MONASH University

In Good Times and in Bad:
Dyadic Sleep Processes in Couples without Sleep Disorders and those Experiencing Insomnia

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Bachelor of Arts (Honours) / Bachelor of Science

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Abstract

Most Australian adults sleep with a bedpartner, yet research into couples' sleep remains limited. Specifically, methods of characterising dyadic sleep are lacking. Most couples' sleep research has been conducted within healthy sleeping populations, despite insomnia patients frequently reporting disturbances related to their bedpartner. The goal of this thesis was to advance understanding of dyadic sleep, in couples with and without insomnia. Specific aims were to: (1) review literature on dyadic sleep, and provide context by considering sleep's close relationship with health (**Paper 1**, literature review); (2) characterise how bedpartners influence each other's sleep (**Papers 2,3**); (3) identify factors predicting vulnerability to wake transmission (**Papers 2,3**); and (4) examine whether anxiety or depression symptoms predict an individual or bedpartner's sleep. All were considered in couples with and without insomnia.

Paper 2 addressed Aims 2 and 3 in couples without sleep disorders. Participants completed the Morningness-Eveningness Questionnaire-reduced version, then monitored habitual sleep/wake for seven nights via actigraphy and sleep diary. We described rates of epoch-by-epoch sleep/wake concordances (shared sleep/wake minutes), number of transmissions received from a bedpartner (i.e., number of awakenings after initiation of sleep immediately preceded by bedpartner wakefulness), percent transmissions received (percentage of total awakenings that were transmissions), transmissibility (percentage of all bedpartner awakenings transmitted), and percent minutes resistant to transmission (percentage of bedpartner's wake minutes that an individual slept). Mixed-effects modeling assessed predictors of dyadic sleep. Average couple-level percent transmissions received were highest and percent minutes resistant lowest in couples who had similar bedtime, compared to couples with greater bedtime differences.

Paper 3 addressed Aims 2 and 3 in couples where one partner experienced insomnia, who also completed the above protocol. Concordance and transmission rates were similar in

patients as non-disordered sleepers. Partners received wake transmissions at 1.25 times that of patients. Similarly to Paper 2, percent transmissions received was increased in couples with concordant bedtimes, and individuals with later chronotype than their bedpartner. Patterns of chronotype and bedtime order predicting percent minutes resistant to transmission differed across the rest interval.

Finally, **Paper 4** combined the samples to address Aim 4. All participants completed the Beck Anxiety Inventory, Patient Health Questionnaire-9, and Insomnia Severity Index (ISI). Actor-Partner Interdependence Models assessed whether anxiety or depression symptoms predicted individual or dyadic sleep (wake transmission). Elevated anxiety in insomnia patients predicted increased vulnerability to wake transmissions they received, and those received by their bedpartner. Neither anxiety nor depression symptoms predicted an individual's or their bedpartner's sleep efficiency, however ISI was positively predicted by own anxiety and depression symptoms.

Results provide evidence couples affect one another's sleep, and many existing measures of sleep do not describe this phenomenon. Further, vulnerability and resistance to transmissions can be predicted by role, bedtime, and mental health characteristics. Insomnia patients were no more likely to be disturbed by a partner than non-sleep-disordered individuals. Couples were generally unaware of the influence of their bedpartner. Findings have methodological and clinical implications for considering a bedpartner's role in sleep. It is hoped future studies will further this line of inquiry.

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Publications Arising from Thesis

Peer reviewed publications

Walters, E.M., Phillips, A.J.K., Mellor, A., Hamill, K., Jenkins, M.M., Norton, P.J., Baucom, D.H., & Drummond, S.P.A. (2020). Sleep and wake are shared and transmitted between individuals with insomnia and their bed-sharing partners. *Sleep,* 43(1):1-12. doi: 10.1093/sleep/zsz206

Walters, E.M., Phillips, A.J.K., Boardman, J.M., Norton, P.J., & Drummond, S.P.A. (in press). Vulnerability and resistance to sleep disruption by a partner: A study of bed-sharing couples. *Sleep Health.*

Walters, E.M., Phillips, A.J.K., Hamill, K., Norton, P.J., & Drummond, S.P.A. (under review). Anxiety predicts dyadic sleep characteristics in couples experiencing insomnia but not in couples without sleep disorders.

Book chapters

Stewart, E.M., Landry, S., Edwards, B.A., & Drummond, S.P.A. (in press). The Bidirectional Relationship between Sleep and Health. In E.A. Klonoff (Ed.), *Wiley Encyclopedia of Health Psychology, Volume IV: Special Issues in Health Psychology*. John Wiley and Sons.

Components of thesis presented at conferences

Drummond, S.P.A., Walters, E.M. (2019, September). *Data from Project REST: Exploring Dyadic Sleep.* Invited presentation at World Sleep Congress 2019. Vancouver, BC.

Walters, E.M., Phillips, A.J.K., Hamill, K., Norton, P.J., & Drummond, S.P.A. (2019, October). *Anxiety and depression predict dyadic sleep characteristics differently in bed-sharing couples with and without insomnia.* Paper presented at Sleep Down Under, Sydney, NSW.

Walters, E.M., Phillips, A.J.K., Boardman, J.M., Mellor, A., Hamill, K., & Drummond, S.P.A. (2019, September). *Sleep and Wake are Shared and Transmitted between Partners of Bed-Sharing Couples.* Paper presented at World Sleep Congress 2019, Vancouver, BC.

Walters, E.M., Kilmartin, C., Norton, P.J., & Drummond, S. P. A. (2018, October). *Sleep and Wake are Shared and Transmitted between Members of Bed-Sharing Couples.* Paper presented at Sleep Down Under 2018, Brisbane, QLD.

Stewart, E.M., Mellor, A., Jenkins, M.M., Clark, J., Norton, P.J., Baucom, D., & Drummond, S. P. A. (2017, June). *Who are the Partners? A Sleep Profile of Partners of Individuals Seeking Treatment for Insomnia.* Paper presented at SLEEP 2017, Boston, MA.

Peer reviewed publications, describing research beyond this thesis

Hamill, K., Jumabhoy, R., Kahawage, P., de Zambotti, M., Walters.,E.M., & Drummond, S. P. A. (2020). Validity, Potential Clinical Utility, and Comparison of a Consumer Activity Tracker and a Research-Grade Activity Tracker in Insomnia Disorder II: Outside the Laboratory. *Journal of Sleep Research*, 29(1):e12944. doi: 10.1111/JSR.12944.

Walters, E. M., Jenkins, M. M., Nappi, C. M., Clark, J., Lies, J., Norman, S. B., & Drummond, S. P. A. (2019). The impact of prolonged exposure on sleep and enhancing treatment outcomes with evidence-based sleep interventions: A pilot study. *Psychological Trauma: Theory, Research, Practice, and Policy.* Advance online publication. doi: 10.1037/tra0000478.

Abstracts co-authored by the candidate, describing research beyond this thesis

Hamill, K., Jumabhoy, R., Kahawage, P., de Zambotti, M., Walters.,E.M., & Drummond, S. P. A. (2019, October). *Validity and Potential Clinical Utility of a Consumer and Resaerch-Grade Activity Tracker in Insomnia Disorder: Outside the Laboratory.* Paper presented at Sleep Down Under, Sydney, NSW.

Lebel, J., Bastien, C.H., Mellor, A., Walters, E.M., Hamill, K., & Drummond, S.P.A. (2019, September). *Insomnia and Bed Partner Accommodation: Preliminary Exploration.* Paper presented at World Sleep Congress, Vancouver BC.

Hamill, K., Jumabhoy, R., Kahawage, P., de Zambotti, M., Walters.,E.M., & Drummond, S. P. A. (2019, September). *Validity and Potential Clinical Utility of a Consumer and Resaerch-Grade Activity Tracker in Insomnia Disorder: Outside the Laboratory.* Paper presented at World Sleep Congress, Vancouver BC.

Mellor, A., Stewart, E.M., Jenkins, M. M., Hamill, K., Norton, P.J., Baucom, D., & Drummond, S.P.A. (2017, June). *Bedpartner Accommodation of Insomnia in Treatment-Seeking Couples.* Paper presented at SLEEP 2017, Boston, MA.

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 a peer reviewed journal, one book chapter accepted for publication, and two submitted publications (one of which was accepted after revisions while the thesis was under examination). The core theme of the thesis is the dyadic nature of sleep in couples with and without sleep disorders. The ideas, development and writing up of all the papers in the thesis were the principal responsibility of myself, the student, working within the Turner Institute for Brain and Mental Health and School of Psychological Sciences, Monash University, under the supervision of Professor Sean P A Drummond and Professor Peter J Norton.

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 Chapters 1, 3-5, my contribution to the work involved the following:

Thesis Chapter	Publication Title	Publication Status	Nature and % of student contribution	Co-author name/nature of contribution	Co-author(s), Monash student Y/N
1	The bidirectional relationship between sleep and health	In press	50% contribution by student. This included writing the manuscript and editing of co-author sections	2. Shane Landry, 10% input 3. Brad A Edwards, 10% input 4. Sean PA Drummond, 30% input	2. No 3. No 4. No

3	Vulnerability and resistance to sleep disruption by a partner: A study of bed-sharing couples	Submitted, under review at time of thesis submission. At time of submitting final corrected thesis, a revised version of this manuscript is in press.	60% contribution by student. This included formulation of experimental design, data collection, analysis, and writing the manuscript	2. Andrew JK Phillips, 10% input 3. Johanna M Boardman, 7% input 4. Peter J Norton, 8% input 5. Sean PA Drummond, 15% input	2. No 3. Yes 4. No 5. No
4	Sleep and wake are shared and transmitted between individuals with insomnia and their bed-sharing partners	Published	60% contribution by student. This included formulation of experimental design, data collection, analysis, and writing the manuscript	2. Andrew JK Phillips, 10% input 3. Alix Mellor, 3% input 4. Kellie Hamill, 3% input 5. Melissa M Jenkins, 3% input 6. Peter J Norton, 3% input 7. Donald H Baucom, 3% input 8. Sean PA Drummond, 15% input	2. No 3. No 4. No 5. No 6. No 7. No 8. No
5	Anxiety predicts dyadic sleep characteristics in couples experiencing insomnia but not in couples without sleep disorders	Submitted, under review	60% contribution by student. This included formulation of experimental design, data collection, analysis, and writing the manuscript	2. Andrew JK Phillips, 10% input 3. Kellie Hamill, 7% input 4. Peter J Norton, 8% input 5. Sean PA Drummond, 15% input	2. No 3. No 4. No 5. No

I have not renumbered sections of submitted or published papers in order to generate a consistent presentation within the thesis but rather have left them as they intend to be published.
Elizabeth M Walters

Signed:

Date:

I, the undersigned, 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.
Primary Supervisor: Sean P A Drummond

Signed:

Date:

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Finally, a dedication: To Long Nguyen, who made starting work on this project infinitely less daunting and who provided the biggest learning curve of all.

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List of Tables and Figures

Chapter 3:

Chapter 4:

List of Abbreviations

AIC:	Akaike's Information Criterion
APIM:	Actor-Partner Interdependence Model
BAI:	Beck Anxiety Inventory
BMI:	Body Mass Index
CBTI:	Cognitive Behavioural Therapy for Insomnia
CSA:	Central Sleep Apnea
CPAP:	Continuous Positive Airway Pressure
CPQ:	Communications Patterns Questionnaire
DAS:	Dyadic Adjustment Scale
DSISD:	Duke Structured Interview for Sleep Disorders
GAD:	Generalized Anxiety Disorder
ISI:	Insomnia Severity index
MAS:	Mandibular Advancement Splint
MDD:	Major Depressive Disorder
MEQr:	Morningness-Eveningness Questionnaire, reduced form
OSA:	Obstructive Sleep Apnea
PHQ-9:	Patient Health Questionnaire – 9
PSG:	Polysomnography
REM:	Rapid Eye Movement
RLS:	Restless Legs Syndrome
SDB:	Sleep Disordered Breathing
SE:	Sleep Efficiency
SL:	Sleep Latency
SWS:	Slow Wave Sleep

TST: Total Sleep Time

WASO: Wake After Sleep Onset

CHAPTER 1

The Bidirectional Relationship between Sleep and Health

Chapter 1 is a book chapter accepted for publication in the *Wiley Encyclopedia of Health Psychology*,

Stewart, E.M., Landry, S., Edwards, B.A., & Drummond, S.P.A. (in press). The Bidirectional Relationship between Sleep and Health. In E.A. Klonoff (Ed.), *Wiley Encyclopedia of Health Psychology, Volume IV: Special Issues in Health Psychology*. John Wiley and Sons.

Foreword to Chapter 1

Sleep has traditionally been viewed as an individual behaviour, both in scientific literature as well as in sleep clinics. The field of couples and sleep is relatively new. Yet over half of Australian adults report living and sleeping with a romantic partner (De Vaus, 2004). For these people, sleep is a shared activity. The available evidence supports the notion couples influence one another's sleep, and this may have broader implications than simply sleep: this may also play a role in individual and bedpartner well-being and relationship functioning.

Examining sleep at the level of the dyad will improve our understanding of the role partners play in precipitating and perpetuating sleep disorders, particularly insomnia. To date there has been little acknowledgement of the couple process in the sleep disorder insomnia, despite its high prevalence.

