015).

In the current study we examined the association between habitual sleep, as measured by one week of actigraphy, and stress reactivity, induced by a strong stress procedure (Trier Social Stress Test; Kirschbaum et al., 1993). In order to examine the consistency between different physiological outcome measures, we measured both cardiovascular (BP, heart rate) and HPA reactivity (salivary cortisol). In light of prior total sleep deprivation studies, and studies on habitual sleep in adolescents and children, we hypothesized that indices of poor sleep (i.e. short sleep duration, low sleep efficiency) would be associated with exaggerated reactivity to stress (Mezick et al., 2014).

2. Methods

2.1. Participants & procedure

Fifty-nine male volunteers were recruited from the university population (mean age [sd] = 22.83 [2.49]). Initial selection based on a web-based questionnaire. In order to obtain a sufficient numbers of persons with short and longer habitual sleep durations, approximately equal numbers of persons who slept 7–8 h and less than 6 h per night, were enrolled from a larger sample of screened participants (Mezick et al., 2014). Habitual sleeping patterns were then objectively measured using wrist actigraphy (ActiWatch2; Philips Respironics, Andover, MA, USA). Analyses were based on these objective recordings of habitual sleep. To minimize variance in physiological stress responsiveness, the sample was restricted to male subjects of Chinese ethnicity. Participants involved in the study had to be free from a history of psychiatric, neurological or sleep disorders, not take long-term medication, and not be on prescription medicines during the whole of the sleep monitoring period. Furthermore habitual sleep patterns should be restricted to nocturnal sleep. Participants were therefore not allowed to work night shifts or expose themselves to bouts of total sleep deprivation during the period of sleep monitoring.

Habitual sleep was monitored at home through wrist actigraphy for 7 consecutive days. Participants were instructed to follow their natural sleep/wake schedules with the restriction that sleep should be confined to nocturnal sleep (i.e. refrain from day time naps and from nights of total sleep deprivation). Actigraphy data were visually inspected and sleep intervals were corrected based on light sensor and sleep diary data (i.e. if clear changes in light intensity and activity coincided, bedtime and wake time were scored accordingly. In cases where this was less clear, sleep diary data were used as guide). Total sleep time (TST), total time in bed (TIB), and sleep efficiency (SE = TST/TIB × 100%) determined as the average across the 7 days using Actiware Version 6.0.0 software (Philips Respironics, Andover, MA, USA). Prior to the sleep monitoring period participants filled out the Pittsburg Sleep Quality Inventory (PSQI; Buysse et al., 1989), which is 24-item questionnaire measuring subjective sleep quality over the past month. A global summary score (ranging from 0 to 21) is good (low scores) and poor (high score) habitual sleepers.

Following this period they came in to the lab where they were subjected to an experimental stress induction. Upon arrival, participants rested in a seated position for about one hour before commencing the stress procedure. Participants were instructed to abstain from caffeine, nicotine, and alcohol a day prior to the visit and to refrain from eating for at least 1 h before the visit. The procedure was approved by the National University Singapore Institutional Review Board (IRB). Participants signed informed consent upon briefing in the first session Fig. 1.

2.2. Stress procedure

Participants were exposed to the Trier Social Stress Test (TSST), a widely used experimental paradigm for eliciting moderate psychosocial stress and inducing activation of the HPA axis stress responses (Kirschbaum et al., 1993). The stress protocol comprised an anticipation phase (5 min) where participants were instructed to prepare a speech, and a test phase in which participants had to deliver the speech (5 min) followed by a mental arithmetic test (5 min). The speech was delivered in front of a stern, two-person panel, consisting of one male and one female. Participants were told that the speech would be video-recorded for subsequent behavioral analysis and that panel members are trained behavioral analysts who will be taking notes during the interview. The stress procedure was preceded and followed by 5-min of task-free baseline and recovery periods, during which heart rate, blood pressure and salivary cortisol were measured. These were collected with the participant in isolation. Throughout the baseline, stress and recovery phases the participant was seated in comfortable chair with both arms rested on a desk. Although the procedure was slightly modified from the original TSST (i.e. two panel members instead of three; one baseline physiology measurement instead of two), these variations of the protocol are quite common throughout the literature and are shown to produce desired stress effects (e.g. Bershad et al., 2015; Creswell et al., 2014).

