Daily Associations between Stress and Sleep: Extending the Knowledge Base using Intensive Longitudinal Designs with Repeated Ecological Momentary Assessments and Objective Measures

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(none)

List of Abbreviations

ACES	Activity, Coping, Emotion, Stress and Sleep Study
AIC	Akaike Information Criterion
ASA-24	Automated Self-Administered 24-hour
AUSNUT	Australian Food and Nutrient Database
AVC	Avoidance-Oriented Coping
BAVC	Between-Person Avoidance-Oriented Coping
BEAC	Between-Person Emotional-Approach Coping
BIC	Bayesian Information Criterion
BMI	Body Mass Index
BPFC	Between-Person Problem-Focused Coping
BSTRESS	Between-Person Stress
COVID-19	Corona Virus Disease 2019
DESTRESS	Diet, Exercise, Stress, Emotions, Speech, and Sleep
EAC	Emotional-Approach Coping
EEG	Electroencephalography
EMA	Ecological Momentary Assessments
HPA	Hypothalamic-Pituitary-Adrenal
ICC	Intraclass Correlations
JITAI	Just-In-Time Adaptive Interventions
No of Obs.	Number of Observations

PFC	Problem-Focused Coping
PSG	Polysomnography
REM	Rapid Eye Movement
SE	Sleep Efficiency
SHS	Stress and Health Study
SOL	Sleep Onset Latency
SWS	Slow Wave Sleep
TST	Total Sleep Time
WASO	Wake After Sleep Onset
WAVC	Within-Person Avoidance-Oriented Coping
WEAC	Within-Person Emotional-Approach Coping
WHO	World Health Organization
WPFC	Within-Person Problem-Focused Coping
WSTRESS	Within-Person Stress

Abstract

Stress and sleep are two important determinants of health, and the current research suggests a possible bi-directional association. However, the current understanding of stress-sleep associations is primarily based on single measures of a group of individuals in a given moment in time or from highly controlled laboratory settings. Stress exposures or experiences and sleep can vary daily, and naturalistic experiences may not be equivalent to those from controlled environments. Thus, the single time-point measures or laboratory designs cannot accurately capture the fluctuating nature of stress and sleep in daily, naturalistic settings. The main aim of this thesis is to advance the stress-sleep associations knowledge base using a daily intensive longitudinal design with repeated ecological momentary assessments (across 7-15 days), as well as integrating both subjective and objective measures of stress (i.e., perceived stress; salivary cortisol) and sleep (i.e., sleep diary; actigraphy; electroencephalography [EEG]). Specifically, this thesis tested the bi-directional and temporal associations between daily stress and the multi-faceted sleep (e.g., total sleep time; sleep onset latency; sleep efficiency; sleep architecture) at the between- and within-person level in young adults.

Overall, there was a more consistent direction of shorter sleep duration and poorer sleep quality, as well as shorter Rapid Eye Movement (REM) sleep and Slow-Wave Sleep (SWS), predicting next-day psychological stress compared to the opposite direction (i.e., psychological stress predicting subsequent sleep). These findings were within-person effects, meaning that regardless of an individual's average sleep, when individuals had nights with shorter or poorer than their *own* usual sleep duration or quality, they experienced higher stress levels the following day. Most between-person effects (i.e., individual differences) were non-significant. When examining cortisol, a marker of physiological stress, higher within-person cortisol levels around pre-bedtime predicted subsequent shorter and poorer

sleep. Although within-person sleep did not predict next-day diurnal cortisol slope, a consistent pattern of between-person associations emerged. Specifically, individuals with poorer or shorter average sleep had a flatter diurnal cortisol slope, indicating a possible dysregulated stress response system. Collectively, these findings highlight the complexity of the temporal associations between stress and sleep in day-to-day settings, which differed across the sleep parameters and measures used.

This thesis also explored several theoretical and novel concepts to expand the current stress-sleep literature, given that stress and sleep are not only associated with one another but also with other factors that can impact health. This thesis had two exploratory aims: 1) to explore modifiable behaviours that may mitigate the associations between daily stress and subsequent sleep, and 2) to explore beyond testing stress or sleep as the outcome by examining their synergistic associations on another health behaviour. To test the first exploratory aim, this thesis examined *whether* and *which* daily coping strategies mitigated the associations of daily stress and subsequent sleep, which can further the understanding of potentially modifiable daytime behaviours that may moderate the impact of stress on sleep on a day-to-day basis. These findings showed that engaging in either problem-focused coping, emotional-approach coping, or emotional-avoidance coping in face of stress was associated with shorter sleep duration. However, engaging in high emotional-approach coping or low in emotional-avoidance coping when experiencing high stress was associated with higher sleep efficiency. Together, these findings show that coping strategies can differentially moderate the associations between stress and sleep.

To test the second exploratory aim, this thesis examined the synergistic predictions of daily stress and nightly sleep on dietary intake. The findings showed that individuals with poorer and shorter sleep had higher total daily energy intake on days when they experienced higher than usual stress levels. There was also within-person evidence showing that shorter

sleep duration and REM sleep duration the previous night predicted a higher percentage of energy intake from discretionary food (i.e., junk food).

Taken together, this thesis advanced the current understanding of the stress-sleep associations in naturalistic settings using rigorous designs and measures, as well as testing theoretical and novel concepts. These findings provide valuable evidence to advance the field of health psychology, methodological implications for future research, and evidence for interventions at the societal and individual level to improve health education delivery programs and incorporate daily strategies into everyday life.

Publications and Presentations during Enrolment

Published Peer Reviewed Journal Articles

Yap, Y., Slavish, D. C., Taylor, D. J., Bei, B., & Wiley, J. F. (2020). Bi-directional Relations between Stress and Self-Reported and Actigraphy-Assessed Sleep: A Daily Intensive Longitudinal Study. *Sleep*. **doi:10.1093/sleep/zsz250**

Submitted Manuscripts in Review

Le, F., **Yap, Y.,** Tung, Y. C., Bei, B., Wiley, J. F. The associations between daily activities and affect: A compositional isotemporal substitution analysis.

Slavish, D. C., Dietch, J. R., Messman, B. A., Garcia, O., Wiley, J. F., **Yap, Y.,** Kelly, K., Ruggero, C., Taylor, D. J. Daily stress and sleep associations vary by work schedule: A between- and within-person analysis in nurses.

Yap, Y., Bei, B., Wiley, J. F. Daily coping moderates the relations between stress and actigraphic sleep: A daily intensive longitudinal study with ecological momentary assessments

Yap, Y., Tung, Y. C., Collins, J., Phillips, A., Bei, B., Wiley, J. F. Daily relations between stress and EEG-assessed sleep: A 15-day intensive longitudinal design with ecological momentary assessments

Yap, Y., Tung, Y. C., Phillips, A., Bei, B., Collins, J., Wiley, J. F. Sleep moderates the association between next-day stress and dietary intake: A 7-day intensive longitudinal design with ecological momentary assessments

Published Conference Abstracts and Presentations

Chachos, E., Shen, L., Maskevich, S., **Yap, Y.,** Stone, J. E., Wiley, J. F., Bei, B (2021). Exploring sleep-related affective vulnerability: the role of dysfunctional beliefs and attitudes about sleep as moderators of associations between daily sleep and affect in adolescents and emerging adults. Oral Presentation at Sleep DownUnder 2021 –

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Shen, L., **Yap, Y. Y**., Rice-Lacey, R. C., Bei, B., & Wiley J. F. (2018). *Daily Sleep and Affect: Examining the bidirectional relationship through ecological momentary assessment.* Poster Discussion and Presentation at Sleep DownUnder 2018 - Australasian Sleep Association, Brisbane Convention and Exhibition Centre, Brisbane, Australia

Wiley, J. F., Rice-Lacy, R. C., **Yap, Y. Y.,** Tuck, D., Stanton, A. L., & Bei, B. (2018). *Coping Moderates the Relationship between Stress and Negative Affect: A 12-day Diary Study*. Poster presentation at the Annual Meeting of the American Psychosomatic Society.

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Yap, Y., Rice-Lacey, R. C., Bei, B., & Wiley, J. F. (2018). *Bidirectional Relations between Stress and Sleep: An Intensive Daily Study.* Poster presentation at the Annual Meeting

of the Associated Professional Sleep Societies, Baltimore Maryland **https://doi.org/10.1093/sleep/zsy061.177**

Yap, Y., Rice-Lacey, R. C., Bei, B., & Wiley, J. F. (2018). *Daily Coping Moderates the Relations of Stress and Sleep: A Daily Study Over 12 Days.* Oral Presentation at Sleep DownUnder 2018 – Australasian Sleep Association, Brisbane Convention and Exhibition Centre, Brisbane, Australia **https://doi.org/10.1111/jsr.63_12765**

Thesis including published works declaration

I hereby declare that this thesis contains no material which has been accepted for the award of any other degree or diploma at any university or equivalent institution and that, to the best of my knowledge and belief, this thesis contains no material previously published or written by another person, except where due reference is made in the text of the thesis.

This thesis includes **one** original paper published in peer-reviewed journals and **three** submitted publications. The core theme of the thesis is examining the daily associations between stress and sleep in naturalistic settings. The ideas, development and writing up of all the papers in the thesis were the principal responsibility of myself, the student, working within the School of Psychological Sciences, under the supervision of Dr. Joshua F. Wiley, and co-supervisor Dr. Andrew Phillips, and Dr. Jorja Collins. The inclusion of co-authors reflects the fact that the work came from active collaboration between researchers and acknowledges input into team-based research. In the case of Chapter 2, 3, 4, and 6, my contribution to the work involved the following:

Thesis Chapter	Publication Title	Status	Nature and % of student contribution	Co-author name(s), Nature and % of Co-author's contribution	Co-author(s), Monash student Y/N
2	Bi-Directional Relations Between Stress and Self-Reported and Actigraphy-Assessed Sleep: A Daily Intensive Longitudinal Study	**Published**	**70%.** Conceptualisation (development of study design), data collection, data analysis, and write-up of manuscript	**1. Dr. Danica Slavish (5%)** Revision and input into manuscript **2. Dr. Daniel Taylor (5%)** Revision and input into manuscript **3. Dr. Bei Bei (10%)** Revision and input into manuscript **4. Dr. Joshua Wiley (10%)** Input into conceptualisation (development of study design), data analysis, revision of manuscript, and funding	**1. N** **2. N** **3. N** **4. N**
3	Daily Coping Moderates the Relations between Stress and Actigraphic Sleep: A Daily Intensive Longitudinal Study with Ecological Momentary Assessments	**Revise and resubmit**	**70%.** Conceptualisation (development of study design), data collection, data analysis, and write-up of manuscript	**1. Dr. Bei Bei (10%)** Revision and input into manuscript **2. Dr. Joshua Wiley (20%)** Input into conceptualisation (development of study design), data analysis, revision of manuscript, and funding	**1. N** **2. N**
4	Daily Relations between Stress and EEG-Assessed Sleep: A 15-day Intensive Longitudinal Design with Ecological Momentary Assessments	**Revise and resubmit**	**70%.** Conceptualisation (development of study design), data collection, data analysis, and write-up of manuscript	**1. Natasha Yan Chi Tung (5%)** Conceptualisation (development of study design), data collection, and input into manuscript **2. Dr. Jorja Collins (5%)** Input into study design and manuscript revision **3. Dr. Andrew Phillips (5%)** Input into study design and manuscript revision **4. Dr. Bei Bei (5%)** Revision and input into manuscript **5. Dr. Joshua Wiley (10%)** Input into conceptualisation (development of study design), data analysis, revision of manuscript, and funding	**1. Y** **2. N** **3. N** **4. N** **5. N**

Thesis Chapter	Publication Title	Status	Nature and % of student contribution	Co-author name(s), Nature and % of Co-author's contribution	Co-author(s), Monash student Y/N
6	Sleep Moderates the Association Between Next-Day Stress and Dietary Intake: A 7-Day Intensive Longitudinal Design with Ecological Momentary Assessments	**Submitted**	**70%.** Conceptualisation (development of study design), data collection, data analysis, and write-up of manuscript	**1. Natasha Yan Chi Tung (5%)** Conceptualisation (development of study design), data collection, and input into manuscript **2. Dr. Jorja Collins (5%)** Input into study design and manuscript revision **3. Dr. Andrew Phillips (5%)** Input into study design and manuscript revision **4. Dr. Bei Bei (5%)** Revision and input into manuscript **5. Dr. Joshua Wiley (10%)** Input into conceptualisation (development of study design), data analysis, revision of manuscript, and funding	**1. Y** **2. N** **3. N** **4. N** **5. N**

I have not renumbered sections of submitted or published papers in order to generate a consistent presentation within the thesis.

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I hereby certify that the above declaration correctly reflects the nature and extent of the student's and co-authors' contributions to this work. In instances where I am not the responsible author I have consulted with the responsible author to agree on the respective contributions of the authors.

Main Supervisor name: DR JOSHUA F. WILEY

Main Supervisor signature: **Date:** 26/08/2021

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CHAPTER 1: GENERAL INTRODUCTION

Stress and sleep are linked, and they are important determinants of health (Benca, Obermeyer, Thisted, & Gillin, 1992; Cappuccio, Cooper, D'Elia, Strazzullo, & Miller, 2011; Cappuccio, D'Elia, Strazzullo, & Miller, 2010; Hall, Fernandez-Mendoza, Kline, & Vgontzas, 2017; Kendler, Hettema, Butera, Gardner, & Prescott, 2003; Kim & Dimsdale, 2007; Richardson et al., 2012; Slopen, Lewis, & Williams, 2016). However, the current knowledge base of the stress-sleep associations is primarily based on cross-sectional or between-person evidence, limiting the understanding of several important concepts. Stress and sleep can fluctuate across days, and the cross-sectional evidence, usually based on single time-point measures, cannot examine how these associations vary within individuals' daily, naturalistic life. Furthermore, sleep is a multi-dimensional construct that includes sleep duration, sleep continuity (e.g., sleep onset latency; wake after sleep onset; sleep efficiency), and sleep architecture (e.g., slow-wave sleep; rapid-eye-movement sleep), and these specific dimensions are associated with mental and physical health outcomes and mortality (Buysse, 2014); however, whether each of these sleep dimensions relate to daily stress on a daily basis remains unclear. Thus, this thesis aimed to extend the current knowledge base using an intensive longitudinal design with repeated ecological momentary assessments (EMA) and incorporating objective stress and sleep measures. In addition to using a rigorous design and objective measures, this thesis also explored several theoretical and novel concepts to expand the current stress-sleep literature. Specifically, this thesis explored whether and which coping strategies mitigate (or exacerbate) the associations of daily stress and sleep, as well as the interaction effects of stress and sleep on dietary intake.

Associations between Stress and Sleep

Cross-sectional research has established a robust association between high perceived stress and self-reported poor, disturbed, or short sleep (Akerstedt, Kecklund, & Axelsson,

2007; Akerstedt et al., 2002; Alsaggaf, Wali, Merdad, & Merdad, 2016; Buxton et al., 2016; Martica Hall et al., 2000; Jerlock, Gaston-Johansson, Kjellgren, & Welin, 2006; Kashani, Eliasson, & Vernalis, 2012; Kim & Dimsdale, 2007; Lemma, Gelaye, Berhane, Worku, & Williams, 2012; Lund, Reider, Whiting, & Prichard, 2010; Palagini et al., 2016a; Palagini et al., 2016b; Slopen et al., 2016; Wiklund, Malmgren-Olsson, Öhman, Bergström, & Fjellman-Wiklund, 2012). However, the directionality of these associations (i.e., whether stress predicts sleep or vice versa) cannot be drawn from cross-sectional evidence. A few longitudinal studies on the stress-sleep associations have shed light on these directions. For example, higher stress levels, stress-related intrusion (e.g., ruminating about the stressor), and work-related stressors were associated with subsequent development and maintenance of insomnia (Bernert, Merrill, Braithwaite, Van Orden, & Joiner Jr, 2007; Drake, Pillai, & Roth, 2014; Jansson & Linton, 2006).

Most of these studies have relied on group-level estimates or recalling experiences over a period of time (e.g., asking participants' average sleep quality or stress levels over the past week or month). Group-level estimates are usually derived from cross-sectional, single time-point measurements, which cannot be applied to understanding the *intraindividual* processes and the directionality of the stress-sleep associations (Fisher, Medaglia, & Jeronimus, 2018). Although longitudinal studies provide a stronger test of directionality compared to cross-sectional designs, they often use few and long interval time points (e.g., two time-points one year apart). Thus, most longitudinal studies cannot examine how daily dynamic changes in both stress and sleep predict one another. Given the variable nature of stress and sleep, examining them over discrete time points with long intervals would not provide insight regarding any potential immediate effects that they have on one another. For example, whether experiencing higher stress *today* predicts subsequent sleep *that night*, or having poor or short sleep predicts *next-day* stress levels, cannot be answered from

longitudinal designs. Thus, examining these variables over continuous time may be more appropriate (Bolger & Laurenceau, 2013; Collins, 2006). Moreover, recalling an experience over a period of time may be subjected to recall bias. It is difficult to report an estimate of the *average* experience over a period; instead, individuals may retrieve and report the most memorable experiences, such as the night with the worst sleep quality or the day with the highest stress (Shiffman, Stone, & Hufford, 2008). Additionally, individuals may be affected by their mental state at the time of recall (Shiffman et al., 2008). Thus, these reports are subjected to systematic biases and may not accurately reflect the individual's actual experiences.

Daily studies, or intensive longitudinal designs, can provide a stronger estimate of the direction of associations and temporal unfolding of daily stress and sleep in naturalistic settings. This design allows using repeated ecological momentary assessments (EMA), examining within-person effects, and accounting for lagged outcomes (Bolger & Laurenceau, 2013; Shiffman et al., 2008). However, it is worth noting that not all daily studies use all these approaches. EMA are usually used in naturalistic settings, which maximises ecological observations that acknowledge the daily changes in an individual's environment (e.g., at home, school, work, or social events). The repeated assessments within and across multiple days capture the individual's *current* and variations in their experiences over time and minimise recall biases. Within-person differences, also known as intra-individual differences, are the deviations of scores from an individual's own mean (e.g., days with higher stress levels compared to their own average). These processes emphasise how experiences change over time, rather than how individuals are different from one another (i.e., between-person effects). Examining within-person effects also are less affected by individual differences, as the individuals served as their own control. The use of daily studies and repeated EMA also allow for statistical analyses to account for lagged outcomes to account for temporal

dependence and strengthen the test of directionality. For example, in models testing whether today's stress predicts *tonight's* sleep, while controlling for *previous* night sleep, the prediction of sleep by stress cannot be attributed to sustained or carry-over effects from sleep the previous night, thus strengthening the directionality of the analyses.

Comparatively, fewer but emerging daily studies on the stress-sleep associations have been conducted (Akerstedt et al., 2012; Doane & Thurston, 2014; Garde, Albertsen, Persson, Hansen, & Rugulies, 2011; Hanson & Chen, 2010; Lee, Crain, McHale, Almeida, & Buxton, 2017; Morin, Rodrigue, & Ivers, 2003; Philbrook & Macdonald-Gagnon, 2021; Sin et al., 2017; Slavish et al., 2020; Winzeler et al., 2014). These studies showed that higher stress levels during the day or at bedtime predicted poorer self-reported sleep quality (Akerstedt et al., 2012; Garde et al., 2011; Lee et al., 2017; Morin et al., 2003; Winzeler et al., 2014) and actigraphic sleep duration that night (Hanson & Chen, 2010; Slavish et al., 2020). Only a few daily studies have explicitly and rigorously tested the bi-directional and temporal associations by including lagged outcomes (Lee et al., 2017; Sin et al., 2017), with even fewer studies using objective sleep measures (Doane & Thurston, 2014). For example, in working adults, higher than usual work-related stress predicted longer self-reported sleep onset latency (SOL), but that longer SOL did not predict higher next-day stress. However, self-reported poorer sleep quality and shorter sleep duration predicted higher next-day stress, whereas the opposite direction was non-significant (Lee et al., 2017). Likewise, Sin et al. (2017) found that poorer subjective sleep quality predicted higher odds of experiencing stressors the next day in working adults; however, the reverse direction did not emerge.

Using actigraphic measures, Doane and Thurston (2014) found a significant bi-directional relationship between stress and sleep duration in adolescents. Specifically, higher within-person daily stress predicted shorter sleep duration that night, and shorter within-person sleep duration predicted higher next-day stress. Moreover, lower sleep efficiency (SE)

that night predicted higher next-day stress, but the daily stress was not associated with SE that night. To date, only one study examined the daily stress-sleep relations using a 7-day daily diary design and electroencephalography (EEG) sleep measures (Slavish et al., 2020). EEG sleep measures may offer a more accurate measure of sleep compared to sleep diary or actigraphy measures. However, the findings showed that stress did not predict any of the subsequent EEG sleep variables, and the EEG sleep measures did not significantly predict next-day stress (Slavish et al., 2020). It is worth noting that this study did not account for lagged outcomes.

Taken together, the evidence from daily studies highlight the complex, bi-directional associations between stress and sleep, which differed across sleep parameters (i.e., duration; quality) and measurements (i.e., self-report vs actigraphic vs EEG). Although significant, self-reported sleep measures often overestimate TST and SOL compared to PSG estimates in healthy adults (Silva et al., 2007). The individuals' self-reported sleep quality and behaviours also may be affected by their current state (e.g., expectancy effects of high stress or negative mood); for example, individuals with insomnia and/or mood symptoms tend to underestimate TST and overestimate wake (Fernandez-Mendoza et al., 2011). Differences in these findings also could be due to the time when stress was measured (e.g., recalling stress levels the previous day; during bedtime; averaged across the day). Nonetheless, these findings indicate that stress and sleep are likely to have bi-directional associations, and more rigorous daily studies incorporating subjective and objective sleep measures are needed to clarify these associations.

Experimental designs provide strong causal associations and interpretations. A systematic review by Kim and Dimsdale (2007) showed that individuals exposed to experimentally manipulated stressors, including an indwelling catheter, first-night effects in a sleep laboratory, and psycho-emotional stress, had significantly lower SE, longer SOL, more

frequent night-time awakenings, and shorter Rapid Eye Movement (REM) and Slow Wave Sleep (SWS). More recent studies also showed that individuals exposed to emotional stress (e.g., failure feedback with difficult/unsolvable tasks; distressing films) showed significantly decreased TST and SE (Vandekerckhove et al., 2011) as well as less REM sleep (Talamini, Bringmann, de Boer, & Hofman, 2013; Vandekerckhove et al., 2011). Similarly, individuals had significantly lower SE, but not TST, during high-stress periods compared with low-stress periods (Petersen, Kecklund, D'onofrio, Nilsson, & Åkerstedt, 2013). These findings show that experimentally induced stressors can affect sleep parameters and architecture. It is worthwhile highlighting that there are also several null and contradictory findings, such that there were increases, decreases, or no changes in SWS or sleep duration following stressful experiences (Kim & Dimsdale, 2007; Petersen et al., 2013; Talamini et al., 2013; Vandekerckhove et al., 2011). These contradictory findings may be due to the experimental stressors not producing the experience of anticipation that reflects real-world stressors, and they may not be of high significance for the individual (Akerstedt, 2006), thus highlighting the need to examine stress in naturalistic settings.

Research on the effects of experimentally manipulated sleep on stress is scarce. One study found that experimentally sleep-deprived individuals responded with greater psychological distress than well-rested individuals after experiencing minor stressors (Minkel et al., 2012). Neuroimaging evidence also showed increases in amygdala reactivity and reduced functional connectivity between the amygdala and medial prefrontal cortex when presented with negative stimuli in sleep-deprived individuals compared to controls (Yoo, Gujar, Hu, Jolesz, & Walker, 2007). These studies indicate that sleep deprivation can impair the emotional regulatory systems, which led individuals to perceive stressors as more intense. Several neuroimaging studies also have proposed the emotional regulatory role of SWS and REM sleep (Ben Simon, Rossi, Harvey, & Walker, 2020; van der Helm et al., 2011). For

example, individuals who had a night of sleep deprivation reported higher anxiety levels the following morning compared to the previous night and to well-rested individuals; within well-rested individuals, longer SWS was associated with lower levels of next-day anxiety (Ben Simon et al., 2020). Another study showed that well-rested individuals with 8 hours of sleep opportunity had decreased amygdala and emotional reactivity towards affective images, compared with sleep-deprived individuals (van der Helm et al., 2011). Within well-rested individuals, low EEG gamma activity (a biomarker of adrenergic activity that plays a role in emotional regulation and amygdala activity) during REM sleep was associated with reductions in both amygdala activity and emotional reactivity towards the affective stimuli (van der Helm et al., 2011). Together, these findings suggest that SWS and REM sleep play a role in emotional regulation.

Collectively, these findings show that sleep deprivation affects our appraisal of stress and emotional reactivity, which could potentially be due to shorter SWS and REM sleep that play a role in emotion regulation. Although existing neuroimaging studies have only examined SWS or REM sleep predicting *affect* reactivity, it is possible that they also may play a role in regulating the appraisal and perception of stress given the inextricable link between emotions and stress. Furthermore, whether these findings translate into naturalistic settings remain unclear, especially where total sleep deprivation in daily life is rare, and the day-to-day fluctuations in sleep architecture remain underexplored. Lastly, individuals in these studies cannot be blinded to the sleep deprivation conditions, wherein their reported experiences following sleep deprivation could partially be due to expectancy effects. Thus, more research is needed to explicitly test whether SWS and REM sleep are associated with next-day stress experiences.

Associations between Cortisol and Sleep

One daily study showed that self-reported somatic arousals (e.g., sweating; heart racing) mediated the associations between daily stress and subjective sleep quality (Winzeler et al., 2014), suggesting that the activation of the Hypothalamus-Pituitary-Adrenal (HPA) axis prior to sleep could be at play (Hirotsu, Tufik, & Andersen, 2015). The HPA axis is a central stress response system, with cortisol being the primary product and one biomarker of stress. Cortisol functions to regulate various physiological processes to help individuals adapt to environmental changes, such as energy and metabolic processes, blood pressure, and inflammatory and immune functioning. Cortisol often is used to assess HPA activity, given its responses to acute and chronic stressors (Adam et al., 2017). When encountering stressors, the body releases more cortisol to support catecholamine activity (e.g., the fight-or-flight response) and effectively cope with the stressor. However, prolonged exposure to stress can repeatedly activate the HPA axis and dysregulate the initiation and termination of the stress response system, thus leading to excessive, flattened, or blunted cortisol response (Adam et al., 2017; McEwen, 2004; Staufenbiel, Penninx, Spijker, Elzinga, & van Rossum, 2013). A flattened diurnal cortisol rhythm is indicative of dysregulated HPA activity. Cortisol secretion follows a diurnal rhythm; levels are high at awakening, peaking at about 30-40 minutes post-awakening, and gradually decrease across the day, with the lowest levels around bedtime. A flatter diurnal cortisol slope, which could be due to lower awakening or higher evening cortisol level or both, is indicative of a dysregulated HPA functioning and is associated with poorer mental and physical health outcomes (Adam et al., 2017).

The evidence for the associations between sleep and cortisol is comparatively more limited, and whether they mutually predict each other, especially on a day-to-day level, is still unclear. There is some cross-sectional (Backhaus, Junghanns, & Hohagen, 2004; Castro-Diehl et al., 2015; Garde, Karlson, Hansen, Persson, & Åkerstedt, 2012; Kumari et al., 2009)

and experimentally-restricted sleep studies (Garde et al., 2012; Guyon et al., 2014; Omisade, Buxton, & Rusak, 2010) linking poor sleep quality or short sleep duration to lower cortisol at awakening, higher evening cortisol, and a flatter diurnal slope. However, the temporal order between sleep and cortisol could not be established from cross-sectional evidence, and that experimentally restricted sleep in laboratory settings may not be ecologically valid. Little research has examined the daily variation in sleep and diurnal cortisol rhythm. For example, a 3-day study in young adults found that shorter between-person sleep duration was associated with flatter diurnal cortisol slope, and that within- and between-person shorter sleep duration predicted cortisol at awakening (Van Laethem, Beckers, van Hooff, Dijksterhuis, & Geurts, 2016). Another 3-day study also found similar findings, where shorter between- and within-person actigraphic sleep duration predicted flatter diurnal cortisol slope, and vice versa (Zeiders, Doane Sampey, & Adam, 2011).

Together, these studies show that short sleep duration and poor sleep quality are associated with lower cortisol at awakening, higher evening or bedtime cortisol, and a flatter diurnal cortisol slope. There is also some evidence suggesting that a flatter slope is associated with shorter sleep duration. However, only a few daily studies on cortisol and sleep in naturalistic environments have been conducted, and the existing evidence is limited to 3-days of cortisol sampling. At least 10-days of cortisol sampling has been recommended to provide better reliability to detect between- and within-person person differences in diurnal cortisol (Segerstrom, Boggero, Smith, & Sephton, 2014). Furthermore, a minimum of one week, including weekends, is needed to capture variation in sleep (Wohlgemuth, Edinger, Fins, & Sullivan JR., 1999). Additionally, whether cortisol levels during pre-sleep or bedtime periods predict subsequent sleep remains unclear from these studies. Furthermore, the daily studies that exist relied on self-reported or actigraphic measures of sleep, which may not be as

accurate as EEG measures to capture the multiple facets of sleep. Thus, more research is needed to clarify these findings and to extend the current stress-sleep knowledge base.

Explore to Extend: Modifiable Behaviours that Moderate the Stress-Sleep Association

Although the stress-sleep association is established in cross-sectional studies (Akerstedt et al., 2007; Akerstedt et al., 2002; Alsaggaf et al., 2016; Buxton et al., 2016; Martica Hall et al., 2000; Jerlock et al., 2006; Kashani et al., 2012; Kim & Dimsdale, 2007; Lemma et al., 2012; Lund et al., 2010; Palagini et al., 2016a; Palagini et al., 2016b; Slopen et al., 2016; Wiklund et al., 2012), with emerging research extending the knowledge base to examine how stress affects sleep on a day-to-day basis (Doane & Thurston, 2014; Lee et al., 2017; Philbrook & Macdonald-Gagnon, 2021; Sin et al., 2017; Slavish et al., 2020), it is still unclear what modifiable factors may moderate the effects of daily stress on sleep. Identifying and testing these factors may further the understanding of the stress-sleep associations, such as what and how these factors can mitigate or exacerbate the effects of sleep on stress. Understanding these factors also may inform interventions on how to mitigate the effects of stress on sleep.

One potential moderator would be coping. Theoretical models highlight the role of coping strategies in moderating stress responses, such that individuals may cope by directly approaching (e.g., directly addressing the stressor or managing the emotional responses to the stressor) or avoiding (e.g., mental or behavioural disengagement) the stressor (Lazarus & Folkman, 1984; Snyder et al., 2017; Stanton, Kirk, Cameron, & Danoff-Burg, 2000; Stanton, Sullivan, & Austenfeld, 2009). Reviews of empirical studies show that approach-oriented coping often is associated with better, whereas avoidance-oriented coping with worse psychological well-being (Compas et al., 2017; Folkman, 2013; Snyder et al., 2017). These findings suggest that different coping strategies used in response to stress can have an impact on sleep. However, this concept remains relatively unexplored, especially in daily settings.

Few studies have examined the associations between coping and sleep. Cross-sectional and longitudinal studies have consistently linked avoidance-oriented coping with poorer or shorter sleep, whereas results for approach-oriented coping remain inconclusive (Hall et al., 1997; Hicks, Marical, & Conti, 1991; Hoyt, Thomas, Epstein, & Dirksen, 2009; Jerlock et al., 2006; Matthews, Hall, Cousins, & Lee, 2016; Morin et al., 2003; Palagini et al., 2016b; Taylor et al., 2015). Only two studies to date examined the moderating role of coping on the stress-sleep association (Maskevich, Cassanet, Allen, Trinder, & Bei, 2020; Sadeh, Keinan, & Daon, 2004). During a high-stress period, university students who used high vs low emotion-focused coping had shorter actigraphic sleep duration and reported poorer sleep quality (Sadeh et al., 2004). Furthermore, individuals who used higher problem-focused coping had longer actigraphic sleep duration regardless of high or low stress periods (Sadeh et al., 2004). Similarly, in older adolescents, problem-focused coping mitigated the associations of stress on both actigraphic and self-reported sleep initiation (Maskevich et al., 2020).

These findings suggest that approach- and avoidance-oriented coping may differentially moderate the associations between stress and sleep. However, these findings cannot inform how the daily dynamic interactions between stress, coping, and sleep unfold over time. Furthermore, most studies have examined only the broad constructs of coping (i.e., approach vs avoidance) or often equated emotion-focused coping to emotional *avoidance*, neglecting emotional *approach* coping. Emotional approach coping involves expressing and processing emotions. In the face of stress, individuals engaging in emotional approach coping would spend time understanding their own emotions and are likely to share or express them with other individuals, rather than avoiding or suppressing them. Hence, more research is needed to examine different coping strategies (i.e., problem-focused, emotional-approach, and emotional-avoidance) simultaneously, so their respective roles are better understood in

the same daily context. Understanding the role of these coping strategies would extend the stress-sleep knowledge base and inform interventions on coping strategies that may ameliorate or worsen the effects of stress on subsequent sleep.

The Synergistic Effects of Stress and Sleep on Dietary Intake: Looking Beyond Stress or Sleep as Outcome

High stress and poor sleep are associated with negative health outcomes, including cardiovascular diseases and diabetes (Cappuccio et al., 2010; Cappuccio et al., 2011; Richardson et al., 2012). It is possible that stress and sleep may increase the risk of developing these health conditions through their independent effects or partially through other health behaviours. One potential affected health behaviour is diet. It is well established that a healthy diet plays an essential role in bodily functions, health, and well-being, which reduces the risk of developing diet-related and chronic diseases such as obesity, hypertension, cardiovascular disease, and type-2 diabetes (Esposito, Maiorino, Ceriello, & Giugliano, 2010; Hooper et al., 2001; Ness & Powles, 1997; Threapleton et al., 2013). It is possible that stress and sleep may synergistically affect one's dietary intake, given that current evidence suggests that stress and sleep are bi-directionally associated (Doane & Thurston, 2014; Kim & Dimsdale, 2007; Minkel et al., 2012), and both are associated with diet (Chaput, 2014; Dashti, Scheer, Jacques, Lamon-Fava, & Ordovás, 2015; Yau & Potenza, 2013). However, the synergistic predictions of stress and sleep on diet remains relatively underexamined in the current literature. More research is needed to advance the current understanding of the stress-sleep literature by looking beyond these factors as the outcome and examine their synergistic effects on other health behaviours.

A healthy diet, as defined by the Australian Dietary Guidelines, includes consumption of various foods from each of the five food groups: 1) vegetables and legumes/beans, 2) fruit, 3) grains and cereals, 4) lean meat, poultry, fish, eggs, legumes, tofu, nuts, and seeds, and 5) milk, yoghurt, cheese, or alternatives (National Health and Medical Research Council, 2013). Foods that are not part of these groups or not required for a healthy diet are considered as discretionary food, or also known as junk food. These foods and drinks are typically energy-dense but nutrient-poor, and often contain excess or high saturated fat, sugar, salt, and/or alcohol, as well as low fibre (National Health and Medical Research Council, 2013). These foods include, but are not limited to, ice-cream, cakes, sugar-sweetened drinks, chocolate and confectionary, potato crisps and similar, and processed meats (Australian Bureau of Statistics, 2014a; National Health and Medical Research Council, 2013). Consumption of excessive discretionary food is associated with an increased risk of developing diet-related and chronic health conditions (Gadiraju, Patel, Gaziano, & Djoussé, 2015; Payab et al., 2015). Approximately 35% of the total daily energy intake in Australian adults aged 19 – 30 is contributed from discretionary foods (Australian Bureau of Statistics, 2014b).

Stress plays a role in our dietary intake and behaviours. Cross-sectional and longitudinal research has linked higher perceived stress with increased food intake, increased snacking, decreased intake of fruits and vegetables, and increased discretionary foods, particularly foods high in fat and/or sugar (Barrington, Beresford, McGregor, & White, 2014; Cartwright et al., 2003; Errisuriz, Pasch, & Perry, 2016; Groesz et al., 2012; Kandiah, Yake, Jones, & Meyer, 2006; Kim, Yang, Kim, & Lim, 2013; Wardle, Steptoe, Oliver, & Lipsey, 2000). A few daily studies also have supported these results. Days with higher than usual daily stress or hassles predicted greater snack intake of foods high in fat and sugar (Conner, Fitter, & Fletcher, 1999; O'Connor, Jones, Conner, McMillan, & Ferguson, 2008; Zenk et al., 2014). Experimental studies showed that individuals under high-stress conditions, such as

being given unsolvable puzzles or difficult arithmetic tasks, consumed more foods high in fat and sugar compared to those under low-stress conditions (Habhab, Sheldon, & Loeb, 2009; Kistenmacher et al., 2018; Wallis & Hetherington, 2004).

However, there also are experimental and daily studies linking higher stress with decreased food intake or null findings (Oliver, Wardle, & Gibson, 2000; Stone & Brownell, 1994). Inconsistency in these findings could be due to individual differences, such as different eating styles, cortisol reactivity status, stressor types, as well as methodological differences in measuring dietary intake (Epel, Lapidus, McEwen, & Brownell, 2001; Hill et al., 2021; O'Connor et al., 2008; Oliver et al., 2000; Wallis & Hetherington, 2004). For instance, while there were no main effects of stress on total energy intake and food preferences, individuals who reported high stress and high emotional eating consumed more high-fat and high-sugar snacks (i.e., cakes and chocolate snacks) as well as energy-dense meals following a laboratory stress task (Oliver et al., 2000). Individuals with high cortisol reactivity also reported increased snacking when experiencing higher daily hassles or consuming more calories following a stress task (Epel et al., 2001; Newman et al., 2007). These findings highlight the heterogeneity of the stress-dietary intake associations.