Given the importance of sleep for health and cognitive function, this thesis will explore sleep as a dyadic process. More specifically, it will investigate the ways in which couples engage in sleeping behaviours together in contexts where one partner experiences insomnia disorder, compared with situations where neither partner experiences disordered sleep. In doing so, we hope to uncover how bedsharing may act as an interpersonal precipitating or perpetuating factor of insomnia.

Before reviewing the available research on dyadic sleep, it is important to understand the role sleep plays for health and well-being. Understanding the importance of sleep is vital for considering the impact of external and internal factors which can affect sleep (including the bedpartner). Therefore, the first paper presented in this thesis is a book chapter describing the bidirectional relationship between sleep and health. This provides context for why psychosocial factors such as a bedpartner are important to consider in research as well as clinically.

Note, this chapter was submitted for publication to *Wiley Encyclopedia of Health Psychology* under the author's maiden name. It has been formatted in compliance with

encyclopedia requirements. The only ways it has been modified is (1) to insert additional references throughout the text, as the original submission requirement had a limit of 15 references, and (2) to provide acknowledgement to the thesis reader of relevant points which will be explored in more depth later in the thesis.

Declaration

Declaration of the Candidate: In the case of Chapter 1, the nature and extent of my contribution to the work was the following:

Nature of Contribution	Extent of Contribution
Writing of manuscript, and editing of co-author sections	50%

The following co-authors contributed to the work:

Name	Nature of Contribution
Shane Landry	Writing manuscript and critical review of manuscript
Bradley A Edwards	Writing manuscript and critical review of manuscript
Sean PA Drummond	Writing manuscript and critical review of manuscript

The undersigned hereby certify that the above declaration correctly reflects the nature and extent of the candidate and co-authors' contributions to this work.

Candidate's Signature:

Primary Supervisor's Signature:

Chapter 1: The bidirectional relationship between sleep and health (Paper 1)

The Bidirectional Relationship between Sleep and Health

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Abstract

Sleep is vital to overall health, with links to many physical and mental health disorders. This entry provides an overview to sleep and common sleep measurement strategies, as well as illustrating how sleep and health affect one another in a bidirectional manner, including how treatment improves health domains. Three examples of disrupted sleep and effect on health are presented: excessively long or short sleep duration ; insomnia; and sleep disordered breathing, particularly obstructive sleep apnea (OSA). Mechanisms linking sleep and health are often complex and bidirectional in nature, however treatment outcomes tend to be unidirectional: chronic sleep problems require direct treatment for improvement. Insomnia and OSA can be effectively treated using psychological and medical treatments. These treatments then contribute to subsequent improvements in health problems.

Key words: Sleep; Sleep Duration; Insomnia; Obstructive Sleep Apnea; Breathing Disorders

Sleep impacts myriad aspects of physical and mental health, daytime function, and quality of life. While we have solid hypotheses regarding the specific functions of sleep, there is no simple answer to the question, "Why do we sleep?" This may help explain why society has less regard for sufficient good-quality sleep than other health behaviours, such as exercise, proper diet, and the need to stop smoking. Sleep is a highly complex behaviour, arising from interactions between brain stem, mid brain, and cortex, neurotransmitter pathways, and hormones. It is this complexity which makes sleep so vulnerable to disruption. Sleep is vital to overall health, with links to many physical and mental health disorders. The aim of this chapter is to provide an overview of sleep and common strategies for measuring it, as well as illustrate how sleep and health affect one another in a bidirectional manner using three examples.

Introduction to Sleep

Sleep is "a reversible behavioural state of perceptual disengagement from and unresponsiveness to the environment" (Carskadon & Dement, 2011). Sleep is composed of two distinct states: non rapid eye movement (NREM) and rapid eye movement (REM) sleep. NREM sleep is further composed of 3 stages: N1, N2, and N3. Each successive stage represents a deeper stage of sleep. Sleep oscillates among the stages, with NREM stages followed by REM stages. This oscillation takes about 80-100 minutes and is repeated 3-4 times each night. The exact timing and composition of sleep episodes are complex and affected by a number of factors. A full explanation is beyond the scope of this chapter, and the interested reader is referred to several excellent sources for more information (Borbély, 1982; Carskadon & Dement, 2011; McCarley, 2007; Ohayon, Carskadon, Guilleminault, & Vitiello, 2004).

Sleep is essential for health and well-being. A recent expert consensus conference convened by The American Academy of Sleep Medicine and Sleep Research Society in 2015 determined, based on an extensive literature review, that adults should regularly obtain at least 7 hours of sleep per night to promote optimal health in a variety of key areas. Less than 7 hours

of sleep per night increases likelihood of obesity, diabetes, cardiovascular disease, depression, and mortality, among other negative consequences (Watson et al., 2015).

Inadequate nightly sleep has major consequences for daytime functioning, resulting in reduced alertness, poor attention and concentration, daytime sleepiness, and increased risk of accidents, as well as compromising immune, cardiovascular, and metabolic systems (Besedovsky, Lange, & Born, 2012; Ferrie et al., 2007; Knutson, Spiegel, Penev, & Van Cauter, 2007). Cognitive performance is impaired in domains including executive function, processing speed, working memory, visual learning, memory, decision-making and judgment (Czisch et al., 2012; Diekelmann & Born, 2010; Drummond, Anderson, Straus, Vogel, & Perez, 2012; Fischer, Diekelmann, & Born, 2011; Killgore, Balkin, & Wesensten, 2006; Lim & Dinges, 2008; Wolfson & Carskadon, 1998). These deficits cannot always be compensated for by use of stimulants (Killgore, Grugle, & Balkin, 2012).

Assessing Sleep

Sleep can be assessed in a variety of ways, including objectively and subjectively, retrospectively and prospectively. Naturally, some methods are more labour intensive than others. A brief overview of the major assessment tools are presented here, from most to least labour intensive and costly.

Polysomnography (PSG). PSG is the gold standard of objective sleep measurement. It uses electroencephalography (EEG) electrodes attached to the scalp to measure electrical brain activity, from which sleep and wake states can be distinguished. Other information about the body is measured concurrently: eye and leg movements, muscle tone, heart rate, oxygen levels in the blood and breathing patterns (Berry et al., 2012). This provides highly accurate and clinically relevant information for diagnosis of many sleep disorders, and can be conducted either in-lab or at home, using fewer numbers of electrodes (Geyer, Talathi, & Carney, 2009; Vaughn & Giallanza, 2008).

Multiple Sleep Latency Test (MSLT). MSLT is an objective measure of daytime sleepiness. Standard MSLT protocols exist and are widely used in both clinical and research settings (Arand et al., 2005). Individuals have 4-5 designated sleep opportunities throughout the day after a typical night's sleep, while connected to PSG. Each nap opportunity lasts 20 minutes, and occurs every 2 hours. Level of sleepiness due to sleep deprivation can be assessed, as well as some sleep disorders (Carskadon & Dement, 1982; Carskadon et al., 1987; Littner et al., 2005). Analogous protocols exist for the Maintenance of Wakefulness Test (MWT), which assesses ability to stay awake, rather than fall asleep, during the day (Littner et al., 2005).

Actigraphy. Sleep can be profiled objectively without PSG. Actigraphy uses a wristwatch-like device that includes an accelerometer, and often a light sensor, to measure movement as a proxy for sleep (Sadeh, 2011). This is less invasive and has greater ecological validity than an in laboratory PSG as the device can be worn at home, for many nights in a row. Practice guidelines suggest it be used in combination with a sleep diary (Kawada, 2013; Sadeh, 2011). Information collected between actigraphy and PSG is different. No brain activity or sleep architecture is recorded with actigraphy. Instead, movement allows the determination of sleep and wake (Ancoli-Israel et al., 2015; Sadeh & Acebo, 2002). Compared with PSG, actigraphy demonstrated high sensitivity (ability to score sleep as sleep; >90%) and low specificity (ability to score wake as wake; <40%; Marino et al., 2013; Sivertsen et al., 2006; Taibi et al., 2013; Toon et al., 2016) in sleep-disordered populations.

Sleep Diary. The most detailed way to assess subjective sleep is with a daily sleep diary. While many investigators and clinicians use a diary of their own design, there is a consensus diary recommending the minimum information one should obtain (Carney et al., 2012). Typical measures collected with a sleep diary include bed time and wake time, length of time to fall asleep, number and length of nocturnal awakenings, and time out of bed. Naps, caffeine and alcohol intake, and a variety of other variables are also commonly obtained. It is

highly useful, particularly in diagnosis and treatment of sleep disorders such as insomnia (Morin & Espie, 2003). Indeed, sleep diaries form one of the key underpinnings of Cognitive Behavioural Therapy for Insomnia (CBTI), discussed later in this chapter.

Self-Report Questionnaires. Validated questionnaires are widely utilised in both healthy populations and those with sleep, psychiatric, and medical disorders. Some of the most common include the Pittsburgh Sleep Quality Index (global sleep quality), the Epworth Sleepiness Scale (trait sleepiness), Karolinska Sleepiness Scale (state sleepiness), the Insomnia Severity Index (insomnia symptoms), the STOP-BANG (sleep apnea risk), and the Horne-Ostberg Morningness-Eveningness Questionnaire (chronotype). Psychometrics of sleep assessments will be explored in more depth later in the thesis.

The Bidirectional Relationship between Sleep and Health

There is a bidirectional relationship between sleep and health. Sleep problems increase the risk of developing various physical and mental health disorders, and in turn, many disorders have characteristic sleep sequelae.

Poor sleep comes in many forms. Within this chapter, three major categories of poor sleep will be discussed: a) excessively long or short sleep duration; b) insomnia; and c) sleep disordered breathing (SDB), in particular obstructive sleep apnea. All three of these categories have direct links with physical and mental health, and carry huge economic burdens (National Institutes of Health, 2005; Sleep Health Foundation, 2011). Targeting sleep as a health behaviour has downstream effects for other health domains. By treating the sleep problems, many comorbid health problems improve too. The following sections provide an overview of the relationship between sleep and health in each of these 3 areas, greater detail on one specific aspect of health within each, and an overview of how treating sleep can improve health.

Excessively Long or Short Sleep Duration

Like many things in health, sleep works on the principle of optimization, as opposed to the principle that more is better. Epidemiological studies show there is an inverted U-shaped curve describing the relationship between sleep and health (Kojima et al., 2000; Kripke, Garfinkel, Wingard, Klauber, & Marler, 2002; Kripke, Simons, Garfinkel, & Hammond, 1979; Patel et al., 2004). Research has consistently found that 7-7.5h sleep is optimal for a variety of health outcomes (Cai et al., 2015; Ferrie et al., 2007; Hublin, Partinen, Koskenvuo, & Kaprio, 2007; Kripke et al., 2002; Patel et al., 2004). As sleep duration becomes progressively further away from this in either direction, risk of poor health outcomes, including mortality, increases. Interestingly, though, the specific negative health outcomes of short vs. long sleep duration often differ.

Sleep Duration and Health

The most obvious consequence of short sleep duration is daytime sleepiness. However, short sleep also has more dangerous health effects. Sleep loss causes characteristic neurological (EEG) changes, alterations in autonomic functions including cardiovascular function, respiration rates, biochemical and metabolic changes, as well as altered gene expression, due to stress placed on the body by prolonged wakefulness with insufficient recovery sleep between wake periods. These underlying physiological processes then lead to poor health outcomes. Bidirectional relationships exist between short sleep (<6 hours) and weight gain/obesity, impaired glucose tolerance, Type 2 Diabetes Mellitus, hypertension, cardiovascular disease, stroke, anxiety symptoms, depression, impaired immunity, increased pain, and increased risk of accidents and mortality (Dew et al., 2003; Ferrie et al., 2007; Karimi, Hedner, Häbel, Nerman, & Grote, 2015; Prather, Janicki-Deverts, Hall, & Cohen, 2015; Sivertsen et al., 2015; Watson et al., 2015; Zhang & Chan, 2014). Additionally, animal studies have shown that

complete sleep loss kills animals in approximately the same amount of time as total food deprivation (Rechtschaffen & Bergmann, 1995).

Long sleep is also associated with increased mortality, perhaps to an even greater extent than short sleep. Long sleepers have increased likelihood of depression, lack of physical exercise, and lower socioeconomic status, all of which are independently associated with poor health. Long sleepers were also more likely to have a diagnosis of medical and psychiatric disorders including diabetes, obesity, hypothyroidism, seizures, multiple sclerosis, stroke, cardiovascular disease, hypertension, rheumatoid arthritis, osteoarthritis, herniated disc, asthma, chronic obstructive pulmonary disease, cancer, inflammatory bowel disease, anxiety disorders, depression, restless legs syndrome, and snoring (Ohayon, 2004; Patel et al., 2006).

The reasons long sleep is associated with increased mortality are less clear than the well-established links between short sleep/sleep deprivation and health. Grandner and Drummond (2007) identified six main contributors to the link between long sleep and poor health: sleep fragmentation, fatigue, altered immune function, circadian abnormalities, lack of physiological challenge, depression, or underlying disease processes (e.g. OSA, heart disease, or failing health). In addition, reports of sleep duration may actually represent total time in bed, rather than truly time asleep (Ferrie et al., 2007). Long time in bed often reflects increased sleep fragmentation, which in its own right has significant associations with poor health (Bennett, Barbour, Langford, Stradling, & Davies, 1999; Dew et al., 2003). In essence, long sleep may be problematic because it is a proxy for general poor or declining health, rather than mechanistically responsible for poor health outcomes.