2.3. Cardiovascular measurement

Heart Rate (HR) was monitored continuously using electrocardiography (ECG; Grass Technologies, Natus Neurology, Warwick, RI, USA). Average HR was calculated over each of the 5-min stress phases (baseline, anticipation, speech, arithmetic, recovery). Blood pressure was measured using a blood pressure cuff around the left arm (Dinamap, GE Healthcare, Milwaukee, MI, USA). The BP cuff inflated automatically at 1-min intervals, resulting in five measurements per stress phase. Average values for systolic BP, diastolic BP and mean arterial pressure (MAP) were calculated for each stress phase. ECG electrodes and BP cuff were placed by a research assistant, prior to the baseline phase and were removed after the recovery period.

2.4. Salivary cortisol measurement

Saliva samples for cortisol determination were collected during the pre-stress baseline phase (T = 0-min), during the post-stress recovery phase (T = +15-min) and at three more post-stress follow-ups (+30 min, +60-min, +90-min), using Salivette cotton swabs (Sarstedt AG & Co., Nümbrecht, Germany). Samples were centrifuged and stored at −80 °C. Prior to further analysis samples were thawed and centrifuged. Competitive enzyme immuno-assay (ELISA, NovaTec Immunodiagnostica GmbH, Dietzenbach, Germany) was used to determine cortisol concentrations. Mean intra and inter-assay CV's were 13.6% and 15.5% respectively with a range of 0.05–10 μg/dl. Concentration values were log transformed to correct for skewness of the distribution.

2.5. Statistical analysis

One subject did not produce sufficient saliva for cortisol measurement. Three participants had missing blood pressure data and four participants had missing HR data due to technical error. These subjects were excluded from the respective analyses. Initial analyses of stress related changes in cortisol, HR and BP were performed by repeated measures ANOVA. Cortisol response was analyzed using ANOVA with time as within-subjects factor (T0, +15, +30, +60, +90). HR and BP were analyzed separately using ANOVA with stress phase (baseline, anticipation, speech, arithmetic, recovery) as within-subjects factor.

The main aim of the study was to investigate whether actigraphy

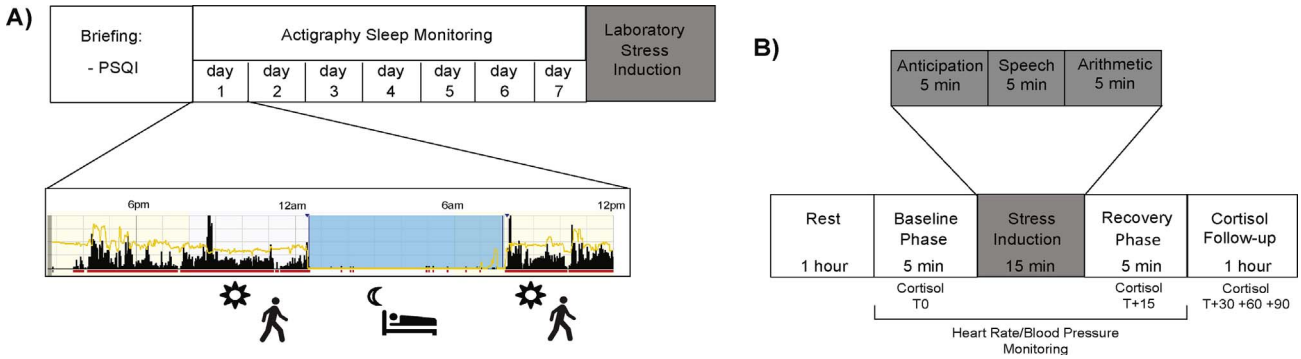

Fig. 1. A) Schematic of study procedures, B) schematic of stress induction procedures.

sleep metrics were associated with stress reactivity. Stress reactivity indices were calculated for all outcome measures. For cortisol, baseline corrected area under the response curve (Area Under the Curve – with respect to increment: AUCi; Pruessner et al., 2003) was calculated. A heart rate reactivity index was defined as the average HR during all stress phases (anticipation, speech, arithmetic) minus mean HR during baseline (Mezick et al., 2014). Blood pressure reactivity was calculated similarly for all BP metrics (systolic BP, diastolic BP, mean arterial pressure) separately. In a similar way, recovery indices were calculated for HR and BP as the mean scores during the 5-min recovery period directly following stress minus the mean baseline scores. One participant with extremely high (> 3× standard deviation above mean) BP reactivity scores was excluded from further BP analysis as outlier. The association between sleep metric and stress reactivity was determined via regression analyses using stress reactivity indices as dependent variables and sleep efficiency or total sleep time as regressors of interest. Subject age, wake time and experiment start time were included as control regressors. For all dependent and independent variables, the Kolmogorov-Smirnov test indicated no significant violations of normality (all p's > 0.05).