A recent meta-analysis recommended future studies to examine potential moderators of the stress-dietary intake relationship that may help to explain these inconsistent findings (Hill et al., 2021). Sleep may be one key moderator of the stress-diet relationship. Emerging evidence from cross-sectional research shows that shorter self-reported sleep duration or poorer sleep quality is associated with higher energy intake and poorer diet quality (Dashti et al., 2015; Grandner, Jackson, Gerstner, & Knutson, 2013). From a hormonal perspective, evidence from laboratory findings demonstrated that individuals with a restricted time in bed of approximately 4-hours of bed time increased ghrelin levels (i.e., hunger hormone) by 28% and decreased Leptin levels by 19% (i.e., satiety hormone) compared to well-rested

individuals (Spiegel, Tasali, Penev, & Van Cauter, 2004). This indicates that short sleep duration can disrupt the appetite-regulating hormones that can influence dietary intake and behaviours. Sleep manipulation studies support this interpretation, such that individuals with restricted sleep opportunity (4.5 – 5.5 hours) eat more energy-dense snacks and have higher total energy intake compared to individuals with 8.5 – 9 hours of sleep opportunity (Broussard et al., 2016; Markwald et al., 2013; Nedeltcheva et al., 2008). It is worthwhile noting that although results showed similar caloric intake for lunch and dinner meals for both conditions, those with restricted sleep had increased ghrelin levels (i.e., hunger regulating hormone) and ate more energy-dense snacks (Broussard et al., 2016) with an average of extra 328±140 kcal (or 1372±585 kJ) between meals. Only a few studies have examined the associations between sleep architecture and dietary intake (Crispim et al., 2011; Shechter et al., 2012). For example, a cross-over sleep restriction study found that lower SWS and REM sleep percentage were associated with higher fat intake, and higher REM sleep percentage was associated with lower carbohydrate intake (Shechter et al., 2012). Furthermore, shorter REM sleep duration was also associated with higher perceived hunger (Shechter et al., 2012). Although there were no changes in overall energy intake, these findings suggest that changes in sleep architecture are associated with dietary intake.

The studies discussed have only tested the associations of stress or sleep independently on dietary intake or behaviours. Whether high stress and poor sleep combined can predict worse diet, or good sleep can buffer the associations of stress and diet, is still unclear. To our knowledge, only one study has examined the moderating role of sleep on the stress-diet association on a daily level (Liu et al., 2017). A 21-day study found that higher work-related stress in the morning predicted lower number of healthy food (e.g., fruits; vegetables) and higher number of unhealthy foods (e.g., soda; sugary drinks) consumed in the evening. However, on days when participants reported better sleep quality, the relationship

between stress and unhealthy food consumption was weaker than days with poorer sleep quality. This suggests that good sleep quality may mitigate (or worse sleep exacerbates) the impact of stress on discretionary food intake. However, it is worth noting that the consumption of healthy vs unhealthy foods was assessed using a checklist of specified foods consumed. It is still unclear whether other objectively-measured sleep parameters (e.g., EEG sleep duration; sleep efficiency) and sleep architecture may mitigate the association of stress with dietary intake. Given that studies have shown the emotional regulatory benefits of SWS and REM sleep (Ben Simon et al., 2020; van der Helm et al., 2011), it is possible that they can ameliorate the impact of stress on dietary intake.

Collectively, these findings show that high stress and poor sleep are associated with poor diet, and one study showed the moderating role of sleep on the stress-diet association. However, more research is needed to test the stress-sleep interactions on diet, especially on a daily basis, to examine how these processes unfold over time in naturalistic settings. Additionally, most studies only measured limited to types of foods or drinks consumed (e.g., pre-specified, limited checklist of foods), which cannot capture the variety of foods consumed, especially in day-to-day, naturalistic settings, and the energy intake at the daily level.

Summary and Overview of Thesis Aims and Structure

The current understanding of the stress-sleep associations is largely based on cross-sectional or between-person evidence, which cannot be generalised to our understanding of the intra-individual differences in stress and sleep. Comparatively fewer daily studies in naturalistic environments have been conducted, with even fewer studies used repeated EMA of daily stress and objectively measured sleep and explicitly testing their bi-directional, temporal associations. Furthermore, nightly variations in sleep architecture and its association with daily stress remain underexamined. Thus, the main aim of this thesis is to examine the

stress-sleep associations using a daily intensive longitudinal design with repeated EMA, as well as integrating both subjective and objective measures of stress and sleep. **Paper 1 (Chapter 2)** examined the bi-directional associations between daily stress and self-reported and actigraphic sleep. **Paper 3 (Chapter 4)** extended **Paper 1** by testing the bi-directional associations between daily stress and EEG sleep, including sleep architecture. **Paper 4 (Chapter 5)** extended **Paper 1 and 3** by examining the associations between cortisol and EEG sleep. Together, these chapters advance the current stress-sleep knowledge base using one of the strongest available measures and designs possible for observational studies in naturalistic settings.

This thesis also explored theoretical and novel concepts that can strengthen and deepen the understanding of the stress-sleep associations. **Paper 2 (Chapter 3)** tested *whether* and *which* daily coping strategies mitigate (or exacerbate) the associations of daily stress on subsequent sleep. These findings can further the understanding of potential modifiable, daytime behaviours that may moderate the impact of stress on sleep on a day-to-day basis. **Paper 5 (Chapter 6)** moved beyond examining stress or sleep as the outcome. This chapter explored the synergistic predictions of stress and sleep on dietary intake. A summary of the overview of the thesis structure is summarised in Figure 1.

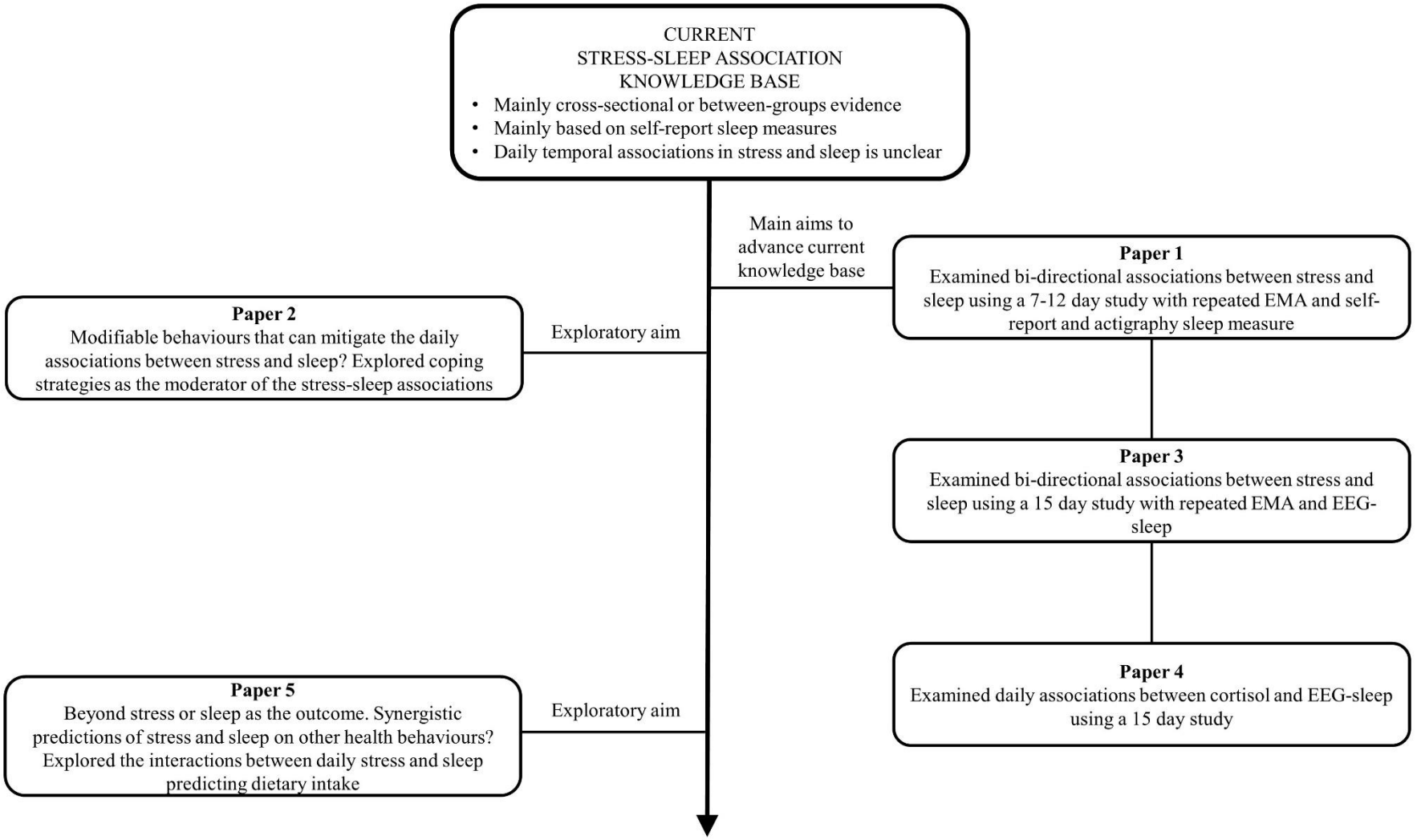

Figure 1. Overview of Core Thesis Theme and Structure

CHAPTER 2:

Bi-Directional Relations Between Stress and Self-Reported and Actigraphy-Assessed Sleep:

A Daily Intensive Longitudinal Study

Preface to Chapter 2

As highlighted in **Chapter 1: Introduction,** the current understanding of the association between perceived stress and sleep is limited to cross-sectional or between-person evidence. How daily fluctuations in stress predict sleep and vice versa remains unclear.

This chapter aims to extend the current stress-sleep association knowledge base by examining the bi-directional and temporal associations using a daily intensive longitudinal design with repeated ecological momentary assessments, as well as self-reported and actigraphic sleep measures. Data for this paper came from two existing daily datasets – 1) Activity, Coping, Emotions, Stress & Sleep (ACES) study (conducted between April – December 2017) and 2) Diet, Exercise, Stress, Emotions, Speech, and Sleep (DESTRESS) study (conducted between May – August 2018). Both studies used intensive longitudinal designs with repeated EMA to measure self-reported stress and sleep, and actigraphy to estimate objective sleep.

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ORIGINAL ARTICLE

Bi-directional relations between stress and self-reported and actigraphy-assessed sleep: a daily intensive longitudinal study

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Abstract

Study Objectives: Stress is associated with poor and short sleep, but the temporal order of these variables remains unclear. This study examined the temporal and bi-directional associations between stress and sleep and explored the moderating role of baseline sleep complaints, using daily, intensive longitudinal designs.

Methods: Participants were 326 young adults (M_{age} = 23.24 ± 5.46), providing >2,500 nights of sleep altogether. Prospective total sleep time (TST), sleep onset latency (SOL), wake after sleep onset (WASO), and sleep efficiency (SE) were measured using actigraphy and sleep diaries. Perceived stress was reported three times daily between: 11:00–15:00, 15:30–19:30, and 20:00–02:00. Sleep complaints were measured at baseline using the PROMIS sleep disturbance scale. Within- and between-person sleep and stress variables were tested using cross-lagged multilevel models.

Results: Controlling for covariates and lagged outcomes, within-person effects showed that higher evening stress predicted shorter actigraphic and self-reported TST (both $p < .01$). Conversely, shorter actigraphic and self-reported TST predicted higher next-day stress (both $p < .001$). Longer self-reported SOL and WASO (both $p < .001$), as well as lower actigraphic ($p < .01$) and self-reported SE ($p < .001$), predicted higher next-day stress. Between-person effects emerged only for self-reported TST predicting stress ($p < .01$). No significant results were found for the moderating role of baseline sleep complaints.

Conclusions: Results demonstrated bi-directional relations between stress and sleep quantity, and a consistent direction of worse sleep quantity and continuity predicting higher next-day stress. Results highlighted within-individual daily variation as being more important than between-individual differences when examining sleep and daytime functioning associations.

Statement of Significance

This study examined the temporal and bi-directional associations between stress and sleep (self-report and objective estimates) in a large sample of young adults using one of the strongest tests of directionality possible in observational designs (daily, intensive longitudinal design). Even after accounting for covariates and previous-night outcomes, higher evening stress predicted subsequent shorter sleep quantity, and shorter sleep quantity and continuity predicted higher next-day stress. These findings highlight the vicious daily cycle between high stress and short or discontinuous sleep, which may increase the risk or accelerate the progression of mental and physical disorders. In addition to the behavioral indices of sleep, future research should explore the associations between daily stress and sleep architecture.

Key words: stress; sleep continuity; sleep quantity; daily design

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Introduction

Stress and sleep are two important determinants of health and well-being, both linked with health outcomes including cardiovascular diseases, diabetes, depression, and anxiety [1–6]. Current understanding of the associations between perceived stress and sleep is primarily based on studies that examined between-person differences, such as cross-sectional associations and between-group comparisons [7–13]. Despite daily variations in both stress and sleep within individuals, whether stress and sleep bi-directionally influence each other on a day-to-day basis is unclear. This study aimed to test the temporal and bi-directional associations between stress and both self-reported and actigraphic sleep across 12 days. Daily repeated ecological momentary assessments (EMA) used in this study provide a stronger test of directionality between daily stress and sleep in a naturalistic environment than cross-sectional studies [14].

Stress and sleep

Cross-sectional studies show a consistent association between high perceived stress and poor, disturbed, or short sleep, particularly self-reported sleep [7–13]. For example, employees who experienced higher work-related stressors that affected their family relationships reported lower sleep sufficiency, poorer sleep quality, more insomnia symptoms, and had shorter actigraphic sleep duration [9]. Similarly, some prospective longitudinal studies have demonstrated that greater psychosocial stress, such as more stressful life events, stress-related cognitive intrusion, and work-related stressors, is associated with the development and maintenance of insomnia [15, 16]. However, due to the lack of daily stress and sleep, these studies do not inform whether daily, dynamic changes in stress influence sleep or vice versa.

To date, only a few studies examined the stress–sleep association on a daily basis. On stress predicting subsequent sleep, a 42-day study showed that higher severity of stress or worries at bedtime, but not the average stress across the day, predicted poorer self-reported sleep quality that night [17]. Similarly, a seven-day study using actigraphic sleep measures in young adults found that on days of higher-than-usual stress, those with high childhood family risk had shorter actigraphic total sleep time (TST) [18].

Few studies tested the bi-directional associations between stress and sleep [19], with even fewer studies using objective estimates of sleep [20]. In an 8-day study in working parents [19], within-person effects showed that greater work-related stress predicted longer self-reported sleep onset latency (SOL) that night, but the reverse direction was nonsignificant (i.e. longer SOL the previous night did not predict higher next-day stress). Further, self-reported poorer sleep quality and shorter duration predicted higher next-day work-related stress, whereas the opposite direction was nonsignificant. When actigraphic sleep measures were utilized, Doane and Thurston [20] found a significant bi-directional association between perceived stress and TST in adolescents. Specifically, higher perceived stress predicted shorter TST that night, and shorter TST predicted higher next-day perceived stress. Finally, lower sleep efficiency (SE) predicted higher next-day stress, but not the reverse direction.

These inconsistent findings show the complexity of the bi-directional associations and temporal order between stress and sleep, which may differ across sleep parameters (i.e. SOL, TST, and SE) and measurement (i.e. self-report vs. actigraphy). Despite differences in self-reported and objectively measured sleep parameters [21], current literature increasingly recognizes both as representing unique features of sleep and its associated experiences. Thus, there is a need for daily studies on stress and sleep that incorporate both objective and self-reported sleep measures.

Current study

The primary aim of this study is to examine the bi-directional, temporal associations between daily stress and sleep across 12 days, using both objective actigraphic and self-report measures of sleep. It was hypothesized that: (1) Higher evening stress would predict shorter sleep duration (TST) and worse sleep continuity (i.e. longer SOL, higher wake after sleep onset [WASO], and lower SE [22]) on the same night. (2) Shorter sleep duration and worse sleep continuity would predict higher next-day stress. These a priori hypotheses were preregistered on the Open Science Framework on July 7, 2017, prior to the completion of data collection (https://osf.io/h47yb/).

As a secondary exploratory aim, this study examined whether baseline levels of sleep complaints would moderate the prospective associations between nightly sleep and next-day stress. We hypothesized that individuals with greater sleep complaints at baseline would report higher stress following a night of shorter or lower than usual sleep duration and continuity. This hypothesis was made based on the consistent associations between sleep complaints and unhelpful thoughts and beliefs about sleep (e.g. catastrophizing about the consequences of poor sleep on daytime functioning) [23, 24]. Thus, individuals with high sleep complaints may be more vulnerable to the effects of discontinuous or short sleep the previous night and experience higher stress the following day.

Methods

Participants

Participants in this study were drawn from two studies: (1) The Activity, Coping, Emotions, Stress, and Sleep (ACES) study and (2) Diet, Exercise, Stress, Emotion, Speech, and Sleep (DESTRESS) study. Both studies utilized similar eligibility criteria, daily EMA designs, and recruitment strategies (see Figure 1). The main differences were the number of days observed (12 vs. 7 days for ACES and DESTRESS, respectively) and the age range (see Figure 1). The final sample consisted of 326 participants (191 from ACES and 135 from DESTRESS). A priori power analyses conducted in G*Power [25] showed a sample of 60 participants (assuming 75% completion rate) provide 80% power to detect a small-medium effect size at the within-person level. A larger sample was collected to support other aims and subgroup analyses not related to the current paper. ACES was conducted from April 2017 to December 2017, whereas DESTRESS was conducted from May 2018 to August 2018. Figure 1 shows a participant flow chart.

Design and procedure

All procedures were approved by the Monash University Human Research Ethics Committee. Both studies employed daily,

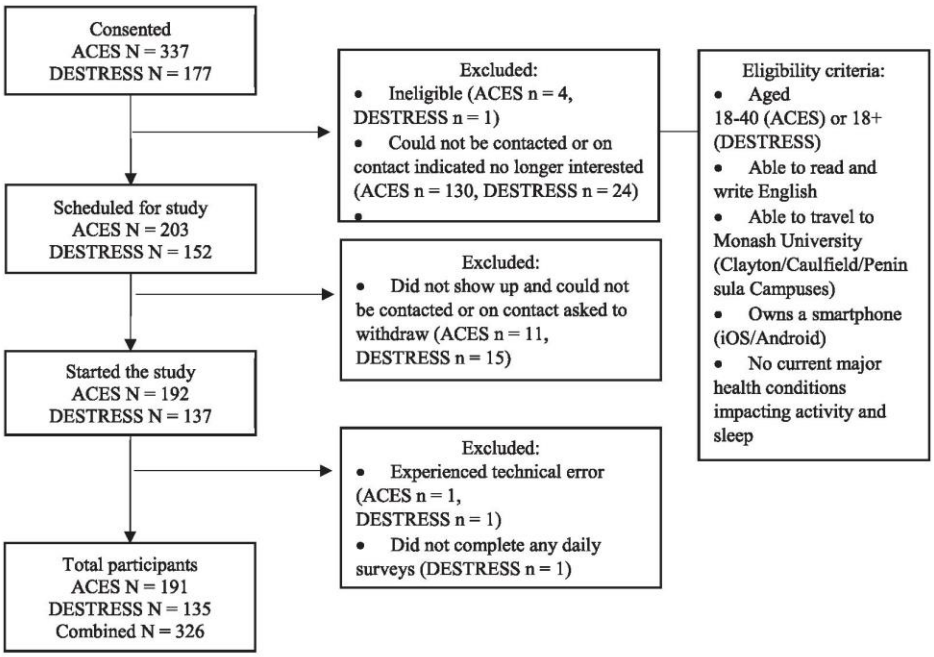

Figure 1. Summary of recruitment process. The ACES study recruited from April 13, 2017, to December 5, 2017. The DESTRESS study recruited from May 22, 2018, to August 13, 2018.

intensive, longitudinal design with repeated EMA. Through EMA, participants report their real-time experiences in their natural environment, which maximizes ecological validity and reduces recall biases [26]. By centering within-person data on an individual's average across time, repeated assessments across days allowed participants to serve as their own control, providing rigorous testing of the temporal relations between stress and sleep.

Most participants participated in both studies during school or semester periods. Participants first completed a baseline questionnaire consisting of demographic information, covariates, and other measures related to the overall study. For ACES, participants started on a Thursday or Friday and end on a Monday or Tuesday, whereas DESTRESS started on any weekday. Participants then completed the daily EMA component where they wore an actigraphy device throughout the study period and completed three surveys per day in the mornings (11:00–15:00), afternoons (15:30–19:30), and evenings (20:00–02:00) via a mobile application (MetricWire). Specifically, participants reported their stress levels in the morning, afternoon, and evening surveys and completed sleep diary in the morning surveys within the stated time windows (see Figure 2). Automated reminders were sent to participants when surveys were available, and all surveys were closed outside their respective time windows to ensure participants reported their real-time experiences.

Measures

Sleep

Objective estimates of sleep were determined at 60-s epoch using ActiGraph wGT3X-BT, an actigraphy-device with good validity and reliability against polysomnography estimates [27]. The sleep data were scored using the ActiLife software (v.6.13.3) following an established protocol based on activity, light, and sleep diary as well as integrated approximations from the Cole–Kripke scoring algorithm [28]. The parameters included TST, SOL, WASO, and SE. For self-reported sleep, participants reported bed and rise times, SOL, the number of and total time of WASO as part of the daily morning survey. These items were adapted from the Consensus Sleep Diary [29].

Perceived Daily Stress

Perceived daily stress was measured three times a day (mornings, afternoons, and evenings as described above) using a single item ranging from 0 (Not at all stressful) to 10 (Very stressful) adapted from the Daily Inventory of Stressful Events scale [30, 31]. For example, "Since the afternoon survey (or since 3:30 pm if you did not do the afternoon survey), how stressful has your day been?". Morning and afternoon stress were averaged to create the composite of next-day stress. Evening stress was reported an average of 3.30 (*SD* = 1.60) h prior to actigraphic bedtime.

Sleep Complaints

Sleep complaints, or self-reported sleep disturbance symptoms within the past 7 days were measured at baseline using the eight-item, Patient-Reported Outcomes Measurement Information System (PROMIS) Sleep Disturbance Short-Form 8a scale [32]. Example item includes "My sleep was restless," and responses were rated on a 5-point Likert scale. The raw sum scores were converted to a standardized *T*-score following PROMIS guidelines which have a population mean of 50 and standard deviation of 10, with higher scores indicating greater sleep complaints or disturbance. This scale showed strong internal consistency reliability (Cronbach's α = .89 and α = .90 for ACES and DESTRESS, respectively).

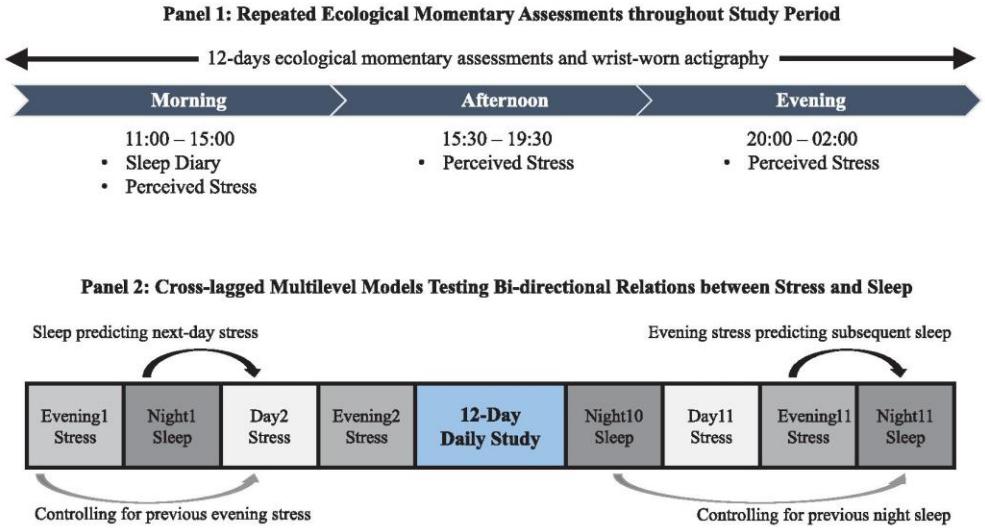

Figure 2. Panel 1 shows the repeated ecological momentary assessments throughout the study period, with participants completing three surveys a day and wearing an actigraphy watch. Panel 2 illustrates the cross-lagged multilevel models testing bi-directional relations of stress and sleep. All relationships were tested prospectively and controlled for lagged outcomes (i.e. previous evening stress or previous night sleep).

Covariates

Given that sociodemographic characteristics, smoking, and alcohol consumption may relate to stress and sleep, age (years) [33], sex (male/female) [33–35], education level (university graduate and below/postgraduate) [36, 37], race/ethnicity (White/Asian/other) [37], body mass index (BMI) [38, 39], employment status (working/not working) [36, 37], school status (in school/not in school) [36, 37, 40], smoking (current/former vs. never) [39, 41], and alcohol consumption (abstainers/moderate/at-risk) [39, 42] assessed at baseline were included as covariates. Alcohol consumption was measured using the World Health Organization alcohol use identification test [43]. Questions 9 and 10 were removed to exclude probing potentially sensitive questions regarding harms caused by participants' alcohol use. The first three items were used to classify participants as abstainers, moderate, or at-risk based on the National Institute on Alcohol Abuse and Alcoholism recommendations [44]. Daily covariates included study day and day of the week (weekend/weekday), given that individuals reported longer sleep duration, higher positive and lower negative affect during weekends as compared to weekdays [45, 46].

Analytic approach

Cross-lagged multilevel linear models were used to examine the bi-directional relationship between stress and sleep, which tested between- and within-person effects as well as fixed and random effects. All models were estimated using restricted maximum likelihood [47]. A homogenous, independent, residual covariance matrix was used given autoregressive effects were explicitly modeled by including lagged variables as described below. Previous study has shown the association between sleep and waking health behaviors (e.g. caffeine and alcohol consumption) was the strongest in weekly patterns compared to immediate influence [48]. Thus, this study validated the appropriate number of lags to be included in the models through step-wise addition of lags (e.g. first to fourth order lags stress and sleep variables) and compared the models through Bayesian information criterion (BIC). All analyses were run in R software (v.3.4.4) [49], using lme4 v1.1–13 (to estimate the models) [50] and lmerTest v2.0–33 (to estimate degrees of freedom and *p*-values) [51].

To provide a strong test of directionality, all relationships were tested prospectively and controlled for lagged outcomes (see Figure 2). Specifically, the first set of models tested daily evening stress as a predictor of both actigraphic and self-reported TST, SOL, WASO, and SE, while controlling for the previous night sleep outcomes. The second set of models tested actigraphic and self-reported TST, SOL, WASO, and SE as predictors of next-day stress (average from morning and afternoon surveys), while controlling for previous evening stress. All within-person variables were centered on the individual's own average. Effect sizes were calculated as follows. For every model, marginal and conditional R^2 values were calculated [52, 53]. The marginal R^2 is the proportion of total variance explained by the fixed effects, while the conditional R^2 is the proportion of total variance explained by both the fixed and random effects combined. For each predictor, nested models were run dropping predictors one at a time, and these were used to calculate the unique change in marginal and conditional R^2 values attributable to each predictor, which were then used to calculate a Cohen's f^2 type effect size for mixed models as:

$$\frac{R^2_{AB} - R^2_{A}}{1 - R^2_{AB}}$$

where R^2_{AB} is the marginal R^2 from the full model, and R^2_{AB} is the marginal R^2 from the restricted model dropping the relevant predictor.

For the exploratory analysis, the third set of models tested the interaction effects of baseline sleep complaint and daily sleep on next-day stress. Follow-up analyses for significant interactions were then examined using simple slopes tests for high (+1 SD from the mean) and low (–1 SD from the mean) sleep complaint [54].

Separate models were tested for each sleep parameter and type of sleep measure (i.e, actigraphic and self-reported). All

covariates and between- and within-person predictors were included as fixed effects. Intercepts, lagged-dependent variables, and within-persons predictors were included as random effects. All repeatedly measured predictors were separated into between- and within-person levels of analyses. Between-person levels examined the differences between individuals (i.e. the participants' own mean), whereas within-person levels tested deviations from the individual's own average levels calculated across the 7 or 12 days. Alpha was set at .01 to reduce false discovery and control Type 1 errors. All dependent variables were examined for normality violations. Both actigraphic and self-reported SOL, WASO, and SE showed skew, and they were winsorized and all but actigraphic SE were square-root transformed for all subsequent analyses.

The number of daily observations varied from 2040 to 2610. Sample sizes and number of observations varied across the models due to missing data, type of sleep measures (i.e. actigraphic and self-reported), and the cross-lagged design. For instance, when testing stress predicting same night sleep, evening stress on the last study day (i.e. day 12) was excluded due to missing same-night sleep variable. Likewise, when testing sleep predicting next-day stress, reports of next-day stress (morning and afternoon stress on the first study day) were excluded due to missing previous day stress and prior night sleep.

Results

Descriptive results

Table 1 shows the descriptive sociodemographic profile of the participants from ACES and DESTRESS. The participants were mostly young adult females, with a majority of university students and of Asian descent. Overall, the sample was healthy, with most participants never having smoked, being moderate drinkers, and having an average BMI within the healthy range for adults.

Examining the daily study variables (Table 2), there were low missing daily surveys on average. Comparing across days and surveys, there were more missing evening surveys compared to mornings and afternoons (Figure S1). Rates of missing surveys were consistent across days except for Wednesday and Thursday evenings for ACES due to the lagged design.

On average, participants reported relatively low stress levels throughout the mornings, afternoons, and evenings. On average, participants slept 7.32 and 7.80 h based on actigraphic and self-reported, respectively, both of which are within the recommended hours of sleep duration for adults [55]. Large discrepancies were observed between actigraphic and self-reported SOL and WASO. Nonetheless, both actigraphic and self-reported SE were above the 85% threshold, indicating that participants, on average, were sleeping well [56].

Evening stress predicting same-night sleep

All models with step-wise addition of evening stress lags were compared through BIC, and results showed that the first-order lag model was the most appropriate model with lowest BIC values. Table 3 shows the adjusted cross-lagged multilevel models of evening stress predicting same-night actigraphic and self-reported TST, SOL, WASO, and SE. Even after controlling for 11 covariates and lagged outcomes, within-person effects showed that one-unit higher evening stress (out of a 0 to 10-point scale) than average significantly predicted a –0.05 h (3-min) shorter actigraphic and self-reported TST. However, evening stress was not a significant predictor of actigraphic and self-reported SOL, WASO, and SE. No significant relations were found for between-person stress and actigraphic and self-reported sleep.

Table 1. Descriptive results for demographic variables by study

Participant characteristic	ACES (N = 191)	DESTRESS (N = 135)	P-value
Age, M (SD)	22.55 (4.13)	24.76 (7.51)	<.001
Body mass index (BMI), M (SD)	22.30 (3.59)	22.63 (3.51)	.40
Female, N (%)	127 (66.50)	102 (75.60)	.86
Race/ethnicity, N (%)			.16
White/European	44 (23.20)	42 (31.10)	
Asian	111 (58.40)	65 (48.10)	
Others	35 (18.40)	28 (20.70)	
Level of education, N (%)			.02
Undergraduate and below	139 (72.80)	82 (60.70)	
Postgraduate	52 (27.20)	53 (39.3)	
School status, N (%)			<.001
In school	175 (91.60)	100 (76.30)	
Not in school	16 (8.40)	31 (23.70)	
Work status, N (%)			.08
Working	61 (31.90)	55 (42.00)	
Not Working	130 (68.10)	76 (58.00)	
Smoking status, N (%)			.04
Current	2 (1.10)	2 (1.50)	
Former	5 (2.60)	12 (8.90)	
Never	183 (96.30)	121 (89.60)	
Alcohol risk, N (%)			.05
Abstainer	35 (18.40)	39 (29.10)	
Moderate	128 (67.40)	74 (55.20)	
At-risk	27 (14.20)	21 (15.70)	

Table 2. Means and standard deviations for main variables by study

Variables	ACES (N = 191)	DESTRESS (N = 135)	p-value
Stress			
Morning	2.08 (1.50)	2.06 (1.65)	.91
Afternoon	2.47 (1.65)	2.22 (1.54)	.17
Evening	2.32 (1.60)	2.10 (1.61)	.22
Actigraphic sleep			
Total sleep time (hours)	7.32 (0.97)	7.33 (0.92)	.91
Sleep onset latency (mins)	7.14 (4.24)	5.61 (4.66)	.01
Wake after sleep onset (mins)	57.55 (28.19)	52.95 (24.18)	.22
Sleep efficiency (%)	87.27 (4.98)	88.52 (4.45)	.90
Time in bed (hours)	8.40 (1.11)	8.31 (1.03)	.56
Self-reported sleep			
Total sleep time (hours)	7.86 (0.94)	7.75 (1.14)	.34
Sleep onset latency (mins)	28.72 (35.36)	27.68 (34.26)	.79
Wake after sleep onset (mins)	9.64 (12.14)	9.84 (14.30)	.22
Sleep efficiency (%)	93.00 (0.05)	93.00 (0.06)	.06
Time in bed (hours)	8.46 (1.15)	8.32 (1.08)	.27
Sleep Disturbance Index (T-score)	46.89 (8.15)	48.50 (6.10)	.05
Proportion of Missing Daily Surveys	0.08 (0.09)	0.13 (0.13)	<.001

Note. The actigraphic and self-reported sleep efficiency, sleep onset latency, and wake after sleep onset presented are raw and untransformed values. *p*-Values are based on independent samples *t*-tests after first averaging values for each participant.

Table 3. Multilevel modeling examining evening stress as a predictor of actigraphic and self-reported sleep (*N* = 326)

	TST	SOL	WASO	SE
Between-person effects				
Actigraphic	−0.03, < .01	−0.01, < .01	−0.14, 0.01	0.39, 0.01
	[−0.10, 0.05]	[−0.09, 0.07]	[−0.29, 0.01]	[−0.02, 0.08]
Self-reported	−0.06, <.01	0.07, <.01	−0.04, <.01	−0.27, <.01
	[−0.14, 0.02]	[−0.09, 0.24]	[−0.16, 0.07]	[−1.00, 0.46]
Within-person effects				
Actigraphic	−0.05, 0.01*	−0.01, 0.02	−0.02, 0.01	−0.02, 0.01
	[−0.08, −0.01]	[−0.05, 0.02]	[−0.07, 0.03]	[−0.15, 0.11]
Self-reported	−0.05, 0.02 *	0.02, < .01	0.01, 0.01	−0.22, 0.01
	[−0.08, −0.01]	[-0.02, 0.06]	[−0.02, 0.06]	[−0.44, −0.01]

Note. Results are unstandardized regression coefficients, Cohen's f^2, [95% confidence intervals]. * $p < .01$, ** $p < .001$. TST, Total Sleep Time; SOL, Sleep Onset Latency (square-root transformed); WASO, Wake After Sleep Onset (square-root transformed); SE, Sleep Efficiency (square-root transformed). Covariates were adjusted in all models including age, sex, body mass index, race, alcohol use, smoking status, education level, school status, employment status, day of week, and study days 1 to 12. The predictor for all models is within-person centered evening stress.

Table 4. Multilevel modeling examining actigraphic and self-reported sleep as predictors of next-day stress (N = 326)

	TST	SOL	WASO	SE
Between-person effects				
Actigraphic	−0.15, <.01	−0.08, < .01	−0.12, 0.01	0.04, 0.01
	[−0.37, 0.07]	[−0.30, 0.14]	[−0.23, 0.00]	[−0.01, 0.08]
Self-Reported	−0.23, 0.01*	0.04, <.01	−0.05, <.01	−0.01, <.01
	[−0.40, −0.06]	[−0.04, 0.12]	[−0.17, 0.08]	[−0.03, 0.01]
Within-person effects				
Actigraphic	−0.13, 0.04**	0.04, 0.01	0.02, 0.01	−0.02, 0.02*
	[−0.08, −0.01]	[0.00, 0.08]	[−0.01, 0.06]	[−0.04, −0.01]
Self-Reported	−0.15, 0.07**	0.07, 0.05**	0.07, 0.01**	−0.02, 0.06**
	[−0.20, −0.10]	[0.03, 0.11]	[0.03, 0.10]	[−0.03, −0.02]

Note. Results are unstandardized regression coefficients, Cohen's f^2, [95% confidence intervals]. Covariates were adjusted in all models including age, sex, body mass index, race, alcohol use, smoking status, education level, school status, employment status, day of week, and study days 1 to 12. The outcome in all models is next-day stress.
TST, total sleep time (square-root transformed); SOL, Sleep Onset Latency (square-root transformed); WASO, Wake After Sleep Onset (square-root transformed); SE, sleep efficiency (square-root transformed).
* $p < .01$, ** $p < .001$.