Based on the large sleep and health literature, sleeping 7-7.5 hours per night is thought to be optimal for health, and as cited above, the current recommendation is that adults 18-64 years old sleep 7-9 hours per night (Hirshkowitz et al., 2015). Worryingly, fewer hours of sleep and greater levels of sleep disturbance have become widespread in industrialised societies,

largely due to trading off sleep to fit in more work and leisure activities (Bartlett, Marshall, Williams, & Grunstein, 2008; Ferrie et al., 2007). Such voluntary restricted sleep duration is a common occurrence in modern society. In Australia, a 2010 community study found 18.4% of adults slept less than 6.5 hours per night, and 11.7% reported chronic sleepiness (Bartlett et al., 2008). This prevalence is even higher in the United States, 30% of all workers and 44% of night shift workers reported sleeping less than 6 hours per night (Luckhaupt, 2012). Some populations are more prone to obtaining insufficient sleep and/or experiencing sleep disruption than others, including shift workers, physicians, truck drivers, parents, and teenagers. This increases risk of many health problems and also directly impacts daytime functioning the day after a night of short sleep.

Treatment Effects on Health

Despite the major health consequences of sleep loss (which may be seen in chronic restricted sleep duration) and overly long sleep, treatment and management of excessively long or short sleep duration in "an otherwise healthy" individual is rarely addressed by clinicians. There are no formal treatment guidelines for managing short sleep duration. The most popular professional advice is to take a short nap (Veasey, Rosen, Barzansky, Rosen, & Owens, 2002), and to sleep longer on a regular basis. These may be perceived as impractical advice. Furthermore, catching up on sleep during the weekends is not sufficient to return functioning to baseline levels.

While there are no evidence based guidelines for managing sleep duration in healthy populations, health improvements seen with improved sleep duration have been investigated in two ways: longitudinal epidemiological studies, and experimental studies which systematically restrict the sleep of individuals in a laboratory setting, then observe over a period of recovery. A recent longitudinal epidemiological study investigated how increasing sleep duration to the healthy range improved mortality risk (Ferrie et al., 2007). When regular sleep

length increased from 5-6 h to 7-8 h over the 3 year follow-up period, there were decreases in risk of both cardiovascular and non-cardiovascular disease-related mortality. This has not been consistently replicated though (Dew et al., 2003; Hublin et al., 2007), suggesting the relationships observed by between sleep duration and mortality may be mediated or moderated by other specific health improvements, which were not explicitly reported.

Insomnia

The current Diagnostic and Statistical Manual (DSM-5) defines Insomnia Disorder as a combination of dissatisfaction with sleep quality or quantity, characterised by: a) difficulty initiating sleep, maintaining sleep, or early-morning awakening with inability to return back to sleep; and b) a negative impairment in some aspect of daytime functioning, which the individual relates back to poor sleep (i.e., they believe the poor sleep causes or exacerbates the daytime impairment). The insomnia occurs on at least 3 nights per week for a minimum of 3 months, occurs despite adequate opportunity for sleep, and is not fully explained by another sleep-wake disorder or other mental or medical disorder, nor is it attributable to physiological effects of a substance (American Psychiatric Association, 2013). It is important to note the concept of "secondary insomnia" is no longer considered valid. A diagnosis of Insomnia Disorder can occur in the context any other physical or mental health disorder and is considered a comorbid diagnosis, not a secondary diagnosis. This is because once insomnia becomes chronic (i.e., ≥3 months in duration), it typically needs specific and independent treatment and does not respond adequately to treating the "other" diagnosis. As a result, insomnia can be conceptualised as a chronic health disorder. Approximately 80% of individuals with insomnia experience it for at least one year, and 40% for 5 years (Bixler, Kales, Soldatos, Kales, & Healey, 1979; Hohagen et al., 1993; Morin, LeBlanc, Daley, Gregoire, & Merette, 2006).

Insomnia is one of the most common sleep disorders, and is among the most prevalent of all mental health disorders, affecting 10-15% of adults worldwide (Bartlett et al., 2008; Ford & Kamerow, 1989; National Institute of Health, 2005; Morin, LeBlanc, et al., 2011; Ohayon & Bader, 2010). Insomnia commonly presents with a range of psychiatric and medical comorbidities. As such, insomnia represents a huge health burden. A 2010 report estimated that in Australia, insomnia and attributable disorders cost the economy A$10.9 billion (Deloitte Access Economics, 2011). This is minor in comparison with other places around the world. In the province of Québec alone, the total annual cost of insomnia and associated health concerns was estimated at C$6.6 billion (Daley, Morin, LeBlanc, Gregoire, & Savard, 2009). Despite the chronic nature and extensive impact, insomnia is vastly undertreated, and primarily addressed within general practice/primary care provider clinics, where few resources other than sleep pills are available.

Insomnia and health

Given the high rates of comorbidity and the chronic nature of the disorder, chronic insomnia is linked with substantial decreases in quality of life, including by measures of absenteeism, accidents, and decreased vitality and social functioning. Insomnia plays key roles in the course of many psychological problems, contributing to their development or relapse, and exacerbating symptoms. These disorders include anxiety, depression, bipolar disorder, post-traumatic stress disorder (PTSD), adjustment disorder, panic disorder, somatoform disorder, schizophrenia, neurocognitive disorders, eating disorders, and substance use disorders, and physical health problems including arthritis, cancer, hypertension, chronic pain, cardiovascular disease, and Type 2 Diabetes Mellitus (T2DM;American Psychiatric Association, 2013; Buysse et al., 1994; Foley et al., 1995; Ohayon, Caulet, & Lemoine, 1998).

As an illustrative example, depression is highly comorbid with insomnia. About three quarters of all depressed patients complain of insomnia symptoms, and close to half report

sufficient symptoms to warrant an insomnia diagnosis. Until recently, when the two disorders were comorbid, insomnia was viewed as a symptom of depression and so was not considered a focus of treatment. More recent research indicates a more complex, bidirectional, relationship. While insomnia typically has its origins before depression develops, high levels of depressive symptoms are classified as a premorbid psychological vulnerability to insomnia (Ford & Kamerow, 1989; LeBlanc et al., 2009).

Insomnia impacts not only the development, but also the course of depression once developed. It can perpetuate depression, exacerbate depression, and increase risk of its recurrence. In a multisite randomised control trial (RCT) of elderly depressed patients, participants were randomised to receive either specialist depression treatment or treatment as usual. After 3 months of therapy, those with persistent insomnia were 3.5 times more likely to remain depressed at 12 months, compared to those without insomnia, showing how insomnia perpetuates depression (Pigeon et al., 2008). Similarly, in a 9-week RCT where depressed participants were treated with either 3 mg fluoxetine or placebo, participants with higher levels of insomnia experienced significantly greater intensity of suicidal ideation, indicating insomnia exacerbates depressive symptoms (McCall et al., 2010). After an RCT treating depression with interpersonal psychotherapy and/or pharmacotherapy, residual sleep disturbance was the strongest independent predictor of depression recurrence (Perlis, Giles, Buysse, Tu, & Kupfer, 1997).

Treatment Effects on Health

Cognitive Behavioural Therapy for Insomnia (CBTI) is currently the gold standard insomnia intervention. It is highly effective, and has been extensively researched: it has a 70-80% success rate in efficacy studies for chronic insomnia(Irwin, Cole, & Nicassio, 2006; Morin, Culbert, & Schwartz, 1994). Efficacy remains even when insomnia is comorbid with a range of other medical and psychiatric disorders (Geiger-Brown et al., 2015; Haynes et al.,

2016; Mundt et al., 2016; Talbot et al., 2014; Trauer, Qian, Doyle, Rajaratnam, & Cunnington, 2015; Ye et al., 2015). Research consistently shows that CBTI is, at minimum, equally as effective as pharmacological treatment, and has significantly longer duration of benefits after treatment ceases.

CBTI is a multimodal therapy, typically provided over a 4-8 session course of treatment. It has two main foci: altering behaviours to improve sleep patterns (behavioural therapy), and altering the unhelpful and/or inaccurate thoughts, beliefs and attitudes people hold regarding sleep (cognitive therapy). The ultimate goals of CBTI are to: 1) consolidate sleep (behavioural); 2) break associations between sleep and anxiety and/or arousal (behavioural); and 3) to develop a more realistic picture of sleep and sleeping habits (cognitive). It is effective for reducing insomnia symptoms under many conditions, including when delivered in group settings, over the phone or internet, and in brief 1-2 session treatments, though different patient populations benefit most from different conditions (Cape, Leibowitz, Whittington, Espie, & Pilling, 2015; Ellis, Cushing, & Germain, 2015; Ho, Chung, Yeung, Ng, & Cheng, 2014; Norell-Clarke, Jansson-Fröjmark, Tillfors, Holländare, & Engström, 2015; Ye et al., 2015; Zachariae, Lyby, Ritterband, & O'Toole, 2016).

While experiencing insomnia, individuals report lower quality of life and many negative health outcomes (American Psychiatric Association, 2013; Buysse et al., 1994; Foley et al., 1995; Fortier-Brochu, Beaulieu-Bonneau, Ivers, & Morin, 2010; Ohayon et al., 1998). Many health domains have been found to improve significantly when insomnia is treated with CBTI (Manber et al., 2011a; McCall et al., 2010). The direct impact on depression is discussed in detail. While the relationship between insomnia and depression is bidirectional, when it comes to improving health, the effect is unidirectional: improving insomnia alleviates depression symptoms. When patients with comorbid insomnia and depression are treated with CBTI, whether individually or in group format, both insomnia and depression symptoms

improve, and remission rates for depression are higher in CBTI than in control groups (Bei, Ong, Rajaratnam, & Manber, 2015; Manber et al., 2011b; Manber et al., 2008; Norell-Clarke et al., 2015; Trockel, Manber, Chang, Thurston, & Tailor, 2011).

Importantly, these therapeutic effects are not bidirectional. When depression is treated with CBT or with antidepressants, while depressive symptoms improve, sleep problems are not consistently alleviated. In essence, once insomnia develops, it must be treated in its own right. This is also true for other mental health disorders, including PTSD, bipolar disorder and substance use disorders (Belleville, Guay, & Marchand, 2011; Brower, Krentzman, & Robinson, 2011; St-Amand, Provencher, Bélanger, & Morin, 2013; Zayfert & De Viva, 2004), indicating the importance for treating insomnia symptoms independently and early. These findings also underscore the reason "secondary insomnia" is no longer considered a valid diagnosis. Even when another health condition is the precipitating event initially triggering the insomnia, once chronic, the insomnia has developed into its own disorder.

Sleep disordered breathing

Sleep disordered breathing (SDB) refers to a range of sleep disorders characterised by insufficient or abnormal ventilation during sleep. Importantly, disordered breathing is specific to the sleep state, with respiration during wake being normal. The most common form of SDB is sleep apnea whereby the individual suffers from repetitive episodes of reductions (hypopnea) or cessation (apnea) in airflow during sleep. Sleep apnea is typically divided into two main forms, obstructive sleep apnea (OSA) where apneas/hypopneas occur due to complete or partial collapse of the upper airway, or central sleep apnea (CSA), where apneas/hypopneas occur due to a reduction in brain stem respiratory motor output (i.e. no effort is made to breathe). These repeated respiratory events are associated with reductions in blood oxygen saturation (hypoxemia), rises in blood carbon dioxide (hypercapnia) and surges in sympathetic activation. Unfortunately, in many instances regular breathing only resumes upon arousal from sleep.

Individuals with a SDB diagnosis stop breathing at least 5 times an hour, with 30 or more events per hour considered severe. The cumulative impact of repeated respiratory events (i.e., reduction/cessation of breathing followed by an awakening) is significant sleep fragmentation and reductions in both deep stage N3 sleep and REM sleep (Lamphere et al., 1989; Quan et al., 2011; Waldhorn et al., 1990).

Of the two types of SDB, OSA is by far the most common, with prevalence rates ranging between 9-38% of the general population. There are a number of factors which put people at higher risk for developing OSA. The most common risk factor is obesity, specifically central obesity. In both sexes, OSA is more prevalent with increasing age (Young et al., 1993), reaching very high rates (70%) in patients over 65 years (Ancoli-Israel, Klauber, Kripke, Parker, & Cobarrubias, 1989). Males appear to have a higher rate of sleep apnea than women, with the ratio varying from 2:1 to 10:1 depending on the study (Strohl & Redline, 1996). In addition, any condition resulting in a smaller airway can predispose towards the development of OSA. Family history is also a strong risk factor for OSA whereby the risk significantly increases based on the number of affected relatives (Redline et al., 1995). There are also several environmental factors which can worsen OSA symptoms. Use of alcohol or sedative medication can contribute to development of OSA through their relaxant effect on the upper airway muscles (Issa & Sullivan, 1982). Finally, smokers are at an increased risk for developing OSA with current smokers at greater risk than nonsmokers and heavy smokers having the greatest risk (Wetter, Young, Bidwell, Badr, & Palta, 1994).