3. Results

3.1. Sample characteristics

By design, participants were exclusively males of Chinese ethnicity. They aged between 20–33 years. Resting physiological measurements taken during the baseline before the start of the stress induction indicated no clinically significant elevation of blood pressure and normal levels of salivary cortisol (Table 1). Furthermore, physiological measures at baseline were not correlated with habitual sleep metrics total sleep time (TST: all p's > 0.05) and sleep efficiency (SE: all p's > 0.05).

3.2. Sleep metrics

Time-in-Bed (TIB), Total-Sleep-Time (TST) and Sleep Efficiency (SE) were extracted from actigraphic recordings over the seven nights prior to the stress induction session. Average TIB was 6:48 (hours:minutes) per night (stdev = 59 min), average TST was 5:25 (hours:minutes; stdev = 54 min), average SE was 79.8% (stdev = 6.4%). TIB and TST (but not SE) significantly correlated with self-reported sleep duration and PSQI global score, collected prior to the actigraphy sleep monitoring period (see also Supplemental Materials).

Table 1
Baseline Physiology.

		Correlations	
	Mean (*SD*)	TST	SE
HR (bpm)	64.5 (8.23)	0.13 (n.s.)	−0.01 (n.s.)
BP systolic (mmHg)	109.5 (7.85)	0.05 (n.s.)	−0.09 (n.s.)
BP diastolic (mmHg)	64.5 (6.71)	0.05 (n.s.)	−0.11 (n.s.)
Mean arterial pressure (mmHg)	81.0 (6.77)	0.04 (n.s.)	−0.1 (n.s.)
Salivary cortisol (ng/ml)	6.0 (3.12)	0.05 (n.s.)	0.16 (n.s.)

HR = Heart Rate, BP = Blood Pressure, TST = Total Sleep Time, SE = Sleep Efficiency.

3.3. Stress reactivity

All physiological measures showed significant elevations due to the stress induction (See Fig. 2; Stress main effect: Cortisol $F(4, 228) = 41.62$, $p < 0.0001$; $B_{systolic}$ $F(4, 216) = 247.9$, $p < 0.0001$; $BP_{diastolic}$ $F(4, 220) = 217.5$, $p < 0.0001$; $BP_{MeanArterialPressure}$: $F(4, 220) = 318.6$, $p < 0.0001$; HR $F(4, 216) = 118.0$, $p < 0.001$). Planned contrast showed that HR returned to baseline, but BP remained elevated during the recovery period directly following the termination of the stress induction (all p's < 0.0001).

3.4. Associations between sleep metrics and stress reactivity

The relationship between sleep metrics and stress reactivity indices was analyzed via multiple regression analysis. Negative regression weights indicated that low sleep efficiency was associated with higher cortisol and blood pressure responses to stress (Table 2). Furthermore, low sleep efficiency was associated with blood pressure recovery index, indicating that those participants who had poor habitual sleep efficiency still had elevated blood pressure during the recovery period, while blood pressure returned to baseline for those participants who had high sleep efficiency (see Fig. 3). No significant associations between heart rate reactivity/recovery and sleep metrics were found. Associations between sleep efficiency and stress reactivity indices are illustrated in Fig. 3.

Regression analyses with total sleep time as the regressor of interest showed trends towards the same relationships between sleep and stress, however these models did not reach significance (Table 2).

4. Discussion

The current findings demonstrate that poor sleep efficiency is associated with exaggerated reactivity to acute psychosocial stress. In our sample of young adult male participants, cortisol reactivity was inversely correlated with habitual sleep efficiency, objectively measured through seven days of actigraphy. This finding concurs with data from a sample of children (Räikkönen et al., 2010), in which individuals with poor actigraphically recorded sleep efficiency showed increased HPA-axis responses to an age-appropriate version of the Trier Social Stress Test. Furthermore, in parallel with the cortisol findings, blood pressure reactivity during stress was negatively correlated with sleep efficiency. Moreover, this association remained present during the recovery phase when the stressor was already terminated. Taken together, the current findings indicate that poor sleep efficiency exacerbates the effects of acute psychosocial stress on HPA-axis and