Sleep predicting next-day stress

All models with step-wise addition of sleep variable lags were compared through BIC, and results showed that the first-order lag was the most appropriate model with lowest BIC values. The adjusted models of actigraphic and self-reported TST, SOL, WASO, and SE predicting next-day stress are summarized in Table 4. At the between-person level, shorter self-reported TST significantly predicted higher next-day stress ($b = -0.23$). No significant relations were found between other actigraphic and self-reported sleep parameters and next-day stress. Similarly, at the within-person level, shorter actigraphic and self-reported TST predicted higher next-day stress ($b = -0.13$ and -0.15, respectively). Longer self-reported SOL and WASO (both $b = 0.07$) predicted higher stress the next day. Lower actigraphic and self-reported SE also predicted higher next-day stress (both $b = -0.02$). In additional exploratory analyses, a quadratic relationship between TST (actigraphic and self-reported) and next-day stress was tested to examine whether sleep duration demonstrated a J-shaped association with stress. All quadratic effects were not significant and are not reported. Further, sensitivity analyses revealed that within-person effects of the relations between self-reported WASO and next-day stress, as well as actigraphic SE and next-day stress, were significantly stronger during weekends compared to weekdays (both $p < .01$). All other stress–sleep relations were not significantly different during weekdays vs. weekends.

Moderating role of baseline sleep complaint

Overall, baseline sleep complaint did not significantly moderate the effects of either actigraphic or self-reported sleep on next-day stress. However, we did observe an interaction between baseline sleep complaint and actigraphic and self-reported TST on next-day stress in the hypothesized direction (both $b = -0.01$, $p = .03$, nonsignificant on a priori alpha of .01). Figure 3 shows the simple slopes of the relations between actigraphic TST and next-day stress at high and low baseline sleep complaint. For those with greater baseline sleep complaint, days with shorter-than-average actigraphic TST were associated with higher perceived stress (compared to those with lower levels of baseline sleep complaint).

Discussion

This study investigated the bi-directional, temporal associations between daily stress and sleep, with sleep measured using both actigraphy and self-report measures. We also explored the moderating role of baseline sleep complaint on the daily sleep–stress

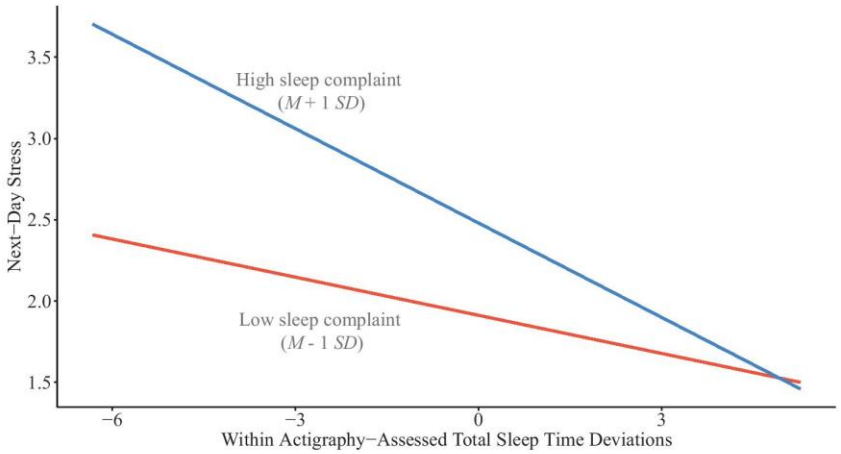

Figure 3. Simple slopes plot for next-day stress by within-person actigraphic total sleep time for high and low baseline sleep complaint.

relationships. Findings showed a bi-directional relationship between sleep duration and stress: higher evening stress predicted shorter actigraphic and self-reported TST, whereas shorter actigraphic and self-reported TST predicted higher next-day stress. Data in this study supported poorer sleep continuity (i.e. longer self-reported SOL and WASO, as well as lower actigraphic and self-reported SE) as predictors of higher next-day stress, but evening stress did not predict same-night sleep continuity. No significant relations were found for the moderating role of baseline sleep complaint on the relations between sleep and next-day stress, although some trend in the hypothesized direction was noted.

Between- versus within-person effects

First and foremost, in this study, only shorter self-reported TST significantly predicted higher next-day stress on the average levels (i.e. between-person effects). The weak between-person stress–sleep relationship highlighted the importance of considering each individual's daily within-person experience. These within- and between-person effects are entirely independent of each other, but each provides unique information. The lack of between-person effects suggests that those who typically experience higher stress compared to others do not necessarily have more disturbed sleep on average; and vice versa. The within-person effects discussed below should be interpreted, such that regardless of a person's *average* sleep and stress levels, days with greater *changes* in sleep or stress were associated with greater changes in the other.

Daily evening stress predicting sleep

Comparing among the number of evening stress lags (i.e. first- to fourth order lags), results indicated that first-order evening stress was the best predictor of subsequent sleep. Individuals who experience higher than usual stress on a given evening had shorter sleep that night (both actigraphic and self-reported), even after controlling for covariates and previous night TST. These results support previous daily studies showing decreased objectively measured sleep duration following higher than usual stress days [20] and strengthened the temporal directionality between evening stress and sleep. In contrast to the study by Lee and colleagues in working adults [19], the current findings showed evening stress as a significant predictor of shorter self-reported TST. Differences between our results and Lee and colleagues' could be due to the sampled population, such that working adults have fixed wake and sleep schedule and different types of stressors as compared to mostly university students in this study [19]. Further, in Lee and colleagues, participants reported their prior-night sleep on the following evening (vs. the following morning in this study), which may have influenced the accuracy of report [19].

The effects of stress on sleep could be explained through the framework of hyperactivation of the hypothalamic–pituitary–adrenal axis and presleep cognitive arousals. For example, previous research has demonstrated that elevated evening cortisol and flatter diurnal cortisol slopes are associated with shorter sleep duration [57–59]. Experiencing high psychological stress in the evening or near bedtime can cause spikes in evening cortisol, which are associated with physiological arousal that can impair sleep. Further, the experience of evening stress may cause emotional and cognitive arousal to affect sleep. Previous studies have demonstrated an association between preoccupation with stress at bedtime and subsequent poorer sleep, such as rumination and "not letting go of problems" [10, 11], and presleep arousal significantly mediated the stress–sleep relationship [15]. It is also possible that experiencing higher evening stress (e.g. before an upcoming examination) may require greater effort and time to manage or accommodate the increased demands (e.g. spending time resolving the issue), thus delaying bedtime and reducing sleep duration. Together, preoccupation with the stressor may amplify physiological activation, emotional reactivity, and presleep arousals, thus delaying sleep onset and reducing sleep duration.

Surprisingly, higher evening stress did not predict subsequent actigraphic and self-reported sleep continuity. These findings differed from previous cross-sectional [9] and daily-diary [19, 20] studies demonstrating the association between higher stress and subsequent poorer sleep quality or continuity. As the previous studies examined the stress–sleep relationship in working adults [9, 19] or adolescents [20], the types of stressor

experienced may be different and may affect sleep quality or continuity differently compared to university students. As previously discussed, experiencing higher evening stress may require greater efforts to accommodate the increased demands and subsequently delay bedtime. For instance, a student who is stressed about completing assignments may stay up late resulting in a delayed bedtime and sleep deprivation. A delayed bedtime could result in a stronger sleep drive and reduced circadian alerting signal, thus leading to a shorter time to fall asleep and possibly a shorter sleep duration. Consistent with this interpretation, the findings showed that evening stress did indeed predict shorter TST. Another explanation may be that evening stress was reported on an average of 3 hours before bedtime, which may not capture the rumination of stressful experiences during or pre-bedtime that can prolong SOL.

Poor Sleep Predicting Next-Day Stress

Similar to the stress as predictor models, results showed that the first-order sleep lag was the best predictor of next-day stress. Findings showed a consistent relationship of shorter sleep duration and worse sleep continuity (actigraphic and self-reported) predicting next-day stress. These results are in accordance with previous daily studies using objective [20] and subjective measures of sleep [19] linking shorter TST with higher stress. In this study, self-reported longer SOL and WASO, as well as lower actigraphic and self-reported SE, predicted significantly higher stress the next day, extending the null findings from previous research [19].

One potential underlying mechanism is that discontinuous and short sleep may impair the emotional regulatory system, which is critical for regulating the negative emotional experiences caused by stressors. For instance, neuroimaging evidence shows decreased capacity in regulating negative emotional responses following sleep deprivation [60]. Specifically, there is significantly greater amygdala reactivity toward negative stimuli and lower functional connectivity between the amygdala and the medial prefrontal cortex (an area with projections to the amygdala which inhibits amygdala reactivity) in sleep-deprived individuals compared to controls [60]. Supporting this explanation, research has also shown that sleep-deprived individuals respond with greater psychological distress than well-rested individuals following exposure to minor (but not high-intensity) stressors [61]. This suggests that poor or discontinuous sleep may impair the emotional regulatory system and increase the likelihood of perceiving events or demands as stressful [61]. Hence, individuals with discontinuous and short sleep may react more strongly to daily stressors and perceive them as more severe.

Moderating role of sleep complaint

Results did not support the exploratory hypothesis that baseline levels of sleep complaint would moderate the sleep and next-day stress within-person relationships. However, we did observe a tendency for individuals with higher baseline sleep complaints to report higher stress the following day when they had shorter than usual TST (on both actigraphic and self-reported, both $p = .03$), compared to individuals with lower baseline sleep complaints. Although nonsignificant at the conservative alpha of .01, the results suggest that individuals with greater sleep complaints may be somewhat more vulnerable to the effects of short sleep on their stress levels following day. As the current sample consisted of relatively healthy individuals with low sleep complaints, these effects may be stronger in populations with higher sleep complaints and concerns (e.g. those with insomnia) and should be explored in future studies.

Limitations and strengths

Findings from this study should be interpreted considering several limitations. First, the nonsignificant findings could be partially due to a relatively low stress level in this sample, thus causing a floor effect. Second, like all other actigraphy-based studies, quiet wakefulness (i.e. lying on bed with eyes closed without activity) may be counted toward sleep, thus underestimating SOL and affect its associated results. Although partially addressed by including self-reported measures of sleep, self-reported SOL and TST are often overestimated [21]. Third, this study included mainly young, healthy university students with relatively low stress levels. Thus, these findings may not generalize to other individuals experiencing high stress (e.g. people with cancer), working adults with relatively fixed sleep and wake schedule, or individuals with more severe sleep problems (e.g. people with insomnia). Fourth, it is not possible to examine seasonal influence due to data collection was mostly carried out in one season: for ACES, most participants (85%) completed the study from April to October (mostly winter in Australia), while all participants completed the study in winter for DESTRESS. Finally, the lack of in-home electroencephalography sleep monitoring means that it is not possible to explore how stress and sleep architecture may be related on a daily basis. Future daily studies are needed that explicitly measure and compare sleep during restricted (e.g. semester term) and unrestricted (e.g. vacation; weekend), which may influence sleep/wake schedule and stress levels [62].

Despite these limitations, this study had notable strengths. The core strength of this study is the use of intensive, longitudinal daily design with EMA as well as both objective and subjective estimates of sleep, which extended findings from previous cross-sectional and daily studies. The use of repeated, real-time assessments maximize ecological validity and reduce recall biases [26]. Further, this study employed vigorous methodologies to test the temporal and bi-directional relations between stress and sleep by including lagged outcomes and separating between- and within-person effects in the analyses along with effect sizes, thus strengthening the confidence in the findings [14]. Together, this study provides one of the strongest tests of directionality and causality possible in observational designs, extending the current literature by demonstrating the bi-directionality between daily stress and nightly sleep in a large sample of young adults.

Conclusion

Using a daily repeated measures design with rigorous analytical methods, this study demonstrated a bi-directional relationship between stress and TST for both objective and subjective sleep measures. Although stress did not predict subsequent sleep continuity, the opposite direction emerged, such that longer SOL,

WASO, and poorer SE predicted higher next-day stress. The weak between-person effects highlighted the importance of considering the daily variations of stress and sleep of each individual.

The magnitude of bi-directional effects between stress and sleep may be relatively small from one day to the next. However, the cumulative impact of potentially vicious cycles of high stress and short/poor sleep is not to be underestimated given the importance of both stress and sleep to physical and mental health [1–6]. On a positive note, the bi-directional links between lower daily stress with longer sleep duration are consistent with the notion of good sleep as a source of resilience and replenishment of energy and emotional regulation [19, 63, 64].

Considering these findings, behavioral interventions that can be embedded and applied in daily routines to either reduce modifiable stressors or improve sleep could help break the vicious cycles. Encouraging awareness of stress levels and sleep quantity/continuity in everyday life may help adopt timely countermeasures and coping, which may be especially helpful for individuals who often experience significant stressors or changes in sleep/wake routines.

Supplementary Material

Supplementary material is available at *SLEEP* online.

Acknowledgments

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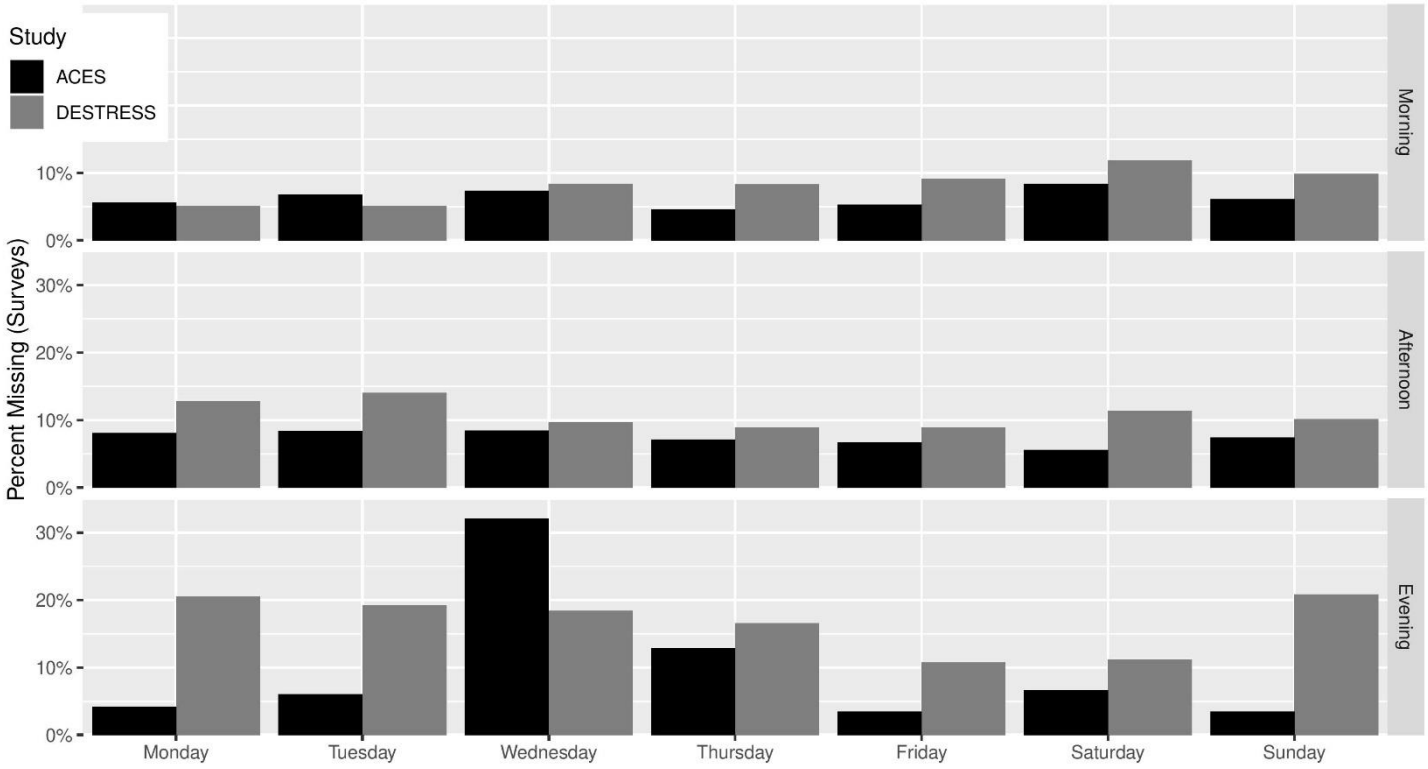
Study
ACES
DESTRESS
Percent Missing (Surveys)
Morning
Afternoon
Evening
0%
10%
20%
30%
Monday
Tuesday
Wednesday
Thursday
Friday
Saturday
Sunday

CHAPTER 3:

Daily Coping Moderates the Relations Between Stress and Actigraphic Sleep: A Daily Intensive Longitudinal Study with Ecological Momentary Assessments

Preface to Chapter 3

Chapter 2 demonstrated that higher within-person evening stress levels (i.e., higher than usual evening stress) predicted shorter self-reported and actigraphic sleep that night. This chapter builds upon the findings from **Chapter 2** by exploring modifiable factors that can moderate the prediction of sleep by stress, thus deepening our understanding of the stress-sleep associations.

This chapter explored *whether* and *which* daily coping strategies (i.e., problem-focused coping, emotional approach coping, and avoidance coping) moderate the associations of evening stress with sleep duration and quality, using a daily, intensive longitudinal design with repeated ecological momentary assessments. Data for this paper came from ACES and DESTRESS studies.

This chapter has been submitted to ***Sleep Medicine*** and is presented in manuscript form. The status of this manuscript is currently a revise and resubmit.

Daily Coping Moderates the Relations between Stress and Actigraphic Sleep: A Daily Intensive Longitudinal Study with Ecological Momentary Assessments

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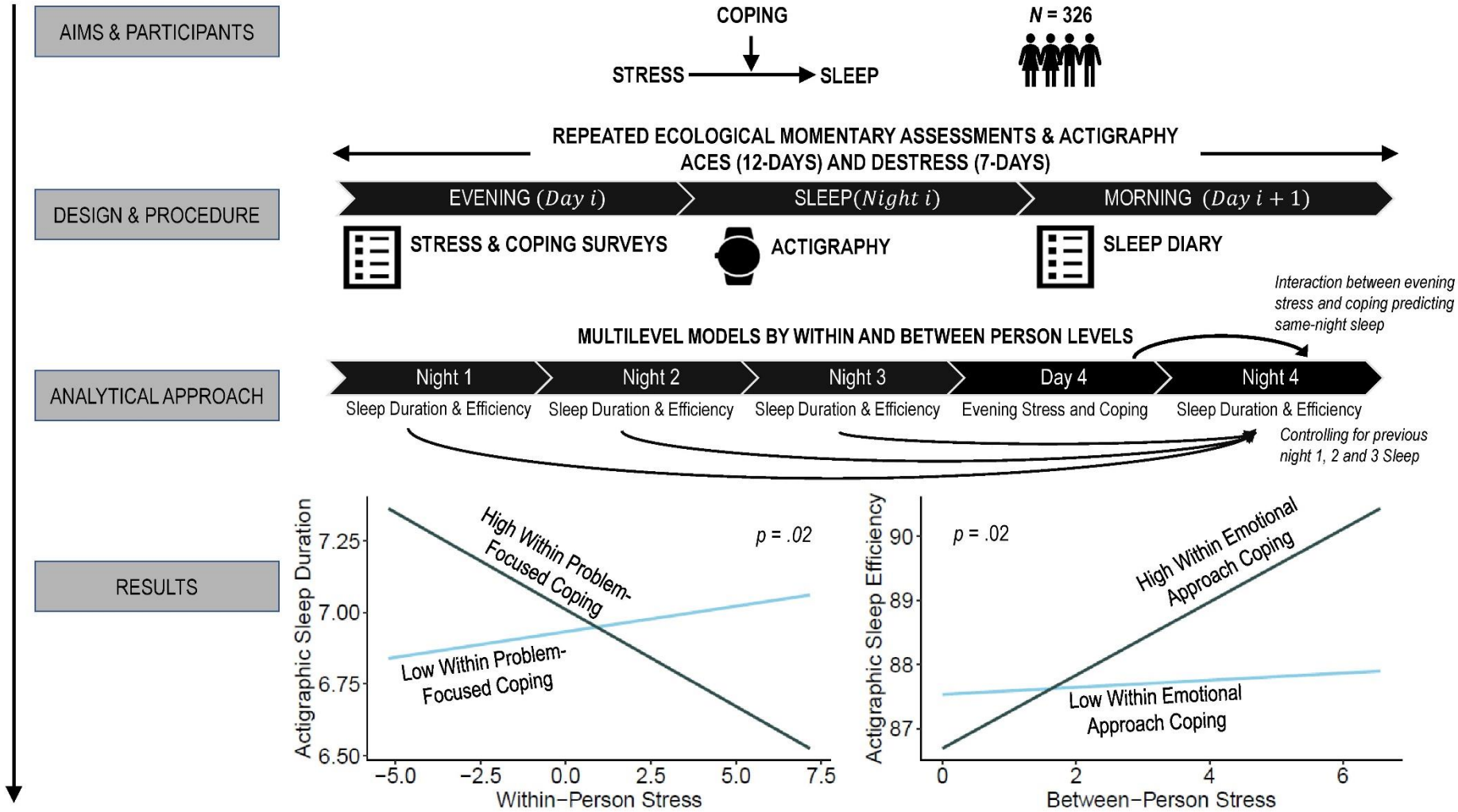
Graphical Abstract



Abstract

Background: Theoretical models argue that coping reduces stress responses, yet no studies have tested whether coping moderates the prospective stress effects on sleep in daily life.

Purpose: This study tested if coping moderates the stress-sleep association using a daily, intensive longitudinal design.

Methods: 326 young adults (M_{age}=23.24±5.46) reported perceived stress and coping (problem-focused, emotional-approach, and avoidance) every evening between 20:00–02:00, providing over 2400 nights of sleep data and 3000 stress surveys from all participants. Actigraphy and sleep diaries measured total-sleep-time and sleep efficiency. Multilevel models tested the interaction effects of within- and between-person stress and coping on sleep.

Results: Within-person problem-focused and emotional-approach coping moderated the within-person stress effects on actigraphic total-sleep-time (both p=.02); higher stress predicted shorter total-sleep-time only during high use of problem-focused coping or emotional-approach coping (both p=.01). Between-person avoidance moderated the between-person stress effect on actigraphic total-sleep-time (p=.04); higher stress predicted shorter total-sleep-time for high avoidance coping (p=.03). Within-person emotional-approach coping buffered the between-person stress effect on actigraphic sleep efficiency (p=.02); higher stress predicted higher sleep efficiency for high emotional-approach coping (p=.04).

Conclusions: This study showed that daily coping moderates the effects of evening stress on sleep that night. More efforts to cope with stress before bedtime had a short-term cost of shorter sleep that night. However, high use of emotional-approach coping buffered the impact of stress to promote sleep quality.

Key Words: Multilevel Model; Daily Stress; Daily Sleep; Daily Coping; Ecological Momentary Assessments

Introduction

The stress-sleep relationship is well established in cross-sectional studies[1] with emerging research extending the knowledge base to examine how stress affects sleep on a day-to-day basis. Using ecological momentary assessments, a few recent studies showed higher daily stress predicted shorter self-report and actigraphic sleep duration.[2,3] However, what modifiable factors may moderate the effects of daily stress on sleep are poorly understood.

When faced with stress, people often engage coping strategies—behaviors or strategies that attempt to address real or perceived demands and stressors.[4] Coping strategies can be broadly categorized as aiming to approach or avoid a stressor, paralleling behavioral approach/inhibition systems work.[5] Approach-oriented coping can be further distinguished as coping strategies aimed at directly resolving the stressor (i.e., problem-focused coping [PFC]) or strategies that actively address emotional responses to the stressor (i.e., emotional-approach coping [EAC]).[4,6] Theoretical models highlight the role of coping in moderating stress responses,[4] and reviews of empirical studies demonstrate that often approach-oriented coping is associated with better and avoidance-oriented coping (AVC) with worse psychological well-being.[7-9] Thus, coping may moderate how stress affects sleep, an important aspect of well-being, which could inform interventions on what coping strategies may ameliorate or worsen the effects of stress on subsequent sleep.

Few studies have examined the association between coping and sleep. Cross-sectional studies show a consistent link between AVC with poor or short sleep, while results for approach-oriented coping and sleep are inconclusive.[10-12] For instance, a 7-day study in adolescents show that approach-oriented coping was associated with delayed bedtime and increased daytime sleepiness, and AVC was associated with shorter actigraphic sleep duration, more wake after sleep onset, and more daytime sleepiness.[10] Currently available prospective studies also showed similar results, where higher use of AVC predicted poorer

sleep quality or higher sleep interference, whereas results for approach-oriented coping were inconclusive.[13,14] For example, in patients with breast cancer, higher use of AVC was associated with more sleep problems and longer sleep onset latency, whereas approach-oriented coping was unrelated to sleep.[13] In patients with prostate cancer, higher AVC and lower approach-coping were related to higher self-reported sleep problems, and higher approach-coping was related to shorter self-reported sleep onset latency.[13]

To our knowledge, only two studies examined whether coping moderates the effects of stress on sleep, and neither utilized daily measures of stress or coping.[15,16] During a high-stress period, university students who used high emotion-focused coping had shorter actigraphic total sleep time and reported poorer sleep quality compared to low emotion-focused coping; further, in both high- and low-stress periods, individuals who used higher PFC had longer actigraphic total sleep time.[15] Similarly, in older adolescents, PFC buffered the effects of stress on sleep initiation measured using both actigraphy and self-report.[16] This study also reported that EAC was associated with lower, whilst AVC was associated with higher pre-sleep arousal, a construct that mediated the effects of stress on sleep onset difficulties.[16] These findings suggest that high PFC and EAC and low AVC may be optimal for good sleep. However, research also shows that acute engagement with a task or stressor can be associated with at least a short-term physiological toll, with studies showing optimistic people both engaged more with difficult tasks[17] and short-term had worse immune function during conflicts.[18] Thus, it is possible that PFC and EAC, which promote engagement with stressors, may result in at least short-term costs to sleep.

Current study

The current literature has several gaps. First, there is a lack of understanding whether engaging in any coping strategies in response to daily stress can mitigate the deleterious effects of stress on sleep, especially in a naturalistic, day-to-day setting. Second, most studies are cross-sectional in nature. Stress, coping, and sleep are dynamic processes, and a cross-

sectional design cannot provide information regarding how these processes unfold over time. As noted, although PFC and EAC have been found to be associated with better sleep, no existing studies to our knowledge have examined daily stress and daily coping, which are needed to understand the immediate effects of coping and to examine not only differences between people but also within people across time. Finally, most studies examined only the broad construct of coping, so there is a need to examine different coping strategies (e.g., PFC, EAC, AVC) at the same time so their respective roles are better understood in the same context.

Given the aforementioned limitations, this study aimed to test whether and which daily coping strategies (i.e., PFC, EAC, and AVC) moderate the effects of evening stress on sleep duration and quality, using a daily, intensive longitudinal design with repeated ecological momentary assessments. It was hypothesized that on days where individuals used higher approach-oriented coping (i.e., PFC and EAC) and lower AVC, the effects of evening stress on subsequent sleep duration and quality (i.e., longer total sleep time and higher sleep efficiency, respectively) would be weaker compared to days of low approach-oriented or high AVC coping.

Methods

Participants

The current study follows the Strengthening The Reporting of Observational Studies[19] and Checklist for Reporting Ecological Momentary Assessments Studies [20] reporting guidelines. Participants were drawn from two studies with overlapping eligibility criteria, design, and recruitment strategies – 1) The Activity, Emotions, Stress, and Sleep (ACES) study, and 2) Diet, Exercise, Stress, Emotion, Speech, and Sleep (DESTRESS) study. DESTRESS was an extension of ACES, where it was conducted to recruit a larger sample to support other aims and subgroup analyses not related to this paper. The number of days observed on ACES and DESTRESS were 12 and 7 days, respectively. ACES was conducted

from April 2017 to December 2017, while DESTRESS was conducted from May 2018 to August 2018. Among 514 participants who consented (for both studies), 329 of them started the daily study (after excluding participants who are ineligible, could not be contacted, and asked to withdraw). The final sample consisted of 326 participants (ACES N = 191, DESTRESS N = 135), after excluding those who experienced technical errors and did not complete any of the daily surveys. Further details on the sample and flowchart are reported in our previous work.[3] *A priori* power analyses indicated that 60 participants (assuming 75% completion rate) provide 80% power to detect a small-medium effect size at the within-person level.

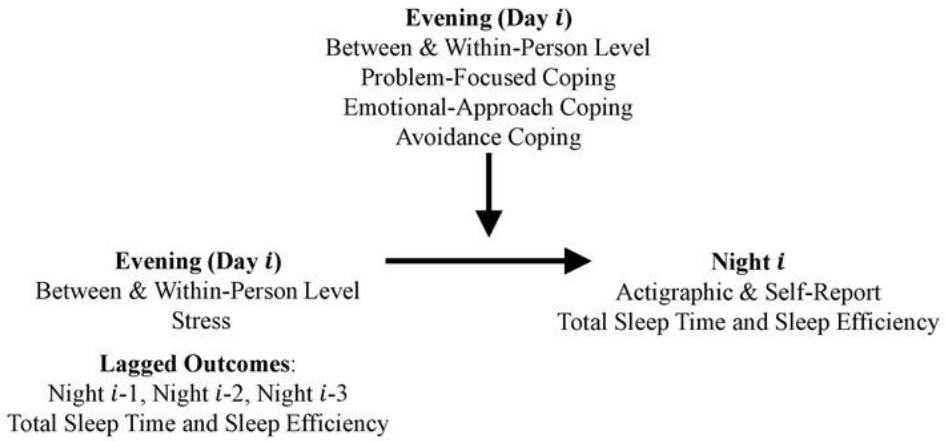
Design and Procedure

Monash University Human Research Ethics Committee approved all procedures (Project IDs #8245 & #12637) and all participants provided consent. Both studies used daily, intensive, longitudinal designs with repeated ecological momentary assessments, where participants can report their real-time experiences in their natural setting. This helps to maximize external validity, reduce recall biases, and provide strong tests of directionality in a naturalistic environment.[21] Further, through repeated assessments across the days and creating within-person data centered on the individual's average, allowed participants to serve as their own control (i.e., controlling for individual differences) and provided a strong and rigorous test of directionality.

First, participants completed a baseline survey consisting of demographic characteristics, covariates, and other measures related to the studies. Participants then attended an introductory session where they were demonstrated how to wear the actigraphy device and complete the daily surveys via a mobile application (i.e., MetricWire). Throughout the daily study period, participants completed their sleep diaries in the mornings when surveys are opened (anytime between 11:00 – 15:00) and reported their coping strategies for the day and perceived stress levels over the last few hours in the evenings

(20:00 – 02:00). Automated push notifications were sent to participants when surveys were available and reminders after every hour until completed. On average, participants completed the morning surveys 0.80 hours after the morning surveys are available and took approximately 2 minutes to complete. Evening surveys were completed approximately 3.06 hours prior to bedtime and took approximately 4 minutes to complete. Participants were only able to complete the surveys during the specified time-window to ensure real-time experiences are captured. Participants also wore the actigraphy device throughout the study period. Hypothesized moderating pathways, study design, and procedure are summarized in *Figure* 1.

Panel A



Panel B

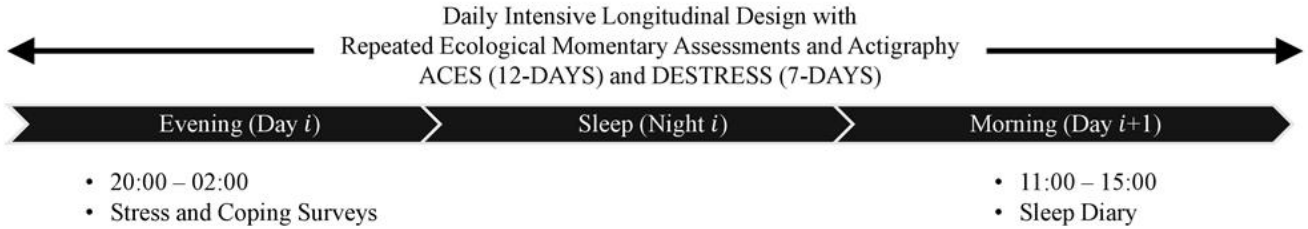


Figure 1

Panel A. The interaction between daily coping and evening stress predicting subsequent sleep, accounting for lagged outcomes (i.e., up to three previous nights of sleep).

Panel B. Repeated ecological momentary assessments and wrist-worn actigraphy throughout study period (ACES = 12 days; DESTRESS = 7 days). Participants report stress and coping strategies in the evening and complete sleep diary the next morning.

Measures

Sleep. Actigraphic sleep estimates (i.e., total sleep time, sleep onset latency, wake after sleep onset, and sleep efficiency) were scored in 60-second epochs collected from ActiGraph wGT3X-BT wrist-worn accelerometers, which have good validity and reliability against polysomnography estimates.[22] The sleep data were scored using the ActiLife software (v.6.13.3) with the integrated Cole-Kripke scoring algorithm,[23] as well as following an established protocol based on activity, light, and sleep diary. Self-report sleep items were adapted from the Consensus Sleep Diary,[24] where participants reported bed and rise times, sleep onset latency, and the number of and total time of wake after sleep onset.

Evening Stress. A single item ranging from 0 (Not at all stressful) to 10 (Very stressful), adapted from the Daily Inventory of Stressful Events scale,[25,26] was used. Specifically, "Since the afternoon survey (or since 3:30pm if you did not do the afternoon survey), how stressful has your day been?". We referred this as evening stress given that the survey covers primarily the evening period (and a small part of late afternoon), and surveys were opened at 20:00. To strengthen directionality of stress predicting *subsequent* sleep, only evening stress was examined in this study given the closest proximity to sleep.

Coping strategies. Coping strategies were measured daily in the evening surveys, asking participants what they have been doing to cope with any stress or hassles experienced *today*. PFC and AVC strategies were assessed using items from the active, planning, and mental disengagement subscales from the COPE inventory.[27] For PFC, the subscale consisted of 4-items in ACES (a subset of active and planning) and 8-items in DESTRESS, the full active and planning subscales. An example item is, "I tried to come up with a strategy about what to do". AVC was measured using the mental disengagement subscale consisting of 4 items from the COPE, e.g., "I daydreamed about other things than this". EAC strategies were measured using the Emotional Approach Coping Scale,[6] which combined two subscales with 4 items each assessing emotional processing and expression; for instance, "I take time to

figure out what I am really feeling" and "I let my feelings come out freely". All responses were recorded on a 4- and 7-point scale in ACES and DESTRESS, respectively. Given the difference in the units of measurement, the scores were z-scored in each study. For both studies, the omega coefficient for internal consistency reliability for all coping strategies at the between-person level were ≥ .84; within-person level reliability for all coping strategies is summarized in Table 1.

Covariates. Covariates were selected based on previous studies demonstrating their associations with stress and sleep.[28-32] Between-person covariates assessed at baseline included age (years), sex (female/male), education level (university graduate and below/postgraduate), race/ethnicity (White/Asian/Other), body mass index (BMI; kg/m^2), employment status (working/not working), school status (in school/not in school), smoking (current or former/ never), and alcohol consumption (abstainers/moderate/at-risk). Alcohol consumption was measured using the WHO Alcohol Use Identification Test,[33] using the first three items to classify participants as abstainers, moderate, or at-risk based on the National Institute on Alcohol Abuse and Alcoholism guidelines.[34] Daily covariates included study day and day of the week (weekend/weekday), as individuals tend to sleep shorter and report higher negative affect during weekdays compared to weekends.[35,36]

Analytic Approach

Multilevel linear models tested the interaction effects of evening stress and coping on sleep at the between- and within-person level, alongside fixed and random effects and effect sizes in R v.3.4.4.[37] Evening stress and coping strategies were separated into between-person (i.e., interindividual differences or the participants' own average) and within-person (i.e., deviations from participants' own average) levels of analyses. Fixed effects included all covariates and between- and within-person predictors, whereas random effects included intercepts, lagged outcome variables (i.e., sleep variables on the previous nights), and within-persons predictors.

All associations were tested prospectively and accounted for lagged outcomes (previous night sleep) to ensure a strong test of directionality. We tested the appropriate number of lagged nights to be included in the models (i.e., one to three lagged sleep outcomes) through model comparisons and Bayesian Information Criterion; the results showed that the model with up to three nights of lagged sleep was the best model. Thus, all models included previous night 1, 2 and 3 sleep outcomes. Our previous work showed that the first order lagged evening stress was the most appropriate model.[3] Non-significant interactions were excluded from the analyses, and significant interactions were further tested using simple slopes tests through ±1 SD from the mean of between or within-person coping.[38] Given the low within-person reliability for AVC (i.e., .52 and .36 for ACES and DESTRESS, respectively), within-person levels of AVC were dropped from all analyses and included only between-person level of AVC. Results from the unadjusted models are included in the electronic supplementary material 1.

Due to skewness, several variables were winsorized and transformed for subsequent analyses. Specifically, both actigraphic and self-reported sleep onset latency, wake after sleep onset were winsorized and square-root transformed. Self-reported sleep efficiency was winsorized and square-root transformed, and actigraphic sleep efficiency was winsorized.

Results

Descriptive Results

The sociodemographic profile and daily study variables from ACES and DESTRESS are summarized in Table 1. For baseline sociodemographic characteristics, participants were mostly Asian undergraduate students, with over half being female. Most participants reported never having smoked, being either abstainers or moderate drinkers, and having an average BMI within the healthy range for adults, suggesting an overall healthy sample.