In comparison to OSA, CSA is much less prevalent, however it is commonly found in certain populations such as in preterm and term infants, patients with heart failure (~30%; Sin et al., 1999), in most adults when ascending to high altitude (Burgess, Johnson, & Edwards, 2004), and can also be a side effect of opioids. Given that this review will largely focus on OSA, a more detailed description of CSA can be found in a recent review by Sands et al. (2017).

Sleep Disordered Breathing and Health

One of the most commonly reported symptoms of SDB is excessive daytime sleepiness (AASM, 2014), and this may explain the increased risk of motor vehicles accidents seen in this population (Terán-Santos, Jiménez-Gómez, & Cordero-Guevara, 1999; Young, Blustein, Finn, & Palta, 1997). However, the relationship between crash risk and measures of sleep apnea severity and subjective sleepiness have not always been consistently demonstrated (Ellen et al., 2006).

Sleep apnea has also been associated with a range of cognitive impairments, most particularly in attention, executive function, and memory (Ayalon, Ancoli-Israel, & Drummond, 2010; Bedard, Montplaisir, Malo, Richer, & Rouleau, 1993; Bedard, Montplaisir, Richer, Rouleau, & Malo, 1991; Canessa et al., 2011), executive function (Canessa et al., 2011; Naëgelé et al., 1998; Saunamäki, Himanen, Polo, & Jehkonen, 2009), and memory (Canessa et al., 2011; Daurat, Foret, Bret-Dibat, Fureix, & Tiberge, 2008; Salorio, White, Piccirillo, Duntley, & Uhles, 2002). Systematic reviews and meta-analyses have confirmed the consistency of these findings. These impairments in cognition are also consistent with imaging data showing significant cerebral grey matter loss in OSA patients (Yaouhi et al., 2009). Nonetheless, cognitive deficits are often not as extreme as one might expect given the severity of sleep disruption and hypoxia, as well as gray matter abnormalities experienced, suggesting that some compensatory mechanisms may be at work in these individuals (Ayalon, Ancoli-Israel, Klemfuss, Shalauta, & Drummond, 2006; Castronovo et al., 2009). Based on a range of converging evidence from animal and human studies, it would appear that sleep fragmentation, intermittent hypoxia, or both, play causal roles in the pathogenesis of these cognitive deficits (Beebe & Gozal, 2002; Knoepke & Aloia, 2009; Verstraeten, 2007).

An important health domain where SDB is implicated is cardiovascular and metabolic health. Population based studies have demonstrated SDB to be associated with metabolic

abnormalities including glucose intolerance and insulin resistance, with risk increasing with increasing hypoxemic and SDB severity indexes (Punjabi et al., 2004). Furthermore, SDB been shown to be an independent risk factor for diabetes (Botros et al., 2009; Reichmuth, Austin, Skatrud, & Young, 2005; Wang, Bi, Zhang, & Pan, 2013). Hypertension is extremely prevalent in SDB populations. Numerous data from animal models and cross sectional studies have demonstrated significant associations between SDB and blood pressure (Brooks, Horner, Kozar, Render-Teixeira, & Phillipson, 1997; Fletcher, DeBehnke, Lovoi, & Gorin, 1985; Nieto, 2000; Stoohs et al., 1996). Prospective longitudinal studies have demonstrated patients with SDB have increased risk of developing hypertension, with increasing severity of SDB associated with increasing risk (O'Connor et al., 2009; Peppard, Young, Palta, & Skatrud, 2000). Although SDB has been shown to be an independent risk factor for hypertension, comorbid obesity has previously been shown to mediate some of this dose-response relationship (Nieto, 2000; O'Connor et al., 2009). SDB has also been associated with a range of other cardiovascular morbidities including, but not limited to, atherosclerosis, atrial fibrillation, stroke, and heart failure (Gottlieb et al., 2010; Hoffstein & Mateika, 1994; Hung, Whitford, Parsons, & Hillman, 1990; Neilan et al., 2013; Redline et al., 2010; Shahar et al., 2001; Somers, Dyken, Clary, & Abboud, 1995; Wessendorf, 2000; Yaggi et al., 2005). Importantly, longitudinal studies have shown untreated OSA patients are at increased risk of both fatal and non-fatal cardiovascular events compared to controls (Marin, Carrizo, Vicente, & Agusti, 2005), as well as an increased risk of all-cause mortality (Punjabi et al., 2009).

Treatment Effects on Health

The current gold standard treatment for OSA in adults is Continuous Positive Airway Pressure (CPAP) therapy which works by producing a positive pressure in a patient's airway in order to "splint" the airway open such that obstruction is no longer possible. CPAP is exceedingly efficacious in reducing OSA severity and improving hypoxemic and sleep quality

parameters (Issa & Sullivan, 1986; Kakkar & Berry, 2007; Sullivan, Issa, Berthon-Jones, & Eves, 1981). However clinical effectiveness is mainly limited by its tolerability. Approximately 30% of patients fail to initiate treatment and long term usage studies suggest that of those that start therapy, only half of patients continue to use therapy after 3 months. Even in patients using CPAP, many use it for less than half the night and are thus undertreated (Weaver et al., 1997). The second major treatment modality for OSA is mandibular advancement splits (MAS, also referred to as oral appliances). These devices reposition the tongue and protrude the mandible forward in order to tighten several soft tissue structures and as such, increase the overall size of the upper airway (Chan et al., 2010). A variety of surgical procedures are also utilized for OSA treatment, however these are typically undertaken as a last resort option, often when patients are unable to adhere to these non-invasive treatments. Importantly, the treatment success for both the MAS and surgical procedures varies considerably in the literature (Caples et al., 2010). Part of this variability is likely due to differences in the way treatment successes have been defined in these studies as well as differences in the way OSA severity/burden was assessed.

Although there is a high degree of variability between studies, meta-analyses show treatment with CPAP can significantly reduce subjective sleepiness complaints and, to a lesser degree, reduce objective sleepiness parameters (Patel, White, Malhotra, Stanchina, & Ayas, 2003). These effects are typically stronger in patients with more severe SDB and those with a greater degree of baseline sleepiness. A reduction in motor vehicle crash risk, as well as improved driving simulator performance has also been demonstrated after successful CPAP treatment (Antonopoulos, Sergentanis, Daskalopoulou, & Petridou, 2011; Tregear, Reston, Schoelles, & Phillips, 2010). While a number of studies have demonstrated the success of CPAP in normalizing various aspects of neurocognitive function (Borak, Cieślicki, Koziej, Matuszewski, & Zieliński, 1996; Canessa et al., 2011; Engleman, Martin, Deary, & Douglas,

1994), others have demonstrated enduring impairments in some cognitive domains after treatment (Bedard et al., 1993; Ferini Strambi et al., 2003; Kotterba et al., 1998). Available meta-analyses suggest that deficits in attention are most effectively resolved by CPAP treatment (Kylstra, Aaronson, Hofman, & Schmand, 2013), whereas improvements in executive functions and memory are less consistently reported (Aloia, Arnedt, Davis, Riggs, & Byrd, 2004; Sánchez, Martínez, Miró, Bardwell, & Buela-Casal, 2009).

In terms of the effect of treatment on cardiovascular sequelae, several meta-analyses have demonstrated that CPAP usage significantly reduces blood pressure. However, the effect size is small in comparison to anti-hypertensive medications (Fava et al., 2014; Montesi, Edwards, Malhotra, & Bakker, 2012). While observational evidence suggests that treatment with CPAP is associated with a reduced risk of fatal and non-fatal cardiovascular events (Doherty, Kiely, Swan, & McNicholas, 2005; Marin et al., 2005), randomised control trials have not replicated these findings (Yu et al., 2017). At present the majority of the available treatment data currently available applies to the use of CPAP, treatment data from patients using a MAS device generally demonstrates improvement in health outcomes equivalent to those observed with CPAP (Bratton, Gaisl, Wons, & Kohler, 2015; Phillips et al., 2013).

Summary

This chapter has emphasized the critical importance of achieving healthy sleep for a diverse set of physical and mental health morbidities. The mechanisms linking sleep and health are often complex and bidirectional in nature, however treatment outcomes tend to be unidirectional. Insomnia and OSA can be highly effectively treated using psychological and medical treatments. These treatments then contribute to subsequent improvements in health problems. However, the unidirectionality of these improvements means that chronic sleep problems require direct treatment for improvement. This reflects the special role that sleep

plays in health, and the attention that needs to be paid to healthy sleep for overall physical and mental health.

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CHAPTER 2

Literature Review

Chapter 2: Literature Review

Overview

Given the influence of sleep on health and well-being presented in the previous chapter, it is important to understand factors which can promote or impair an individual's sleep. One such factor, which is the focus of this thesis, is the presence of a bedpartner. The field of sleep in couples is very much in its infancy. This literature review will present an exploration of what is currently known about sleep as a dyadic process, and present an overview of the aims of the present body of research.

Over half of Australian adults share a bed each night, suggesting adult bed-sharing is an intrinsically social behaviour that extends far into the community. For most adults, romantic relationships are the primary source of social contact. Falling asleep beside a partner may promote a couple's emotional connection and relationship functioning. However, bedpartners can have potential negative impacts on an individual's sleep structure and circadian rhythms (Dittami et al., 2007).

Literature to date has been published from medical, psychological, and sociological perspectives. All have suggested that one individual's sleep has potential to significantly, and negatively, influence their bedpartner's sleep (Baron et al., 2011; Baron, Smith, Czajkowski, Gunn, & Jones, 2009; Beninati, Harris, Herold, & Shepard Jr, 1999; Rosenblatt, 2006; Strawbridge, Shema, & Roberts, 2004; Venn, 2007). As such, the presence of a bedpartner can lead to partner inconvenience, sleep disruption, or distress.

This review aims to address three key questions regarding how bedsharing may impact on an individual's sleep experience:

(1) How and why do bedpartners influence one another's sleep? (i.e., what is the theoretical basis for studying adult bedsharing, and what measures exist to assess this influence?)

(2) What factors are currently known to influence dyadic sleep? (e.g., relationship quality, circadian considerations, physical and mental health?).

(3) How does presence of a sleep disorder impact the bedpartner, as well as the patient?

How and why do bedpartners influence one another' sleep?

On average, couples prefer to sleep in the same bed. The fact that sleep is often shared with a bedpartner was first recognised in the literature 50 years ago, when Monroe (1969) recorded simultaneous polysomnography (PSG) in heterosexual dyads. Changes occurred in sleep architecture when bedsharing: Slow Wave Sleep (SWS) decreased in both sexes and Rapid Eye Movement (REM) sleep increased in men when couples slept together compared to sleeping alone. The researchers noted sleep architecture and sleep efficiency changes were not related to the usual psychological changes that accompany poor sleep (i.e., they did not self-report poor sleep quality or feeling unrested afterwards; Monroe, 1969). This discrepancy raises questions regarding why sharing a bed with an intimate partner may alter objective sleep but not an individual's perception of their sleep, and indicates a potentially complex and conflicting effect between sleep, individual daytime functioning, and relationship functioning. Unfortunately, the field of dyadic sleep has remained relatively limited, and due to limited research conducted in this area, Monroe's (1969) PSG results have not been replicated.

Understanding why bedpartners matter: Attachment theory.

Sleep is a physiologically vulnerable state. To fall asleep, an individual must feel safe and secure enough to down-regulate vigilance and alertness levels (Dahl & Lewin, 2002). This is most likely to occur in social and environmental contexts where an individual is comfortable feeling vulnerable. Given this, sleep in couples can be understood through the theoretical framework of attachment (Feeney & Kirkpatrick, 1996; Troxel, Cyranowski, Hall, Frank, & Buysse, 2007).

Attachment refers to the emotional bonds formed between children and caregivers early in their lives. Interactions with early caregivers influence expectations surrounding how warm, nurturing, and consistently responsive to their needs a caretaker should be (Bowlby, 1973). This same framework can be applied to different life stages, including how attachment style and expectations learned in early life can manifest in adult romantic partnerships (Hazan & Shaver, 1994; Troxel, Robles, Hall, & Buysse, 2007).

Attachment-related concerns become particularly apparent while falling asleep (Carmichael & Reis, 2005). If an individual is concerned about their partner's physical or emotional availability at bedtime, this can interfere with sleep by preventing down-regulation of vigilance processes (Carmichael & Reis, 2005), and by creating anxiety leading to hypervigilance and alertness (Dahl & El-Sheikh, 2007; Troxel, 2010). In contrast, securely attached individuals may feel a sense of safety and security going to bed with their partner, thereby providing the optimal environment in which to achieve consolidated, high-quality sleep. This theory is supported by data (Elsey, Keller, & El-Sheikh, 2019; Sloan, Maunder, Hunter, & Moldofsky, 2007). Individuals with anxious-insecure attachment styles have been found to have a greater proportion of EEG in the alpha frequency range during sleep, and report poorer quality sleep than those with secure attachment styles (Sloan et al., 2007). These markers indicate a hyperaroused sleep state, making the individual more sensitive to external stimuli and increasing the likelihood of arousals from sleep (Perlis, Giles, Bootzin, et al., 1997). Notably, in Sloan's (2007) study participants' sleep was recorded when sleeping alone and in a sleep clinic which may have influenced outcomes. Thus, sleep may be less restorative for those with an insecure attachment style, especially while in the vulnerable position of sharing a bed with their romantic partner.

Understanding why bedpartners matter: The Social Role of the Bed.