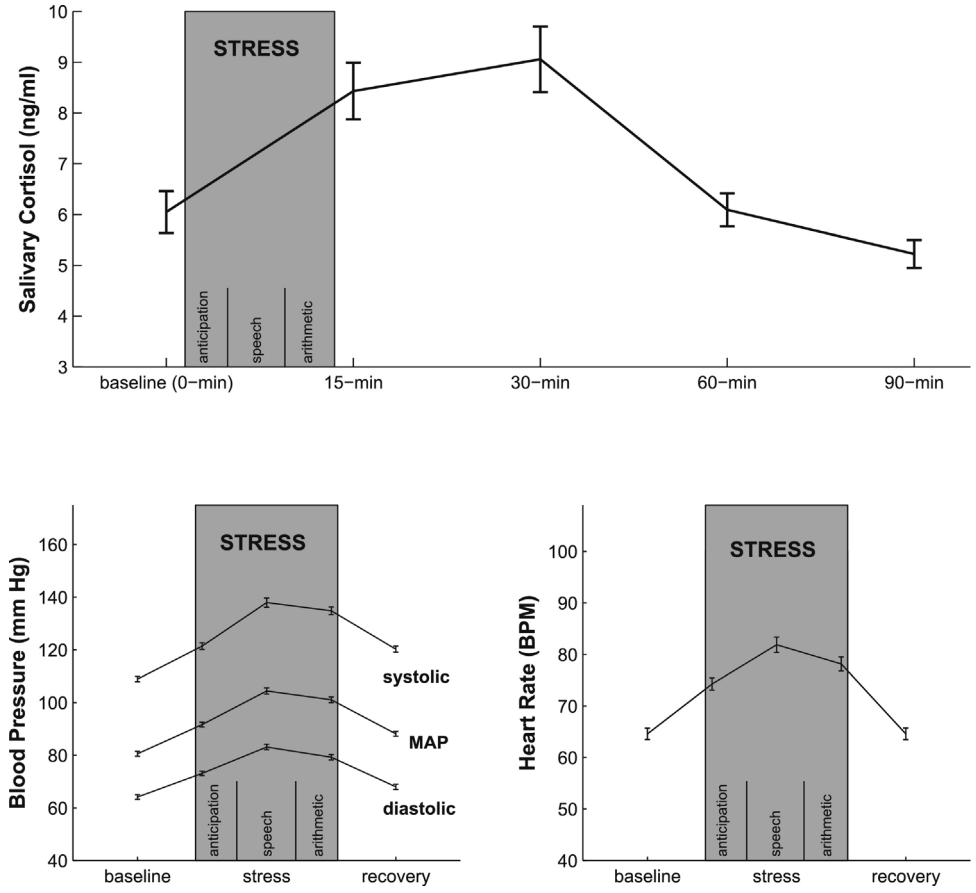

Fig. 2. Physiological reactivity during the stress protocol for salivary cortisol (top panel), systolic and diastolic blood pressure and mean arterial pressure: MAP (bottom left), and heart rate (bottom right).

cardiovascular reactivity.

Although, physiological responses to stressful situations may have short-term adaptive value, prolonged or repeated exposure to stress may result in physiological changes that ultimately lead to adverse health outcomes (McEwen, 2005). By potentiating the effects of acute stress, poor sleep efficiency may hasten the development of cardiovascular and metabolic diseases. This interaction between stress and poor sleep may therefore represent a particular risk factor contributing to conditions that are associated with stress and poor sleep separately (e.g. cardiovascular disease, diabetes, depression; Cappuccio et al., 2011; Cappuccio et al., 2010; Hamer et al., 2010; Hamer and Steptoe, 2012; Maglione et al., 2014; Susman et al., 1997).

It should be noted that in our data, the relationship between stress and sleep was present for sleep efficiency but not for sleep duration. This seems to be somewhat at odds with epidemiological findings that link abnormal sleep duration to negative health outcomes. Moreover, a significant negative associations between BP stress reactivity and habitual sleep duration (one week of actigraphically measured total sleep time) have been reported in a previous study (Mezick et al., 2014). In the current data, regression analyses using total sleep time as a predictor yielded associations that trended in the expected direction but which did not reach significance.

A recent study demonstrated that experimental manipulation of sleep duration and sleep fragmentation have similar negative effects on mood, but these effects were significantly stronger for sleep fragmentation than for restricted sleep without fragmentation (Finan et al., 1995). Therefore, an association between sleep duration and physiological stress reactivity is suggested, albeit with smaller effect size.

On the other hand however, a similar dissociation between sleep duration and sleep efficiency has been reported in a study that included a large sample of children (Räikkönen et al., 2010). An alternative possibility therefore, could be that sleep efficiency and sleep duration have separate influences, and the influence of habitual short sleep duration on stress reactivity, in the absence of low seep efficiency, is only minimal. A growing body of literature implicates sleep fragmentation as a separate risk factor for negative health outcomes and

Table 2
Regression coefficients (β) for models with sleep metrics as main regressor and stress reactivity as dependent variable.