For the daily variables, on average, participants reported low evening stress levels, and their actigraphic and self-reported total sleep time were within the recommended 7 to 9

hours of sleep duration for adults.[39] Further, both actigraphic and self-reported sleep efficiency were above 85%, indicating that participants were sleeping well on average.[40] There were low missing daily surveys on average.

Table 1. Descriptive *Results for Demographic and Daily Study Variables by Study (N=326)*

	ACES (*N*=191)	**DESTRESS (*N*=135)**	**ICC**	**No. of Obs**	***P***
Participant Characteristic	*M* (SD) / N (%)	*M* (SD) / N (%)			
Age	22.55 (4.13)	24.76 (7.51)	—	325	< .001
BMI	22.30 (3.59)	22.63 (3.51)	—	325	.40
Female (%)	127 (66.50)	102 (75.60)	—	326	.86
Race/Ethnicity (%)			—	325	.16
White/European	44 (23.20)	42 (31.10)	—		
Asian	111 (58.40)	65 (48.10)	—		
Others	35 (18.40)	28 (20.70)	—		
Undergrad and below (vs postgrad %)	139 (72.80)	82 (60.70)	—	326	.02
In school (vs not in school %)	175 (91.60)	100 (76.30)	—	322	<.001
Working (vs not working %)	61 (31.90)	55 (42.00)	—	298	.08
Smoking Status (%)			—	325	.04
Current	2 (1.10)	2 (1.50)	—		
Former	5 (2.60)	12 (8.90)	—		
Never	183 (96.30)	121 (89.60)	—		
Alcohol risk (%)			—	324	.05
Abstainer	35 (18.40)	39 (29.10)	—		
Moderate	128 (67.40)	74 (55.20)	—		
At-risk	27 (14.20)	21 (15.70)	—		
Daily Study Variables					
Evening stress	2.32 (1.60)	2.10 (1.61)	.34(66%)	3017	.22
Actigraphic Sleep					
Total sleep time (hours)	7.32 (0.97)	7.33 (0.92)	.27(73%)	2442	.91
Sleep onset latency (mins)	7.14 (4.24)	5.61 (4.66)	.17(82%)	2442	.01
Wake after sleep onset (mins)	57.55 (28.19)	52.95 (24.18)	.40(60%)	2442	.22
Sleep efficiency (%)	87.27 (4.98)	88.52 (4.45)	.42(58%)	2442	.06
Time in bed (hours)	8.40 (1.11)	8.31 (1.03)	.30(70%)	2442	.56
Bedtime (hours from 18:00)	6.61 (1.52)	6.41(1.51)	.56(44%)	2442	.33
Rise time (hours from 00:00)	9.01 (1.27)	8.72 (1.36)	.46(54%)	2442	.10
Self-Reported Sleep					
Total sleep time (hours)	7.86 (0.94)	7.75 (1.14)	.23(76%)	2966	.34
Sleep onset latency (mins)	28.72 (35.36)	27.68 (34.26)	.51(49%)	2983	.79
Wake after sleep onset (mins)	9.64 (12.14)	9.84 (14.30)	.33(67%)	2982	.90
Sleep efficiency (%)	0.93(0.05)	0.93 (0.06)	.44(55%)	2966	.91
Time in bed (hours)	8.46 (1.15)	8.32 (1.08)	.31(69%)	3003	.27
Bedtime (hours from 18:00)	6.58 (1.53)	6.44 (1.41)	.55(45%)	3004	.43
Rise time (hours from 00:00)	9.04 (1.25)	8.76 (1.38)	.46(54%)	3004	.05
Coping Strategies					
Problem-Focused (.78/.73)†	2.15 (0.62)	3.89 (1.09)	.55(45%)	2961	—
Emotional-Approach (.81/.80)†	2.32 (0.56)	3.79 (1.10)	.61(38%)	2971	—
Avoidance (.52/.36)†	2.02 (0.55)	3.04 (1.09)	.58(42%)	2968	—
Missing Daily Surveys (%)	0.08 (0.09)	0.13 (0.13)	—	—	<.001

Note. ICC = Intraclass Correlations. No. of Obs = Number of observations. Coping, actigraphic and self-reported sleep efficiency, sleep onset latency, and wake after sleep onset presented are raw values. P-values are based on independent samples *t*-tests after first averaging values for each participant. P-values for coping strategies were not included given the different units of measurement (1-4 in ACES; 1-7 in DESCTRESS). †= omega coefficient for internal consistency reliability at within-level by study (ACES/DESTRESS).

Moderating Role of Coping Strategies on Evening Stress and Total Sleep Time

The adjusted models of the interactions between coping and evening stress on actigraphic and self-reported total sleep time are summarized in the first two columns of Tables 2 (PFC), 3 (EAC), and 4 (AVC).

After controlling for covariates and lagged outcomes, there was a significant interaction between within-person PFC and within-person evening stress on actigraphic total sleep time (p = .02). Simple slopes analyses showed evening stress predicted actigraphic total sleep time on nights when participants used high levels of PFC (*Figure* 2, panel A). This indicates that on evenings with higher than *usual* stress, individuals who use higher than usual PFC (compared to their own means) had shorter subsequent actigraphic total sleep time. Although low PFC was not significantly different than zero, it was significantly different from high PFC.

Similarly, within-person EAC also moderated the relations between within-person evening stress and actigraphic total sleep time (p = .02). Simple slopes analyses (*Figure* 2, panel B) indicated a significant relationship between higher within-stress and lower actigraphic total sleep time for those who used high within-person EAC.

At the between-person level, there was a significant interaction between AVC and evening stress on actigraphic total sleep time (p = .04). Simple slopes analyses showed (*Figure* 2, panel C) a significant relationship between higher stress and shorter actigraphic total sleep time for those with overall high AVC. This indicates that for those who are generally stressed and typically use AVC generally had shorter actigraphic total sleep time.

No significant interactions between any coping strategies and stress levels on self-reported total sleep time were found on both between and within-person levels.

Moderating Role of Coping Strategies on Evening Stress and Sleep Efficiency

There was a significant within-person EAC and between-person evening stress interaction effect on actigraphic sleep efficiency (p=.02; Table 3). Simple slope analyses

revealed that individuals with higher average stress had higher actigraphic sleep efficiency than individuals with lower average stress on days they used higher than usual EAC (*Figure* 2, Panel D). No significant interaction effects between other coping strategies and stress emerged for both self-reported and actigraphic sleep efficiency at either between or within-person levels.

Exploratory Analyses

Given the significant interactions of PFC, EAC, and AVC and evening stress on actigraphic total sleep time, further exploratory analyses were conducted to examine bedtime. There was a significant interaction between evening stress and EAC on actigraphic bedtime at the within-person level (b = 0.07, p = .02, CI [0.01, 0.12], Cohen's f^2 < .01). Specifically, higher within-person stress predicted earlier bedtime for low within-person EAC (simple slope b = -0.05, p = .03). Although high EAC (simple slope b = 0.02, p = .31) was not significantly different than zero, it was significantly different from low EAC. No significant interaction effects were found for other coping strategies on either actigraphic or self-reported bedtime.

Further exploratory analyses were conducted on actigraphic sleep onset latency and wake after sleep onset for EAC, given its significant interactions with evening stress on actigraphic SE. There was a significant interaction between EAC and evening stress on actigraphic sleep onset latency (b = -0.15, p = .04, 95% CI = [-0.30, -0.01], Cohen's f^2 = 0.01) at the between-person level. Simple slopes analyses showed a significant negative relationship between evening stress and actigraphic sleep onset latency for those with high EAC (b = -0.14, p =.07), but a positive relationship for those with low EAC (b = 0.10, p = .21). Although neither of the simple slopes was significantly different from zero, they were significantly different from each other.

Further, there was a significant within-person EAC and between-person evening stress interaction effect on actigraphic wake after sleep onset (b = -0.17, p = .01, 95% CI = [-

0.30, -0.04], Cohen's f^2 <.01). Simple slopes analyses indicated that people with higher average evening stress had shorter actigraphic wake after sleep onset on days they used higher than average EAC (b = -0.22, p =.01), but not on days they used lower than average EAC (b = -0.04, p = .67).

Table 2. Multilevel Models Examining the Interaction Effects between Problem-Focused Coping and Evening Stress Predicting Actigraphic and Self-Reported TST and SE

	Actigraphic TST	**Self-Report TST**	**Actigraphic SE**	**Self-Report SE**
Between Person Effects				
PFC	-0.10, <.01 [-0.28, 0.07]	0.06, <.01 [-0.11, 0.23]	0.23, <.01 [-0.71, 1.16]	1.44, 0.01 [-0.17, 3.05]
Evening stress	-0.01, <.01 [-0.11, 0.08]	-0.05, <.01 [-0.15, 0.05]	0.29, <.01 [-0.23, 0.80]	0.10, <.01 [-0.81, 1.02]
Within Person Effects				
PFC	0.06, 0.04 [-0.07, 0.19]	-0.001, <.01 [-0.12, 0.12]	0.32, <.01 [-0.13, 0.78]	0.50, <.01 [-0.26, 1.27]
Evening Stress	-0.02, <.01 [-0.06, 0.01]	-0.05, 0.01** [-0.29, -0.16]	-0.03, <.01 [-0.13, 0.19]	-0.19, <.01 [-0.45, 0.07]
Lag1 TST/SE	-0.19, 0.05*** [-0.25, -0.13]	-0.23, 0.19*** [-0.28, -0.17]	-0.13, 0.02*** [-0.19, -0.07]	-0.09, 0.02** [-0.14, -0.03]
Lag2 TST/SE	-0.19, 0.05*** [-0.25, -0.13]	-0.23, 0.10*** [-0.26, -0.16]	-0.15, 0.03*** [-0.21, -0.09]	-0.09, 0.01** [-0.14, -0.03]
Lag3 TST/SE	-0.14, 0.02*** [-0.20, -0.08]	-0.15, 0.03*** [-0.21, -0.09]	-0.08, 0.01** [-0.14, -0.02]	-0.14, 0.02*** [-0.20, -0.09]
Interaction Effects				
WSTRESS:WPFC	-0.07, 0.01* [-0.12, -0.01]	—	—	—

Note. Results are coefficients, Cohen's f^2, [95% confidence intervals]. * p <.05, ** p <.01. *** p<.001. PFC = Problem-Focused Coping. BSTRESS = Between-person evening Stress. WSTRESS = Within-person evening Stress. BPFC = Between-person Problem-Focused Coping. WPFC= Within-person Problem-Focused Coping. TST = Total Sleep Time. SE = Sleep Efficiency. Covariates were adjusted in all models including age, sex, body mass index, race, alcohol use, smoking status, education level, school status, employment status, day of week, and study day 1 to 12. Non-significant interactions were dropped from analyses.

Table 3. Multilevel Models Examining the Interaction Effects between Emotional-Approach Coping and Evening Stress Predicting Actigraphic and Self-Reported TST and SE

	Actigraphic TST	**Self-Reported TST**	**Actigraphic SE**	**Self-Report SE**
Between Person Effects				
EAC	-0.10, <.01 [-0.28, 0.08]	0.03, <.01 [-0.14, 0.21]	0.20, <.01 [-0.78, 1.18]	1.25, 0.01 [-0.38, 2.88]
Evening stress	-0.02, <.01 [-0.12, 0.07]	-0.04, <.01 [-0.13, 0.06]	0.31, 0.01 [-0.19, 0.81]	0.29, <.01 [-0.60, 1.18]
Within Person Effects				
EAC	0.05, <.01 [-0.08, 0.19]	0.01, <.01 [-0.13, 0.15]	-0.76, <.01 [-1.74, 0.22]	0.76, 0.03 [-0.23, 1.74]
Evening stress	-0.02, <.01 [-0.06, 0.02]	-0.05, 0.01** [-0.09, -0.01]	0.04, <.01 [-0.12, 0.20]	-0.16, <.01* [-0.41, 0.10]
Lag1 TST/SE	-0.19, 0.05*** [-0.24, -0.13]	-0.22, 0.06*** [-0.28, -0.17]	-0.13, 0.02*** [-0.19, -0.07]	-0.09, 0.02 ** [-0.14, -0.03]
Lag2 TST/SE	-0.18, 0.04*** [-0.24, -0.12]	-0.17, 0.04*** [-0.23, -0.11]	-0.15, 0.03*** [-0.21, -0.09]	-0.09, 0.01 ** [-0.14, -0.03]
Lag3 TST/SE	-0.14, 0.02*** [-0.20, -0.08]	-0.15, 0.02*** [-0.20, -0.09]	-0.08, 0.01** [-0.15, -0.02]	-0.15, 0.01 *** [-0.20, -0.09]
Interaction Effects				
WSTRESS:WEAC	-0.08, 0.01* [-0.14, -0.01]	—	—	—
BSTRESS:WEAC	—	—	0.47, 0.01* [0.06, 0.87]	—

Note. Results are coefficients, Cohen's f^2, [95% confidence intervals]. * p<.05, ** p< .01. *** p<.001. EAC = Emotional-Approach Coping. BSTRESS = Between-person evening Stress. WSTRESS = Within-person evening Stress. BEAC = Between-person Emotional-Approach Coping. WEAC= Within-person Emotional-Approach Coping. TST = Total Sleep Time. SE = Sleep Efficiency. Covariates were adjusted in all models including age, sex, body mass index, race, alcohol use, smoking status, education level, school status, employment status, day of week, and study day 1 to 12. Non-significant interactions were dropped from analyses.

Table 4. Multilevel Models Examining the Interaction Effects between Avoidance Coping and Evening Stress Predicting Actigraphic and Self-Reported TST and SE

	Actigraphic TST	**Self-Reported TST**	**Actigraphic SE**	**Self-Report SE**
Between Person Effects				
AVC	0.37, 0.01* [0.07, 0.66]	0.02, <.01 [-0.15, 0.20]	0.47, <.01 [-0.44, 1.37]	-1.56, 0.01 [-3.15, 0.03]
Evening stress	-0.04, <.01 [-0.14, 0.06]	-0.07, <.01 [-0.17, 0.04]	0.23, <.01 [-0.28, 0.74]	0.47, <.01 [-0.45, 1.40]
Within Person Effects				
Evening Stress	-0.02, <.01 [-0.06, 0.02]	-0.05, 0.06 [-0.09, 0.00]	0.03, <.01 [-0.13, 0.19]	-0.16, <.01 [-0.42, 0.09]
Lag1 TST/SE	-0.21, 0.12*** [-0.27, -0.14]	-0.23, 0.21*** [-0.30, -0.17]	-0.13, 0.02*** [-0.19, -0.07]	-0.08, 0.01 ** [-0.14, -0.03]
Lag2 TST/SE	-0.23, 0.09*** [-0.28, -0.17]	-0.22, 0.10*** [-0.28, -0.17]	-0.15, 0.02*** [-0.21, -0.09]	-0.09, 0.01 *** [-0.15, -0.04]
Lag3 TST/SE	-0.14, 0.02*** [-0.20, -0.08]	-0.15, 0.03*** [-0.20, -0.09]	-0.08, <.01** [-0.14, -0.02]	-0.15, 0.02 *** [-0.20, -0.09]
Interaction Effects				
BSTRESS:BAVC	-0.12, 0.01* [-0.23, 0.00]	—	—	—

Note. Results are coefficients, Cohen's f^2, [95% confidence intervals]. * p<.05, ** p< .01. *** p<.001. AVC = Avoidance Coping. BSTRESS = Between-person evening Stress. WSTRESS = Within-person evening Stress. BAVC = Between-person Avoidance Coping. TST = Total Sleep Time. SE = Sleep Efficiency. Covariates were adjusted in all models including age, sex, body mass index, race, alcohol use, smoking status, education level, school status, employment status, day of week, and study day 1 to 12. Non-significant interactions were dropped from analyses.

Discussion

This study examined the moderating role of daily coping strategies on the relationship between evening stress and self-report and actigraphic sleep. Findings indicated that high engagement in PFC, EAC, or AVC was associated with stronger negative association between evening stress on subsequent actigraphic total sleep time, which was contrary to the hypothesized direction for PFC and EAC. Results partially supported the hypothesis for sleep efficiency. Specifically, engaging in high EAC strategies buffered the effects of evening stress on subsequent actigraphic sleep efficiency. Further exploratory analyses showed that higher evening stress predicted longer actigraphic bedtime for high EAC compared to low EAC. Engaging in high EAC buffered the effects of evening stress on subsequent actigraphic sleep onset latency and wake after sleep onset.

Moderating Role of Coping Strategies on Total Sleep Time

Within-person effects showed that on evenings with higher than *usual* stress, individuals who used more than *usual* levels of PFC or EAC had shorter actigraphic total sleep time that night. Similar results emerged for PFC and self-report total sleep time. Further, between-person effects indicated that individuals who *generally* experience high levels of evening stress and use high AVC on *average* also had shorter actigraphic total sleep time, which is consistent with previous studies.[10,13,14]

Although the direction of results for PFC and EAC were contrary to hypotheses, previous research on the relations between approach-oriented or PFC and sleep has been inconsistent, with higher levels of PFC related to longer actigraphic total sleep time,[15] delayed bedtime,[10] or null findings.[12,14] Inconsistencies in these findings may be due to methodological differences in assessing coping, such as daily versus single-timepoint assessments, and different coping measurements.

Both PFC and EAC involve actively engaging in addressing the demands of the stressor, either directly to resolve it or to address the emotional sequelae. Either form of

coping requires time, which may delay bedtime and reduce sleep duration. For example, additional study or work time (actively addressing the stressor) for an upcoming test or deadline (on days with higher than usual stress) can come at the expense of sleep. Similarly, actively expressing emotions (e.g., communicating and sharing feelings) when experiencing an interpersonal argument in the evening can also delay bedtime and result in shorter sleep. In adolescents, Matthews and colleagues[10] showed that high use of approach-coping strategies was associated with delayed bedtime. To explore this speculation in our own data, we carried out exploratory analyses on bedtime and found that higher within-person stress predicted later bedtime for high within-person EAC compared to low within-person EAC. These findings support our interpretation that high engagement in EAC is associated with a delayed bedtime in the presence of higher than usual stress.

Besides potential behavioral mechanisms via delayed bedtime, increased arousal may be another pathway to shorter total sleep time with increased coping. A small literature suggests that although coping may have beneficial effects in the following months, arousal is acutely increased while actively recalling or expressing emotions regarding a stressor.[41] Relatedly, the literature showing the benefits of PFC and EAC associated with better psychological adjustment and well-being has primarily relied on cross-sectional or longitudinal designs.[8,42,43] Over these broader time frames (compared to acute, daily stress, and coping), there may indeed be beneficial effects, yet engaging in these approach-oriented coping strategies may have short-term costs, including sleep duration when such relationships are examined on a daily basis.

Results also showed that individuals who experienced overall high evening stress and generally engaged in high AVC had shorter sleep duration. This is consistent with previous literature that linked AVC to poor or short sleep.[10-14] Although avoidance may appear not to require time or resources, research has long shown paradoxical effects, whereby attempting to

avoid or suppress thoughts backfires and results in greater pre-occupation.[44] Avoiding the problems also may reduce efforts in finding effective resolutions, thereby exacerbating the problem and ruminations that can interfere with sleep quantity. Thus, avoiding the stressor and the possibility of increased pre-occupation and rumination regarding stressors are plausible pathways linking greater use of AVC to shorter sleep duration in the presence of stress.

Moderating Role of Coping Strategies on Actigraphic Sleep Efficiency

For individuals who *generally* experience high evening stress, they had better sleep quality (higher actigraphic sleep efficiency) on days when they engaged in higher than *usual* EAC. Exploratory analyses suggest this was due to reduced shorter time taken to fall asleep (lower actigraphic sleep onset latency) and night-time awakening (lower actigraphic wake after sleep onset). Previous studies that examined the broad construct of approach coping (the combination of both PFC and EAC) showed inconsistent results.[13,14] Our findings extend the current literature by distinguishing PFC and EAC and showing the benefits of EAC on sleep quality. Engaging in EAC may improve sleep quality through processing and expressing the negative emotions related to the stressors. This process may include positive cognitive reappraisals of the stressor that can reduce rumination and enhance perceived resources in managing the increased demands, and thereby reducing night-time awakenings and improving sleep quality.

Limitations and Strengths

Several limitations should be acknowledged. First, as with all EMA methods, it is possible that participants' report on their stress levels or coping strategies may be affected (e.g., reflecting more on these experiences) when asked to report over multiple days, leading to reactivity effects and potential biases.[20,45] Second, the low reliability for within-person AVC limits the examination of its interactions with stress on sleep. AVC measures are known to have poor reliability,[27,46] highlighting a need for further psychometric work to develop

valid and reliable measures of AVC. Third, the coping strategies measured may not represent how participants coped with stress in the *evening*, given that they were asked to think about how they coped throughout the day. Future studies are recommended to measure participants' coping strategies at multiple time points within the day to strengthen the temporal inferences. Third, periods of quiet wakefulness (i.e., lying in bed with eyes rested without activity) may be scored as sleep in actigraphy data, thus underestimating sleep onset latency and its associated results (e.g., sleep efficiency and total sleep time). Although partially accounted by including self-reported sleep measures, self-reported sleep onset latency and total sleep time are often overestimated.[47] Fourth, the sample in this study reported relatively low average stress levels (M = 2.23 on a 10-point scale), which may lead to floor effects or reduced variability. Given the low stress levels, these findings cannot be generalized to populations experiencing high stress levels (e.g., clinical populations), and require further validation in these populations. Lastly, the types and perceived control of stress were not tested in this study. Research including experimental studies show that the effectiveness of coping strategies depends on the perceived controllability of the stressor (e.g., upcoming examination for high perceived control; major physical illness for low perceived control).[8,48] Given the significant findings using the general perceived overall evening stress in our study, future research should extend these findings by examining the severity of different stressors and their associated perceived controllability.

Our study also had notable strengths. To our knowledge, this is the first study that examined the moderating role of coping on the stress-sleep relationship on a daily basis in a large sample of young adults with diverse gender and race/ethnicity. The use of a daily, intensive longitudinal design with repeated ecological momentary assessments in naturalistic environments, as well as the examination of specific types of approach-coping strategies (i.e., PFC and EAC), extended the findings of previous cross-sectional and prospective studies by

capturing the daily interaction of these variables. Additionally, this study employed rigorous analyses to examine the moderating role of daily coping on the relations between evening stress and *subsequent* sleep (self-reported and actigraphic measures), as well as accounting for the effects of three previous nights of sleep. Confidence in our findings was strengthened by separating between and within-person effects in all analyses alongside effect sizes.

Conclusions and Implications

We found that daily coping moderated the relations between evening stress and subsequent sleep duration and quality. Engaging in either PFC, EAC, or AVC in the presence of high stress was associated with shorter sleep duration. Direct attempts to address the emotional sequalae of stress were associated with better sleep quality.

Coping strategies are routinely incorporated and promoted in the management of high stress. Despite research showing the long-term benefits of approach coping in well-being and health, our study suggests that even what is typically considered "helpful" coping (e.g., actively communicating and sharing feelings) may reduce sleep duration in the short-term. Stress management could consider ways to offset the reduced sleep duration associated with coping (e.g., encourage problem-solving during the day, wind down before bedtime). This may be particularly important for individuals who may be overly concerned about sleep loss (e.g., individuals with insomnia). Future studies should extend these findings by examining the long-term effects of these coping strategies on sleep.

Results from this intensive longitudinal study further support findings in the literature that approach-oriented coping processes improve sleep quality, and that the use of avoidance-oriented coping worsens sleep duration. Reducing the use of avoidance-oriented coping (e.g., disengaging and distracting oneself from the stressor) and replacing with either more approach-oriented coping (e.g., actively process or communicate emotions) may be a profitable direction for future research to support good sleep.

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Electronic Supplementary Material

Table S1. *Unadjusted Multilevel Models Examining the Interaction Effects between Problem-Focused Coping and Evening Stress Predicting Actigraphic and Self-Reported TST and SE*

	Actigraphic TST	**Self-Report TST**	**Actigraphic SE**	**Self-Report SE**
Between Person Effects				
PFC	-0.06, <.01 [-0.23, 0.10]	0.07, <.01 [-0.09, 0.23]	0.39, <.01 [-0.51, 1.28]	1.64, 0.01* [0.15, 3.14]
Evening stress	-0.02, <.01 [-0.11, 0.06]	-0.09, 0.01* [-0.17, 0.00]	0.34, <.01 [-0.12, 0.80]	-0.24, <.01 [-1.03, 0.55]
Within Person Effects				
PFC	0.03, 0.02 [-0.09, 0.15]	-0.01, <.01 [-0.12, 0.09]	0.18, <.01 [-0.24, 0.61]	0.50, <.01 [-0.20, 1.20]
Evening Stress	-0.03, <.01 [-0.07, 0.00]	-0.05, 0.01** [-0.09, -0.02]	-0.005, <.01 [-0.15, 0.14]	-0.18, <.01 [-0.42, 0.06]
Lag1 TST/SE	-0.18, 0.04*** [-0.24, -0.13]	-0.22, 0.17*** [-0.29, -0.16]	-0.12, 0.02*** [-0.17, -0.07]	-0.07, 0.01** [-0.12, -0.02]
Lag2 TST/SE	-0.19, 0.05*** [-0.24, -0.13]	-0.21, 0.09*** [-0.26, -0.16]	-0.15, 0.03*** [-0.21, -0.10]	-0.08, 0.01** [-0.13, -0.02]
Lag3 TST/SE	-0.14, 0.02*** [-0.19, -0.09]	-0.15, 0.03*** [-0.20, -0.10]	-0.11, 0.01*** [-0.16, -0.05]	-0.14, 0.02*** [-0.19, -0.09]
Interaction Effects				
WSTRESS:WPFC	-0.06, 0.01* [-0.12, -0.01]	-—	—	—

Note. Results are coefficients, Cohen's f^2, [95% confidence intervals]. * p <.05, ** p <.01. *** p<.001. PFC = Problem-Focused Coping. BSTRESS = Between-person evening Stress. WSTRESS = Within-person evening Stress. BPFC = Between-person Problem-Focused Coping. WPFC= Within-person Problem-Focused Coping. TST = Total Sleep Time. SE = Sleep Efficiency.

Table S2. *Unadjusted Multilevel Models Examining the Interaction Effects between Emotional-Approach Coping and Evening Stress Predicting Actigraphic and Self-Reported TST and SE*

	Actigraphic TST	**Self-Reported TST**	**Actigraphic SE**	**Self-Report SE**
Between Person Effects				
EAC	-0.05, <.01 [-0.22, 0.12]	0.03, <.01 [-0.13, 0.19]	0.46, <.01 [-0.46, 1.38]	1.66, 0.01* [-0.16, 3.15]
Evening stress	-0.03, <.01 [-0.11, 0.06]	-0.07, <.01 [-0.15, 0.01]	0.37, 0.01 [-0.08, 0.82]	-0.09, <.01 [-0.86, 0.68]
Within Person Effects				
EAC	0.01, <.01 [-0.12, 0.13]	-0.03, <.01 [-0.15, 0.10]	0.16, <.01 [-0.34, 0.66]	0.84, 0.02 [-0.04, 1.71]
Evening stress	-0.03, <.01 [-0.07, 0.01]	-0.06, 0.01** [-0.09, -0.02]	-0.002, <.01 [-0.15, 0.14]	-0.15, <.01* [-0.38, 0.09]
Lag1 TST/SE	-0.19, 0.05*** [-0.24, -0.14]	-0.22, 0.07*** [-0.27, -0.17]	-0.12, 0.02*** [-0.18, -0.07]	-0.07, 0.01 ** [-0.12, -0.01]
Lag2 TST/SE	-0.19, 0.04*** [-0.24, -0.13]	-0.17, 0.04*** [-0.22, -0.11]	-0.16, 0.03*** [-0.21, -0.10]	-0.08, 0.01 ** [-0.13, -0.03]
Lag3 TST/SE	-0.14, 0.02*** [-0.19, -0.08]	-0.14, 0.02*** [-0.20, -0.09]	-0.11, 0.01*** [-0.16, -0.05]	-0.14, 0.02 *** [-0.19, -0.09]
Interaction Effects				
WSTRESS:WEAC	-0.07, <.01* [-0.13, -0.01]	—	—	—

Note. Results are coefficients, Cohen's f^2, [95% confidence intervals]. * p<.05, ** p< .01. *** p<.001. EAC = Emotional-Approach Coping. BSTRESS = Between-person evening Stress. WSTRESS = Within-person evening Stress. BEAC = Between-person Emotional-Approach Coping. WEAC= Within-person Emotional-Approach Coping. TST = Total Sleep Time. SE = Sleep Efficiency.

Table S3. *Unadjusted Multilevel Models Examining the Interaction Effects between Avoidance Coping and Evening Stress Predicting Actigraphic and Self-Reported TST and SE*

	Actigraphic TST	**Self-Reported TST**	**Actigraphic SE**	**Self-Report SE**
Between Person Effects				
EAVC	0.15, <.01 [-0.02, 0.32]	0.07, <.01 [-0.09, 0.23]	0.73, 0.01 [-0.15, 1.61]	-0.98, <.01 [-2.46, 0.49]
Evening stress	-0.08, <.01 [-0.17, 0.01]	-0.11, 0.01* [-0.20, -0.02]	0.24, <.01 [-0.23, 0.70]	0.07, <.01 [-0.75, 0.89]
Within Person Effects				
Evening Stress	-0.03, <.01 [-0.07, 0.01]	-0.05, 0.04* [-0.09, -0.01]	-0.0005, <.01 [-0.15, 0.15]	-0.16, <.01 [-0.40, 0.07]
Lag1 TST/SE	-0.20, 0.10*** [-0.26, -0.14]	-0.23, 0.18*** [-0.29, -0.17]	-0.12, 0.02*** [-0.18, -0.07]	-0.07, 0.01 * [-0.12, -0.02]
Lag2 TST/SE	-0.22, 0.09*** [-0.28, -0.17]	-0.21, 0.09*** [-0.26, -0.16]	-0.15, 0.03*** [-0.20, -0.10]	-0.08, 0.01 ** [-0.13, -0.03]
Lag3 TST/SE	-0.14, 0.02*** [-0.19, -0.09]	-0.15, 0.03*** [-0.20, -0.10]	-0.10, 0.01*** [-0.16, -0.05]	-0.14, 0.02 *** [-0.19, -0.10]
Interaction Effects				
	—	—	—	—

Note. Results are coefficients, Cohen's f^2, [95% confidence intervals]. * p<.05, ** p< .01. *** p<.001. AVC = Avoidance Coping. BSTRESS = Between-person evening Stress. WSTRESS = Within-person evening Stress. BAVC = Between-person Avoidance Coping. TST = Total Sleep Time. SE = Sleep Efficiency.

CHAPTER 4:

Daily Relations Between Stress and EEG-Assessed Sleep: A 15-Day Intensive Longitudinal Design with Ecological Momentary Assessments

Preface to Chapter 4

To address the limitations highlighted and extend the findings shown in **Chapter 2,** this chapter examined the bi-directional associations between daily stress and EEG-assessed sleep using a 15-day intensive longitudinal design in a sample of young adults who relocated from a different state or country for undergraduate studies. This population is assumed to experience higher stress levels, as the normal transitional challenges may be compounded by relocation and losing existing social support. Furthermore, international students may experience additional acculturative stressors, such as language barriers and foreign educational systems. The use of EEG-sleep allows the examination of a more accurate estimation of sleep parameters compared to actigraphy or self-report measures, as well as sleep architecture in relation to stress on a daily basis. This chapter also examined *pre-bedtime* stress in relation to subsequent sleep (a closer proximity to sleep), rather than *evening* stress as examined in chapter 2. Data for this paper came from the Stress and Health Study (conducted between February 2019 – June 2020), which used a 15-day intensive longitudinal design.

This chapter has been submitted to ***Annals of Behavioral Medicine*** and is presented in manuscript form. The status of this manuscript is currently a revise and resubmit.

Daily Relations between Stress and EEG-Assessed Sleep:

A 15-day Intensive Longitudinal Design with Ecological Momentary Assessments

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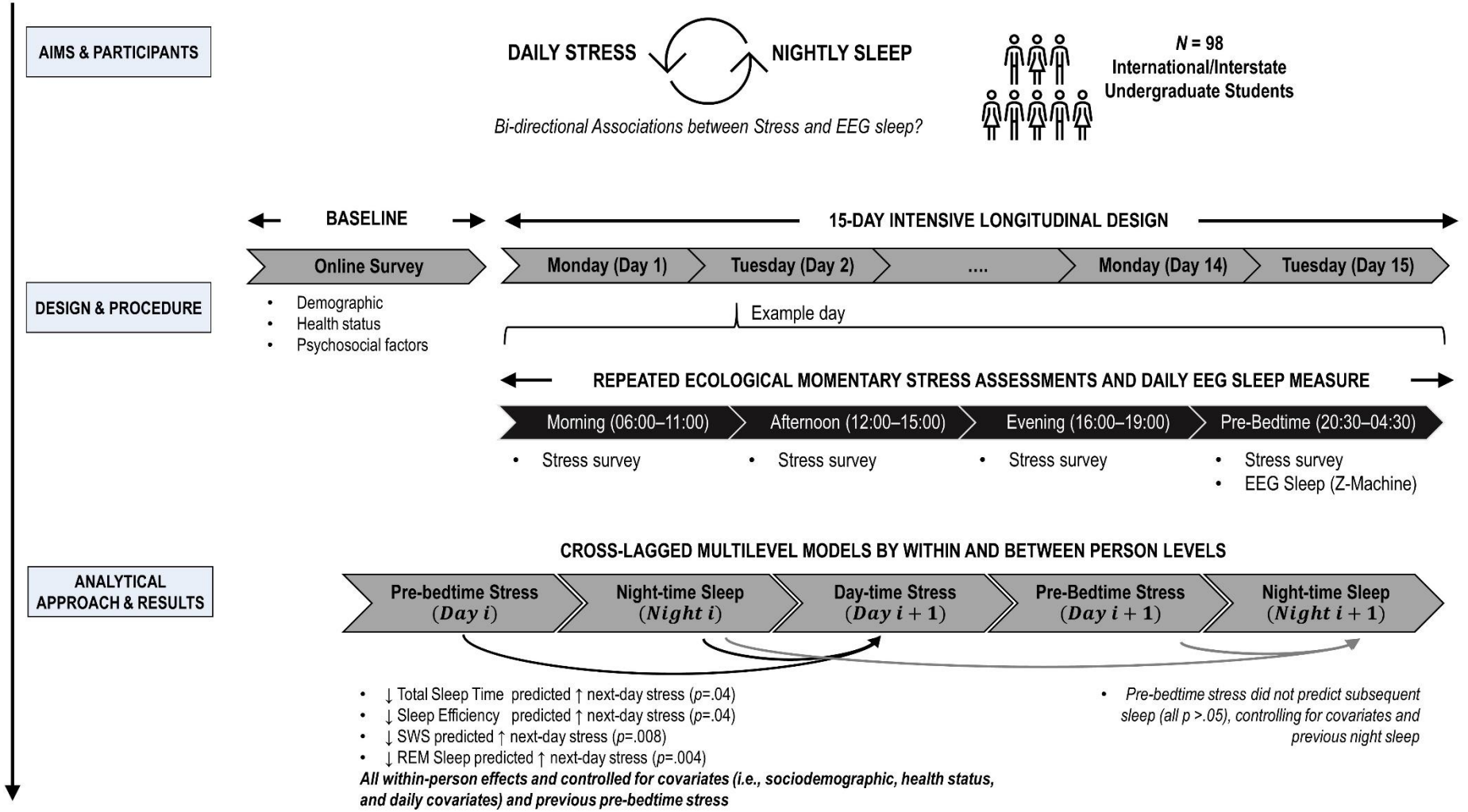
Graphical Abstract



Abstract

Background: Recent studies have found bi-directional relations between stress and sleep. However, few studies have examined the daily associations between stress and electroencephalography (EEG) measured sleep.

Purpose: This study examined the temporal associations between repeated ecological momentary assessments of stress and EEG-estimated sleep.

Methods: 98 international or interstate undergraduate students (M_{age}=20.54±1.64, 76.5% Female, 84.7% Asian) reported their stress levels four times daily at morning awakening, afternoon, evening, and pre-bedtime across 15 consecutive days (>4,000 total observations). Next-day stress was coded as an average of morning, afternoon, and evening stress. Z-Machine Insight+ recorded over 1,000 nights EEG total sleep time (TST), sleep onset latency, wake after sleep onset, sleep efficiency (SE), slow-wave sleep (SWS) and rapid eye movement (REM) sleep duration. Cross-lagged multilevel models, adjusted for covariates (i.e., sociodemographic, health factors, and daily covariates) and lagged outcomes, tested the daily within- and between-level stress-sleep associations.

Results: Within-person shorter TST (*b*=-0.11[-0.21,-0.01], *p*=.04), lower SE (*b*=-0.02[-0.03, 0.00], *p*=.04), less SWS (*b*=-0.38[-0.66, -0.10], *p* =.008), and less REM sleep (*b*=-0.32[-0.53, -0.10], *p* =.004) predicted higher next-day stress. Pre-bedtime stress did not predict same-night sleep. No significant results emerged at the between-person level.**Conclusions:** These findings demonstrate that poor or short sleep, measured by EEG, is predictive of higher next-day stress. Results for sleep architecture support the role of SWS and REM sleep in regulating the perception of stress. Given that only within-person effects were significant, these findings highlight the importance of examining night-to-night fluctuations in sleep affecting next-day stress and its impact on daytime functioning.