The bed and sleep play an important role in maintaining a couple's relationship, and presence of a bedpartner may affect the sleep of both partners in a couple. For cohabitating adult couples with at least one partner in full-time employment, sleep comprises the majority of time spent together (Rosenblatt, 2006). Bedtime and the sleep routine are thus not solely about sleep, but also about renewing and maintaining the relationship. Because of the importance many couples place on time together at bedtime, it is unsurprising positive or negative sleep experiences interact with relationship satisfaction. Feeling satisfied and supported in a romantic relationship, and having a positive outlook on life, can lead to better subjective and objective sleep quality (El-Sheikh, Kelly, & Rauer, 2013; Kane, Slatcher, Reynolds, Repetti, & Robles, 2014; Troxel, Buysse, Hall, & Matthews, 2009). On the other hand, negative daytime emotions, mood and experiences are associated with sleep disturbance (Åkerstedt et al., 2012; Brissette & Cohen, 2002; Morin, 2003).

The bed's social meaning may depend on an individual's sex. In Pankhurst and Horne's (1994) seminal paper, females reported the reason they preferred to share a bed was that they felt greater levels of security when sleeping with their partner. This finding is supported by survey findings that over half of women prefer their romantic partner share their own desired bedtime (Randler, Barrenstein, Vollmer, Díaz-Morales, & Jankowski, 2014). In contrast, males reported their reason for preferring to share a bed was because cosleeping was simply a habit. Sex differences in the social role of the bed are further demonstrated when examining couples' alignment of sleep schedules. Looking at sleep timing from an individual perspective, influences considered are individual, i.e., one's own circadian phase, and one's own daytime schedule. However, adult bedsharing involves negotiation around bedtimes, including the choice whether or not to have a concordant bedtime. Hida et al. (2012) investigated factors that predicted sleep timing for members of heterosexual couples, and found women typically take

their male partner's sleep timing into account when selecting their schedule. When looking at sleep timing, women's schedules were associated both with her own chronotype, as well as her husband's sleep schedule. Interestingly, men's sleep timing was not associated with their wife's sleep schedule.

Negotiating sleep schedules in romantic relationships demonstrates Hislop's (2007) suggestion that bed-sharing requires compromise from partners. Bedpartners must take into account their partner's sleep schedule in order to choose a time that might be optimal for the relationship, even while it may not be optimal for their own sleep quality. Social compromises go further than simply selecting sleep timing, also including phenomena such as choosing sides of the bed, room lights, noise, and temperature. In this way, bedpartners act as social influences on the timing of sleep/wake cycles (Hasler & Troxel, 2010; Revenson, Marín-Chollom, Rundle, Wisnivesky, & Neugut, 2015).

The chosen bedtime is an important reflection of relationship functioning for many couples. There is evidence suggesting spouses who are mismatched in sleep patterns report poorer marital functioning than those with concordant patterns (Hasler & Troxel, 2010; Larson, Crane, & Smith, 1991; Revenson et al., 2015). Specifically, mismatched couples have reported lower self-reported relationship satisfaction, greater levels of conflict, less time spent conversing and participating in shared activities, and less frequent sex (Larson et al., 1991). It is likely this effect is bidirectional (Hasler & Troxel, 2010). Couples who have greater relationship satisfaction are more likely to desire and orchestrate concordant bedtimes, in order to spend time together. Conversely, individuals in unhappy relationships may mismatch sleep patterns in order to avoid intimacy (Revenson et al., 2015; Troxel, 2010). Of course, this is not deterministic. There are couples who maintain high relationship satisfaction while having discordant sleep times. Characteristics of these couples include higher levels of flexibility, adaptability, and problem-solving ability, compared with happy couples with concordant sleep

schedules (Gunn, Buysse, Hasler, Begley, & Troxel, 2015; Larson et al., 1991). This is an important reminder that while overall relationship satisfaction is frequently positively associated with concordant sleep timing, there are other factors important to understanding the dyadic sleep experience.

Viewing dyadic sleep through lenses both of attachment theory and the social role played by the bed in a romantic relationship, are popular frameworks to characterise the influence of a bedpartner, particular as it relates to relationship functioning. This has given rise to one of the only truly dyadic sleep measures currently in use: sleep/wake concordance.

Understanding how bedpartners matter: What measures exist to assess dyadic sleep?

Ways in which couples influence one another's sleep include: altering timing of the sleep period, waking one another with noise or movement, and influencing the environment in which they sleep. However, the extent to which couples influence one another is as yet unknown. A review of the literature indicates that dyadic sleep is currently measured using either an individual's full-night sleep parameters, or a novel metric known as sleep/wake concordance.

Individual Sleep Parameters.

The most common method to assess dyadic sleep to date has been by using individual sleep parameters (Dittami et al., 2007; Hislop, 2007; Lee et al., 2018; Spiegelhalder et al., 2015), and investigating how these change between nights spent with and without bedpartners. One of the first studies which thoroughly investigated dyadic sleep was conducted by Pankhurst and Horne (1994), examining the association of body movements between bedpartners. Regularly sharing a bed was associated with having shorter sleep latency, greater total sleep time, and earlier bedtime. This supports research finding couples frequently alter their sleep schedules to achieve matched schedules, which many couples view as desirable (Hislop, 2007;

Lee et al., 2018; Randler et al., 2014; Spiegelhalder et al., 2015). Using actigraphy, Meadows et al. (2009) found some individual sleep parameters were more relevant to understanding couples' sleep than others. Specifically, bedtime, sleep latency, the light/dark ratio within the room, and number of wake bouts all significantly clustered at the couple level; while other parameters did not. A significant limitation of using individual sleep parameters to measure a dyadic construct is the lack of sensitivity of these measures. There are likely many reasons why individual sleep parameters change on a night-to-night basis, making the bedpartner signal-to-noise ratio small so it is difficult to see the specific effect of a bedpartner. While these findings provide insight into the change in sleep behaviours by bedpartners, measures designed to assess the specific dyadic nature of sleep are required in order to understand the dyadic sleep experience more fully, .

Concordance.

The only specifically dyadic measure developed is that of sleep/wake concordance (Drews et al., 2017; Gunn et al., 2015; Pankhurst & Horne, 1994). Concordance assesses the extent to which couples are in the same sleep/wake state, or differing states, using an epoch-by-epoch analysis. Sleep/wake concordance refers to couples' coregulation of physiological states through the rest interval. Coregulation has been described as couples being "in tune" and matching each other's physiological states, and has been documented in sleep, cortisol, and mood, among others (Ditzen, Hoppmann, & Klumb, 2008; Saxbe & Repetti, 2010; Timmons, Margolin, & Saxbe, 2015). Concordance has also been considered in terms of coordination of couples' sleep/wake schedules (Elsey et al., 2019; Meadows, Arber, Venn, Hislop, & Stanley, 2009).

Findings from Gunn et al. (2015) showed 48 heterosexual married couples who regularly shared a bed with their spouse had minute-by-minute sleep/wake concordance rates significantly higher than that of randomly matched pseudocouples. Using a dyadic rest interval

beginning when the first partner went to bed and ending the time the latest partner arose, a concordance rate for each couple was determined. Sleep/wake concordance ranged between 53-88% of the night (*M* = 75% rest interval epochs for couples were spent in matched sleep or wake states). This means on average, 25% of any given night is spent in discordant states, i.e., one partner asleep and one awake. A subsequent study analysing the same dataset reported that, as expected because in healthy sleepers much more of the rest interval is spent sleeping than awake, sleep concordance comprised more of the concordant time (*M* = 66%) than wake concordance (*M* = 9%; Gunn et al., 2017). This variable can be combined with individual full-night sleep parameters to provide a richer description of dyadic sleep. This ground-breaking research prompts questions such as what role couples synchronising their rest intervals may play on sleep/wake concordance or other aspects of sleep disruption by a bedpartner.

Concordance may also extend to synchronization of sleep stages. A pilot investigation of *n* = 4 heterosexual couples was recently published by Drews et al (2017). In this study, couples underwent a laboratory stay where they slept either alone or shared a bed with their bedpartner. This study found bed-sharing was associated with a number of sleep benefits: increased objective and subjective sleep duration, improved sleep efficiency, increased REM sleep, and increased Stage N3 (i.e., “deep sleep”) sleep duration. Interestingly, synchronization of these stages was higher in couples sleeping together compared with when they slept apart – which may have been due to synchronised sleep schedules, as it was not reported whether on ‘sleep-apart’ nights schedules were synchronised. Additionally, participants who slept with their bedpartner subjectively reported feeling significantly more relaxed than when they slept alone. The pilot study therefore proposes a link between stage N3, REM sleep, and the emotional features of bedsharing. However, replication with larger sample size is critical before drawing strong conclusions, particularly as these results are differ somewhat from Monroe’s (1969) study. It is difficult to draw direct comparisons between Drews et al. (2017) and Monroe

(1969), due to difference scoring classification systems and different definitions of “deep sleep”. However, it is worth noting differences in subjective data. During Drews et al.’s (2017) study, partners reported feeling much more relaxed after spending the night with their bedpartner, whereas in Monroe’s (1969) study, partners reported no difference between their subjective sleep and how rested they felt in either condition.

To our knowledge, concordance and coregulation of sleep have never been documented in the context of sleep disorders. As such, our knowledge in this field is limited to good sleeping couples. Additionally, while sleep/wake concordance provides a tool to describe how similar bedpartner’s sleep are to one another, it falls short of describing any potential mechanism of sleep disruption. Whether or not there are other processes involved, and how to improve tools to measure and describe dyadic sleep, will be the first aim of this thesis.

What Factors are Currently Known to Influence Dyadic Sleep?

Much of the research in the field of dyadic sleep has considered the interaction between bedpartners and relationship functioning. However, sleep for individuals is known to be influenced by a range of biopsychosocial factors. It is likely several factors related to individual sleep also have a dyadic component. This next section will review factors that have been identified as important for the dyadic sleep experience, including: relationship quality; a circadian perspective of the bedpartner as time cue; and two important determinants of individual sleep that have been identified as important for dyadic sleep: physical and mental health.

Relationship Quality.

Relationship quality is the most thoroughly investigated factor of the dyadic sleep experience. An individual’s relationship dissatisfaction can influence both bedpartners’ sleep quality (Cartwright & Knight, 1987; Hasler & Troxel, 2010; Strawbridge et al., 2004; Troxel

et al., 2009). This influence is thought to be mediated by several processes, including social support, conflict, and the role of the partner as a social time cue.

Social Support.

Social support is a protective factor for sleep in adults, and according to attachment theory, is the primary reason partners prefer to sleep together (Eshkoor, Hamid, Nudin, & Mun, 2013; Troxel, Buysse, Monk, Begley, & Hall, 2010). Social support is positively related to sleep quality, while social strain worsens sleep quality and can increase self-reported sleep problems (Ailshire & Burgard, 2012; Brummett et al., 2006; Kent, Uchino, Cribbet, Bowen, & Smith, 2015). Higher rates of insomnia symptoms are associated with lower perceived social support, even after controlling for other factors such as physical health and sexual activity (Strawbridge et al., 2004; Troxel et al., 2009). When the social support/sleep relationship was assessed by Troxel et al. (2010) on a day-by-day basis, higher levels of perceived social support predicted better subjective and objective sleep efficiency, with less time spent awake throughout the night, in both healthy and sleep-disordered (insomnia) populations. Additionally, subjective sleep latency was lowest in individuals with highest levels of perceived social support, which indicates greater ease of falling asleep each night when feeling supported. As such, social support, which in many cases includes support offered by a bedpartner, has benefits for individuals' sleep that are visible in both healthy and insomnia populations (Troxel et al., 2010).

Interpersonal Conflict.

It has been hypothesised that greater levels of interpersonal conflict during the day correlate with poorer quality sleep at night (Fillo et al., 2017; Gordon & Chen, 2014; Hasler & Troxel, 2010; Hicks & Diamond, 2011). Daytime conflict could plausibly cause several sleep changes. Conflict may cause difficulty falling asleep (due to post-conflict arousal), alter the sleep schedule (by going to bed at different times to avoid one another), or by changing setting

(one partner sleeping in a different room; Hasler & Troxel, 2010). Additionally, conflict can lead to distress. Individuals experiencing more interpersonal distress demonstrate altered sleep architecture, specifically, a greater percentage of REM sleep (Carmichael & Reis, 2005; Diamond, Hicks, & Otter-Henderson, 2008; Gunn, Troxel, Hall, & Buysse, 2014; Hicks & Diamond, 2011). Finally, interpersonal conflict may demonstrate a negative relationship with social support. That is, those who lack social support from their partners experience higher levels of interpersonal conflict.

Bedpartner as time cue.

An additional explanation for the positive relationship between sleep and marital happiness is one's bedpartner acting as a social '*zeitgeber*' – an external time cue which helps regulate the body's sleep-wake rhythms. As discussed above, couples frequently accommodate their sleep habits and timing to be concordant. Couples with higher sleep concordance, especially shared bedtime and shared chronotype (i.e., the time of day when an individual is naturally inclined to be either more or less physically and mentally alert), have been found to have higher marital satisfaction reported by both wives and husbands (Lai, Ip, Lam, Weaver, & Fong, 2015; Lange, Waterman, & Kerkhof, 1998; Larson et al., 1991). If this concordance is due to circadian rhythms adjusting over time to become more similar, a hypothesis as yet untested in the literature, this may indicate a potential biological basis to relationship satisfaction.