	Cortisol AUCi	BP reactivity			BP recovery			HR reactivity	HR recovery
Independent Variable		*Systolic*	*Diastolic*	*MAP*	*Systolic*	*Diastolic*	*MAP*		
	n = 58	n = 55	n = 55	n = 55	n = 56	n = 56	n = 56	n = 55	n = 57
Sleep Efficiency (SE)	−0.27*	−0.27*	−0.29*	−0.34**	−0.26^{+}	−0.26^{+}	−0.31*	−0.05	−0.12
Total Sleep Time (TST)	−0.22	−0.16	−0.18	−0.19	−0.22	−0.15	−0.20	−0.03	0.01

Regression models controlled for Age, Wake Time & Experiment Time. $^{+}$ $p < 0.06$, * $p < 0.05$, ** $p < 0.01$; AUCi = Area Under the Curve – with respect to increment; BP = Blood Pressure; HR = Heart Rate.

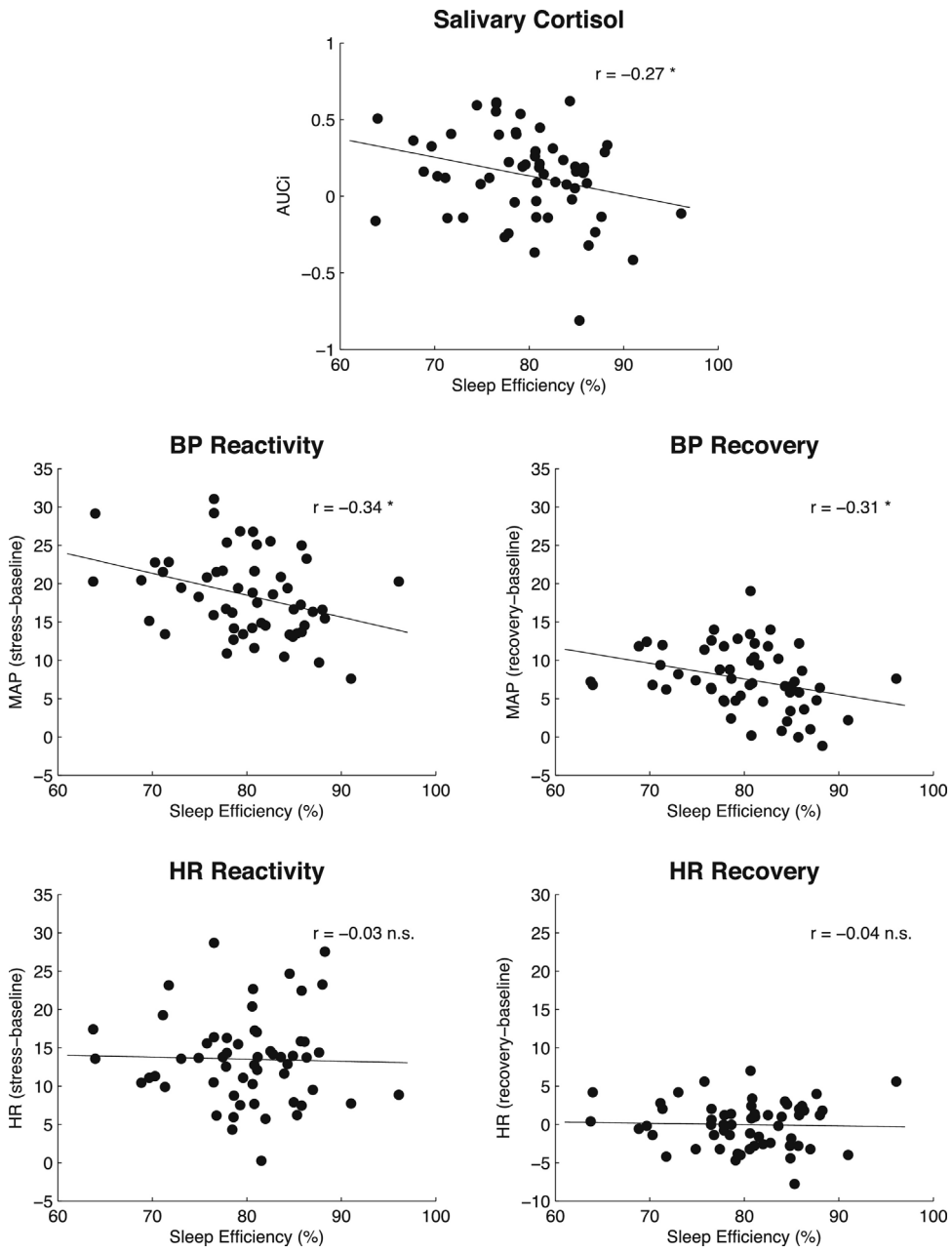

Fig. 3. Correlations between physiological stress reactivity and recovery indices and actigraphically assessed sleep efficiency (* $p < 0.05$).

behaviors (Dashti et al., 2016; Lim et al., 2016; Smagula et al., 2016).