Key Words

stress, sleep, EEG, EMA, daily, international students

Introduction

Previous research has established cross-sectional associations between high stress and poor or short sleep [1], with both contributing to higher risks of poor health outcomes [2-5]. Recent studies have extended these findings by determining bi-directional or temporal associations between stress and sleep using daily sleep diary or actigraphy measures [6-11]. However, few studies to date have examined the relationship between stress and sleep using objective, electroencephalography (EEG) measures on a daily basis. Laboratory studies have shown restorative and emotional regulative benefits of Slow Wave Sleep (SWS) and rapid eye moment (REM) sleep [12-15]. We sought to determine whether laboratory findings translate to naturalistic conditions, and whether stress and sleep architecture bi-directionally influence each other on a daily basis in young adult undergraduate students. In recent years in developed countries over half of young adults pursue tertiary education, making this transitional period from high school to university a normative developmental period applying to most young adults. Within tertiary education students, those who move from a different state or country for the first time are particularly vulnerable as the normal transitional challenges may be compounded by relocation (e.g., adapt to living independently in new environments) and losing existing routine, community, and social support [16]. Furthermore, international students may experience additional acculturative stressors, such as language barriers and foreign educational systems [17, 18]. Although there is an increasing number of international and interstate students worldwide [19], and that relocated students represent an at-risk subset of tertiary education students, no known studies have explicitly examined the day-to-day stress and sleep associations in this population.

Studies have examined daily stress-sleep relations, with those that explicitly tested the temporal order of the stress-sleep associations finding complex, bi-directional and temporal associations [6-9]. Results differ across different aspects (e.g., duration, quality) and

measurement of sleep (e.g., self-report, actigraphy) [6-9]. For instance, a 12-day study found that evenings with higher than usual stress levels predicted both shorter actigraphic and self-reported total sleep time (TST) that night, and that shorter actigraphic and self-reported TST predicted higher stress the next day [6]. Furthermore, worse than usual sleep quality (i.e., self-reported sleep onset latency [SOL], self-reported wake after sleep onset [WASO], and actigraphic and self-reported sleep efficiency [SE]) predicted higher next-day stress [6]. However, there is contrary evidence, showing that stress did not predict same-night self-reported TST, whereas self-reported TST and sleep quality predicted higher next-day stress [8]. Inconsistent findings may be due to differences in sleep measures. Self-reported sleep is susceptible to individuals' mood state and perception about sleep; for example, those with insomnia and/or mood symptoms tend to under-estimate TST and over-estimate wake [20]. Movement-based actigraphy on the other hand, may underestimate SOL compared to polysomnography (PSG) [21].

Neither self-reported nor actigraphic measures of sleep can accurately assess sleep architecture, which requires measurements of EEG. The sleep architecture consists of three non-rapid eye movement (NREM) sleep stages, i.e., stage N1, N2 and N3, and rapid eye movement (REM) sleep. A typical sleep cycle starts with entering the NREM sleep stages, followed by REM sleep. Each NREM sleep stage is progressively deeper and has unique brain waves, e.g., rhythmic alpha waves in N1, sleep spindles and K-complexes in N2, and high-voltage, slow-wave-activity in N3. REM sleep, on the other hand, has low-voltage, high-frequency brain wave activity.[22] Few studies have demonstrated the effects of both experimentally induced and naturally occurring stress on subsequent EEG-assessed sleep. Individuals exposed to emotional stress showed significantly decreased TST and SE [23], as well as less REM sleep [23-25]. Similarly, individuals had significantly lower SE, but not TST, during high-stress periods compared with low-stress periods [25]. Reported findings for

SWS are inconsistent, as individuals exposed to emotional stress or experiencing high stress periods have shown an increase, decrease, or no change in SWS [23-26].

When people experience sleep loss, they tend to exhibit greater psychological stress and emotional reactivity [12, 27]. Evidence from neuroimaging studies suggests that SWS and REM sleep may play an emotional regulatory role, which may potentially explain these findings [14, 15]. For example, individuals who had a night of sleep deprivation reported higher anxiety levels the following morning compared to the previous night and to well-rested individuals; within well-rested individuals, longer SWS was associated with lower levels of next-day anxiety [15]. Another study showed that well-rested individuals with 8 hours of sleep opportunity had decreased amygdala and emotional reactivity towards affective images, compared with sleep-deprived individuals. Within well-rested individuals, low EEG gamma activity (a biomarker of adrenergic activity that plays a role in emotional regulation and amygdala activity) during REM sleep was associated with reductions in both amygdala activity and emotional reactivity towards the affective stimuli [14]. These findings have demonstrated that SWS and REM sleep are associated with mood and emotional reactivity. Although perceived stress was not examined in these studies, given the inextricable link between stress and emotions, it is possible that SWS and REM sleep also may regulate one's perception and appraisal of stress severity in addition to emotional responses. However, this notion remains untested, especially in daily settings.

The use of daily diary designs with repeated ecological momentary assessments (EMAs) allows daily variations in stress to be related to subsequent or previous sleep architecture. Furthermore, these designs allow ecological changes in stress or sleep to be studied in naturalistic settings, rather than relying on extreme manipulations used in experimental studies (e.g., sleep deprivation). Only one study to date examined the daily stress-sleep relations, using a 7-day daily diary design and EEG sleep measures [28]. The

findings showed that stress did not predict any of the subsequent EEG sleep variables, and the EEG sleep measures did not significantly predict next-day stress.

The current study aimed to examine the bi-directional and temporal associations between stress and sleep across 15 days, using an intensive longitudinal design with repeated EMAs and a single-channel EEG sleep measure in young adults who are international or interstate undergraduate students. Specific hypotheses were: 1) higher pre-bedtime stress will predict subsequent shorter sleep duration (TST), worse sleep quality (i.e., longer SOL, higher WASO, and lower SE), and less SWS and REM sleep duration. 2) Shorter sleep duration, worse sleep quality, and less SWS and REM sleep will predict higher next-day stress.

Methods

Participants

The Stress and Health Study was conducted from February 2019 to June 2020 and recruited participants who had moved from a different state or country for undergraduate studies in Victoria, Australia. All participants moved to Victoria for the first time and had not previously spent more than six months in Victoria in the past 10 years. Participants were recruited through the XXXXXXX Research Participation Platform, social media (e.g., Facebook), word-of-mouth, in-class presentations, and learning management systems (e.g., Moodle posts). Figure 1 summarizes the participant flow chart and eligibility criteria of the Stress and Health Study. *A priori* power analysis indicated that 68 participants, with the assumption of a 75% completion rate, would provide 80% power to detect small-to-medium effect sizes at the within-person level for stress and sleep. Additional participants were recruited to account for potential attrition, missing data, and other aims not related to the current paper.

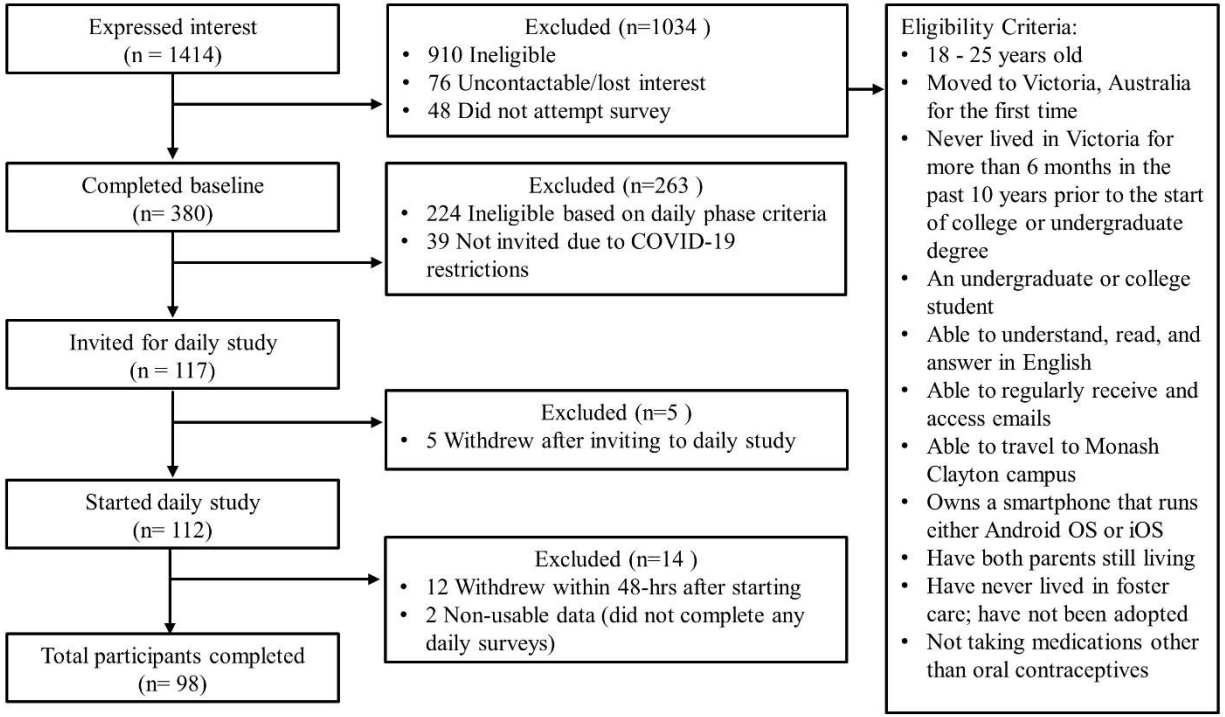


Figure 1. Summary of the Stress and Health Study recruitment process. The recruitment period was between February 2019 and June 2020

Design and Procedure

XXXXXX (project ID: XXXX) approved all procedures, and all participants provided consent. This study used an intensive longitudinal design with repeated EMA for 15 consecutive days. This approach captures real-time variability of experiences in naturalistic settings, maximizing external validity and reducing memory and other biases related to conventional retrospective recall methods [29, 30]. Participants also served as their own control through the repeated assessments across days. These methods provide a rigorous test of directionality and temporal order between stress and sleep (e.g., examining pre-bedtime stress collected *during* pre-bedtime predicting subsequent sleep that night) [30]. Detailed procedures are in Figure 2. This study follows the Strengthening The Reporting of Observational Studies in Epidemiology and Checklist for Reporting EMA Studies reporting guidelines [31, 32].

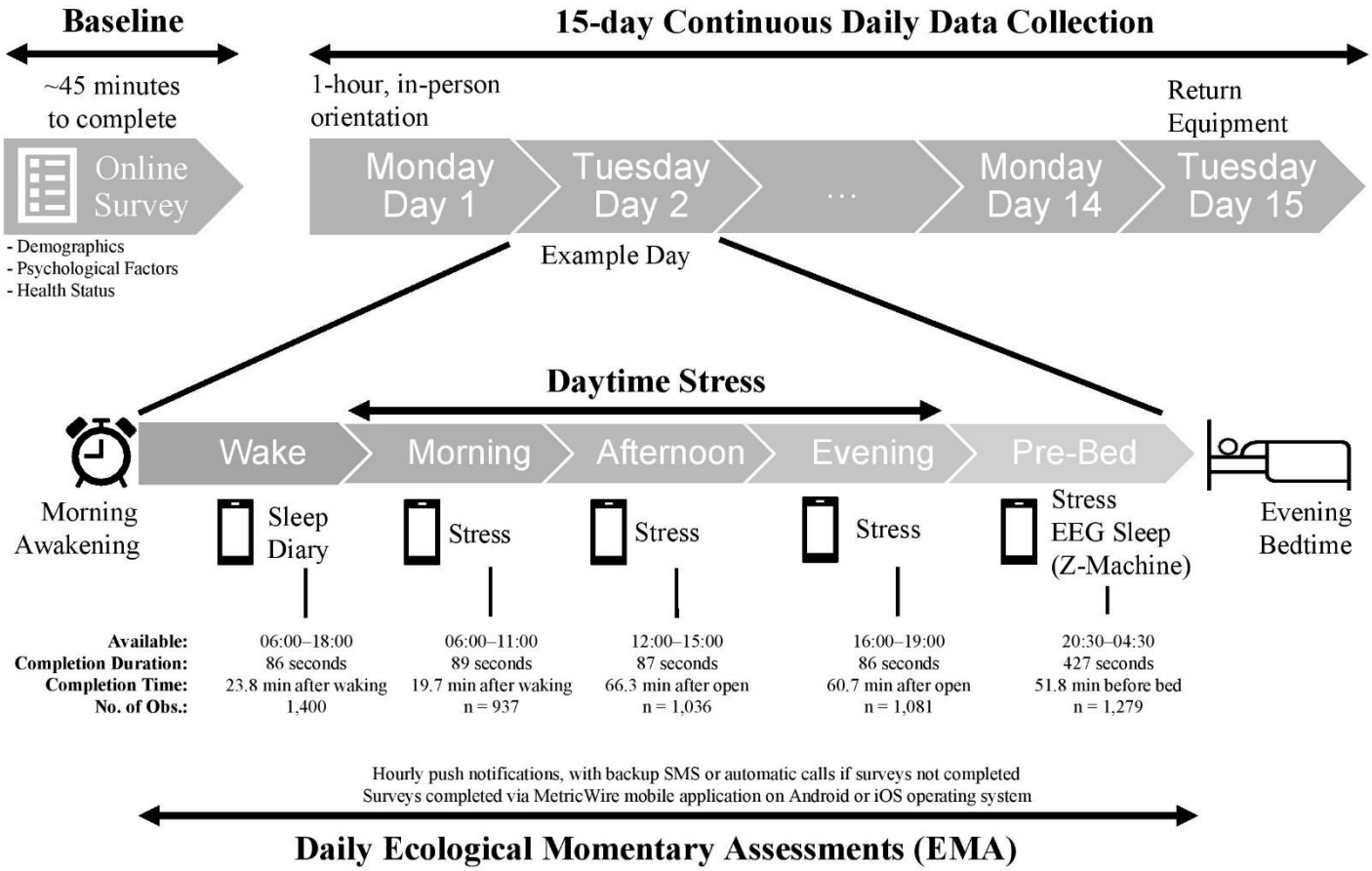


Figure 2. Diagram of the Stress and Health Study procedures. The top of the diagram shows the initial, baseline online survey phase after which eligible participants were invited to 15-day daily phase of the study, which always commenced on a Monday and completed on a Tuesday two weeks later. Daily measures were completed via the MetricWire mobile phone application and night-time EEG sleep via the Z-Machine Insight+. During the daily phase, participants completed five surveys per day via MetricWire (middle of diagram) assessing self-reported sleep via sleep diary and daytime and evening stress. Participants were provided training on how to complete the surveys and wear the Z-Machine Insight+ during the 1-hour, in-person orientation. Participants were instructed to clean their skin (with the provided alcohol wipes) and attach the disposable sensors (i.e., one-time use) approximately 30-minutes before their bedtime. Participants were instructed to only attach the cables to the EEG device when attempting to sleep. Surveys were available during a broad window with the median number of seconds to complete each survey shown in the figure as well as the median timing in minutes (min) of each survey, relative to wake, open of the available period, or bedtime. No. of Obs. is the number of observations available for each survey.

Measures

Sleep. Daily objective estimates of sleep (i.e., TST, SOL, WASO, SE, SWS, and REM sleep) were measured in 30-second epochs using the Z-Machine Insight+, a portable, single-channel EEG sleep-monitoring device. There were three sensors: one sensor (signal) was placed behind each ear (i.e., differential mastoids A_1 and A_2), and one on the center of the neck below the hairline (ground). Raw EEG signals were automatically scored as sleep or wake through the Z-ALG13 (for more information on the Z-ALG algorithm and scoring methods, see original publication by Kaplan and colleagues [33]). The scored data were then processed by the Z-PLUS [34] algorithm to determine sleep stages as light (N1 and N2), SWS or REM sleep. Previous studies showed that the Z-ALG13 has high sensitivity and specificity for determining sleep (95.5% and 92.5%, respectively), and the Z-PLUS has positive predictive values of 0.85 for light sleep, 0.83 for SWS, and 0.76 for REM sleep in normal, healthy sleepers compared to PSG consensus [33, 34]. Reported sensitivities of Z-PLUS are 0.83 for light sleep, 0.77 for SWS, and 0.74 for REM sleep [34]. Overall kappa agreement is 0.85 and 0.72 for Z-ALG13 and Z-PLUS, respectively [34]. Self-report sleep measures in our study were adapted from the Consensus Sleep Diary [35] and included bedtime, rise time, SOL, number of awakenings, and WASO.

***Daily Stress*.** Daily stress was measured using a self-reported adaptation from the Daily Inventory of Stressful Events scale, [36, 37] completed four times each day (i.e., mornings, afternoons, evenings, and pre-bedtime). The number of surveys completed for each timepoint, alongside completion duration and time, are summarized in Figure 1. In this study we focused on a single item ranging from 0 (Not at all stressful) to 10 (Very stressful), i.e., "Since the previous survey, how stressful has your day been?". Stress values from the morning, afternoon, and evening surveys (See Figure 2 for survey periods) were averaged by participant and day to create a composite of daily stress with $\omega_{within} = 0.635$, $\omega_{between} = 0.98$,

indicating adequate and excellent reliability at the within and between levels, respectively. In analysis, daily stress was examined following sleep, thus named as next-day stress.

Covariates. Covariates were determined *apriori* based on research demonstrating their association with stress and sleep. Previous studies have shown several sociodemographic variables that are associated with stress or sleep. For example, individuals who are females (vs males) [38, 39], non-White (vs White) [40, 41], or reported lower subjective social [42] had reported higher stress or poorer sleep. Thus, the sociodemographic variables included as covariates in our model include: age (years) [38], sex (coded as male/female) [38, 39], race/ethnicity (coded as white/Asian/other) [40, 41], employment status (coded as working/not working)[40, 43], student status (coded as international/interstate) [18], time spent in Victoria (years), English language acculturation (using the adapted Short Acculturation Scale for Hispanics to refer to participants' native language instead of Spanish) [17, 44], and subjective social status [42, 45, 46]. Furthermore, COVID-19 period (coded as pre [before Victoria lockdown 2020 March 8] vs. during) were included as recent study has shown the impact of the pandemic on psychological well-being and sleep [47]. Several confounding health factors that can impact stress or sleep were also included. For example, higher body mass index (BMI), smoking, and alcohol consumption are associated with higher stress and poorer sleep [48-51]. Thus, BMI (kg/m2 from self-reported height and weight), smoking (coded current/former vs never), and alcohol risk (coded as abstainers/moderate/at-risk based on the National Institute on Alcohol Abuse and Alcoholism guidelines [52] using the first three items of the World Health Organization Alcohol Use Identification Test [53]) were included as covariates. Daily covariates that may impact experiences of stress and sleep included day of the week, such that individuals have longer sleep and experience lower negative affect during weekends, as well as daily circadian misalignment (measured using Composite Phase Deviation) [54], as it is associated with affect and sleep architecture [55-57]

Analytic Approach

Cross-lagged multilevel linear models were run in R (v.4.0.3), using restricted maximum likelihood and lme4 v1.1-13 to estimate the models, and lmerTest v2.0-33 to estimate degrees of freedom and *p*-values. Cohen's f^2 type effect size for all predictors was also calculated. These models, separated by sleep variable, tested the temporal order and bi-directional associations between stress and sleep at between (i.e., interindividual differences; the participants' own average) and within-person levels (i.e., deviations from the individual's own average across the 15 days), and included lagged outcomes to allow for a rigorous test of directionality. The first set of models tested daily pre-bedtime stress levels as predictors of sleep that night (TST, SOL, WASO, SE, SWS, and REM sleep), controlling for previous night sleep. The second set of models tested next-day stress levels as the outcome of previous night sleep, controlling for previous night pre-bedtime stress. The number of lagged variables to be included in these models was determined through stepwise addition (i.e., first to fourth order stress and sleep lags) and model comparisons using the Bayesian Information Criterion (BIC). All models showed that the first-order lag was the most appropriate model (i.e., lowest BIC value). Fixed effects included all covariates and between- and within-person predictors, whereas random effects included intercepts, lagged outcome variables, and within-person predictors. These models were also applied to self-reported sleep variables; these results are reported in Electronic Supplementary Material (Table S2 and S3). Intra-class correlations (ICC; between-person level variance/total variance) for the stress and sleep variables (reported in Table 1 showed that a high proportion of the variance is within-person (45 – 74%), justifying the use of cross-lagged multilevel models.

All dependent variables and model diagnostics were checked for relevant assumption violations. Due to skewness, SOL and WASO were square-root transformed and winsorized and SE was winsorized (top and bottom 0.5%). For nights with EEG sleep recordings that

were identified as sensor errors or battery issues after the first sleep epoch, TST, WASO, SE, SWS, and REM were set as missing; SOL was retained given that it occurred before sleep. Model convergence failure was addressed using the Nelder-Mead algorithm and tightening tolerance values. If the singularity persisted, the random effect variable with the lowest variance was dropped from the model.

Results

Descriptive

Figure 1 summarizes the participant flow chart and eligibility criteria of the current study. From 117 participants who were invited to the daily study, 5 withdrew prior to starting, and 12 withdrew within 48 hours after starting the daily study. Two participants were excluded due to not completing any daily surveys. The final sample consisted of 98 participants (M_{age}= 20.54, *SD* = 1.64 years). Most participants were female (76%), of Asian descent (84%), and were international students (91%) who had spent less than a year in Melbourne. Only 5% of the sample is currently taking oral contraceptives, and the others were currently not taking any medications. Most participants had a BMI within the healthy adult range (18.5 – 24.9 kg/m^2), were moderate drinkers (65%), and had never smoked (95%). Table 1 shows the number of observations, descriptive statistics for demographic and daily variables, alongside ICCs for all daily variables.

On average, participants' pre-bedtime and next-day stress level throughout the study period were 2.49±1.63 and 1.93±1.49 (M±SD; possible range 0–10), respectively, representing normative stress levels comparable to daily stress levels reported in other studies including healthy undergraduate students in Australia [6] and adults [10]. Participants reported the highest number of work or university related stressors (13.94%) and lowest discrimination-related stressors (0.55%) (see Table S2). Participants' average self-reported TST was 7.44±0.96 h, within the recommended sleep duration for adults, and the average

self-reported SE was 94±8%, indicative of good sleep quality [58, 59]. However, EEG-estimated average TST was 6.23±0.90 h, below the recommended sleep duration for adults, and average SE was 83±6%. The proportion of SWS and REM sleep were typical of the healthy young adult population without sleep complaints [60]. On average, participants completed 73% of all possible stress surveys (i.e., across morning, afternoon, evening, and pre-bedtime). Across all nights, 74% of the EEG TST, WASO, SE, SWS, and REM, as well as 86% SOL, were usable for analysis.

There were no significant differences in stress levels or EEG sleep variables between international and interstate students (all $p \geq .37$). Comparing pre and during the COVID-19 period, there were no significant differences in stress levels, or in self-reported or EEG-estimated TST and SE. However, participants during the COVID-19 period had significantly shorter overall SWS (1.22±0.28 hr) compared to individuals during pre-COVID-19 (1.50±0.28 hr), $p<.001$. These results are reported in Electronic Supplementary Material (Table S1).

Table 1. *Descriptive Statistics for Demographic and Daily Variables (N =98)*

	M (SD) / N (%)	No. of Obs	ICC
Participant Characteristics			
Age (years)	20.54 (1.64)	98	—
Time Spent in Melbourne (years)	0.73 (0.94)	98	—
Body Mass Index (kg/m^2)	21.94 (3.48)	98	—
Language Acculturation	3.85 (1.02)	98	—
Subjective Social Status	5.52 (1.44)	98	—
Sex		98	
Male	20 (20.50%)	—	—
Female	75 (76.50%)	—	—
Others	3 (3.00%)	—	—
Race/Ethnicity		98	
Asian	83 (84.70%)	—	—
White/European	9 (9.20%)	—	—
Others	6 (6.10%)	—	—
International Student (vs. Interstate)	90 (91.80%)	98	—
Working (vs. not working)	22 (22.40%)	98	—
Never smoked (vs. Current/Former)	94 (95.90%)	98	—
Before COVID-19 Period (vs. During)	72 (73.50%)	98	—
Alcohol risk		98	
Abstainer	23 (23.50%)	—	—
Moderate	64 (65.30%)	—	—
At risk	11 (11.20%)	—	—
Daily Study Variables			
Stress levels			
Pre-Bed	2.49 (1.63)	1279	.38 (61%)
Next-Day	1.93 (1.49)	1359	.50 (50%)
Self-reported sleep			
Total sleep time (h)	7.44 (0.96)	1379	.27 (73%)
Sleep onset latency (min)	25.83 (43.59)	1394	.55 (45%)
Wake after sleep onset (min)	5.77 (7.63)	1396	.29 (71%)
Sleep efficiency (%)	93.69 (7.81)	1379	.48 (52%)
EEG-estimate sleep			
Total sleep time (h)	6.23 (0.90)	1086	.27 (73%)
Sleep onset latency (min)	22.74 (11.53)	1272	.26 (74%)
Wake after sleep onset (min)	47.64 (21.86)	1086	.37 (63%)
Sleep efficiency (%)	83.31 (5.84)	1086	.32 (68%)
SWS (h)	1.43 (0.30)	1086	.31 (69%)
REM sleep (h)	1.64 (0.44)	1086	.40 (60%)

Note. ICC = Intraclass Correlations, the proportion of total variance between people. Values in parentheses in the ICC column are the remaining variance, that is, the percent of variance within individuals. No. of Obs = Number of observations. EEG = Electroencephalogram. SWS = Slow Wave Sleep. REM = Rapid Eye Movement Sleep. EEG-estimated and self-reported sleep efficiency, sleep onset latency, and wake after sleep onset presented are raw values.

Pre-bedtime Stress Predicting EEG-Estimated Sleep

Table 2 shows the unadjusted and adjusted cross-lagged multilevel models of pre-bedtime stress predicting EEG-estimated sleep, showing the between-person and within-person effects. For the unadjusted models, between-person effects showed that individuals with generally higher pre-bedtime stress had longer SOL (b = 0.17, 95% CI [0.02, 0.32], p = .03), lower SE (b= -0.87, 95% CI [-1.62, -0.12], p = .03), and less REM sleep (b = -4.51, [-8.12, -0.91], p = .02), adjusting for previous night sleep. However, after adjusting for covariates, pre-bedtime stress did not significantly predict sleep at either the between- or within-person levels. Similarly, the post-hoc exploratory analyses also showed that pre-bedtime stress did not predict self-reported sleep at either the between or within-person levels (Supplementary Table S3).

Table 2. *Cross-lagged Multilevel Model Testing Pre-Bedtime Stress as Predictor of EEG-Estimated Sleep*

	Between-Person Unadjusted	**Within-Person Unadjusted**	**Between-Person Adjusted**	**Within-Person Adjusted**
TST (min)	-1.58, <0.01 [-8.67, 5.51]	-1.44, <0.01 [-4.24, 1.36]	3.36, <0.01 [-5.09, 11.81]	-0.25, <0.01 [-3.14, 2.64]
SOL ($\sqrt{\text{min}}$)	0.17, 0.02* [0.02, 0.32]	-0.03, <0.01 [-0.08, 0.02]	0.16, 0.01 [-0.04, 0.35]	-0.03, <0.01 [-0.09, 0.04]
WASO ($\sqrt{\text{min}}$)	0.20, 0.02 [0.00, 0.39]	-0.005, <0.01 [-0.07, 0.06]	0.09, <0.01 [-0.15, 0.33]	0.005, <0.01 [-0.06, 0.07]
SE (%)	-0.87, 0.03* [-1.62, -0.12]	0.08, <0.01 [-0.18, 0.35]	-0.30, <0.01 [-1.20, 0.61]	0.09, <0.01 [-0.17, 0.36]
SWS (min)	-0.98, <0.01 [-3.36, 1.40]	0.35, <0.01 [-0.51, 1.22]	-0.07, <0.01 [-3.33, 1.90]	0.44, <0.01 [-0.48, 1.36]
REM (min)	-4.51, 0.03* [-8.12, -0.91]	-0.84, <0.01 [-1.99, 0.30]	-1.92, 0.01 [-6.20, 2.36]	-0.76, <0.01 [-1.94, 0.42]

Note. Results are unstandardized regression coefficients, Cohen's f^2, [95% confidence intervals]. * $p < .05$. TST = Total Sleep Time; SOL = Sleep Onset Latency (square-root transformed); WASO = Wake After Sleep Onset (square-root transformed), SE = Sleep Efficiency (winsorized); SWS = Slow Wave Sleep. REM = Rapid Eye Movement. Adjusted models included baseline and daily covariates: age; sex; race/ethnicity; body mass index; employment status; English language acculturation; subjective social status; time spent in Melbourne; COVID-19 period; student status; smoking status; alcohol consumption; day of week; composite phase deviation.

EEG-estimated Sleep Predicting Next-day Stress

Adjusted and unadjusted models of EEG-estimated sleep predicting next-day stress are summarized in Table 3. In the unadjusted models, between-person effects showed that longer SOL (b = 0.35, 95% CI [0.09, 0.61], p = .009), lower SE (b = -0.07, 95% CI [-0.13, -0.02], p = .02), and less REM sleep (b = -0.84, 95% CI [-1.55, -0.16], p = .02) predicted higher next-day stress, adjusting for previous pre-bedtime stress. Within-person effects showed similar trends, with longer SOL (b = 0.06, 95% CI [0.01, 0.11], p = .02), lower SE (b = -0.01, 95% CI [-0.02, 0.00], p =.047), less REM sleep (b = -0.21, 95% CI [-0.39, -0.03], p = .02), and less SWS (b = -0.36, 95% CI [-0.59, -0.13], p = .002) predicting higher next-day stress.

Adjusting for covariates, within-person effects showed that when individuals had shorter TST (b = -0.11, 95% CI [-0.21, -0.01], p = .04), lower SE (b = -0.02, 95% CI [-0.03, 0.00], p =.04), less SWS (b = -0.38, 95% CI [-0.66, -0.10], p = .008), and less REM sleep (b = -0.32, 95% CI [-0.53, -0.10], p = .004) relative to their average levels, they had higher stress the following day. No significant associations were found for between-person sleep and next-day stress. For self-reported sleep, shorter within-person TST predicted higher next-day stress (p = .041); no other significant results emerged (Supplementary Table S4).

Table 3. *Cross-lagged Multilevel Model Testing EEG-Estimated Sleep as a Predictor of Next-Day Stress*

	Between-Person Unadjusted	**Within-Person Unadjusted**	**Between-Person Adjusted**	**Within-Person Adjusted**
TST (h)	-0.08, <.01 [-0.45, 0.29]	-0.06, 0.04 [-0.15, 0.03]	0.22, 0.01 [-0.21, 0.65]	-0.11, 0.03* [-0.21, -0.01]
SOL ($\sqrt{min}$)	0.35, 0.04** [0.09, 0.61]	0.06, <.01* [0.01, 0.11]	0.32, 0.04 [0.00, 0.64]	0.05, <.01 [-0.02, 0.11]
WASO ($\sqrt{min}$)	0.19, 0.02 [-0.02, 0.40]	-0.01, <.01 [-0.06, 0.04]	0.19, 0.02 [-0.01, 0.40]	-0.02, <.01 [-0.07, 0.03]
SE (%)	-0.07, 0.04* [-0.13, -0.02]	-0.01, <.01* [-0.02, 0.00]	-0.03, 0.01 [-0.10, 0.04]	-0.02, 0.01* [-0.03, 0.00]
SWS (h)	-0.80, 0.01 [-1.90, 0.30]	-0.36, 0.01** [-0.59, -0.13]	-0.92, .01 [-2.38, 0.54]	-0.38, 0.02** [-0.66, -0.10]
REM (h)	-0.84, 0.04* [-1.55, -0.16]	-0.21, 0.01* [-0.39, -0.03]	-0.44, 0.01 [-1.28, 0.40]	-0.32, 0.02** [-0.53, -0.10]

Note. Results are unstandardized regression coefficients, Cohen's f^2, [95% confidence intervals]. * $p < .05$, ** $p < .01$. TST = Total Sleep Time; SOL = Sleep Onset Latency (square-root transformed); WASO = Wake After Sleep Onset (square-root transformed), SE = Sleep Efficiency (winsorized); SWS = Slow Wave Sleep. REM = Rapid Eye Movement. Adjusted models included baseline and daily covariates: age; sex; race/ethnicity; body mass index; employment status; English language acculturation; subjective social status; time spent in Melbourne; COVID-19 period; student status; smoking status; alcohol consumption; day of week; composite phase deviation.

Discussion

This study examined the temporal relations between daily stress and EEG-measured sleep across a 15-day intensive longitudinal design with repeated EMA, extending the stress-sleep literature that is primarily based on cross-sectional evidence. All models controlled for lagged outcomes to provide a rigorous test of directionality. The results showed that pre-bedtime stress did not predict any of the subsequent EEG-sleep variables after accounting for covariates. However, compared to one's own mean, nights with shorter TST, lower SE, less REM sleep, and less SWS predicted higher next-day stress, even after adjusting for covariates. These findings indicate a stronger unidirectional effect from sleep to next-day stress, compared to pre-sleep stress on sleep. Furthermore, after controlling for covariates, stress and EEG-sleep were not significantly associated on the between-person level, underlining the importance of considering day-to-day fluctuations in sleep.

Pre-bedtime Stress Predicting Same-night Sleep

Findings from the unadjusted models, with lagged outcomes included, showed that individuals with generally higher pre-bedtime stress (i.e., between-person effects) had significantly longer SOL, lower SE, and less REM sleep. These findings are consistent with previous studies where individuals exposed to experimentally-manipulated stressors (e.g., emotional stress from a failure experience) or during high-stress periods had significantly lower EEG-estimated SE or less REM sleep compared with neutral groups or during low-stress periods [23-25]. However, these findings became non-significant when adjusted for baseline (e.g., demographics and health factors) and daily (e.g., day of week; circadian misalignment) covariates. These adjusted findings are similar to those of a recent 7-day study showing no association between daily stress and EEG sleep adjusting for day of week, gender, and age [28].

The non-significant findings could be due to our sample not being powered to detect small effect sizes or that in young adults, naturalistic levels of stress do not predict

subsequent sleep. This direction is also observed in other daily studies that showed a more consistent direction of sleep predicting stress, rather than stress predicting sleep [9, 11], although one study found that higher within-person stress predicted longer self-reported sleep onset latency [8]. Differences in these findings could be due timing of the sleep measures, such that participants' sleep was measured in the following evening (vs the following morning in this study) that may have influenced the accuracy of the report. Additionally, it could be that major stressful events (e.g., after traumatic events; severed relationships) may have a larger and more consistent impact on sleep compared to daily stress [26]. Although our sample consisted of international and interstate students, their average daily stress levels were similar to other undergraduate students and international students in Australia [6], suggesting that despite additional demands due to moving and entering another culture, these students have normative stress levels. There also may be differences between perceived stress and physiological stress responses (e.g., cortisol). For example, in a sample of nurses and physicians, increased cortisol responses were associated with occurrences of stressful events (e.g., medical emergency; routine care), but over 70% of these responses occurred without individuals perceiving the events as stressful [61]. Future research should incorporate physiological markers, such as collecting daily cortisol samples or other biomarkers (e.g., skin conductance, heart-rate variability), in addition to perceived stress to understand interactions between psychological and physiological stress on daily sleep.

EEG-estimated Sleep Predicting Next-day Stress

After adjusting covariates, EEG sleep variables did not significantly predict next-day stress on the between-person level. On the within-person level, when individuals had a shorter than their own usual TST, SWS, or REM sleep, or lower than their usual SE, they experienced higher stress levels the following day. These results were adjusted for pre-sleep stress levels of the previous night, so the associations cannot be attributed to sustained or

carry-over stress from the previous day. These results are in line with previous daily studies using actigraphic and self-reported sleep measures that linked shorter and poorer sleep with higher stress [6, 7]. Our findings contrast with the null findings from the previous study examining nightly EEG sleep and daily stress [28]. This could be due to differences in assessing stress levels (e.g., retrospective recall assessing stress once per day in the mornings vs EMA in this study), analytical approach (i.e., we included lagged outcomes and influential covariates), or population as our sample was younger and diverse racially versus middle-aged and predominantly White. Our findings also add to the growing evidence consistent with emotional regulatory benefits of SWS and REM sleep [14, 15].

The observed stress-sleep relationships could be explained by previous studies showing the impact of short and poor sleep on the emotional regulatory system by potentiating emotional responses to stressor intensity [27, 62]. This interpretation is supported by our findings that less SWS and REM sleep predicted higher next-day stress, in accord with studies that have uncovered the functional role of SWS and REM sleep in mood and emotional regulation [12, 14, 15]. For example, a recent study suggested the anxiolytic benefits of SWS, as longer SWS was associated with higher medial pre-frontal cortex activity (important for emotional regulation) and lower self-reported anxiety levels [15]. Furthermore, neurobiological frameworks have indicated the functional benefits of REM sleep in optimal emotional regulation [12]. Although emotional reactivity towards stressors were not tested in this study, given the robust link between stress and emotions, it is possible that short and poor sleep can impact one's regulation of stress perception. In other words, it is possible that on nights when individuals had less SWS or REM sleep, they may have been deprived of the usual regulatory benefits, leading to a stronger reactivity to next-day stressors and thus perceiving them as more intense. Nonetheless, future studies are needed to further explore and clarify whether SWS and REM sleep predicts next-day emotional reactivity (e.g.,

increased negative affect and dampened positive affect) towards daily stressors on a daily, naturalistic basis.