Physical Health.

As with relationship quality, physical health of an individual can influence sleep in both bedpartners. Individuals in romantic relationships are attuned to each other's health and well-being; in particular, their physiological arousal levels (Butler, 2011; Timmons et al., 2015). As was discussed in Chapter 1, sleep is a highly important health behaviour, and for most adults, it is shared between members of couples.

The dyadic influence of physical health on sleep is likely to be due to two factors: physiological linkage, and shared health behaviours.

Physiological Linkage.

Covariation between couples in moment-to-moment physiological states is referred to as physiological linkage. Matched activation has been observed in couples in measures of heart rate, blood pressure, cortisol and other hormones (Ditzen et al., 2008; Timmons et al., 2015). All these measures of arousal are directly linked with sleep; therefore, one partner's activation could directly influence their partner's arousal level, and as such, their propensity to fall asleep.

Sleep linkage may be mediated by physiological changes caused directly by physical intimacy. Increased physical touch by a romantic partner can result in better sleep quality (Field, 2010; Urponen, Vuori, Hasan, & Partinen, 1988). The pathway for this result may be mediated by hormonal changes: physical intimacy by a partner can reduce stress and cortisol levels, which can thereby improve sleep (Burleson, Trevathan, & Todd, 2007; Ditzen et al., 2007). This pathway may be a contributing explanation for two major associations explored in this thesis thus far: the mechanisms by which couples' sleep arousals and sleep states can be linked (i.e., sleep/wake concordance; Gunn et al., 2015; Gunn et al., 2017), and why sleep problems are more common in unhappy or distressed relationships, where both partners experience increased conflict-based arousal (Ditzen et al., 2008; Timmons et al., 2015).

Being attuned to a partner's health can affect both partners' sleep; likewise, being attuned to a partner's sleep/wake state can subsequently affect both partners' health. In non-sleep disordered couples, a spouse's subjective sleep problems were increased if their partner reported "poor" health status (Strawbridge et al., 2004). A similar effect was found in couples where one partner experienced osteoarthritis (Martire et al., 2013). On days where one partner was in greater pain, their bedpartner had poorer sleep that night and reported feeling less refreshed after sleep, regardless of how the patient slept. Equally, in couples without sleep

disorders, having increased sleep/wake concordance (i.e., linkage in sleep states) was associated with individuals having lower blood pressure and less systemic inflammation. These results indicate a novel linkage between health and dyadic sleep, which is likely bidirectional in nature. This link may be present in many elements of health and illness.

Shared Health Behaviours.

Couples in romantic relationships tend to participate in shared health behaviours – many of which either promote or impair sleep. Couples share similar health behaviours at rates much higher than expected by chance (Jackson, Steptoe, & Wardle, 2015; Lewis et al., 2006), with 66% concordance for physical inactivity, and up to 83% concordance for smoking (Graham, Hutchinson, Law, Platt, & Wardle, 2016). Strong concordance exists across the lifespan, demonstrated in both younger and older couples (Graham et al., 2016; Machado et al., 2017).

Importantly, physical, social and mental health are intertwined: when one member of a couple experiences a significant chronic physical illness, partners experience social limitations as a result of the patient's changed sleep habits and sleep requirements. Partners reported no longer being able to take part in evening social engagements due to the patient's increased sleep needs, and reported more tiredness due to disturbed sleep (Broström, Strömberg, Dahlström, & Fridlund, 2003). Further emotional distress is caused by the patient's cognitions about not wanting to be a burden. This cascade of symptoms results in both partners reporting poorer quality of life, including poorer sleep quality (Poloméni, Lapusan, Bompoint, Rubio, & Mohty, 2016). Therefore, it is clear that a couple's health context significantly impacts sleep in complex ways.

Mental Health.

Mental health and sleep are strongly related within an individual. It is likely that the same relationship occurs in bed-sharing couples. On an individual basis, and as explored in

Chapter 1, depression and insomnia are frequently comorbid (Ford & Kamerow, 1989; LeBlanc et al., 2009; Sivertsen et al., 2012). The connection between anxiety and sleep difficulties, particularly sleep onset difficulty, are also well-documented within the literature (Staner, 2003). Mechanisms by which these disorders contribute to an individual's sleep problems are varied, including biological, psychological, and social mechanisms. Biological factors include shared genetic vulnerabilities, changes in cortisol and inflammatory markers, and dysregulation of sleep architecture. Psychological factors include attentional and cognitive biases, and dysfunctional beliefs about self and sleep. Social mechanisms include altered and reduced social interactions, and interpersonal stressors (Blake, Trinder, & Allen, 2018; Carney, Edinger, Meyer, Lindman, & Istre, 2006; Esposito, Di Matteo, & Di Giovanni, 2008; Forbes et al., 2006; Gehrman et al., 2011; Hiller, Johnston, Dohnt, Lovato, & Gradisar, 2015; Lovato & Gradisar, 2014; Perlis et al., 2016).

Within social relationships such as marriage, mood states are contagious – particularly negative moods, such as depression (Hill, Griffiths, & House, 2015; Joiner Jr & Katz, 1999; Kelly, Iannone, & McCarty, 2016; Revenson et al., 2015). Negative mood states may be transferred due to a process known as empathetic distress. Empathetic distress is described as an individual's emotional involvement in the distressed feelings and problems of their intimate partner, to the extent that they begin to adopt the partner's distress and experience it as their own (Smith & Rose, 2011). The mood state can thereby become present, to some extent, in both members of the couple (Joiner Jr & Katz, 1999).

Because there is evidence that separately, each of sleep and affective states are linked within couples (Hill et al., 2015; Joiner Jr & Katz, 1999), and sleep is linked with affective symptoms within an individual, it is logical mental health may also influence dyadic sleep. However, unlike physical health or relationship quality, studies examining the link between mental health and dyadic sleep are scarce. Overall, findings are in the expected direction.

Greater depressive or anxiety symptoms of an individual are associated with poorer sleep outcomes in the bedpartner (Chan et al., 2017; Moturu, Khayal, Aharony, Pan, & Pentland, 2011; Revenson et al., 2015; Strawbridge et al., 2004).

One limitation of these studies is their exclusively subjective assessment of sleep using limited questions, such as average self-reported sleep duration (Moturu et al., 2011; Revenson et al., 2015) or global sleep quality (Chan et al., 2017; Strawbridge et al., 2004). Results from such studies are therefore somewhat lacking in detail, and as such, findings should not be over-interpreted. For example, Revenson et al. (2015) used self-report measures of anxiety and depression, and a single sleep duration question ("how many hours a night do you sleep on average?"). A husband's higher levels of self-reported depression and anxiety symptoms could predict a wife's shorter sleep duration at a one-year follow-up appointment. In a similar study, when couples completed daily prospective sleep diaries and mood ratings, mood was significantly poorer for partners of people who slept less than 7 hours the night before, compared to partners of people who slept more than 7 hours (Moturu et al., 2011). Both pieces of research support the notion that affective symptoms and sleep are connected within a couple, however emphasise the lack of in-depth research in the area. While subjective characterisations of mood and sleep are important, there can be substantial differences when using subjective or objective sleep data, and these differences may be meaningful. Thus, more detailed research with using a combination of subjective and objective measures will be important.

To our knowledge, no studies have assessed sleep, or characterised the impact that an individual's affective symptoms can have on their partner's sleep, using detailed, objective assessment tools. This remains an unexplored and intriguing area.

This section of the review has highlighted that factors known to influence an individual's sleep have also been recognised in light of couple's sleep, to various degrees. By far the most studied area is relationship functioning. Therefore, this thesis will focus on expanding the literature in underdeveloped areas. Additionally, there is a scarcity of research investigating sleep at the level of the dyad: most research has characterised sleep using global sleep measures of an individual, or global measures of subjective sleep quality or duration, rather than acknowledging further dyadic processes. This reinforces the notion that there is a large gap in the field's ability to assess dyadic sleep separately from (or in addition to) individual sleep.

How does presence of a sleep disorder impact on the bedpartner, as well as the patient?

Review of the risks of sharing a bed.

As has been outlined, there are risks to sleep when the decision is made to share a bed with a romantic partner. To recap, these risks can include reduced objective sleep quality, reduced sleep quantity, increased sleep disruption/fragmentation throughout the night, altered sleep architecture, and changes in sleep timing, potentially resulting in an individual sleeping outside of their ideal circadian timing, in order to accommodate a partner's preferences. Overall, if one partner experiences poor sleep, the other partner is at risk of being affected and experiencing poor sleep.

Individual objective sleep deficits and insufficient sleep quality and quantity can have flow-on effects for daytime functioning and well-being, in ways documented extensively through literature. This can include, but is not limited to, low mood, increased anxiety, cognitive difficulties, decreased immune function, increased inflammation, increased pain, hypertension, and decreased glucose tolerance (American Psychiatric Association, 2013; Buysse et al., 1994; Dew et al., 2003; Ferrie et al., 2007; Foley et al., 1995; Karimi, Hedner, Häbel, Nerman, & Grote, 2015; Ohayon, Caulet, & Lemoine, 1998; Prather, Janicki-Deverts,

Hall, & Cohen, 2015; Sivertsen, Harvey, Pallesen, & Hysing, 2015; Watson et al., 2015; Zhang & Chan, 2014).

Despite risks to sleep, even when couples are aware of potential risks or are aware of experiencing negative sleep consequences, they commonly choose to sacrifice sleep in order to derive the potential relationship benefits of bed-sharing (Hislop, 2007). It is therefore important to understand the role of a bedpartner in cases of clinical levels of sleep disruption, where a bedpartner may act as a precipitant or maintaining factor of the disorder. This has been most clearly described in the case of OSA, although a small handful of observational studies have been published investigating REM sleep behaviour disorder and Restless Legs Syndrome (RLS). The precedent for examining dyadic sleep more closely in insomnia derives from observations in these other sleep disorders.

Obstructive Sleep Apnea (OSA)

Impact on Partner's Sleep.

There is a small but growing literature studying the effects of OSA on both the patient and the bedpartner's sleep, health and relationship quality (Beninati et al., 1999; Cartwright & Knight, 1987; Henry & Rosenthal, 2013; McArdle, Kingshott, Engleman, Mackay, & Douglas, 2001; Parish & Lyng, 2003; Ulfberg, Carter, Talbäck, & Edling, 2000; Zarhin, 2016). OSA is the most common form of sleep disordered breathing and is characterised by repeated episodes of reductions or cessations in airflow during sleep. This occurs due to partial or complete collapse of the upper airway. Breathing usually only recommences upon arousal from sleep, leading to highly fragmented sleep with reductions in deep stage N3 and REM sleep every night (Lamphere et al., 1989; Quan et al., 2011; Waldhorn et al., 1990).

OSA affects the sleep of bedpartners so much that frequently it is the bedpartner who recommends the affected individual seek treatment (Henry & Rosenthal, 2013). Studies which have quantified the impact on the bedpartner have recorded the extent in different ways,

however have concluded the same point: bedpartners of individuals with OSA have their sleep compromised by this partner (Luyster, 2017). Specifically, Beninati et al. (1999) studied 10 heterosexual married couples where the husband experienced OSA (median apnea-hypopnea index = 26). Bedpartners were at least partially aroused by their bedpartner's snores a median of 9 times per hour. Separately, moderate to severe sleep disturbances due to partner snoring were reported by 69% of bedpartners (McArdle et al., 2001), while 304 female partners of heavy snorers reported they experienced sleep problems twice as frequently as a general community sample (Ulfberg et al., 2000). These sleep problems included symptoms such as difficulty starting or maintaining sleep, daytime fatigue, or sleepiness. Additionally, this sample reported the sleep they did get was less restorative than reports by the community sample (Ulfberg et al., 2000). On top of these direct sleep consequences, OSA bedpartners are more likely to use sleeping aids known to increase risk of poor health outcomes, including increased use of ear plugs, alcohol, marijuana and sleep medications in attempts to improve sleep (Luyster et al., 2016; Seidel et al., 2012).

Impact on Partner's Well-being.

OSA affects the lives of partners from psychological and medical perspectives as well as sleep. Partners have higher levels of health-related contact, take more medications, and have lower incomes than the general population, differences which become more pronounced as the disorder worsens (Jennum, Ibsen, & Kjellberg, 2014). Consequently, partners are negatively affected in terms of reduced quality of life, daytime functioning, and specific health consequences including hypertension, chronic musculoskeletal pain, and increased morning headaches (Doherty, Kiely, Lawless, & McNicholas, 2003; Jennum et al., 2014; Parish & Lyng, 2003; Seidel et al., 2012; Smith, Togeiro, Tufik, & Roizenblatt, 2009; Uloza, Tomas, Raimundas, Miliauskas, & Ĵemaitiene, 2010).

Furthermore, OSA can alter relationship dynamics within a couple. More than one quarter of couples with at least one heavy snorer reported sleeping in separate bedrooms in order to achieve better sleep (Ulfberg et al., 2000). Wives of men with heavy snoring and/or OSA reported poor marital and social/leisure adjustment, and more frequent reports of insomnia, daytime sleepiness, and fatigue than wives of non-snorers (Cartwright & Knight, 1987; Ulfberg et al., 2000). Relationship dissatisfaction can then serve to maintain or exacerbate some health problems. As an example, OSA patients are at heightened risk of sexual dysfunction (Steinke, Palm Johansen, Fridlund, & Broström, 2016). This may be caused by physical health problems in the individual, but also may be caused or exacerbated by marital conflict. Sexual dysfunction can then increase marital dissatisfaction for the patient and their partner, resulting in a feedback loop (Steinke et al., 2016).