Several limitations of the current study should be noted. Firstly, participants were sampled from a highly homogenous population (young adults, males, college students). Although this approach allowed us to study variations in sleep and stress reactivity, relatively free from moderating factors such as age, gender, and health status, a drawback is that the current findings cannot be directly generalized to a wider population (e.g. females, patients, elderly). Particularly, gender differences in the relationship between sleep and stress reactivity have been reported (Bassett et al., 2015; Mrug et al., 2016; Pesonen et al., 2012). The relationship between habitual sleep and stress reactivity is often more clearly present in males than in females. Moreover one study has reported a reversed association (lower stress reactivity after a night of low sleep efficiency) in females (Wright et al., 2007). Future studies should therefore aim to examine gender differences in the relation between habitual sleep and stress, controlling for moderating factors such as menstrual cycle (Kirschbaum et al., 1999).

A further cautionary note is that the correlational nature of the current investigation precludes strict inferences about the directionality of the found effects. Since sleep was not manipulated in this study, it cannot be concluded that poor sleep efficiency was the cause of the found increases in stress reactivity. Studies on experimental sleep deprivation have demonstrated that insufficient sleep can causally lead to exaggerated stress responses (Franzen et al., 2011; Liu et al., 2015; Minkel et al., 2014). However, a growing body of evidence suggests that reversely, stress also leads to sleep disturbances (Åkerstedt et al., 2015; Hall et al., 2015; Kalmbach et al., 2014; Petersen et al., 2013).

Finally, it should be mentioned that actigraphy provides an indirect estimate of sleep. Sleep and wake periods are inferred from relative activity profiles. Comparison of actigraphy with polysomnography (PSG), the gold standard for sleep recording, demonstrates that total sleep time is generally overestimated in elderly populations (Ancoli-Israel et al., 2003), but underestimated in adolescents and young adults (Lo et al., 2016). This may have contributed to the relatively large difference between time in bed and total sleep time as was found in the current study. Despite these known discrepancies, actigraphy is gen-

erally found to show good correlations with PSG based measures of sleep duration (Ancoli-Israel et al., 2003). Importantly, actigraphy is minimally disruptive to participants' normal sleep and wake routines, and is therefore particularly well suited to monitor natural sleep over multiple nights.

In conclusion, the current study shows that, in line with studies on experimental sleep deprivation (Franzen et al., 2011; Liu et al., 2015; Minkel et al., 2014), habitual poor sleep efficiency is associated with increased physiological reactivity to psychosocial stress, both expressed as HPA-axis responses and cardiovascular reactivity. The combined occurrence of stress and poor sleep may therefore further amplify the deleterious effects that are associated with both conditions separately.

Funding sources

This work was supported by a grant awarded to Michael Chee from the National Medical Research Council Singapore (NMRC/STaR/0015/2013) and the Far East Organization. The authors have not received any financial support from other sources.

Author contribution

SAAM, JCJL and MWLC have designed the study. SAAM, NBM and JCJL have conducted the study. SAAM has analyzed the data. SAAM, JCJL and MWLC have written the manuscript.

Acknowledgments

The authors would like to thank Vinod Khanna for assistance with data collection and analysis. This work was supported by a by a grant awarded to Michael Chee from the National Medical Research Council Singapore (NMRC/STaR/0015/2013) and the Far East Organization.

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at http://dx.doi.org/10.1016/j.psyneuen.2017.04.013.