Strengths and Limitations

This study is one of the first to examine the daily associations between stress and sleep in an understudied population of young adults who relocated from a different state or country for undergraduate studies. Our rigorous study design included repeated EMAs to measure perceived stress levels multiple times across the day, EEG-derived estimates of sleep, and a 15-day intensive longitudinal design. These methods addressed key limitations of previous studies, such as recall biases, under- and overestimations of sleep using actigraphic and self-report measures, and the reliance of single time-point or cross-sectional evidence. This study is one of the first to explore the ecology of nightly variations in EEG-estimated sleep parameters and architecture in relation to next-day stress, extending the findings from sleep-manipulation studies conducted in laboratory settings. Furthermore, our rigorous analytical methods allowed for a robust test of temporal directionality by including both within- and between-person predictors and lagged outcomes in all models. The confidence in our findings and precision estimation are further strengthened by including important covariates, such as daily circadian misalignment.

Nonetheless, several limitations should be acknowledged. First, participants were young adults and relatively healthy, thus the findings cannot be generalized to other age groups (e.g., children, older adults) or populations experiencing clinical conditions (e.g., individuals with insomnia or mood disorders). Future studies are needed to confirm these results in clinical populations and of different age groups, which may show and may show stronger associations and larger effect sizes. Second, the missing data in stress surveys and EEG-sleep measures may have influenced the results. For example, it is possible that participants may not wear the EEG device during nights with more stressful events, e.g.,

working on an assignment until late at night and going to bed immediately after, although we found no significant differences in pre-bedtime stress between nights with missing vs non-missing EEG sleep data. Relatedly, within-person reliability for next-day stress was on the low end of acceptable, which may explain some non-significant findings. It is also important to acknowledge that the current study only used a single-channel EEG to estimate the sleep parameters and architecture, which may still be less accurate than the gold-standard PSG sleep measure. There also may be first-night effects using the Z-Machine Insight+, as with every other in-person PSG studies, such that individual's sleep on the first night may be impacted by wearing the device [63], although this is mitigated by the within-person levels of analyses in our study. It is worthwhile noting that we did not control for multiple comparisons, which may increase our chances for making Type 1 error. Given that this is one of the first studies examining daily EEG sleep in naturalistic settings, we think it is important to examine the multi components of EEG sleep (i.e., parameters and architectures) and their associations with daily stress. Nonetheless, future studies are needed to replicate these findings. Finally, although every effort was made to robustly test the directionality of results through time-lagged predictions and covariates, our findings may still be influenced by unexplored confounds. Future studies involving experimentally-manipulated sleep in naturalistic settings (i.e., at home and, rather than severe sleep deprivation, manipulating sleep by minutes or hours) or populations with greater sleep disruption (e.g., shift workers) could provide evidence for whether there are causal effects of sleep on next-day stress in an ecologically valid context.

Conclusion and Implications

Our findings showed that young adult perceived higher stress the next day after nights with shorter than usual EEG-measured TST, REM sleep, SWS, and poorer than usual sleep quality. However, higher pre-bedtime stress was not associated with same-night sleep over

and above the effects of covariates. There are several implications, particularly for the educational sector and tertiary educational institutions as they relate to international students' daily well-being. First, all significant associations were within-person effects, highlighting the importance of day-to-day fluctuation of sleep on each individual's next-day experiences. The lack of robust between-person association between sleep and stress suggests that simply identifying poor sleepers may not be sufficient. Although the effect sizes observed may be relatively small, which could partly be attributable to the sleep measurement scale (i.e., in minutes, hours, or percentages), these results are still significant as having poor or short nightly sleep and high daily stress can cumulatively impact both physical and mental health [2-5]. Behavioural sleep strategies that are appliable in everyday settings for managing the fluctuating nature of nightly sleep, especially on nights of particularly short or poor sleep is needed. Finally, this study identified sleep architecture (especially REM and SWS) as an aspect of sleep that is relevant to individuals' experiences of stress. With the advancement of portable and consumer EEG, the incorporation of sleep architecture measures (e.g., SWS; REM) could be a fruitful area for future research to better understand sleep and its function in everyday life.

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Electronic Supplementary Materials

Table S1. *Descriptive Statistics for Demographic and Daily Variables by COVID-19 Period*

Participant Characteristics	Pre-COVID Period *M* (SD)	During COVID Period *M* (SD)	*p*-value
Daily Study Variables			
Stress levels			
Pre-Bed	2.40 (1.60)	2.76 (1.71)	.34
Next-Day	1.85 (1.38)	2.15 (1.77)	.37
Self-reported sleep			
Total sleep time (h)	7.40 (1.01)	7.56 (0.82)	.46
Sleep onset latency (min)	25.79 (49.31)	25.94 (21.63)	.99
Wake after sleep onset (min)	5.05 (5.46)	7.76 (11.64)	.12
Sleep efficiency (%)	93.88 (8.65)	93.16 (4.91)	.69
EEG-estimate sleep			
Total sleep time (h)	6.21 (0.96)	6.30 (0.71)	.64
Sleep onset latency (min)	23.19 (11.52)	21.51 (11.70)	.53
Wake after sleep onset (min)	49.24 (24.75)	47.18 (15.12)	.69
Sleep efficiency (%)	83.62 (6.23)	84.37 (4.35)	. 58
SWS (h)	1.50 (0.28)	1.22 (0.28)	<.001
REM sleep (h)	1.62 (0.45)	1.69 (0.43)	.51

Note. EEG = Electroencephalogram. SWS = Slow Wave Sleep. REM = Rapid Eye Movement Sleep. EEG-estimated and self-reported sleep efficiency, sleep onset latency, and wake after sleep onset presented are raw values. Participants' values were first averaged and *p*-values were based on the comparison between pre- and during COVID period using Independent-samples t-tests.

Table S2. Proportion of pre-bedtime stressors reported

Pre-bedtime Stressor Type	*%*
Work/School	13.94%
Health	5.13%
Finance	1.73%
Argument	6.24%
Home	2.68%
Discrimination	0.55%
Relationship	1.42%
Others	1.89%

Table S3. *Cross-lagged Multilevel Model Testing Pre-Bedtime Stress as Predictor of Self-Reported Sleep*

	Between-Person Unadjusted	**Within-Person Unadjusted**	**Between-Person Adjusted**	**Within-Person Adjusted**
TST (min)	-1.66, <.01 [-9.86, 6.55]	-1.44 <.01 [-4.32, 1.45]	-1.55 <.01 [-11.41, 8.30]	-0.77, <.01 [-3.51, 1.97]
SOL ($\sqrt{\text{min}}$)	0.09, <.01 [-0.21, 0.40]	-0.04, <.01 [-0.10, 0.02]	0.17, 0.01 [-0.18, 0.51]	-0.04, <.01 [-0.10, 0.02]
WASO ($\sqrt{\text{min}}$)	-0.01, <0.01 [-0.17, 0.14]	0.01, <0.01 [-0.06 0.07]	0.01, <0.01 [-0.16, 0.19]	0.01, <0.01 [-0.04, 0.07]
SE (%)	-0.33, <0.01 [-1.69, 1.04]	0.10, <0.01 [-0.20, 0.40]	-0.62, <0.01 [-2.17, 0.94]	0.12, <0.01 [-0.20, 0.44]

Note. Results are unstandardized regression coefficients, Cohen's f^2, [95% confidence intervals]. TST = Total Sleep Time; SOL = Sleep Onset Latency (square-root transformed); WASO = Wake After Sleep Onset (square-root transformed), SE = Sleep Efficiency (winzorised). Adjusted models included baseline and daily covariates: age; sex; race/ethnicity; body mass index; employment status; English language acculturation; subjective social status; time spent in Melbourne; COVID-19 period; student status; smoking status; alcohol consumption; day of week; composite phase deviation.

Table S4. *Cross-lagged Multilevel Model Testing Self-Reported Sleep as Predictor of Next-Day Stress*

	Between-Person Unadjusted	**Within-Person Unadjusted**	**Between-Person Adjusted**	**Within-Person Adjusted**
TST (h)	0.04, <.01 [-0.26, 0.34]	-0.04, 0.02 [-0.10, 0.03]	-0.05, <.01 [-0.38, 0.29]	-0.07*, <.01 [-0.13, 0.00]
SOL ($\sqrt{\text{min}}$)	0.03, <.01 [-0.11, 0.16]	0.02, 0.03 [-0.03, 0.07]	0.10, 0.01 [-0.05, 0.25]	0.02, <.01 [-0.03, 0.06]
WASO ($\sqrt{\text{min}}$)	0.10, <.01 [-0.17, 0.37]	0.003, 0.04 [-0.06, 0.07]	0.15, 0.01 [-0.18, 0.48]	0.004, 0.03 [-0.06, 0.07]
SE (%)	-0.01, <.01 [-0.04, 0.02]	0.004, <.01 [-0.01, 0.00]	-0.02, <.01 [-0.06, 0.01]	-0.004, <.01 [-0.01, 0.00]

Note. Results are unstandardized regression coefficients, Cohen's f^2, [95% confidence intervals]. * $p < .05$. TST = Total Sleep Time; SOL = Sleep Onset Latency (square-root transformed); WASO = Wake After Sleep Onset (square-root transformed), SE = Sleep Efficiency (winzorised). Adjusted models included baseline and daily covariates: age; sex; race/ethnicity; body mass index; employment status; English language acculturation; subjective social status; time spent in Melbourne; COVID-19 period; student status; smoking status; alcohol consumption; day of week; composite phase deviation.

CHAPTER 5:

Daily Associations between Saliva Cortisol and EEG-Assessed Sleep: A 15-Day Intensive Longitudinal Study

Preface to Chapter 5

Findings from **Chapter 4** showed that stress level around pre-bedtime was not associated with subsequent sleep. **Chapter 4** suggests that there may be differences in psychological versus physiological stress responses.

Chapter 5 aims to extend the findings of chapter 4 by examining a biomarker of stress, i.e., cortisol, and its association with sleep over 15-days. Specifically, daily diurnal cortisol was collected over 14 consecutive days, and EEG-sleep was recorded over 15 consecutive nights. In addition to extending the findings of **Chapter 4,** this chapter also provide physiological evidence of stress and its association with EEG-sleep, thus advancing and strengthening the stress-sleep knowledge base. Data from this study came from the Stress and Health Study.

This chapter is formatted as a manuscript in preparation for submission to ***SLEEP***

Daily Associations between Saliva Cortisol and EEG-Sleep: A 15-Day Intensive Longitudinal Study

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Abstract

Study Objectives

Current evidence suggests that cortisol levels and trajectories are bi-directionally associated with sleep. However, the daily, naturalistic cortisol-sleep associations remain unclear as the current evidence is mostly cross-sectional. This study tested whether pre-sleep cortisol predicts sleep duration and quality, and whether sleep parameters predict the following day's diurnal cortisol slope using a two-week intensive longitudinal design with electroencephalographic measures and saliva sampling.

Methods

Ninety-five young adults (M_{age}=20.48±1.59) provided saliva samples at awakening and pre-sleep over 14 consecutive days, providing 2345 samples (85% viable). Z-Machine Insight+ recorded over 900 nights of total sleep time (TST) and sleep efficiency (SE). Multilevel models, adjusting for covariates, tested these data at the between- and within-person level.

Results

Higher pre-sleep cortisol predicted shorter TST (p<.001), lower SE (p<.001), and longer SOL (p=.005) at the within-person level. Individuals with shorter average TST or lower average SE had flatter diurnal cortisol slope compared to those with longer average TST or lower average SE (both p<.001). Individuals with shorter average TST (vs longer average TST) had higher pre-sleep cortisol (p=.04), whereas those with lower average SE (vs higher average SE) had lower awakening cortisol (p=.002).

Discussion

Our findings provide evidence that higher pre-sleep cortisol predicts shorter and poorer quality sleep at the within-individual level under naturalistic conditions. Furthermore, individuals with short or poor sleep had flatter diurnal cortisol slope and higher pre-sleep

cortisol levels. These findings suggest that sleep maintains the regulation of the stress-response system, which is protective against mental and physical disorders.

Key Words

Sleep, EEG, Cortisol, Daily

Statement of Significance

This study examined the daily associations between cortisol and sleep using a powerful daily intensive longitudinal design with saliva sampling across 14 consecutive days and electroencephalographic sleep recordings across 15 consecutive nights. We found that, compared to one's own average, higher pre-sleep cortisol levels predicted subsequent shorter and poorer quality sleep that night. Individuals with shorter or poorer sleep on average had significantly flatter diurnal cortisol slope, which is indicative of a dysregulated stress-response system. These findings reinforce the importance of good sleep in regulating the stress-response system and maintaining good health.

Introduction

The Hypothalamus-Pituitary-Adrenal (HPA) axis is the central stress-response system, with cortisol being its primary end product and a biomarker of stress.[1] Cortisol often is used to assess HPA activity, given its responses to acute and chronic stressors.[2] Research suggests a bi-directional association between cortisol and sleep, such that higher cortisol levels can impair sleep, and poor quality or short sleep can lead to dysregulated cortisol levels.[3-5] The associations between day-to-day variations in cortisol and sleep under naturalistic conditions are not well understood because most data are from cross-sectional or laboratory designs. Most field studies of sleep have not used electroencephalographic (EEG) measures, and no studies to date have incorporated daily cortisol and EEG sleep measures across multiple days. Thus, this study examined whether pre-sleep cortisol levels predict subsequent EEG-assessed sleep, and whether EEG-assessed sleep predicts next-day diurnal cortisol, using a 15-day, intensive longitudinal design.

Higher daily stress is linked with shorter sleep and poorer sleep quality.[6-9] One study showed that self-reported pre-sleep somatic symptoms (e.g., sweating, heart racing) mediate this association.[10] One explanation for these findings is that the experience of stressors near bedtime activates HPA axis activity, leading to increased cortisol levels and physiological arousals that disrupt sleep.[11] This interpretation is supported by several cross-sectional studies showing that higher evening or bedtime cortisol levels are associated with shorter and poorer quality sleep from self-report or actigraphy measures.[12,13] Individuals with insomnia have been found to have higher evening and nocturnal cortisol compared to healthy controls;[14,15] higher evening and nocturnal cortisol levels were associated with more awakenings at night in both groups.[14] However, the temporal order of whether increases in cortisol levels during pre-sleep or bedtime can impair subsequent sleep, especially in naturalistic settings, remain unclear from these findings.

Cortisol secretion follows a diurnal rhythm; levels are high at awakening, peaking at about 30-40 minutes post awakening, and gradually decreasing across the day with the lowest levels around bedtime. Short or poor quality sleep has been associated with lower cortisol levels at awakening, higher evening or bedtime cortisol levels, and a flatter diurnal cortisol slope,[5,16-20] suggesting a potential bi-directional association between sleep and cortisol. A flatter diurnal cortisol slope, which could be due to lower awakening cortisol levels, higher evening cortisol levels, or both, is indicative of dysregulated HPA activity and has been associated with poorer mental and physical health outcomes.[2] Only a few studies to date have tested the associations between daily variations in sleep and cortisol in naturalistic conditions. A 3-day study in young adults found that shorter average sleep duration was associated with flatter diurnal cortisol slope, and that between- and within-person shorter sleep duration predicted lower next-day cortisol at awakening.[19] Another 3-day study had similar findings, where shorter between- and within-person actigraphic sleep duration predicted flatter diurnal cortisol slope.[18]

Although these findings show the daily associations between sleep and cortisol in naturalistic settings, they may not fully capture the variations in cortisol and sleep over longer periods. Recent evidence shows that 10 days of cortisol sampling is needed to reliably detect both between- and within-person differences in diurnal cortisol,[21] and a minimum of one week is needed to capture variation in sleep.[22] Furthermore, previous daily studies relied on self-reported or actigraphic measures of sleep, which may not be as accurate as EEG measures. Thus, this study examined the cortisol-sleep associations with 14-days of saliva sampling and 15-nights of EEG-assessed sleep. Specific hypotheses were: 1) higher pre-sleep cortisol levels (at the between- and within-person levels) will predict same-night shorter total sleep time (TST) and lower sleep efficiency (SE), and 2) shorter TST and lower SE (at the between and within-person levels) will predict a flatter next-day diurnal cortisol slope.

Methods

Transparency and Openness

Research materials are available [https://doi.org/10.17605/OSF.IO/TZ48Y]. Data will be made available on reasonable request and are planned for future public sharing in redacted form. The study and analysis plan were not pre-registered. Power analyses with α=0.05, 80% power as the target, and 10 predictors total, testing a single predictor with Cohen's f^2 effect sizes of 0.05 (small-to-medium, approximately equivalent to a correlation of r = .20, a "small" correlation) and 0.15 (the conventional cut off for a medium effect size) required 167 and 63 independent observations, respectively. Assuming a 75% completion rate on average over 14 days and intraclass correlation coefficients of 0.2 or 0.4, 75 participants provide 271 and 164 effective independent observations, respectively, achieving 80% power. Additional participants were recruited to account for potential attrition, missing data, and aims not related to this paper.

Participants

Ninety-eight undergraduate students participated in the Stress and Health Study, conducted between Feb 2019 – June 2020. This study focuses on stress, resilience, and health behaviours in emerging adults who recently relocated for tertiary education, and therefore the sample was predominantly of international descent. Ninety-five participants had usable cortisol samples. Figure 1 summarizes the eligibility criteria and participant flowchart, and Table 1 summarizes the participant characteristics.

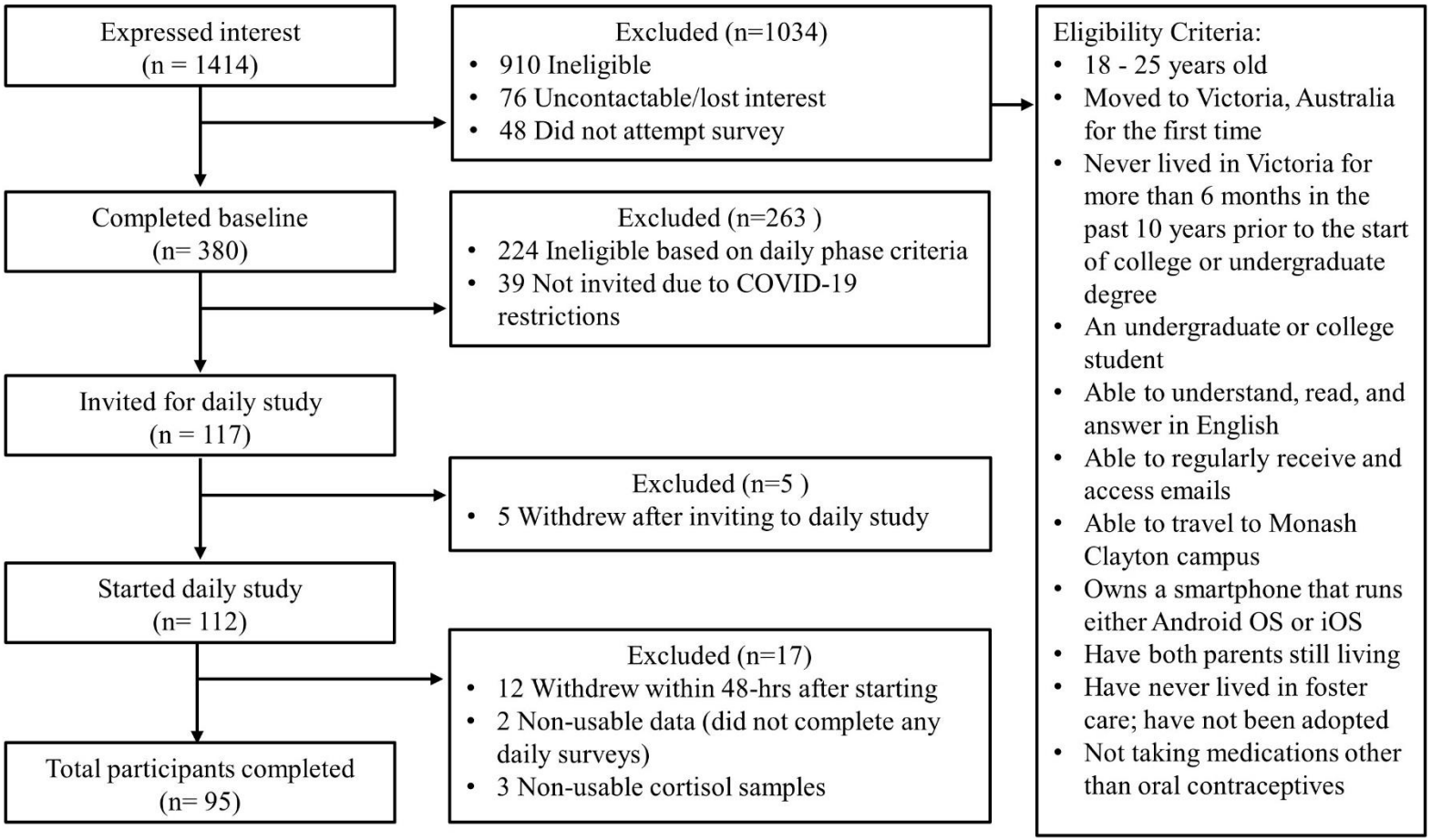

Figure 1. Summary of the Stress and Health Study recruitment process. The recruitment period was between February 2019 and June 2020

Design & Procedure

The study procedures were approved by Monash University Human Research Ethics Committee, and all participants provided informed consent. This study used a daily intensive longitudinal design. First, participants completed a baseline survey (~45 min) consisting of questions related to sociodemographic details, psychosocial factors, and health status. Eligible participants were then invited to the daily phase commencing on a Monday and ending 15 days after (i.e., Tuesday). On the first day, participants attended a 1-hour orientation to the study, where they were shown how to provide their saliva samples and wear the EEG sleep monitoring device (Z-Machine Insight+). For saliva cortisol collection, participants were instructed to provide saliva samples two times (i.e., at awakening and at pre-sleep) per day for 14 consecutive days. Once collected, participants wrote the date and time on the Salivette tubes, took photographs of those tubes, and uploaded them onto the MetricWire app to allow for digital timestamp and cross-examination of the written collection date and time. Participants were instructed not to 1) provide the samples when waking ± 2 hours outside of habitual wake time, or sleeping ± 4 hours outside of habitual bedtime; 2) brush their teeth, eat, or drink within the 30 minutes prior to sampling; and 3) have any dental work done within the 24 hours prior to sampling. Participants also were instructed not to consume any major meals, alcohol, nicotine (smoking), caffeine, and/or medication within the 60 minutes before saliva collection, and not to chew gum, perform vigorous physical activity, or drink water right before saliva collection. Participants were instructed to report any of these violations in the pre-sleep survey via MetricWire each day. For the EEG sleep monitoring device, participants were asked to wear the machine for 15 consecutive nights. Participants were instructed to clean their skin with the provided alcohol wipes and apply the sensors approximately 30 minutes before bedtime. Participants were instructed only to start the recording when they attempt to sleep.

Measures

Salivary cortisol. Salivary samples were collected using Salivettes[23] twice daily for 14 days: 1) at awakening within ±2 hours of habitual wake time, and 2) at bedtime within ±4 hours of habitual sleep time. The time window for saliva collection was based on a previous study to partially control for the influence of circadian phase on cortisol.[24] The decision to collect two samples per day was based on a previous study demonstrating that two cortisol samples taken per day for 10 days can better detect between- and within-person differences in diurnal cortisol slope compared to taking five samples per day for four days.[21] Diurnal slopes measured based on two cortisol samples taken at wake time and 21:00 correlated .97 and .99 with diurnal slopes measured using four and three samples, respectively. Thus, this approach can maximize the power to detect both between- and within-person differences while minimizing the cost of the collection and assay analysis, as well as participant burden. Samples were removed if 1) they were collected outside of the respective habitual wake or sleep time, 2) > 1-hour discrepancy between the digital timestamp and written label, and 3) > 30 min after waking. In total, 85% of the saliva samples were usable for analyses.

Sleep. EEG-estimated TST and SE were measured using the Z-Machine Insight+. Raw EEG signals were automatically scored as sleep or wake through an automated sleep–wake detection algorithm (Z-ALG).[25] Previous studies showed that the Z-ALG has high sensitivity and specificity for determining sleep (95.5% and 92.5%, respectively). High agreement between Z-ALG and polysomnography technologists is observed, with r = .95 for TST and r =.93 for SE.[25] On nights where we identified sensor errors or battery issues, TST, Wake After Sleep Onset (WASO), and SE for that night were set as missing. In total, 66% of EEG recordings for TST, WASO, and SE, and 77% of Sleep Onset Latency (SOL), were usable for analyses.

Covariates. Covariates were determined based on previous studies showing their associations with cortisol and sleep: age (years),[26,27] sex (coded as male/female),[26,27] race/ethnicity (coded as Asian/White/Others),[28,29] alcohol risk (coded as abstainer/moderate/at-risk),[26,30] smoking (coded as never vs current/former),[26,31] COVID-19 period (coded as pre [before Victoria lockdown 2020 March 8] vs. during),[32] work status (coded as working/not working),[29] student status (coded as international/interstate),[33] oral contraceptive use (coded as using vs not using),[26] body mass index (kg/m^2),[31,34] daily stress,[2,6] daily negative affect,[35,36] depressive symptoms,[37,38] anxiety symptoms,[37,38] day of week,[39] and daily circadian misalignment.[40-42] Saliva collection compliance, such as consuming any major meals, alcohol, nicotine (smoking), caffeine, and/or medication 60-minutes before saliva collection, and chewing gum, engaging in vigorous physical activity, or drinking water right before saliva collection, were included (coded as no violations/violations).[26,35]

Analytical Approach

All analyses were conducted in R v4.0.3, and a two-level linear mixed model (with observations nested within participants and using lme4 v1.1-26) was used to analyze these data. Degrees of freedom were tested and significance testing was performed using lmerTest v3.1-3. Pre-sleep cortisol and sleep variables were separated into between-person (i.e., the individual's average cortisol or sleep values across the study period) and within-person levels (i.e., the deviation of the cortisol or sleep values from the individual's average, also called person-mean centered). Given the skewness, cortisol was log transformed, and SE was winsorized (top and bottom 0.5%).

For models testing sleep as the outcome, pre-sleep cortisol, the lagged sleep outcome (to strengthen the test of directionality), and covariates were entered as predictors of sleep that night (i.e., TST and SE). To test diurnal cortisol slope as the outcome, the models included the interaction between time (i.e., awakening and pre-sleep) and sleep (between and

within-person levels), and covariates as predictors. Significant interaction effects were probed using simple slopes tests for long TST or high SE (i.e., *M*+1SD) and short TST or low SE (i.e., *M*-1SD). Mean differences between long/short TST or high/low SE within timepoints also were conducted to determine whether slopes were due to changes in awakening or pre-sleep cortisol levels (using emmeans v1.5.4). Follow-up analyses were planned for SOL and WASO if results for SE were significant. Due to skewness, SOL and WASO were square-root transformed and winsorized (top and bottom 0.5%). Cohen's f^2 effect size was calculated for all predictors. All adjusted models included between- and within-person predictors and covariates as fixed effects, whereas intercepts and within-person predictors were included as random effects. Convergence issues were resolved using the Nelder-Mead algorithm and by tightening tolerance values. Singularity issues were resolved by dropping the random effect variables with the lowest variance. Unadjusted models are reported in Supplementary S1.

Results

Table 1 summarizes the participant characteristics. Participants were mostly females (72%), international students (92.6%), and of Asian descent (85.3%). Participants were generally healthy with most indicating they never smoked (95.8%), were moderate drinkers (65.3%), and were within the healthy BMI range (67.4% within normal range, 16.8% underweight, and 14.7% overweight). Most participants were not using daily medications (94.7%), and about 5% reported using oral contraceptives. Participants' depression and anxiety symptoms were within normal levels (PROMIS T-Scores with a population mean = 50, *SD* = 10).[43] Daily average stress levels and negative affect were similar to young adults and working adults reported in other daily studies.[6,9,44] On average, participants' total sleep time was 6.23 hours (below the recommended 7-9 hours for young adults), and sleep efficiency was 83.9% (slightly below the recommended ≥ 85% cut-off).[45,46]

Table 1. *Sample Characteristics (N = 95)*

Variables	*M* (SD)/N (%)	*n* (ICC)
Age (years)	20.48 (1.59)	95
Female (vs Male)	75 (78.9%)	95
International Student (vs interstate)	88 (92.6%)	95
Race		95
Asian	81 (85.3%)	–
White	8 (8.4%)	–
Others	6 (6.3%)	–
Not working (vs working)	75 (78.9%)	95
Never Smoked (vs Current/Former)	91 (95.8%)	95
Not using oral contraceptive (vs using)	90 (94.7%)	95
Alcohol risk		95
Abstainer	22 (23.2%)	–
Moderate	62 (65.3%)	–
At-risk	11 (11.6%)	–
Body Mass Index (kg/m^2)	21.84 (3.43)	95
Depression (possible range: T-Score 31.7–81.3)	52.75 (9.24)	95
Anxiety (possible range: T-Score 31.7–81.3)	56.07 (9.09)	95
Average Daily Stress (possible range: 0–10)	2.14 (1.49)	95
Average Daily Negative Affect (possible range: 1–5)	1.48 (0.51)	95
Total Sleep Time (h)	6.21 (0.90)	943 (.71)
Sleep Onset Latency (min)	22.48 (11.62)	1100 (.74)
Wake After Sleep Onset (min)	48.36 (22.32)	943 (.64)
Sleep Efficiency (%)	83.90 (5.76)	943 (.65)
Awakening Cortisol (nmol/L)	11.32 (14.68)	1142 (.77)
Pre-Sleep Cortisol (nmol/L)	1.29 (1.93)	1203 (.66)

Note. ICC = Intraclass Correlations. *n* = Number of observations. Values in parenthesis for *n*(ICC) indicate percent of variance within individuals. Sleep Onset Latency, Wake After Sleep Onset, and Sleep Efficiency, and Cortisol presented are raw values.

Pre-sleep Cortisol Predicting Sleep

Table 2 (first four rows) summarises the adjusted models of pre-sleep cortisol (between- and within-person levels) predicting sleep. Within-person effects showed that a higher pre-sleep cortisol level was significantly associated with shorter subsequent TST (p <.001) and lower SE (p <.001). This indicates that on days when participants had higher than usual pre-sleep cortisol levels, they had shorter sleep duration and poorer SE that night. Given the significant association with SE, follow-up analyses were conducted on SOL and WASO. Higher than usual pre-sleep cortisol predicted longer SOL (p = .005) but did not associate with WASO. No significant results emerged at the between-person level. Results for unadjusted models (see Supplementary Table S1) showed that higher pre-sleep cortisol predicted shorter TST and lower SE. However, unadjusted results for SOL were non-significant.

Sleep Predicting Diurnal Cortisol Slope

Table 2 (last four rows) summarizes the adjusted models of the interaction between sleep (between- and within-person levels) and time (awakening and pre-sleep), predicting next-day diurnal cortisol slope. A significant time x TST interaction effect on next-day cortisol (p <.001) emerged at the between-person level (Figure 1, Panel A). Specifically, individuals with shorter average TST had a flatter diurnal cortisol slope (b = -2.24, p <.001) compared to individuals with longer average TST (b = -2.71, p <.001). A similar interaction emerged for SE at the between-person level (b = -0.03, p <.001; Figure 1, Panel B). Individuals with a lower average SE had a significantly flatter diurnal cortisol slope (b = -2.20, p <.001) compared to individuals with higher average SE (b = -2.74, p <.001). Follow-up analyses were conducted to clarify whether the flatter slopes were due to lower cortisol levels in the morning and/or higher cortisol levels during pre-sleep. Individuals with shorter

average TST had significantly higher pre-sleep cortisol levels compared to individuals with longer average TST (M_{diff} = 0.38, p = .04); no significant differences emerged for awakening cortisol levels between long vs short TST. Individuals with lower average SE had significantly lower awakening cortisol levels compared to individuals with higher average SE (M_{diff} = -0.49, p = .002); no significant differences emerged for pre-sleep cortisol levels between high vs low SE.

Follow-up analyses were conducted on SOL and WASO given the significant effects of time x SE on diurnal cortisol. Results showed a significant time x WASO interaction at the between (p <.001) and within-person levels (p = .011). Specifically, longer average WASO (between-person effects; b = -2.36, p <.001) or longer than average WASO (within-person effects; b -2.39, p <.001) on the previous night predicted a flatter diurnal cortisol slope. Similar to SE, longer average WASO (vs shorter average WASO; M_{diff} = 0.25, p = .009) or longer than usual WASO (vs shorter than usual WASO; M_{diff} = 0.14, p = .01) the previous night had lower awakening cortisol. No significant differences emerged for pre-sleep cortisol levels. No significant findings emerged for SOL.

Results for unadjusted models showed a significant interaction of between-person TST, SE, or WASO and Time on diurnal cortisol, and the directions were similar to the adjusted models (see Supplementary Table S1). No significant results emerged for within-person WASO.

Table 2. *Multilevel Model Testing Cortisol as Predictor and Outcome of Sleep (N = 95)*

	Between-Person level	**Within-Person level**
	Pre-Sleep Cortisol as Predictor of Sleep	
TST (h)	-0.44 [-0.91, 0.02] p = .07, f^2<.01	**-0.39 [-0.55, -0.24]** ***p* <.001, f^2=0.04**
SE (%)	-2.08 [-5.12, 0.97] p = .19, f^2<.01	**-1.91 [-2.74, -1.08]** ***p* <.001, f^2=0.04**
SOL ($\sqrt{\text{min}}$)†	0.13 [-0.52, 0.79] p = .69, f^2<.01	**0.30 [0.09, 0.51]** ***p* = .005, f^2=0.02**
WASO ($\sqrt{\text{min}}$)†	0.32 [-0.49, 1.14] p = .44, f^2<.01	0.14 [-0.08, 0.35] p = .22, f^2<.01
	Sleep x Time as Predictor of Diurnal Cortisol Slope	
TST(h) x Time	**-0.16 [-0.25, -0.08]** ***p* <.001, f^2=0.01**	0.03 [-0.03, 0.09] p = .40, f^2<.01
SE (%) x Time	**-0.03 [-0.05, -0.02]** ***p* <.001, f^2 =0.02**	-0.01 [-0.02, 0.00] p = .12, f^2<.01
SOL($\sqrt{\text{min}}$) x Time†	0.01 [-0.06, 0.07] p = .86, f^2<.01	-0.01[-0.06, 0.04] p = .66, f^2<.01
WASO($\sqrt{\text{min}}$) x Time†	**0.09 [0.04, 0.14]** ***p* <.001, f^2=0.01**	**0.05 [0.01, 0.10]** ***p* = .011, f^2=0.01**

Note. Results are reported as unstandardized coefficients, [95% Confidence Interval], *p*-values, f^2 effect size. Values in bold denote significant results. Cortisol values are log transformed. SOL and WASO are square-root transformed. TST = Total Sleep Time. SE = Sleep Efficiency. SOL = Sleep Onset Latency. WASO = Wake After Sleep Onset. † = Follow up analyses given significant results for Sleep Efficiency.

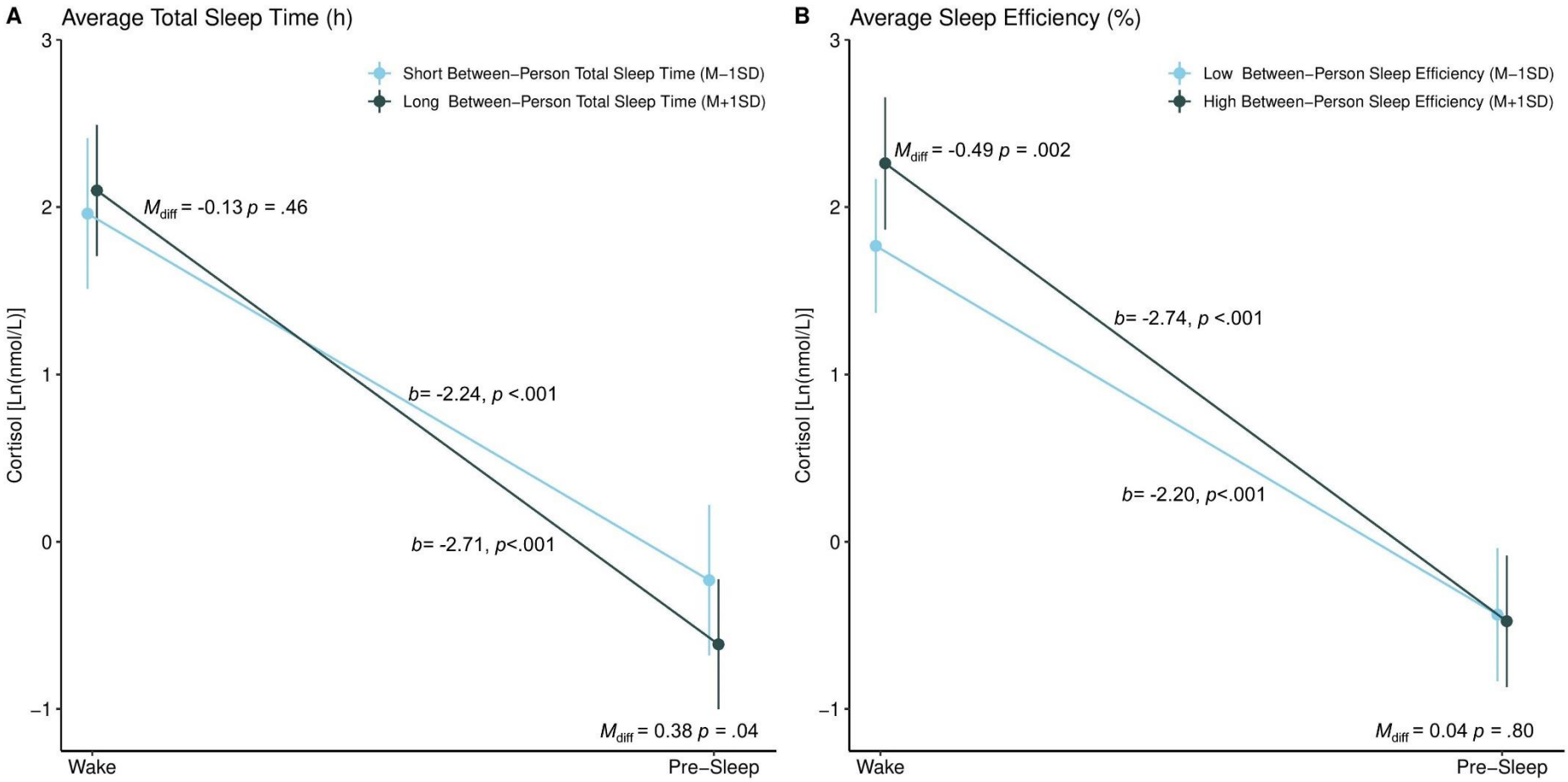

Figure 2.
Panel A shows the interaction of between-person total sleep time and time (wake and pre-sleep) on cortisol.
Panel B shows the interaction of between-person sleep efficiency and time (wake and pre-sleep) on cortisol.