Clearly both the individual with OSA as well as their partners have sleep impairment resulting from the disorder, as well as associated health outcomes.

Other Sleep Disorders.

Research into dyadic effects of other sleep disorders is scarce. A review of the literature revealed only a series of case reports and a case comparison questionnaire study into partner effects of REM sleep behaviour disorder (Ingravallo, Schenck, & Plazzi, 2010; Lam et al., 2016; Olson, Boeve, & Silber, 2000; Schenck, Lee, Bornemann, & Mahowald, 2009; Wing et al., 2008; Yeh & Schenck, 2004), and a single questionnaire study about Restless Legs Syndrome (Ondo, 2018).

Within REM sleep behaviour disorder, 40 patients and their partners reported on how the patient's sleep disorder influence both of their sleep, health and well-being (Lam et al., 2016). Almost all bedpartners (90%) reported the disorder negatively impacted their own sleep, and over half (62.5%) reported at least once being injured by a bedpartner during their sleep. Bedpartners had higher prevalence of anxiety, depression, and insomnia than the general

population. Despite negative effects, nearly two-thirds of the sample continued to bed-share. These findings support previous case reports of couples with REM sleep behaviour disorder which describe high prevalence of sleep-related assaults (Ingravallo et al., 2010; Olson et al., 2000; Schenck et al., 2009; Wing et al., 2008; Yeh & Schenck, 2004).

A similar pattern emerged in the single study published to date on RLS (Ondo, 2018). Almost half (41.2%) of all bedpartners reported their sleep was disturbed at least weekly, and 48.4% of partners reported their next day productivity was impaired at least weekly by the disorder. Bedpartners reported making life accommodations in fields of the way they work, sleep, travel, socialise, and schedule their days.

Insomnia.

Following OSA, insomnia is the second most common sleep disorder in Australia. It affects 10-15% of the global adult population (Bartlett et al., 2008; Ford & Kamerow, 1989; Health, 2005; Morin et al., 2011; Ohayon & Bader, 2010). However, in stark contrast to OSA, and despite some data published in less common disorders, to our knowledge there has been no empirical research conducted examining the effect of insomnia on a bedpartner.

Whilst no studies have been published presenting data in an insomnia disorder population, bedpartners of individuals who self-reported regular “sleep problems” were found to have increased risk of self-reported poor health, depressed mood, unhappiness, feeling left out, and unhappy relationships (Strawbridge et al., 2004). This indicates bedpartners of those experiencing insomnia-like symptoms may experience similar patterns of bedpartner-driven sleep disruption observed in OSA, RLS and REM sleep behaviour disorder populations, but their subjective reports of disturbances are as yet unexplored using objective measures.

A clinical review into insomnia suggested a framework by which bedpartners could be considered within insomnia treatment (Rogojanski, Carney, & Monson, 2013). Indeed, it is commonly accepted interpersonal factors are important contributors to the onset and

maintenance of insomnia. However, as demonstrated here, research supporting this assertion in terms of the bedpartner is largely lacking. Understanding dyadic influence of insomnia is an important step for more thoroughly understanding, and ultimately reducing, burden of disease and suffering. Without understanding of the role of the bedpartner in insomnia, strategies cannot be implemented in order to improve outcomes.

Objectives of this thesis

It is clear that the field of dyadic sleep is in its infancy. There is much work to be done in this area, particularly within populations with sleep disorders other than OSA. Therefore, the aim of this thesis was to further understandings of the dyadic nature of sleep, in couples with and without the sleep disorder insomnia. Within this aim, the thesis had three primary objectives.

The first objective of this thesis was to examine how bedpartners influence each other's sleep, in couples with and without insomnia. While the literature to date has utilised sleep/wake concordance to characterise the dyadic sleep experience, there are almost certainly more processes occurring which will inform our understanding of sleep. Within insomnia populations, the dyadic nature of sleep has been almost entirely ignored. Thus, the first aim was to characterise dyadic sleep processes in both samples. Parallel studies including both objective (actigraphy) and subjective (daily sleep diary) measures of sleep were designed as the most appropriate to answer this objective.

The second objective was to examine sleep-related and demographic factors relevant to understanding variability in dyadic sleep constructs, in couples with and without insomnia. Factors including chronotype, bedtime order, as well as sex or role (i.e., patient or partner), and time of night, were drawn from previous findings in the literature for characteristics which may be important for understanding effects.

Finally, the third objective was to undertake a more thorough examination of mental health symptoms (depression, anxiety) and how they predict an individual or bedpartner's sleep in both the abovementioned populations. As presented within this review of the literature, studies examining how mental health is related to dyadic sleep are scarce, despite the strong accepted understanding of the intraindividual sleep-affect relationship. This study will be the first to more comprehensively examine both sleep (individual and dyadic characteristics) and mental health in couples with and without insomnia.

Thesis organisation

This thesis uses data from two population groups in order to address the thesis objectives: couples where neither partner reports evidence of sleep disorders, and couples where one partner is seeking treatment for insomnia.

Study 1 (Paper 2/Chapter 3; under peer review in the journal *Sleep Health)* presents the results of the first and second thesis objectives; presenting data from the sample where neither couple experiences sleep disorders.

Study 2 (Paper 3/Chapter 4; accepted for publication in the journal *Sleep*) presents parallel results for the first and second thesis objectives; presenting data from the sample where one partner experiences insomnia. For the first time, dyadic sleep characteristics were mapped in a population of couples where one partner sought treatment for insomnia.

Study 3 (Paper 4/Chapter 5; submitted for peer review to the *Journal of Affective Disorders*) presents the results of the third thesis objective; including data from both samples.

The final chapter (Chapter 6) provides a general discussion of the main results, strengths and limitations of the thesis, implications for clinic, theory, and methodology and original contributions of the research, and provides suggestions for future research.

CHAPTER 3

Vulnerability and resistance to sleep disruption by a partner: A study of bed-sharing couples

Chapter 3 is a manuscript which at time of initial thesis submission was under review at *Sleep Health: Journal of the National Sleep Foundation.* While the thesis was under examination, a revised version of this manuscript was accepted for publication so is now in press. This Chapter of the final thesis comprises the earlier version of the manuscript.

Walters, E.M., Phillips, A.J.K., Boardman, J.M., Norton, P.J., & Drummond, S.P.A. (under review). Vulnerability and resistance to sleep disruption by a partner: A study of bed-sharing couples.

Foreword to Chapter 3

This chapter first conceptualises and describes the process of wake transmission, a novel metric for describing sleep in bedpartners, in bedsharing couples aged 18-72 years, then examines whether demographic and sleep-related factors affect dyadic sleep.

A note on the order of the manuscripts presented within the thesis. This thesis was conceptualised with Papers 2 and 3 forming its foundations. The papers therefore mirror one another in methodology and research questions, and both samples were used to develop and characterise concepts of wake transmission and the specific variables described. They are presented in the order in which they were conceptualised. However, due to recruitment and data collection time frames, Paper 3 was prepared and accepted for publication before Paper 2. Thus, the Introductions and Discussions read a little awkwardly when placed sequentially within the thesis. Additionally, the original drafts of each one had identical analytic strategies. Through the peer review process they each have been altered, so while are no longer identical, we still believe they are essentially parallel and are companion papers.

Conceptually, we see them as occurring alongside one another. They are intended to be taken together as a coherent piece.

This manuscript has been submitted to *Sleep Health: Journal of the National Sleep Foundation.* It has been formatted in compliance with journal requirements. At time of initial thesis submission it was under a second round of revisions which were minor. While the thesis was under examination, the thesis was accepted after a third round of revisions which again were minor. This manuscript is presented in the earlier version, which has been altered from journal requirements by changing the in-text references to APA style and in accordance with thesis examiner suggestions.

Declaration

Declaration of the Candidate: In the case of Chapter 3, the nature and extent of my contribution to the work was the following:

Nature of Contribution	Extent of Contribution
Formulation of research questions and experimental design, data collection, analysis, and writing the manuscript	60%

The following co-authors contributed to the work:

Name	Nature of Contribution
Andrew JK Phillips	Input into data analysis, consultation in formulation of experimental design, and critical review of manuscript
Johanna M Boardman	Consultation in experimental design, and critical review of manuscript
Peter J Norton	Critical review of manuscript
Sean PA Drummond	Consultation in formulation of research questions and experimental design, discussion of ideas expressed in manuscript, and critical review of manuscript.

The undersigned hereby certify that the above declaration correctly reflects the nature and extent of the candidate and co-authors' contributions to this work.

Candidate's Signature:

Primary Supervisor's Signature:

Chapter 3: Vulnerability and resistance to sleep disruption by a partner: A study of bed-sharing couples (Paper 2)

Vulnerability and Resistance to Sleep Disruption by a Partner: A Study of Bed-Sharing Couples

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Abstract

Objectives: Methods for analyzing sleep as a dyadic behaviour remain relatively unexplored. We aimed to (1) characterize how bedpartners influence each other's sleep, and (2) identify factors that predict sensitivity to wake transmission between bedpartners.

Design: Cross-sectional study.

Setting: Community members in Melbourne, Australia.

Participants: Fifty-five couples without sleep disorders, aged 18-72 years.

Measurements: Participants completed the Morningness-Eveningness Questionnaire-reduced version. Habitual sleep/wake patterns were monitored for seven nights via actigraphy and sleep diary. Epoch-by-epoch sleep/wake concordances (shared sleep/wake minutes), number of transmissions received (number of awakenings immediately preceded by bedpartner wakefulness), percent transmissions received (percentage of total awakenings that were transmissions), transmissibility (percentage of all bedpartner awakenings transmitted), and percent minutes resistant to transmission (percentage of bedpartner's wake minutes that an individual slept), were calculated. Mixed-effects modeling assessed predictors of dyadic sleep.

Results: We described rates of sleep concordance (M = 66.8±6.8%), wake concordance (M = 6.8±3.1%), number transmissions received (M = 6.0±2.7), percent transmissions received (M = 18.9±7.5%), transmissibility (M = 20.0±6.2%), and percent minutes resistant (M = 52.1±13.6%). Average couple-level percent transmissions received were highest and percent minutes resistant lowest in couples who had similar bedtime (within 30 minutes), compared to couples with greater differences in bedtime.

Conclusions: Wake transmission is a useful metric of dyadic sleep, which varies according to relative bedtimes, and chronotypes of bedpartners. Higher wake transmissions for couples with similar bedtimes suggests dyadic preferences for shared bedtimes may be due to

psychosocial benefits of shared sleep timing, rather than minimisation of bedpartner-driven sleep disruption.

Keywords: couples, concordance, wake transmission, relationships, dyad, bedpartners

Conflicts

The authors have nothing to disclose on receipt of financial support for the research, authorship, and/or publication of this article. EMW, AJKP, JMB, and PJN have no conflicts of interest to declare. The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

SPAD serves on a DSMB for Zelda Therapeutics Limited, and he has provided consulting to Jazz Pharmaceuticals.

Introduction

Sleep quality is important for physical and mental health (Ford & Kamerow, 1989; Sivertsen et al., 2012; Staner, 2003). One important yet frequently overlooked determinant of sleep quality is the presence of a bedpartner. Although most research has examined sleep as an individual behavior, sleep is a shared experience for most adults (De Vaus, 2004). Recent research has begun to consider the role of the bedpartner, documenting how adult bedsharing can be important for both sleep quality (Dittami et al., 2007; Gunn, Buysse, Hasler, Begley, & Troxel, 2015; Pankhurst & Horne, 1994; Troxel, 2010) and health (Gunn et al., 2017; Revenson, Marín-Chollom, Rundle, Wisnivesky, & Neugut, 2015; Strawbridge, Shema, & Roberts, 2004; Uchino et al., 2019).

Couples have previously been shown to coregulate levels of cortisol, blood pressure, and mood (Saxbe & Repetti, 2010; Timmons, Margolin, & Saxbe, 2015). Relatively recent work shows coregulation extends to sleep/wake states. Sleep/wake concordance has been used to describe covariation of sleep and wake states within bedsharing couples (i.e., whether individuals are both awake or asleep at the same time; Gunn et al., 2015; Gunn et al., 2017; Hasler & Troxel, 2010; Meadows, Venn, Hislop, Stanley, & Arber, 2005; Pankhurst & Horne, 1994). For example, in healthy controls, couples have been found to be in the same sleep/wake state for 75% of the night (Gunn et al., 2015), and one third of couples' night-time wrist movements occur in synchrony, suggesting a third of all wake after sleep onset is common between partners (Pankhurst & Horne, 1994). Levels of sleep/wake concordance decrease with increasing age and relationship length (Gunn et al., 2015; Pankhurst & Horne, 1994). Effects of bedsharing may also depend on sex (Dittami et al., 2007; Meadows et al., 2005; Spiegelhalder et al., 2015), with females demonstrating compromised objective sleep efficiency when cosleeping compared with sleeping alone (Dittami et al., 2007), and males demonstrating longer total sleep time when cosleeping (Spiegelhalder et al., 2015).