References

Åkerstedt, T., Garefelt, J., Richter, A., Westerlund, H., Magnusson Hanson, L.L., Sverke, M., Kecklund, G., 2015. Work and sleep – a prospective study of psychosocial work factors, physical work factors and work scheduling. Sleep 38, 1129–1136.
Ancoli-Israel, S., Cole, R., Alessi, C., Chambers, M., Moorcroft, W., Pollak, C.P., 2003. The role of actigraphy in the study of sleep and circadian rhythms. Sleep 26, 342–392.
Bassett, S.M., Lupis, S.B., Gianferante, D., Rohleder, N., Wolf, J.M., 2015. Sleep quality but not sleep quantity effects on cortisol responses to acute psychosocial stress. Stress 18, 638–644.
Bershad, A.K., Jaffe, J.H., Childs, E., Wit, H.d., 2015. Opioid partial agonist buprenorphine dampens responses to psychosocial stress in humans. Psychoneuroendocrinology 51, 281–288.
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 Res. 28, 193–213.
Capaldi II, V.F., Handwerger, K., Richardson, E., Stroud, L.R., 2005. Associations between sleep and cortisol responses to stress in children and adolescents: a pilot study. Behav. Sleep Med. 3, 177–192.
Cappuccio, F.P., D'Elia, L., Strazzullo, P., Miller, M.A., 2010. Quantity and quality of sleep and incidence of type 2 diabetes: a systematic review and meta-analysis. Diabetes Care 33, 414–420.
Cappuccio, F.P., Cooper, D., D'Elia, L., Strazzullo, P., Miller, M.A., 2011. Sleep duration predicts cardiovascular outcomes: a systematic review and meta-analysis of prospective studies. Eur. Heart J. 32, 1484–1492.
Carskadon, M.A., Wolfson, A.R., Acebo, C., Tzischinsky, O., Seifer, R., 1998. Adolescent sleep patterns, circadian timing, and sleepiness at a transition to early school days. Sleep 21, 871–881.
Creswell, J.D., Pacilio, L.E., Lindsay, E.K., Brown, K.W., 2014. Brief mindfulness meditation training alters psychological and neuroendocrine responses to social evaluative stress. Psychoneuroendocrinology 44, 1–12.
Dashti, H.S., Zuurbier, L.A., de Jonge, E., Voortman, T., Jacques, P.F., Lamon-Fava, S., Scheer, F.A.J.L., Kiefte-De Jong, J.C., Hofman, A., Ordovás, J.M., Franco, O.H., Tiemeier, H., 2016. Actigraphic sleep fragmentation, efficiency and duration associate with dietary intake in the Rotterdam Study. J. Sleep Res. 25, 404–411.
Dinges, D.F., Douglas, S.D., Hamarman, S., Zaugg, L., Kapoor, S., 1995. Sleep deprivation and human immune function. Adv. Neuroimmunol. 5, 97–110.
Finan, P.H., Quartana, P.J., Smith, M.T., 1995. The effects of sleep continuity disruption on positive mood and sleep architecture in healthy adults. Sleep 38, 1735–1742.
Franzen, P.L., Gianaros, P.J., Marsland, A.L., Hall, M.H., Siegle, G.J., Dahl, R.E., Buysse, D.J., 2011. Cardiovascular reactivity to acute psychological stress following sleep deprivation. Psychosom. Med. 73, 679–682.
Gunnar, M.R., Wewerka, S., Frenn, K., Long, J.D., Griggs, C., 2009. Developmental changes in hypothalamus–pituitary–adrenal activity over the transition to adolescence: normative changes and associations with puberty. Dev. Psychopathol. 21, 69–85.
Hall, M.H., Casement, M.D., Troxel, W.M., Matthews, K.A., Bromberger, J., Kravitz, H.M., Krafty, R.T., Buysse, D.J., 2015. Chronic stress is prospectively associated with sleep in midlife women: the SWAN sleep study. Sleep 38, 1645–1654.
Hamer, M., Steptoe, A., 2012. Cortisol responses to mental stress and incident hypertension in healthy men and women. J. Clin. Endocrinol. Metab. 97, E29–E34.
Hamer, M., O'Donnell, K., Lahiri, A., Steptoe, A., 2010. Salivary cortisol responses to mental stress are associated with coronary artery calcification in healthy men and women. Eur. Heart J. 31, 424–429.
Kalmbach, D.A., Pillai, V., Roth, T., Drake, C.L., 2014. The interplay between daily affect and sleep: a 2-week study of young women. J. Sleep Res. 23, 636–645.
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.
Kirschbaum, C., Pirke, K.M., Hellhammer, D.H., 1993. The 'Trier Social Stress Test';–a tool for investigating psychobiological stress responses in a laboratory setting. Neuropsychobiology 28, 76–81.