Discussion

This study tested the daily cortisol-sleep associations using an intensive longitudinal design with 14 days of cortisol sampling and 15 nights of EEG-assessed sleep. We found that higher pre-sleep cortisol levels predicted subsequent shorter TST and lower SE at the within-person level. No significant results emerged for average pre-sleep cortisol levels predicting sleep (i.e., between-person effects). Individuals with shorter TST and lower SE on average had a flatter diurnal cortisol slope across the day (i.e., between-person effects). No significant results emerged for nightly variations in TST or SE predicting diurnal cortisol slope, although follow-up analyses showed that longer average WASO and longer than usual WASO predicted a flatter diurnal slope the next day.

The current findings determine the temporal relationships between cortisol and sleep at a daily level, extending on previous cross-sectional studies[12-14]. Specifically, regardless of individuals' own average pre-sleep cortisol levels, on nights where individuals had higher than usual pre-sleep cortisol levels, they had shorter EEG-estimated sleep duration and lower sleep efficiency that night. The directionality of these findings was further strengthened by the inclusion of lagged outcomes, meaning that the effects on sleep observed that night are independent of the previous night's sleep. These associations may be due to a direct effect of cortisol on sleep. Cortisol secretion is associated with physiological arousals (e.g., increased heart rate; temperature) and subsequent increase in feelings of alertness or activeness.[47] These physiological and cognitive arousals can disrupt sleep. This interpretation is supported by our follow-up analyses showing that higher pre-sleep cortisol levels predicted longer time taken to fall asleep. Together, these findings showed that higher than usual cortisol levels around bedtime predicts poorer and shorter subsequent sleep.

Individuals with short or poor sleep on average had flatter diurnal cortisol slope across the day compared to individuals with average long or good sleep. These findings are in

line with previous studies showing shorter average sleep duration or self-reported poorer sleep quality associate with flatter diurnal slope.[5,16-20] However, we did not find any significant within-person effects, which is contrary to previous findings showing shorter than usual sleep duration predicted flatter diurnal slope the following day.[18] Inconsistencies in these findings could be due to the differences in cortisol saliva sampling frequency (14 days in this study vs three days in the previous study) and sleep measurements (EEG vs actigraphy). Our follow-up analyses indicated that individuals with shorter average sleep duration (vs longer average sleep duration) had significantly higher pre-bedtime cortisol, resulting in a flatter diurnal slope. Previous studies have shown that individuals with partial or total sleep restriction have slower decline of cortisol concentrations throughout the day, resulting in higher evening cortisol levels, which reflect dysregulation of the negative feedback regulation of the HPA axis.[48,49] Furthermore, individuals with low average sleep efficiency (vs high average sleep efficiency) had significantly lower awakening cortisol levels. Previous studies show that night-time awakenings are associated with a subsequent increase in cortisol level, followed by a temporary inhibition of cortisol secretion.[3,50] Thus, it is possible that lower awakening cortisol levels could be due to the temporary inhibition of secretion after elevation of night-time cortisol from the night-time awakenings, as hypothesized by Backhaus and colleagues.[16] Supporting this explanation, our follow-up analyses indicated that individuals with high average WASO, as well as on nights with higher than usual WASO, had significantly lower awakening cortisol levels the following day. Nonetheless, this hypothesis cannot be directly tested in our study, since cortisol was not sampled overnight. Collectively, these findings suggest that poor and short sleep are associated with a dysregulated HPA axis, as indicated by a dysregulated diurnal cortisol slope.

A key strength of our study was the collection of salivary cortisol across 14 consecutive days and the use of EEG sleep recordings across 15 consecutive nights, extending previous cross-sectional and daily studies. Specifically, this study was longer than most previous studies examining the cortisol-sleep relationship and used more accurate sleep measures compared to studies using self-report or actigraphic measures.[5,18,19] Together, these methods allowed a more reliable detection of within- and between-person differences in diurnal cortisol slope.[21] Our focus on maximizing compliance (85% usable cortisol data) and minimizing participant burden in cortisol collection also strengthened results.[51] Furthermore, our rigorous analyses examined cortisol and sleep variables at the between- and within-person levels, adjusted for multiple covariates, and accounted for lagged outcomes for models examining cortisol predicting subsequent sleep to strengthen the directionality of effects. Nonetheless, our study only examined cortisol levels twice daily, which cannot provide information on cortisol awakening responses or reliable estimates of total daily cortisol output. Furthermore, these results cannot provide a full understanding of the HPA axis and other stress-response systems (e.g., autonomic nervous system), as other physiological indicators were not examined. Although multiple covariates related to cortisol and sleep were adjusted for in our analyses, these findings may still be impacted by unexplored confounds, such as menstrual cycle phase, and should not be interpreted as causal.[26] Lastly, these findings cannot yet be generalized to clinical populations, individuals on medications, or to older populations that have different cortisol and sleep profiles.[26,52]

In summary, our findings provide a deeper understanding of the cortisol-sleep relationship in a naturalistic setting. Our results provide within-person evidence of higher cortisol levels at pre-sleep time predicting shorter sleep, poorer sleep quality, and longer time to fall asleep that night. These findings support cortisol levels during pre-sleep as a potential mechanism for the association between daily stress and sleep. Moreover, individuals with

shorter or poorer average sleep had a significantly flatter diurnal cortisol slope. These findings could inform daily interventions aiming to reduce cortisol levels to improve sleep. For example, meta-analyses suggest that mindfulness-based interventions can reduce cortisol levels [53,54] and improve sleep duration and quality.[55,56] It is possible that these interventions may improve sleep partly through reducing cortisol levels, although future studies are needed to test these mediating pathways. These findings also reinforce the importance and benefits of good sleep (both duration and quality) on our HPA axis functioning. Flatter diurnal slope is associated with poorer mental and physical health outcomes.[2] Improving sleep duration and quality may help maintain or improve the HPA regulation, thus lowering risks of developing mental and physical disorders.

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Supplementary S1

Table S1. *Multilevel Model Testing Cortisol as Predictor and Outcome of Sleep (N = 95)*

	Between-Person level	**Within-Person level**
	Pre-Sleep Cortisol as Predictor of Sleep	
TST (h)	-0.26 [-0.63, 0.10] p = .16, f^2 <.01	**-0.39 [-0.53, -0.25]** ***p* <.001, f^2=0.03**
SE (%)	-0.47 [-2.87, 1.94] p = .70, f^2<.01	**-1.84 [-2.62, -1.06]** ***p* <.001, f^2=0.04**
SOL ($\sqrt{min}$)†	-0.02 [-0.46, 0.42] p = .92, f^2<.01	0.16 [-0.01, 0.33] p = .08, f^2=0.02
WASO ($\sqrt{min}$)†	-0.12 [-0.76, 0.52] p = .71, f^2 <.01	0.13 [-0.07, 0.33] p = .20, f^2 <.01
	Sleep x Time as Predictor of Diurnal Cortisol Slope	
TST(h) x Time	**-0.15 [-0.23, -0.08]** ***p* <.001, f^2 =0.01**	0.02 [-0.04, 0.07] p = .54, f^2<.01
SE (%) x Time	**-0.03 [-0.04, -0.02]** ***p* <.001, f^2=0.01**	0.004 [-0.01, 0.01] p = .41, f^2<.01
SOL($\sqrt{min}$) x Time†	0.02 [-0.04, 0.07] p = .57, f^2<.01	-0.005 [-0.04, 0.03] p = .81, f^2<.01
WASO($\sqrt{min}$) x Time†	**0.07 [0.03,0.12]** ***p* = .001, f^2=.01**	0.02 [-0.01, 0.06] p = .20, f^2<.01

Note. Results are from unadjusted models. Results are reported as unstandardized coefficients, [95% Confidence Interval], *p*-values, f^2 effect size. Values in bold denote significant results. Cortisol values are log transformed. SOL and WASO are square-root transformed. TST = Total Sleep Time. SE = Sleep Efficiency. SOL = Sleep Onset Latency. WASO = Wake After Sleep Onset. † = Follow up analyses given significant results for Sleep Efficiency

CHAPTER 6:

Sleep Moderates the Association Between Stress and Dietary Intake: A 7-Day Intensive Longitudinal Design With Ecological Momentary Assessments

Preface to Chapter 6

This chapter moves beyond examining stress or sleep as the outcome; instead, the synergistic predictions of sleep and stress on dietary intake were examined. Research shows that poor or short sleep and high psychological stress are associated with increased food intake and poor diet quality. However, the current understanding of the associations of stress and sleep with diet is based mainly on cross-sectional or between-person evidence, and most studies only examined these associations independently. Whether stress and sleep interact to predict dietary intake, especially in a daily, naturalistic setting, remains unclear. Given that **Chapter 2** and **4** showed that longer and better sleep predicted lower stress, it is possible that sleep can mitigate the impact of stress on dietary intake. This study examined whether sleep would moderate the associations between daily stress and dietary intake using a 7-day intensive longitudinal design. Data for this paper came from the Stress and Health Study.

This chapter has been submitted to ***SLEEP*** and is presented in manuscript form.

Sleep Moderates the Association Between Stress and Dietary Intake: A 7-Day Intensive Longitudinal Design with Ecological Momentary Assessments

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Abstract

Study Objectives

When individuals are stressed or have poor sleep, they tend to have poorer diet quality. How sleep, stress, and diet interact in a daily, naturalistic setting remains unclear. This study tested the interaction between electroencephalography (EEG) assessed sleep and daily stress on daily dietary intake using a 7-day intensive longitudinal design in 72 young adults.

Methods

Stress was measured four times daily (morning, afternoon, evening, and pre-bedtime). ZMachine Insight+ recorded EEG total sleep time (TST), sleep efficiency (SE), slow-wave sleep, and rapid eye movement (REM) sleep. Automated Self-Administered 24-hour food records measured daily energy intake and percentage of energy intake from discretionary food. Cross-lagged multilevel models, adjusted for covariates and lagged outcomes, were used to analyze the data.

Results

A crossover interaction emerged for within-person stress with between-person TST (p=.046) and REM sleep (p=.011) predicting energy intake, with higher stress predicting greater energy intake for short TST (p=.068) or REM sleep (p=.053), but lower energy intake for long TST (p =.27) or REM sleep (p =.072). Between-person SE moderated the between-person stress effect on energy intake (p=.041), with higher stress predicting greater energy intake for low SE (p=.014). Main effects showed that shorter within-person TST (p =.030) or REM sleep (p=.008) predicted a higher percentage of energy intake from discretionary food.

Discussion

These findings show that sleep is an important moderator of stress-diet associations. The results suggest that behavioral interventions aiming to improve diet could be enhanced by targeting sleep.

Key Words

Stress, Sleep, Diet, EMA, Daily

Statement of Significance

This study tested the moderating role of sleep on the stress-diet association using a 7-day intensive longitudinal design. Daily stress was measured using repeated ecological sampling, and objective nightly sleep parameters and architecture were measured using electroencephalography. A robust food record captured food and beverage intake, allowing total energy intake and energy intake from discretionary foods to be calculated at the daily level. Findings showed that individuals with overall short sleep duration, poor sleep quality, or short rapid eye movement (REM) sleep duration had significantly more total daily energy intake when experiencing higher daily stress. Furthermore, compared to one's average, nights with shorter sleep duration or REM sleep predicted higher consumption of energy from discretionary foods.

Introduction

Poor diet is associated with negative health outcomes, including obesity, hypertension, diabetes, and cardiovascular diseases.[1,2] Factors that can contribute to poor diet include high psychological stress and poor sleep, which are associated with increased food intake or consuming more unhealthy foods.[3-5] However, most studies have only examined the effects of stress or sleep on diet independently. It remains unclear whether stress and sleep synergistically interact to influence dietary intake, especially in naturalistic settings. Thus, we aimed to test whether poor sleep moderates the associations between daily stress and dietary intake.

Research has linked higher perceived stress with increased food intake and increased intake of unhealthy foods.[4] These unhealthy foods can be categorized as discretionary foods (e.g., ice cream; potato chips), which are not required in a healthy diet. These discretionary foods are usually high in energy, saturated fat, sugar, salt, and/or low fiber and essential nutrients.[2] Stress manipulation studies show that individuals under high-stress conditions (e.g., unsolvable puzzle; difficult arithmetic tasks) consume more high-fat and high-sugar food than individuals under low-stress conditions (e.g., easy Sudoku puzzle; easy arithmetic tasks).[6-8] In naturalistic settings, a few daily studies show that individuals who experience higher than usual daily hassles report greater intake of snacks high in fat and sugar, more unhealthy snacking, and higher perceived food intake.[9-12] However, there is other evidence also linking higher stress with decreased food intake or null findings.[4,13,14] These findings highlight the heterogeneity of the stress-dietary intake associations, perhaps due to individual differences (e.g., eating style behaviors; cortisol reactivity levels).[4,10,13,15]

A recent meta-analysis recommended future studies to examine potential moderators of the stress-dietary intake relationship that could help to explain inconsistent findings.[4] Sleep, a modifiable factor, may be one key moderator of the stress-dietary intake relationship.

Sleep and stress are bi-directionally associated,[16] and poor and short sleep also are linked with poor diet quality.[3,5] Sleep manipulation studies show that individuals with restricted sleep opportunity (4 – 5.5 hours) eat more energy-dense snacks and have higher total energy intake compared to individuals with 8 – 12 hours of sleep opportunity.[17-21] Cross-sectional findings show a similar trend, with shorter self-reported sleep duration and poorer sleep quality associated with higher energy intake and poorer diet quality.[3,22] Only a few studies have examined the associations between sleep architecture and dietary intake.[23,24] One crossover sleep restriction study found that lower slow-wave sleep (SWS) and rapid eye movement (REM) sleep percentages were associated with higher fat intake, and higher REM sleep percentage was associated with lower carbohydrate intake.[24] Although there were no changes in overall energy intake, these findings suggest that changes in SWS and REM sleep are associated with one's dietary intake. However, whether these findings translate into naturalistic settings remains unclear.

To our knowledge, only one study has tested the interaction of stress and sleep on diet.[25] A 21-day study found that higher work-related stress in the morning predicted a lower number of healthy food (e.g., fruits, vegetables) and a higher number of unhealthy foods (e.g., soda, sugary drinks) consumed in the evening. However, on days when participants reported better sleep quality, the relationship between stress and unhealthy food consumption was weaker than on days with poorer sleep quality. This suggests that good sleep quality may mitigate (or poor sleep can exacerbate) the impact of stress on discretionary food intake.[25] Given that studies have shown the emotional regulatory benefits of SWS and REM sleep,[26-28] it is possible that SWS and REM sleep can ameliorate the impact of stress on dietary intake.

Because most studies have examined stress and sleep independently as predictors of dietary intake, the stress x sleep interaction on dietary intake, especially in a naturalistic setting, remains unclear. Thus, this study tested whether sleep moderates the stress-diet intake

association using a seven-day, intensive longitudinal design in young adults. We used ecological momentary assessments (EMA) to measure daily stress, electroencephalography (EEG) measures of sleep, and a rigorous dietary food record to measure dietary intake. We hypothesized that sleep would moderate the associations between daily stress and dietary intake (i.e., total daily energy intake and percentage of total energy intake from discretionary food). Specifically, the positive association between daily stress (at the between and within-person level) and dietary intake would be stronger when individuals had poor sleep (i.e., between-person or within-person *previous* night shorter TST, lower sleep efficiency, less SWS, and less REM sleep) compared to individuals with good sleep.

Methods

Participants

Ninety-eight participants participated in the Stress and Health Study, which aimed to examine the daily interactions between stress, resilience, and health behaviors in young adults who moved from a different state or country for their tertiary studies in Victoria, Australia. The dataset was restricted to 72 participants with plausible average reported energy intake based on the revised Goldberg cut-off method, which is commonly used to identify misreporting of self-reported food intake [29,30] More specifically, participants with an average $\frac{\text{Energy Intake}}{\text{Basal Metabolic Rate}}$ ratio outside of the 95% confidence limits were excluded from analysis (i.e., values below or above the 95% confidence limits indicate under- or over-reporting, respectively), as this is suggestive of misreporting. A total of 26 participants (24 under-reporters, one over-reporter, and one missing diet data) were excluded. Figure 1 illustrates the participant flow diagram and eligibility criteria.

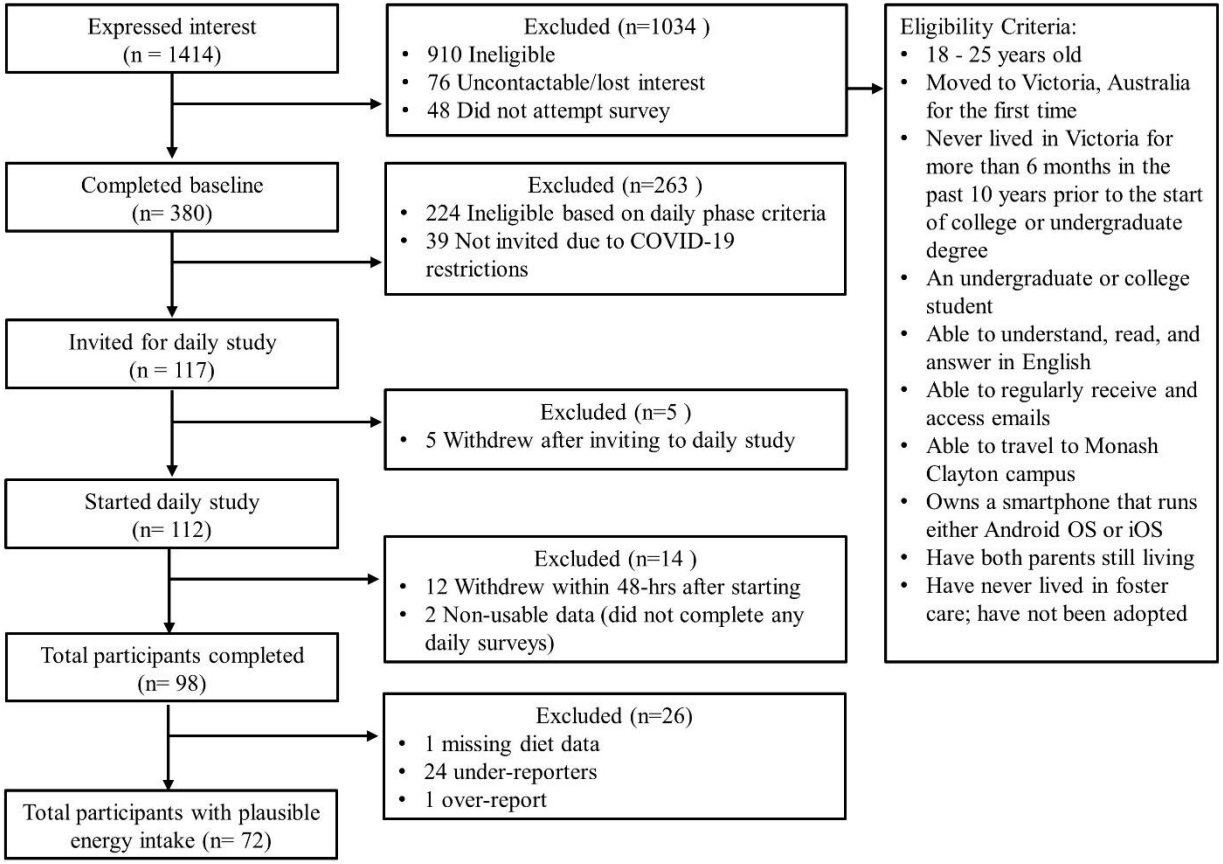

Figure 1. Summary of the Stress and Health Study recruitment process. The recruitment period was between February 2019 and June 2020.

Design & Procedure

All procedures were approved by the Monash University Human Research Ethics Committee (#17281). All participants provided informed consent. The current study used a seven-day intensive longitudinal design with repeated EMA. This approach captures participants' experiences in real-time and naturalistic settings, which enhances external validity while reducing memory biases associated with retrospective recall methods.[31,32] Through repeated assessments, participants also served as their own control.[31]

Participants first completed a baseline survey (approximately 45 minutes) assessing demographics and other self-reported psychological and health measures. Participants then attended a 1-hour, in-person orientation to the study on a Monday, where they collected the research devices and were shown how to apply the EEG sleep device (Z-Machine Insight+), complete the daily surveys via a mobile application (MetricWire), and complete their food record via Automated Self-Administered 24-hour (ASA-24) Dietary Assessment Tool (version 2016), developed by the National Cancer Institute.[33] Participants were instructed to complete the food record immediately after consuming any meals, snacks, or drinks to encourage complete reporting of all foods and drinks, as well as the amounts. Participants also were instructed to maintain their usual dietary intake and habits throughout the study period, and they were provided with contact information to clarify any collection procedures or potential issues. Participants commenced the daily phase on a Tuesday and ended seven days after, providing five weekdays and two weekend days.

Measures

Perceived Daily Stress. Throughout the study period, participants completed four stress surveys per day: morning (06:00–11:00), afternoon (12:00–15:00), evening (16:00–19:00), and during pre-bedtime (20:30–04:30). These surveys were open only during each time window to ensure real-time experiences were captured. Automatic hourly push notifications were sent to participants if surveys were not completed. The median completion time for the surveys were: 19.7 min after waking for morning survey, 66.3 min after open for afternoon survey, 60.7 min after open for evening survey, and 51.8 min before bedtime. Perceived daily stress was self-reported based on an adaptation of the Daily Inventory of Stressful Events scale.[34,35] In this study, we analyze a single item, "Since the previous survey, how stressful has your day been?" ranging from 0 (Not at all stressful) to 10 (Very stressful). The four surveys were averaged each day to create a composite measure of daily stress.

Sleep. Z-Machine Insight+, a single-channel EEG sleep monitoring device manufactured by General Sleep, Inc. Cleveland, OH, recorded daily TST, sleep efficiency, SWS, and REM sleep at 30-seconds epoch. The Z-ALG13 automatically scored EEG signals as sleep or wake, with high sensitivity (95.5%) and specificity (92.5%) for determining sleep.[36] The Z-PLUS algorithm determined sleep stages as light, SWS, or REM sleep, with predictive positive values of 0.85, 0.83, and 0.76, respectively.[37] Overall kappa agreement is 0.85 and 0.72 for Z-ALG13 and Z-PLUS, respectively.[37] Self-reported bed and rise time, adapted from the Consensus Sleep Diary[38], were collected during the wake survey to cross-validate the sleep period. For nights with EEG sensor errors or battery issues TST, sleep efficiency, SWS and REM were set as missing. In total, 81% of the sleep data were usable for analyses.

Dietary Food Record. Dietary records were collected for seven consecutive days using the Australian version of the ASA-24 dietary assessment tool.[33] In its original 24-hour

recall form, this tool has been validated against interviewer-administered 24-hour recall[39] and biomarkers.[40] Participants can select the type of food item eaten from a list, or enter a new food, and pictures of different sized portions are shown to assist with reporting of the amount consumed. The ASA-24 provides estimates for total energy intake (kJ) at the daily level as well as a food code for each item based on the Australian Food, Supplement, and Nutrient (AUSNUT) 2011-2013 database.[41] Discretionary items were determined by matching the food codes from participants' reported intake with the discretionary food items provided by the Australian Bureau of Statistics.[42] Percentage of daily energy intake from discretionary food was determined by dividing daily energy intake (kJ) from discretionary food by total daily energy intake (kJ). Text messages were sent to remind participants to complete their food records, and follow-up phone calls were made on Thursdays to clarify any issues related to the food record. On days where participants' food record was indicative of underreporting (i.e., flagged based on Goldberg's original underreporting cut-off value $\frac{\text{Energy Intake}}{\text{Basal Metabolic Rate}}$ ratio < 0.90),[30,43] participants were followed up via phone call to clarify their food intake.

Covariates. Baseline sociodemographic and daily covariates were selected based on previous studies showing their associations with diet. These included: Age (years),[44] sex (male/female),[45] Body Mass Index (BMI; kg/m^2),[46] race/ethnicity (White/Asian/Other),[47] student status (international/interstate),[48] employment status (working/not working),[49] time spent in Melbourne (years),[47] smoking status (Never/Current/Former),[50] alcohol intake (Abstainer/Moderate/At-Risk),[51-53] COVID-19 period (pre [before the start of Victoria lockdown on 08/03/2020] vs. during lockdown),[54] subjective social status,[55,56] adequacy of factors influencing diet (e.g., cooking skills; appliances for food preparation),[57] day of the week,[58] and daily physical activity (average Metabolic Equivalents during wake hours)[59] using an accelerometer (ActiGraph wGT3X-BT) worn continuously throughout the study.

Analytical Approach

All analyses were run in R (v.4.0.3).[60] Linear mixed models (lme4 v1.1-26), with degrees of freedom and significance testing using lmerTest v3.1-3, tested the cross-level interactions between stress and sleep on dietary intake (i.e., total daily energy intake and percentage of daily energy intake from discretionary food). SE and total daily energy intake were winsorized at the top and bottom 0.5% and 1%, respectively, and percentage of total energy intake from discretionary food was square-root transformed to address skewness. Repeatedly measured predictors were decomposed into between-person (i.e., participants' own average) and within-person (i.e., deviations from the individual's own mean) levels.

In all adjusted models, cross-level interactions between stress and sleep (i.e., between-person daily average stress, between-person average sleep, within-person daily average stress, and within-person *previous* night sleep), lagged outcome (i.e., within-person diet [Day *i*-1]), and covariates were included as fixed effects. Lagged outcome and covariates were included to strengthen the test of directionality and precision of estimates. Random effects included random intercept by ID. Given the singularity issues, within-person predictors were removed from the random effects in all models. Model diagnostics were conducted. Residuals distributions improved after removing extreme values (at 0.5% with 4-17 observations and/or 1-2 IDs removed across all models) for models with total daily energy intake as the outcome. Non-significant interactions were dropped from the model, and significant interactions were followed up using simple slopes tests (using emmeans v1.5.4) for longer/better (+1 SD from the mean) and shorter/poorer (-1 SD from the mean) sleep.[61] Models were separated by each sleep variable. Main effects of daily average stress and sleep on the dietary intake outcomes were tested. Sensitivity analyses are in the supplement, including unadjusted models (Table S1) and results with under- and over-reporters of diet included (Table S2).

Results

Descriptive

Table 1 summarizes participant characteristics and descriptive statistics for study variables. Participants were mostly young adults (M_{age} = 20.66, SD = 1.60 years), female (80%), international students (90.3%), of Asian descent (81.9%), and had spent less than a year in Melbourne on average. Most participants had never smoked (97.2%) and were moderate drinkers (69.4%). On average, participants' BMI was within the healthy range.

On average, participants' daily energy intake was 7454 kJ, and 31% of this intake was discretionary food. Participants' average daily stress level was 2.20±1.52 (*M*±SD, possible range 0 – 10), which is typical for daily stress levels in international students, young adults, and adults reported in other daily studies.[10,16,62] On average, participants slept 6.26 hours, which is below the recommended sleep duration for optimal health in this population (7 – 9 hours). Participants' average sleep efficiency was 84.4%, which is slightly below the ≥85% threshold for good quality sleep.[63,64] The intraclass correlation coefficients for daily study variables showed that 48 – 84% of the variances were attributed to fluctuations within people.

Table 1. Descriptive Results for Demographic and Daily Study Variables (*N*=72)

	M (SD)/ N (%)	ICC	No. of Days
Baseline Participant Characteristics			
Age (years), *M* (SD)	20.66 (1.60)	—	—
Time Spent in Melbourne (years), *M* (SD)	0.75 (1.00)	—	—
Body Mass Index (kg/m^2), *M* (SD)	21.55 (3.11)	—	—
Female (vs. Male), *N* (%)	58 (80.06)	—	—
Race/Ethnicity, *N* (%)		—	—
Asian	59 (81.90)		
White/European	8 (11.10)		
Others	5 (6.90)		
Before COVID-19 period (vs. during), *N* (%)	55 (76.40)	—	—
International Student (vs. Interstate), *N* (%)	65 (90.30)	—	—
Working (vs. not working), *N* (%)	16 (22.20)	—	—
Never smoked (vs. Former), *N* (%)	70 (97.20)	—	—
Alcohol risk, *N* (%)		—	—
Abstainer	14 (19.40)		
Moderate	50 (69.40)		
At risk	8 (11.10)		
Daily Study Variables			
Total daily EI (kJ), *M* (SD)	7454 (1645)	0.20 (80%)	583
Total daily discretionary food EI (%), *M* (SD)	31 (12)	0.17 (83%)	523
Daily Stress level, *M* (SD)	2.20 (1.52)	0.52 (48%)	578
EEG-Estimated Sleep, M (SD)			
TST (h)	6.26 (0.95)	0.27 (73%)	475
Sleep efficiency (%)	84.43 (5.64)	0.36 (64%)	475
Deep sleep (h)	1.46 (0.32)	0.31 (69%)	475
REM sleep (h)	1.68 (0.49)	0.46 (54%)	475
Physical Activity (METs), *M* (SD)	1.73 (0.37)	0.43 (57%)	558

Note. EI = Energy Intake. EEG = Electroencephalography. REM = Rapid Eye Movement. MET = Metabolic Equivalents. ICC = Intraclass Correlation Coefficient, the proportion of total variance explained by between-person differences (% variance within person). No. of Days = Number of days of unique data. Raw values were presented for total daily energy intake, percentage of total daily energy intake from discretionary food, and sleep efficiency.

Primary Results

No significant interactions emerged for percentage of total energy intake from discretionary food. There was a significant cross-level interaction of between-person TST and within-person daily stress on total daily energy intake (b = -229 [-453, -5.00], p = .046, f^2 =0.02). Simple slopes analyses (*Figure* 2, panel A) showed that higher daily stress predicted higher total daily energy intake for individuals with shorter average TST (M–1SD; b = 265 [-19, 550], p = .068), whereas higher stress predicted lower total daily energy intake for those with longer average TST (M+1SD; b = -133 [-371, 105] p = .27). Although neither of the slopes were significantly different from zero, they were significantly different from each other.

A significant interaction of between-person sleep efficiency and between-person daily stress on total daily energy intake emerged (b = -40 [-8.00, -2.80], p = .041, f^2 =0.02). Simple slopes analyses (*Figure 2*, panel B) showed that higher average daily stress was associated with higher total daily energy intake for individuals with lower average sleep efficiency (M-1SD; b = 340 [73, 608], p = .014), whereas higher average daily stress was associated with lower total daily energy intake for those with higher average sleep efficiency (M+1SD; b = -74 [-410, 262], p = .660).

Additionally, there was a significant interaction of between-person REM sleep and within-person stress on total daily energy intake (b = -499 [-883, -115], p = .011, f^2 =0.02). Simple slopes analyses (*Figure* 2, panel C) indicated higher daily stress predicted higher total daily energy intake for individuals with shorter average REM sleep (M–1SD; b = 234 [-3.00, 471], p = .053), whereas higher stress predicted lower total daily energy intake for those with longer average REM sleep (M+1SD; b = -255 [-535, 23], p = .072). Although neither of the slopes were significantly different from zero, they were significantly different from each other.

There were no significant main effects of daily stress levels or sleep variables predicting total daily energy intake. For discretionary food intake, shorter within-person TST (p = .030) and shorter within-person REM sleep duration (p = .008) the previous night predicted higher percentage of total daily energy intake from discretionary foods. Table 2 summarizes the main effects of stress and sleep on total daily energy intake and percentage of total daily energy intake from discretionary food.

Sensitivity and Exploratory Analyses

Results from unadjusted models (see Supplementary S1) and with all participants included (adjusted and unadjusted models; see Supplementary S2) showed a significant interaction of between-person REM sleep and within-person daily stress on total daily energy intake (similar directions), as well as the main effects of shorter within-person REM sleep or within-person TST predicting higher percentage of total daily energy intake from discretionary food.

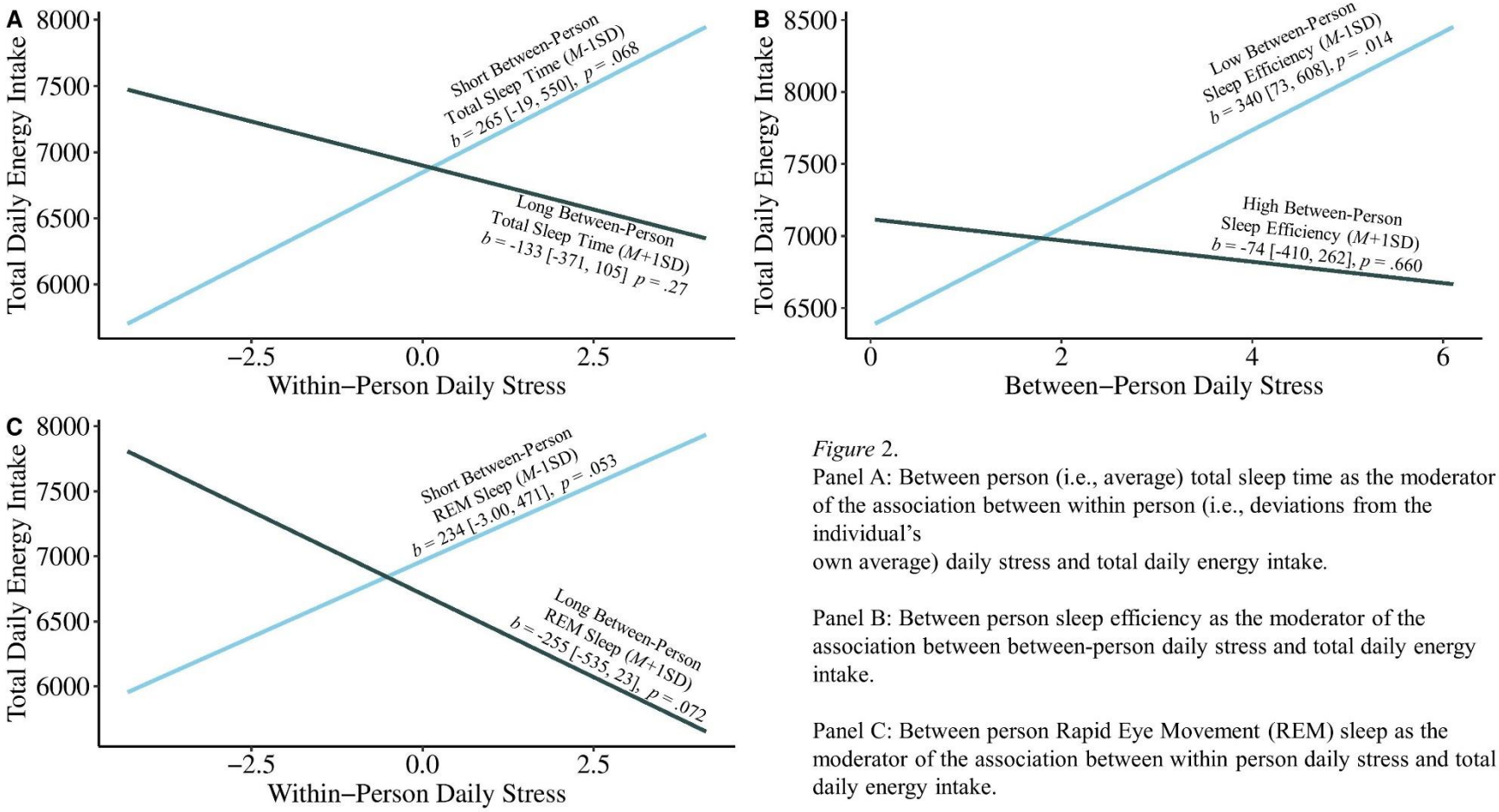

Figure 2.
Panel A: Between person (i.e., average) total sleep time as the moderator of the association between within person (i.e., deviations from the individual's
own average) daily stress and total daily energy intake.