Kirschbaum, C., Kudielka, B.M., Gaab, J., Schommer, N.C., Hellhammer, D.H., 1999. Impact of gender, menstrual cycle phase, and oral contraceptives on the activity of the hypothalamus-pituitary-adrenal axis. Psychosom. Med. 61, 154–162.
Lim, A.S.P., Yu, L., Schneider, J.A., Bennett, D.A., Buchman, A.S., 2016. Sleep fragmentation, cerebral arteriolosclerosis, and brain infarct pathology in community-dwelling older people. Stroke 47, 516–518.
Liu, J.C.J., Verhulst, S., Massar, S.A.A., Chee, M.W.L., 2015. Sleep deprived and sweating it out: the effects of total sleep deprivation on skin conductance reactivity to psychosocial stress. Sleep 38, 155–159.
Lo, J.C., Ong, J.L., Leong, R.L.F., Gooley, J.J., Chee, M.W.L., 2016. Cognitive Performance, Sleepiness, and Mood in Partially Sleep Deprived Adolescents: The Need for Sleep Study. Sleep 39, 687–698.
Maglione, J.E., Ancoli-Israel, S., Peters, K.W., Paudel, M.L., Yaffe, K., Ensrud, K.E., Stone, K.L., 2014. Subjective and objective sleep disturbance and longitudinal risk of depression in a cohort of older women. Sleep 1–9.
McEwen, B.S., 2005. Stressed or stressed out: what is the difference? J. Psychiatry Neurosci. 30, 315.
Mezick, E.J., Matthews, K.A., Hall, M.H., Richard Jennings, J., Kamarck, T.W., 2014. Sleep duration and cardiovascular responses to stress in undergraduate men. Psychophysiology 51, 88–896.
Minkel, J., Moreta, M., Muto, J., Htaik, O., Jones, C., Basner, M., Dinges, D., 2014. Sleep deprivation potentiates HPA axis stress reactivity in healthy adults. Health Psychol. 33, 1430–1434.
Mrug, S., Tyson, A., Turan, B., Granger, D.A., 2016. Sleep problems predict cortisol reactivity to stress in urban adolescents. Physiol. Behav. 155, 95–101.
Pesonen, A.-K., Kajantie, E., Heinonen, K., Pyhälä, R., Lahti, J., Jones, A., Matthews, K.A., Eriksson, J.G., Strandberg, T., Räikkönen, K., 2012. Sex-specific associations between sleep problems and hypothalamic-pituitary-adrenocortical axis activity in children. Psychoneuroendocrinology 37, 238–248.
Petersen, H., Kecklund, G., D'Onofrio, P., Nilsson, J., Åkerstedt, T., 2013. Stress vulnerability and the effects of moderate daily stress on sleep polysomnography and subjective sleepiness. J. Sleep Res. 22, 50–57.
Pruessner, J.C., Kirschbaum, C., Meinlschmid, G., Hellhammer, D.H., 2003. Two formulas for computation of the area under the curve represent measures of total hormone concentration versus time-dependent change. Psychoneuroendocrinology 28, 916–931.
Räikkönen, K., Matthews, K.A., Pesonen, A.-K., Pyhälä, R., Paavonen, E.J., Feldt, K., Jones, A., Phillips, D.I.W., Seckl, J.R., Heinonen, K., Lahti, J., Komsi, N., Järvenpää, A.-L., Eriksson, J.G., Strandberg, T.E., Kajantie, E., 2010. Poor sleep and altered hypothalamic-pituitary-adrenocortical and sympatho-adrenal-medullary system activity in children. J. Clin. Endocrinol. Metab. 95, 2254–2261.
Smagula, S.F., Stone, K.L., Redline, S., Ancoli-Israel, S., Barrett-Connor, E., Lane, N.E., Orwoll, E.S., Cauley, J.A., 2016. Actigraphy- and polysomnography-measured sleep disturbances, inflammation, and mortality among older men. Psychosom. Med. 78, 686–696.
Spiegel, K., Leproult, R., Van Cauter, E., 1999. Impact of sleep debt on metabolic and endocrine function. Lancet 354, 1435–1439.
Susman, E.J., Dorn, L.D., Inoff-Germain, G., Nottelmann, E.D., Chrousos, G.P., 1997. Cortisol reactivity, distress behavior, and behavioral and psychological problems in young adolescents: a longitudinal perspective. J. Res. Adolesc. 7, 81–105.
Vgontzas, A.N., Mastorakos, G., Bixler, E.O., Kales, A., Gold, P.W., Chrousos, G.P., 1999. Sleep deprivation effects on the activity of the hypothalamic-pituitary-adrenal and growth axes: potential clinical implications. Clin. Endocrinol. (Oxf.) 51, 205–215.
Wright, C.E., Valdimarsdottir, H.B., Erblich, J., Bovbjerg, D.H., 2007. Poor sleep the night before an experimental stress task is associated with reduced cortisol reactivity in healthy women. Biol. Psychol. 74, 319–327.
Wright, 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 Behav. Immun. 47, 24–34.