Panel B: Between person sleep efficiency as the moderator of the association between between-person daily stress and total daily energy intake.

Panel C: Between person Rapid Eye Movement (REM) sleep as the moderator of the association between within person daily stress and total daily energy intake.

Table 2. *Main Effects of Previous-Night Sleep and Stress on Total Energy Intake and Percentage of Total Daily Energy Intake from Discretionary Food (N = 72)*

	Between-Person Level	**Within-Person Level**
	Total Daily Energy Intake (kJ)	
Daily Stress	*main effect not shown due to interaction (Figure 2B)*	*main effect not shown due to interaction (Figure 2A & 2C)*
TST (h)	*main effect not shown due to interaction (Figure 2A)*	-95.30 [-288.49, 97.88] f^2 <0.01
SE (%)	*main effect not shown due to interaction (Figure 2B)*	6.10 [-31.96, 44.16] f^2 <0.01
SWS (h)	793.35 [-338.76, 1925.46] f^2 <0.01	-155.39 [-818.16, 507.39] f^2 <0.01
REM (h)	*main effect not shown due to interaction (Figure 2C)*	-249.92 [-755.97, 256.14] f^2 <0.01
	Percentage of Daily Energy Intake from Discretionary Food ($\sqrt{\%}$)	
Daily Stress	-0.06 [-0.24, 0.12] f^2 <0.01	-0.04 [-0.18, 0.09] f^2 <0.01
TST (h)	-0.11 [-0.43, 0.21] f^2 <0.01	-0.18 [-0.35, -0.02]* f^2 =0.02
SE (%)	-0.02 [-0.07, 0.03] f^2 <0.01	-0.01 [-0.04, 0.02] f^2 <0.01
SWS (h)	0.38 [-0.57, 1.33] f^2 <0.01	-0.25 [-0.83, 0.32] f^2 <0.01
REM (h)	0.07 [-0.54, 0.69] f^2 <0.01	-0.59 [-1.02, -0.15]** f^2 =0.02

Note. ** = p <.01. * = p <.05. TST = Total Sleep Time. SE = Sleep Efficiency. SWS = Slow Wave Sleep. REM = Rapid Eye Movement. Results presented are from adjusted models. Results are unstandardized regression coefficient, 95% confidence interval, and Cohen's f^2 type effect size. Extreme values (4-17 observations and 1-2 IDs) were removed for models testing daily energy intake as the outcome. Results for Between-Person TST, SE, and REM, and Between- and Within-Person Stress for Total Daily Energy Intake are not presented given the significant interaction, instead the simple slopes are graphed and simple slope coefficients reported in Figure 2. Percentage of Total Daily Energy Intake from Discretionary Food was square-root transformed. Covariates include age, sex, body mass index, race/ethnicity, student status, employment status, time spent in Melbourne, smoking status, alcohol intake, COVID-19 period, adequacy of factors influencing diet, day of week, and daily physical activity.

Discussion

This study extends current knowledge of the stress-diet relationship by showing that sleep is a potential moderator in a daily, naturalistic setting. Specifically, cross-level interactions emerged, such that individuals with shorter average TST or REM sleep were more likely to have higher total energy intake on days when they experienced higher than usual stress. Conversely, individuals with longer average TST or REM sleep had lower total energy intake on days with higher than usual stress levels. Furthermore, individuals with lower average sleep efficiency and higher average stress levels had higher total energy intake. These findings are in line with previous work showing the moderating role of self-reported sleep quality on the associations between stress and number of self-reported unhealthy foods consumed.[25] Additionally, within-person, shorter TST or REM sleep also predicted higher next-day discretionary food intake. This indicates that after nights with shorter than usual TST or REM sleep, these individuals consumed a higher percentage of their energy intake from discretionary food.

Interaction Effects of Sleep and Stress on Dietary Intake

Theoretical models and research show that in the face of stressful situations, especially when cognitive or self-regulatory resources are overloaded, individuals may avoid the stressor by eating to temporarily alleviate or regulate the negative emotional consequences.[4,25,65] The overloaded capacity for self-regulation also can increase one's impulsivity, which could shift one's priorities towards immediate, temporary reliefs over other long-term goals.[65] The non-significant main effects of daily stress in our findings suggest that the stress levels reported in this sample may not be severe enough, or have enough days with high stress, to overload one's cognitive or self-regulatory capacity. However, when paired with short sleep duration, the positive stress-diet association emerged.

There are several potential mechanisms for the sleep-diet association (discussed below). One possibility is that individuals with generally short sleep or poor sleep quality may lack sufficient replenishment or resources from sleep to regulate emotions effectively.[3,5,66,67] The significant interaction effect of REM sleep duration supports this explanation, as previous studies show that REM sleep plays a role in emotional regulation.[28] Thus, individuals with short sleep, poor sleep, or short REM sleep duration may be more vulnerable to the effects of high stress (e.g., react more strongly to stressors or perceive stressors as more severe) and turn to eating for relief. Conversely, our simple slopes analysis also showed daily higher daily stress and lower total energy intake for individuals with generally long sleep duration, high sleep efficiency, or short REM sleep. These individuals may have better emotional regulatory functioning or sufficient replenishment from sleep, or are less sensitive towards food cues. As such, these individuals may actively engage with the stressor (e.g., active coping strategies), which displaces time spent eating. Another possibility is that other factors may contribute to both average sleep duration and adaptive dietary response to stress. For example, compared to individuals with short average sleep duration, those who typically obtain longer sleep duration may have overall better lifestyle habits, such that they are both more likely to ensure sufficient sleep, and to use other non-dietary related coping in face of stress. Nonetheless, these explanations remain speculative, and future studies are needed to replicate and confirm these findings, especially for the moderating role of sleep architecture.

Main Effects of Sleep on Discretionary Food Intake

There were no significant interaction effects of stress and sleep on discretionary food intake. Findings from main effects show that individuals had a higher percentage of energy intake from discretionary food after nights with shorter than usual TST or REM sleep. These findings extend previous studies linking shorter sleep with poorer diet quality[3,17,22,23] and

shorter REM sleep associated with higher fat and carbohydrate intake.[24] Although there were no total daily energy intake changes, replacing healthy, nutritious foods with discretionary foods can lead to poorer health outcomes.[1,2]

Short sleep can affect reward saliency, inhibitory control, and emotional regulation, which can potentiate the sensitivity and preference for palatable, discretionary foods.[3,5,66,67] For example, individuals who were sleep-deprived showed decreased activity in the frontal and insula cortex (regions associated with cognitive control) and increased amygdala activity during food desirability choice task. Furthermore, individuals who were sleep-deprived also preferred energy-dense foods compared to well-rested individuals.[66] Although the role of REM sleep on dietary intake remains underexamined, previous neuroimaging studies have linked shorter REM sleep with heightened amygdala reactivity towards negative stimuli.[26] Thus, it may be that nights with shorter total or REM sleep potentiate reward sensitivity and impair cognitive control, thus leading to higher consumption of palatable, discretionary foods. From a behavioral perspective, it is possible that nights with shorter sleep duration provide additional opportunity for eating (given the longer hours of wakefulness), particularly during late nights where consumption of convenient, energy-dense foods are likely.[3] Alternatively, young adults may be tired after nights with shorter sleep and opt for more readily accessible foods (which tend to be discretionary) that require minimal or no preparation.

Changes in appetite-related hormones also may explain these results. Higher ghrelin (i.e., appetite-stimulating hormones) and lower leptin (i.e., appetite-suppressing hormones) are linked with disrupted sleep.[3,5] Although there were no significant changes in total energy intake in our findings, elevated levels of ghrelin from short sleep could be associated with the consumption of discretionary foods. For example, Broussard et al.[17] found that individuals restricted to 4.5 hours of sleep had increased levels of ghrelin compared to normal sleepers,

and that higher ghrelin levels were associated with higher consumption of discretionary food. However, it is worthwhile to note that the homeostatic explanations are not well-supported by recent evidence.[5] Taken together, the mechanism for these findings could be a combination of the cognitive, behavioral, and homeostatic perspectives. These explanations could not be directly tested in our study. Future research should examine the interaction of these factors and replicate results, particularly for sleep architecture.

Strengths and limitations

To our knowledge, this study is one of the first to test the interaction of daily stress and EEG-assessed sleep on dietary intake in a naturalistic setting, extending previous studies that relied on between-person or cross-sectional evidence, as well as sleep or stress manipulations and self-reported sleep. The use of detailed dietary food records for seven days, repeated EMA for daily stress levels, and EEG-sleep measures also were the strengths of the study. Specifically, the detailed food record captures a variety of food and drinks, which are connected to the AUSNUT database,[41] consumed in naturalistic settings and provides total energy intake at the daily level. The use of food codes also allowed for classifying discretionary foods more rigorously than previous studies (e.g., studies that relied on pre-specified, limited checklists of unhealthy foods or snacks consumed). As participants were instructed to report their intake immediately after consuming any meals, snacks, or drinks, memory biases are reduced. The follow-up procedures (e.g., text messages and phone calls) also minimized reporting errors and underreporting. Furthermore, the use of daily EEG-sleep measures allowed for exploring the role of nightly variations in sleep architecture on dietary intake. In addition to the use of intensive design and measures, our rigorous analyses also allowed for more precise estimates and testing of temporal directionality by decomposing daily predictors into within- and between-person levels, and including lagged outcomes and important covariates.

Limitations should be acknowledged. Although every effort was made to ensure participants completed their food record accurately, it is possible that participants still did not report all foods consumed (e.g., foods or drinks not available on ASA-24; consciously deciding not to report) or did not report the correct amount consumed, which are inherent issues in dietary intake assessment. Although the ASA-24 food recall has been validated against interviewer-administered 24-hour recall,[39] the food record format has not been validated. Additionally, dietary intake in this study was examined at the nutrient level (i.e., energy intake), and the findings could not be generalized to one's dietary pattern (e.g., eating behaviors; food groups). Future studies are needed to examine how daily stress and sleep can predict both dietary intake and patterns. Given the daily design with repeated EMA, reactivity could have occurred due to the intensity of completing the food record (e.g., participants may change their intake due to the need to report or decided not to report because of the intensity). Furthermore, the temporal associations between daily stress and dietary intake in this study could not be established, given that the values were averaged across the day. Future studies could specifically examine whether variations in stress levels could affect subsequent dietary intake *within* the day. Types of stressors were not examined in this study. Research has shown that emotional and work-related stressors are associated with increased snacking of high fat/sugar food, whereas physical stressors (e.g., feeling anxious/frightened; feeling ill; feeling threatened) are associated with decreased snacking.[10] Future studies can extend these results by examining the severity of different types of stressors as well as the moderating role of sleep on dietary intake. Lastly, given that our participants are generally healthy young people, these findings cannot be generalized to other clinical populations (e.g., individuals with obesity or chronic diseases) or age groups.

Conclusion

Our findings show that shorter sleep duration, poorer sleep quality, and shorter REM sleep duration moderated the associations between daily stress and total daily energy intake. Furthermore, shorter than usual sleep duration or REM sleep duration the previous night predicted higher percentage of total daily energy intake from discretionary food.

Among adults, emerging adults are known to have poor health behaviors including poor sleep[68] and the highest percentage with poor diet.[69,70] Our findings could help improve the delivery of health education by helping emerging adults to understand the connections and importance of both sleep and diet and the role of stress in their health behaviors.

Current behavioral interventions aiming to address diet-related issues in young adults (e.g., changing diet or dietary behaviors; treating or preventing the development of obesity) typically do not include sleep as part of the component.[71,72] Considering our findings, addressing nightly fluctuations in sleep to address poor dietary habits in existing behavioral diet interventions may help bolster their efficacy and warrants further investigation. Additionally, our results suggest that individuals with typically short sleep may be more vulnerable to the effects of stress on their dietary intake. Interventions that only aimed to reduce negative emotions or stress levels to address poor dietary habits are sub-optimal,[71] and these interventions may be more effective if they also address sleep duration. Finally, with the proliferation of consumer-grade sleep tracking wearables, sleep duration is a potential marker for vulnerability that is increasingly accessible to consumers and healthcare providers. More research is needed to explore its roles in domains beyond sleep, such as stress, diet, and other lifestyle factors

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Supplementary S1 – Unadjusted Models

Results presented are unadjusted models. The interaction of between-person TST and within-person stress on total daily energy intake was not significant (b = -209.93 [-427.66, 7.80], p = .059, f^2 =0.02). The interaction of between-person sleep efficiency and between-person stress on total daily energy intake was not significant (b = -4.88 [-44.87, 3.50], p = .812, f^2 <0.01). There was a significant interaction of between-person REM sleep and within-person stress on total daily energy intake (b = -512.58 [-887.37, -137.78], p = .007, f^2 =0.02). Simple slopes analyses indicated higher daily stress was associated with higher total daily energy intake for individuals with shorter average REM sleep (M–1SD; b = 177 [-48, 4.4], p = .12), whereas higher stress was associated with lower total daily energy intake for those with longer average REM sleep (M+1SD; b = -325 [-530, -57.5], p = .017). No significant interactions emerged for percentage of total energy intake from discretionary food. Unadjusted main effects are presented in Table S1.

Table S1. *Main Effects of Previous-Night Sleep and Stress on Total Energy Intake and Percentage of Total Daily Energy Intake from Discretionary Food (N = 72)*

	Between-Person Level	**Within-Person Level**
	Total Daily Energy Intake (kJ)	
Daily Stress	-88.27 [-302.99, 126.46] f^2 <0.01	*Not presented due to significant interaction*
TST (h)	162.28 [-260.44, 584.99] f^2 <0.01	-103.57 [-289.35, 82.20] f^2 <0.01
SE (%)	49.02 [-12.70, 110.74] f^2 = 0.01	15.64 [-20.38, 51.66] f^2 <0.01
SWS (h)	-183.55 [-1368.06, 1000.97] f^2 <0.01	72.59 [-556.74, 701.93] f^2 <0.01
REM (h)	*Not presented due to significant interaction*	-236.96 [-722.35, 248.43] f^2 <0.01
	Percentage of Daily Energy Intake from Discretionary Food ($\sqrt{\%}$)	
Daily Stress	0.02 [-0.15, 0.18] f^2 <0.01	-0.08 [-0.21, 0.05] f^2 =0.01
TST (h)	-0.20 [-0.47, 0.07] f^2 =0.01	-0.20 [-0.36, -0.04]* f^2 =0.02
SE (%)	-0.03 [-0.08, 0.01] f^2 =0.01	0.004 [-0.03, 0.04] f^2 <0.01
SWS (h)	0.14 [-0.68, 0.96] f^2 <0.01	-0.18 [-0.74, 0.38] f^2 <0.01
REM (h)	-0.12 [-0.60, 0.36] f^2 <0.01	-0.57 [-1.00, -0.14]** f^2 =0.01

Note. ** = p <.01. * = p <.05. TST = Total Sleep Time. SE = Sleep Efficiency. SWS = Slow Wave Sleep. REM = Rapid Eye Movement. Results presented are from unadjusted models. Results are unstandardized regression coefficient, 95% confidence interval, and Cohen's f^2 type effect size. Results for Between-Person REM and Within-Person Stress for Total Daily Energy Intake are not presented given the significant interaction. Percentage of Total Daily Energy Intake from Discretionary Food was square-root transformed. Covariates include age, sex, body mass index, race/ethnicity, student status, employment status, time spent in Melbourne, smoking status, alcohol intake, COVID-19 period, adequacy of factors influencing diet, day of week, and daily physical activity.

Supplementary S2 – Full Dataset

No significant interactions emerged for percentage of total energy intake from discretionary food. The interaction of between-person TST and within-person stress on total daily energy intake (adjusted: b = -183.00 [-378.25, 12.25], p = .067, f^2 =0.01; unadjusted: -174.71 [-370.67, 21.25], p = .081, f^2 =0.02). The interaction of between-person sleep efficiency and between-person stress on total daily energy intake was not significant (b adjusted: b= -37.73 [-76.18, 0.72], p = .059, f^2 =0.02; unadjusted: -15.48 [-57.40, 26.44], p = .047, f^2 <0.01). There was a significant interaction of between-person REM sleep and within-person stress on total daily energy intake (adjusted: b = -411.59[-752.12, -71.05], p = .018, f^2 =0.01; unadjusted: b = -426.10 [-760.21, -92.00], p = .013, f^2 =0.02). Simple slopes analyses indicated higher daily stress was associated with higher total daily energy intake for individuals with shorter average REM sleep (adjusted: M–1SD; b = 235 [-35.3, 484.8], p = .02; unadjusted: b = 189 [-6.5, 385.18], p = .058) whereas higher stress was associated with lower total daily energy intake for those with longer average REM sleep (adjusted: M+1SD; b = -160 [-82.5, -1.30], p = .19; unadjusted: -228 [-8.37, -1.896], p = .0586). Table S2 summarizes the main effects of stress and sleep on total daily energy intake and percentage of total daily energy intake from discretionary food.

Table S2. *Main Effects of Previous-Night Sleep and Stress on Total Energy Intake and Percentage of Total Daily Energy Intake from Discretionary Food (N = 98)*

	Between-Person (Unadjusted)	**Within-Person (Unadjusted)**	**Between-Person (Adjusted)**	**Within-Person (Adjusted)**
	Total Daily Energy Intake (kJ)			
Daily Stress	69.00 [-162.27, 300.27] f^2 <0.01	–	103.15 [-101.25, 307.55] f^2 <0.01	–
TST (h)	107.96 [-325.69, 541.62] f^2 <0.01	-70.92 [-232.39, 90.55] f^2 <0.01	-21.69 [-431.67, 388.30] f^2 <0.01	-82.47 [-245.09, 80.15] f^2 <0.01
SE (%)	25.38 [-41.04, 91.81] f^2 <0.01	22.65 [-8.28, 53.57] f^2 <0.01	-25.86 [-91.78, 40.06] f^2 <0.01	15.95 [-15.85, 47.75] f^2 <0.01
SWS (h)	142.33 [-974.29, 1258.95]]f^2 <0.01	164.95 [-363.14, 693.05] f^2 <0.01	564.17 [-614.57, 1742.91] f^2 =0.01	11.46 [-533.83, 556.74] f^2 <0.01
REM (h)	–	-140.09 [-543.41, 263.23] f^2 <0.01	–	-109.88 [-522.46, 302.71] f^2 <0.01
	Percentage of Daily Energy Intake from Discretionary Food ($\sqrt{\%}$)			
Daily Stress	0.02 [-0.14, 0.17] f^2 <0.01	-0.08 [-0.20, 0.05] f^2 <0.01	-0.05 [-0.22, 0.13] f^2 <0.01	-0.03 [-0.16, 0.10] f^2 <0.01
TST (h)	-0.14 [-0.42, 0.13] f^2 <0.01	-0.20 [-0.35, -0.04]* f^2 =0.01	-0.05 [-0.36, 0.25] f^2 <0.01	-0.17 [-0.33, -0.02]* f^2 =0.01
SE (%)	-0.02 [-0.07, 0.02] f^2 <0.01	0.003 [-0.03, 0.03] f^2 <0.01	-0.01 [-0.06, 0.04] f^2 <0.01	-0.01 [-0.04, 0.02] f^2 <0.01
SWS (h)	0.12 [-0.67, 0.92] f^2 <0.01	-0.04 [-0.55, 0.46] f^2 <0.01	|0.44 [-0.48, 1.36] f^2 <0.01	-0.07 [-0.59, 0.45] f^2 <0.01
REM (h)	-0.07 [-0.56, 0.41] f^2 <0.01	-0.44 [-0.83, -0.06]* f^2 =0.01	-0.03 [-0.58, 0.53] f^2 <0.01	-0.42 [-0.81, -0.03]* f^2 =0.01

Note. * = p <.05. TST = Total Sleep Time. SE = Sleep Efficiency. SWS = Slow Wave Sleep. REM = Rapid Eye Movement. Results are unstandardized regression coefficient, 95% confidence interval, and Cohen's f^2 type effect size. Results for Between-Person REM and Within-Person Stress for Total Daily Energy Intake are not presented given the significant interaction. Percentage of Total Daily Energy Intake from Discretionary Food was square-root transformed. Covariates include age, body mass index, race/ethnicity, student status, employment status, time spent in Melbourne, smoking status, alcohol intake, COVID-19 period, adequacy of factors influencing diet, day of week, and daily physical activity.

CHAPTER 7:
GENERAL DISCUSSION

Summary and Synthesis of findings

This thesis examined how stress interacts with health behaviours in everyday life. Sleep and diet are two key pillars of health. Understanding the factors associated with these health pillars, and how changes in these health pillars are associated with daily psychological well-being, is important for maintaining and maximizing both mental and physical health. Our understanding of these associations is primarily based on evidence from a snapshot of a group of individuals' experiences in a single moment in time (i.e., cross-sectional studies) or from highly controlled laboratory settings (e.g., lab-based sleep manipulation studies). Humans are complex, and no two days are the same; therefore, it is important to understand how these daily factors interact in the context of everyday life and how they unfold over time.

Findings from **Paper 1 (Chapter 2)** and **Paper 3 (Chapter 4)** demonstrate a more consistent direction of sleep duration and quality predicting next-day psychological stress compared to psychological stress levels predicting subsequent sleep. These directions are similar to existing daily studies showing more support of sleep as a predictor of next-day psychological stress (Lee, Crain, McHale, Almeida, & Buxton, 2017; Philbrook & Macdonald-Gagnon, 2021; Sin et al., 2017), which is consistent with the notion of good sleep as a source of resilience and replenishment of energy and emotional regulation (Goldstein & Walker, 2014). This notion is further supported by the significant results of longer SWS and REM sleep, which were proposed to have roles in emotional regulation (Ben Simon et al., 2020; van der Helm et al., 2011), predicting next-day psychological stress reported in **Paper 3 (Chapter 4)**. Most of these findings were significant at the within-person level, whereas most between-person effects were non-significant. However, when examining cortisol, a marker of physiological stress, a more consistent pattern of between-person level associations between sleep and diurnal cortisol slope emerged, as shown in **Paper 4 (Chapter 5).** These findings suggest that nightly changes in sleep may have an immediate impact on our next-day

perception of stress but not the regulation of the HPA axis. However, dysregulation of the HPA axis (i.e., a flatter diurnal cortisol slope), rather than psychological stress, can be observed in individuals with generally short or poor sleep.

The lack of consistent findings for daily psychological stress predicting subsequent sleep could be due to various factors, as discussed in **Papers 1 and 3 (Chapters 2 and 4).** One notable aspect could be that the low psychological stress levels reported may not be severe enough to impact sleep (and dietary intake, as shown in **Paper 5 [Chapter 6]**). There also may be differential effects of psychological and physiological stress markers on sleep, as previous studies showed that individuals had increased cortisol responses towards the occurrence of stressful events even without perceiving them as stressful (Fischer, Calame, Dettling, Zeier, & Fanconi, 2000). **Paper 4 (Chapter 5)** also showed that higher within-person cortisol levels during pre-sleep predicted subsequent shorter and poorer sleep. Collectively, these findings show the complex associations between stress and sleep, which differed across sleep parameters and measures used.

Findings from **Paper 2 (Chapter 3)** also may shed light on the inconsistent results of psychological stress predicting sleep. Findings showed that engaging in either problem-focused coping, emotional-approach coping, or avoidance-approach coping in the presence of high daily psychological stress levels was associated with shorter sleep duration. However, direct attempts to manage the emotional consequences of psychological stress were associated with better sleep quality. These findings highlight the differential moderating role of coping in response to daily psychological stress on different sleep parameters.

Beyond examining stress or sleep as the outcome, **Paper 5 (Chapter 6)** explored the synergistic effects of psychological stress and sleep on dietary intake. Findings showed that poorer and shorter sleep exacerbated the effects of daily psychological stress on dietary intake. There is also within-person evidence showing that shorter sleep and REM sleep

duration the previous night predicted higher percentage of energy intake from discretionary food. Similar to sleep, there were no main effects of daily psychological stress on dietary intake.

Contribution of Current Thesis: Significance and Implications

Together, this thesis advanced the current understanding of the stress-sleep associations in naturalistic settings using rigorous designs and measures. These findings also provide valuable evidence to advance the field of health psychology, as well as evidence for interventions incorporating daily strategies into everyday life. The overall significance and implications of this thesis are discussed below.

Firstly, the significance of this thesis is the use of an intensive daily longitudinal design. It moved beyond just examining the average, between-person, or inter-individual differences and examined the within-person or intra-individual differences. Findings from this thesis did not provide strong evidence to support between-person effects of sleep predicting stress and vice versa, suggesting that individuals with generally poor or short sleep do not necessarily experience higher stress levels (and vice versa). These findings differed from previous cross-sectional studies, which could be due to how psychological stress and sleep were measured. The existing cross-sectional evidence is largely based on single time-point measures, which could not determine the directionality of these findings. It is possible that the previous significant findings observed were due to sleep predicting psychological stress, rather than psychological stress predicting sleep. Furthermore, most of the previous cross-sectional studies relied on individuals recalling their *average* psychological stress or sleep in the past week or month. As highlighted in **Introduction (Chapter 1),** these measurements are prone to systematic recall biases. This thesis addressed this issue by incorporating repeated EMAs in measuring daily stress and real-time, objective measures of sleep using actigraphy and EEG. Based on these designs and measures, the results showed

stronger support for within-person effects of sleep predicting next-day psychological stress. These findings support the notion that no two days are the same, wherein psychological stress and sleep are not ergodic processes and can fluctuate across and within days. These findings also highlight the importance of considering the daily, ecological variations in psychological stress and sleep in each individual, and that generalizing the between-person evidence to factors with non-ergodic processes may not be appropriate.

Another significant component of this thesis is the use of objective sleep measures, a biomarker of stress (i.e., cortisol), and a rigorous food record. Most daily field studies on sleep use sleep diary or actigraphy or both, which limits our understanding of how sleep architecture may be related to our daily experiences in naturalistic settings. The use of a portable, single-channel EEG sleep device advanced the current sleep measurement in field studies, which provided a higher accuracy in estimating the multi-faceted sleep parameters (e.g., TST, SOL, WASO, SE) and allowed the examination of the nightly variations in sleep architecture in naturalistic settings. Furthermore, the incorporation of daily cortisol saliva sampling also furthered the understanding of how a biomarker of stress, in addition to psychological stress, is associated with sleep. The repeated saliva sampling over 14 consecutive days provided reliable between- and within-person estimates of the diurnal cortisol slope. With the focus of reducing participant burden and strengthening valid saliva samples, 85% of the samples collected were valid for analyses. The use of a rigorous food record is also a notable strength, as it captured various foods and drinks consumed and provided daily total energy intake and energy intake from discretionary food at the *daily* level.

In addition to using rigorous designs and incorporating objective measures, this thesis strengthened the confidence of results by including *apriori* covariates based on the literature, between- and within-person levels of the predictor in the same model, and lagged outcomes

to strengthen the directionality and temporal order of these associations. Collectively, this thesis advanced the current understanding of stress-sleep associations in daily, naturalistic settings by using one of the most robust tests of directionality possible in observational designs and incorporating repeated experience sampling and rigorous, objective measures.

Findings from this study may benefit public health and the general community, especially young adults, in understanding health behaviours and improving health behaviour change. Young adults are known to have high stress levels and poor health behaviours, including poor sleep and poor diet (Australian Institute of Health and Welfare, 2021; Fayet-Moore et al., 2019; Lund et al., 2010; Rehm, Peñalvo, Afshin, & Mozaffarian, 2016). This may be in part due to young and emerging adults having high perceived invulnerability and downplaying the risks of stress and poor health behaviours (Lao, Tao, & Wu, 2016; Millstein & Halpern-Felsher, 2002). Our findings can benefit public health by helping the delivery of health education for young adults, which may help support their health and well-being. For example, the delivery could emphasise how daily psychological processes and health behaviours are intricately linked, and that they do not only occur in discrete events (e.g., experiencing a major stressful life event) or individuals with high stress or poor sleep; instead, they occur every day and even in relatively healthy individuals. Young adults also may benefit from understanding the vicious daily cycle between stress (physiologically and psychologically) and sleep and what factors can help manage or break this cycle. Furthermore, young adults also can benefit from understanding how their dietary behaviours and intake may be impacted by sleep *and* stress levels. Our findings also may help health delivery programs to tackle young adults' perceived invulnerability. As suggested by Wickman, Anderson, and Smith Greenberg (2008), the programs could focus on current experiences (e.g., poor sleep tonight affects stress the following day) rather than future risks (e.g., poor sleep can increase the risk of developing chronic diseases in the future).

Additionally, the programs also could shift young adults' perception from "I am young and healthy, so it will not happen to me" to "Even though I am young and healthy, it could still happen to me". Together, these findings may benefit health education delivery programs for young adults to improve health literacy and promote health behaviour change.

Additionally, this thesis had many participants who are international university students. These findings may benefit the educational sector and tertiary educational institutions to implement or deliver tailored health programs for this population to help improve their well-being. This is important, especially given that there is comparatively little research in and less support for international students (Forbes-Mewett, 2019), yet the number of international university students in Australia and other developed countries is growing (Organisation for Economic Co-operation and Development, 2021) and make important contributions to the economy (e.g., 40 billion AUD to the Australian economy annually prior to COVID-19 pandemic; Australian Bureau of Statistics, 2021). Thus, improving the knowledge and understanding of the connection of these factors may improve young adults' health behaviours to help prevent or reduce the burden of many preventable, chronic diseases and may reduce the health disparities that exist in international students.

Findings also may benefit clinicians and current interventions aiming to improve health behaviour change to maximize health. Although the current sample is comprised of relatively healthy young adults, these findings may still provide evidence for developing effective strategies for healthcare consumers and non-clinical populations to develop, manage, or maintain healthy behaviours that can prevent the risk of developing or acceleration of chronic diseases. Given the success of using daily diary with EMA and real-time health behaviour tracking devices (with relatively low missing data as demonstrated in the experimental chapters), these findings further support using these strategies for clinicians, as well as the individuals themselves, to better understand their daily psychological well-

being and health behaviours, and in turn provide guidance to adopt timely countermeasures. Additionally, these findings may provide evidence and inform the emerging field of Just-In-Time Adaptive Interventions ([JITAI]; Nahum-Shani et al., 2018). JITAI leverages on the current advancement in technology (e.g., smartphones; wearable health trackers) to deliver personalised support at the right time and context (e.g., the moment and context where the individual would benefit most from the intervention) to improve and promote health behaviour change (Nahum-Shani et al., 2018). The focus on the day-to-day, immediate effects of psychological processes on health behaviours (and vice versa) in this thesis may provide beneficial evidence for JITAIs to target which proximal outcome (i.e., short-term goals of the intervention used for decision-making, such as adapting or optimizing variables) to achieve distal outcomes. Together, these findings provide beneficial evidence for both clinicians and emerging digital health interventions.

Our findings suggest that enhancing emotional-approach coping while reducing emotional-avoidance coping is a potential strategy worth further investigation to improve sleep quality and continuity in face of stress. This would be possible as research has demonstrated the success of reducing one's emotional-avoidance coping while enhancing emotional-approach (e.g., regulation; expression; processing), which led to improvements in depressive and anxiety symptoms (e.g., Unified Protocol for the Transdiagnostic Treatment of Emotional Disorders; Ehrenreich, Goldstein, Wright, & Barlow, 2009). However, it is important to note that engaging in any coping strategies, even the coping strategies that are considered as generally "helpful" and associated with better health outcomes, may have a short-term cost on sleep duration. This highlights that stress management strategies could consider ways to offset the reduced sleep duration associated with coping (e.g., encourage problem-solving during the day, wind down before bedtime). Additionally, behavioural sleep strategies that are appliable in everyday settings for managing the fluctuating nature of

nightly, especially on nights of particularly short or poor sleep, are needed. Our findings also suggest that improving REM and SWS may be a targetable area to improve daytime experiences. Young adults may benefit from adopting good sleep hygiene into their daily and nightly routines; for example, having a consistent sleep/wake schedule and avoiding alcoholic beverages before sleep can help maintain the normal cycle of sleep architecture (Ebrahim, Shapiro, Williams, & Fenwick, 2013). Nevertheless, more research is needed to understand what daily modifiable strategies can improve or maintain sleep architecture, and whether current behavioural sleep interventions can benefit sleep architecture in addition to sleep duration, quality, and insomnia symptoms (Friedrich & Schlarb, 2018; Kodsi, Bullock, Kennedy, & Tirlea, 2021).

In addition to interventions at the individual level, tertiary education institutions also may consider investing in a university-wide program to promote healthy sleep, which may include the delivery of sleep education (e.g., online or workshops) and funded (partially or fully) screening and treatment plans (Hartmann & Prichard, 2018; McCabe, Troy, Patel, Halstead, & Arana, 2018; Prichard & Hartmann, 2019). Investing and funding a healthy sleep program can not only benefit students at the individual level (e.g., better health and academic performance), but also support the university's educational mission, such as improved academic performances, reduced drop-out rates, and increased retention and graduation rate (Hartmann & Prichard, 2018; Prichard & Hartmann, 2019).

Furthermore, findings from this thesis also suggest there may be value in building and optimizing personalised, holistic interventions, as demonstrated by the synergistic effects of stress and sleep on dietary intake. Current interventions aiming to change diet or dietary behaviours in young adults tend to focus on one component at a time (e.g., aiming to reduce negative emotions to improve diet) and do not include sleep as part of the component (Ashton et al., 2019; Kankanhalli, Shin, & Oh, 2019). However, individuals are complex, and

interventions that overlook other factors that influence their feelings or other health behaviours may be less effective. For example, interventions that only aimed to reduce negative emotions to address poor dietary habits are sub-optimal (50% effective; Ashton et al., 2019). Considering our findings that individuals with typically short sleep may be more vulnerable to the effects of stress on their dietary intake, these interventions may be more effective if they also address sleep. These findings suggest that addressing sleep and psychological well-being (e.g., mood, stress, and/or emotions) could be part of a personalized and holistic intervention to target dietary intake or behaviours, and that an individual's poor diet is unlikely to be driven by a single source. Nonetheless, more research and trials are needed to target these components to maximize improvements in dietary intake and behaviours as well as health outcomes.

Limitations and Future Directions

Specific limitations pertaining to each study are discussed in their respective chapters. One common limitation for the overall thesis is the measure of daily stress. Although EMA was used, only perceived overall stress was examined in these studies, providing limited contextual information. Future studies could extend these findings by examining which stressor (e.g., interpersonal; work-related; financial-related) has the strongest (or weakest) impact on sleep or diet. In addition to the types, future research also can delve into the *context* of the stressor, such as the perceived controllability and whether the stressor experienced has been resolved. Given the advancement in smartphones, future studies also can include the location in which the individual reported their perceived stress levels, food consumed, or where they had sleep (e.g., at home vs at work or anywhere else), which may provide additional situational and contextual information of where these psychological processes and health behaviours occur. Understanding the multi-facets and context of these factors can further the understanding the pathways of how daily stress impact sleep, and how

sleep affects the *appraisals* of the stressors, as well as providing contextual evidence of these associations that can inform the emerging field JITAIs and digital health interventions.

Furthermore, our samples were relatively healthy young adults, which could partially explain the low stress levels reported. Thus, these findings cannot be generalised to individuals with known mental or physical health conditions or other age groups. For example, individuals with depression and older adults have different sleep profiles, such as reduced REM sleep latency and increased REM sleep density in individuals with depression and reduced SWS in older populations (Goldstein & Walker, 2014; Ohayon, Carskadon, Guilleminault, & Vitiello, 2004). Thus, future studies are needed to replicate these studies in these populations to not only confirm these findings but also offer interventions critical insights as to how these factors may perpetuate one another and form chronic conditions. Nonetheless, these findings are the first few steps into understanding these daily interactions between psychological processes and health behaviours in everyday life. These findings may still benefit non-clinical populations or healthcare consumers to improve health consciousness and literacy, as well as to develop and maintain healthy lifestyle choices to reduce risks of developing chronic diseases.

Although the number of days and observations sampled in the current studies were longer than many previous daily studies and accounted for weekend and weekday differences, they are still a comparatively short period in an individual's life. The day-to-day experiences and health behaviours during young adulthood are likely to be different when they enter adulthood, and whether they develop diseases in the future is also unknown. Furthermore, the days and observations also were predominantly sampled during semester, which could not offer insights into participants' experiences and health behaviours during holidays. Future studies could consider implementing measurement burst designs, i.e., bursts of daily intensive longitudinal studies over longer intervals (e.g., two daily studies [during

semester and holidays] during adolescence, and repeated daily studies during young adulthood), which may not only provide information about the day-to-day experiences and health behaviours *across*- and *within-days*, but also across- and within *developmental periods* of each individual.

Conclusion

How we feel can affect our health behaviours, and changes in our health behaviours also affect how we feel. This cyclical process occurs every day and plays a role in maintaining a healthy lifestyle and maximizing mental and physical health. Although this process occurs daily, it is different each day and for each individual. This thesis targeted a part of this psychological process and health behaviours relations by examining and advancing the understanding of daily stress-sleep associations in naturalistic settings.

This thesis rigorously tested the temporal, bi-directional associations, examined both the *inter*- and *intraindividual* differences, and used both subjective and objective stress and sleep measures. This thesis furthered the understanding of the stress-sleep associations by exploring coping strategies as potential moderators. Beyond outcomes, this study explored whether stress and sleep synergistically predict dietary intake, one of the key pillars of health. This thesis provided within-person evidence of these daily processes and offered strong methodological, theoretical, and practical implications for future research, the educational sector, clinicians, and interventions.

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