Preliminary research suggests directional effects of one partner physically disrupting the other. In healthy sleepers, overnight awakenings have been documented to frequently occur soon after any body movement or wake was recorded in a bedpartner (Meadows et al., 2005). In obstructive sleep apnea (OSA), noise may be more important than movement: approximately 43% of nocturnal arousals in OSA bedpartners have been found to begin immediately after a bedpartner's snore (Beninati, Harris, Herold, & Shepard Jr, 1999). Within couples where one partner experienced insomnia, Walters et al. (2020) also found directional effects in, as well as other predictors of, bedpartner influence. Overall, non-insomnia bedpartners had their sleep disrupted by their insomnia partner 1.25 times more frequently than vice versa. Some individuals, however, were more vulnerable to transmission than others. Mismatched chronotypes and shared bedtimes were risk factors for more wake transmissions, indicating sleep at a misaligned circadian time, may put couples with insomnia at risk for greater bedpartner-disturbed sleep (Randler, Barrenstein, Vollmer, Díaz-Morales, & Jankowski, 2014; Vetter, Fischer, Matera, & Roenneberg, 2015). Whether these same risk factors exist in couples without sleep disorders has not been explored.

Thus, there is growing evidence that bedpartners influence one another's sleep. Methods for analyzing sleep as a dyadic behaviour, and better understanding which individuals are at greatest risk for disruption by a bedpartner, remain relatively unexplored. The current paper examined dyadic sleep characteristics for couples where neither individual has a diagnosed sleep disorder. Specifically, this study characterized wake concordance and wake transmission in couples without sleep disorders; and identified whether characteristics (individual sex, concordant/discordant bedtime, concordant/discordant chronotype) predict wake transmission.

Participants and Methods

Participants and Procedure

Fifty-five bedsharing adult couples were recruited for participation via print media, and online advertisements. Ethical approval was obtained from Monash University Human Research Ethics Committee. Participants were stable bedpartners (i.e., shared a bed minimum 5 nights per week, for at least one month; *M* = 7.6 years, range 1 month – 46 years). Couples were excluded if either partner (a) displayed evidence of sleep disorders according to the Duke Structured Interview for Sleep Disorders (Edinger et al., 2004), (b) used any medication to assist sleep, (c) had worked night shift in the past 3 months, (d) reported evidence of ongoing intimate partner violence, or (e) in cases of recent transmeridian travel, had returned home less than 3 days per time zone travelled. Eligible participants attended a session where eligibility was confirmed, informed consent gained, and self-report assessments were completed, followed by 7 nights of at-home sleep monitoring.

Measures

The Insomnia Severity Index (ISI) is a 7-item inventory assessing presence and severity of insomnia in the past week (Morin, 1993). Scores of 10 or above were exclusionary (Morin, Belleville, Belanger, & Ivers, 2011).

The STOPBang is an 8-item inventory measuring risk of OSA (Chung et al., 2008). Scores indicating high risk were exclusionary. High risk was defined as either (a) overall score 5 or above, or (b) minimum two endorsements on STOP questions, and BMI > 35 kg/m^2 (Chung, Yang, Brown, & Liao, 2014).

The Morningness-Eveningness Questionnaire, reduced form (MEQr; Adan & Almirall, 1991) is a 5-item self-report assessing chronotype. It has good reliability with the full Morningness-Eveningness Questionnaire (Horne & Ostberg, 1976).

The Dyadic Adjustment Scale (DAS; Spanier, 1976) is a 32-item scale of relationship quality. It is currently considered the gold standard for self-reported relationship functioning, assessing domains of dyadic consensus, satisfaction, cohesion, and affectional expression.

The Communication Patterns Questionnaire (CPQ) is a 33-item self-report assessing couples' communication styles before, during, and following discussion of relationship problems (Christensen & Shenk, 1991; Crenshaw, Christensen, Baucom, Epstein, & Baucom, 2017).

Sleep Monitoring. Participants completed a prospective sleep diary for 7 nights. This included: bed time, sleep latency, number and duration of nocturnal awakenings, wake time, and rise time. From these reports, variables of total sleep time (TST) and sleep efficiency (SE; TST / total rest interval) were calculated. The rest interval from which SE was calculated started the moment the individual reported attempting sleep, and ended the moment the individual woke for the final time (Reed & Sacco, 2016).

Concurrently, participants wore an actigraph. Respironics Actiwatch Spectrum Pro and Actiware software (Respironics, Bend, OR, USA) collected and analysed data in 60-second epochs, with sleep detected at high sensitivity (wake threshold: 20 activity counts per epoch; Maskevich, Jumabhoy, Dao, Stout, & Drummond, 2017). Rest intervals were manually set each night using bed/wake times reported in diaries as guidelines, although intervals could be adjusted up to 60 minutes on either side to account for periods where the actigraph showed clear signs of sleep immediately before or after reported times (Walters et al., 2020). Actigraphs worn by each couple were initialised on the same computer, within a few minutes of one another.

Data Analysis

Calculation of Variables. Outcomes were sleep and wake concordance, and wake transmission (Walters et al., 2020) described below and presented in Figure 1. Sleep and wake

concordance were defined as the percentage of actigraphy epochs within one's own rest interval where both the individual and their bedpartner were in sleep (i.e., shared sleep epochs / total rest interval epochs) or wake (i.e., shared wake epochs / total rest interval epochs), respectively. Epochs when the bedpartner was not in bed at the start and end of the rest interval are included in the total number of epochs for the individual who is in bed, and the bedpartner was assumed to be awake when not in bed. All epochs within an individual's reported rest interval were included in the actigraphy analyses, including any times one partner was out of bed during the night. This is similar to previous studies of sleep/wake concordance (Gunn et al., 2015; Gunn et al., 2017).

Wake transmission was calculated by identifying occurrences throughout the rest interval where an individual experienced an actigraphically determined awakening – after they initiated sleep for the first time - that began when their bedpartner was awake in the preceding minute. For example, if person A is awake during the prior epoch and person B wakes up in the current epoch, A has transmitted wake to B and B has received a wake transmission from A. A transmission could occur only once per each new awakening, i.e., contiguous wake epochs only count as one transmission. Four transmission variables were calculated – three previously described in an insomnia population (Walters et al., 2020), and one novel to this study. (1) **Percent transmissions received**: the percentage of an individual's distinct awakenings in a given time interval classified as a wake transmission (i.e., total number of transmitted wakes received by individual / total awakenings in individual). (2) **Percent minutes resistant** to transmission: percentage of a bedpartner's nocturnal wake minutes during which an individual slept. (3) **Number of transmissions received**: the total number of transmission events in a given time interval. (4) **Transmissibility**: the percentage of the bedpartner's awakenings in a given time interval that were classified as wake transmissions (i.e., number wake transmissions received / total bedpartner awakenings: a measure of vulnerability to a bedpartner's

awakenings). It is worth noting the difference between variable (1) and (4) is the denominator of the equation. For (1) Percent transmissions received, the denominator is the total number of one's own awakenings, whereas for (4) Transmissibility, the denominator is the total number of the bedpartner's awakenings. All variables were calculated within the individual's own rest interval, at two levels: full-night statistics, and by hour where relevant. Percent transmissions received and percent minutes resistant were the primary outcome variables for this paper, as these variables represent the two key elements of transmission (i.e., rate transmissions occur, and amount of time resistance was demonstrated), while removing the impact of individual sleep parameters from the dyadic sleep variable calculation. Number of transmissions received and transmissibility are novel metrics used to describe the sample's dyadic sleep more comprehensively.

Two additional variables were computed: (a) Chronotype difference was determined based on MEQr category (definitely morning, moderately morning, neither type, moderately evening, or definitely evening type; Adan & Almirall (1991)). Individuals were coded as concordant chronotype category, more morning-type than bedpartner, or more evening-type than bedpartner. (b) Bedtime difference was calculated using actigraphy rest intervals on a nightly basis, with each night categorized as: individual in bed before their bedpartner, concordant bedtime (within 30 minutes), or individual in bed later than their bedpartner.

Definition of Terms

Number Transmissions Received: Number of times wake was transmitted by a bedpartner to the individual in question per night.

Percent Transmissions Received: The percentage of all awakenings by an individual that were transmitted by the bedpartner.

Transmissibility: The percentage of all awakenings in a bedpartner that were transmitted to the individual in question.

Percent Minutes Resistant to Transmission: The percentage of a bedpartner's overnight wake epochs where the individual in question was asleep.

Figure 1. Key variable definitions

Missing data. Nights where couples reported that they did not share a bed were excluded (n = 15 of 385; 3.9%). Additionally, there were two nights not available due to non-adherence. This resulted in n = 368 sleep diary nights available for relevant analyses. For actigraphy, in addition to the 15 nights where couples slept separately and the two nights without sleep diary data, which were excluded, there were six nights where actigraphy data were not available due to participant non-adherence or technical issues. This resulted in n = 364 actigraphy nights available for dyadic data analysis (94.5% of total nights).

Statistics. Data was analysed via SPSS v.24 except where otherwise specified. All data were checked for statistical assumptions. To meet the assumption of normality, one univariate outlier on transmissibility (z = 4.96) was adjusted to the next most extreme score +1 (Tabachnick & Fidell, 2013). Pearson's correlations assessed relationships between dyadic sleep and age and sleep disorder symptoms (ISI, STOPBang, diary-based sleep efficiency). Age was significantly associated with several dyadic sleep variables, and thus was used as a covariate for multi-level models. Relationship length was not also included as a covariate as it was highly correlated with age (r = .92). Bivariate correlations and t-tests where appropriate determined the association of age with other predictors. A chi-square test of independence determined whether within-couple chronotype order and bedtime order were related.

To determine whether dyadic sleep variable rates occurred at levels greater than those observed in randomly matched pairs of individuals, a bootstrap distribution of 100,000 randomly paired nights was calculated by randomly sampling nights with replacement using MATLAB R2018b (The MathWorks, Inc., Natick, MA, USA). Significant differences between true and pseudo-couples distributions were then analysed using t-tests. All p values were < .001, indicating all dyadic sleep variables occurred at levels greater than chance. Although we cannot determine whether any isolated wake transmission event was causal, this implies that wake transmission cannot overall be accounted for by chance.

To assess nightly predictors of percent transmissions received and percent minutes resistant, multi-level mixed effects modeling was conducted. Mixed models could incorporate differences by hour, night, individual, and dyad: thereby allowing interpretation for factors predicting protection or disruption of sleep for specific individuals within the dyads. Data was collected for each individual every hour into the rest interval, up to maximum 9 hours due to small sample size beyond 9 hours; and repeated for maximum 7 nights per individual. The data was structured such that time (night by hour) was fully crossed with dyad (n = 55). This structure allowed for non-independence of time measurements between partners of dyads. Dyads were modeled as indistinguishable. Dyad was entered as a random variable; time variables (Hour and Night) were entered as repeated-measures variables within individual. Intercepts were allowed to vary randomly whereas slopes were fixed for all analyses. Only dyads with both individuals' data available were selected for analysis. The models included age, sex, chronotype difference, bedtime difference, and all two-way interactions among sex, chronotype difference, bedtime difference, and hour of the rest interval as fixed effects. Model parameters were fit using a full information maximum likelihood estimation method. As we are modelling more parameters at a greater temporal resolution than prior studies, we have presented model syntax in Supplemental Figure 4a. Akaike's Information Criterion (AIC) determined the most parsimonious models. Post-hoc analyses for significant effects were conducted via *t*-tests and one-way ANOVA as appropriate.

Models with the same predictors were run with dyad-level percent transmissions received, and percent minutes resistant to transmission. As this assessed data at the level of the dyad, these models were not cross-classified (syntax presented in Supplemental Figure 4b). For the percent transmissions received model, entering night of sleep monitoring as a nesting level failed to converge; thus night of monitoring was entered as a random effect (Supplemental Figure 4c). Dyad-level variables were defined as average values of the two individual

bedpartner scores. Bedtime and chronotype differences were recoded for dyad-level models to "discordant" and "concordant" categories.

Models were also run for the additional two wake transmission variables: number of transmissions received, and transmissibility. Measuring transmission in different ways provided the same basic answer; therefore, we have included these data within the Supplement (see Supplemental Tables 1-5, Supplementary Figures 1-3), and do not refer to these in detail within the Results or Discussion.

Results

Sample Characteristics

Couples shared 91.8% (*SD* = 7.1%) of their rest interval minutes. Table 1 presents sample demographics, and individual and dyadic sleep parameters. Couples with bedtimes that differed by more than 30 minutes were older, relative to couples with bedtimes within 30 minutes (mean age of couples with mean bedtime difference more than 30 minutes = 37.9 years, mean age of couples with mean bedtime difference less than 30 minutes = 30.4 years; $p = .02$). There was no significant difference between males and females related to who was more likely to go to bed later than their bedpartners (mean bedtime difference = 8.5 minutes, $p = .11$). The association between within-couple chronotype difference and the order in which bedtime occurred compared with their bedpartner did not reach significance (e.g., if they were more evening type than their partner, this did not mean they necessarily went to bed later than their bedpartner), $X^2(4) = 8.89$, $p = .064$, $V = .08$ (small effect).

Table 1.
Description of Sample: Demographics, Individual and Dyadic Sleep Parameters (n = 55 couples).

Variable	M ± SD
Sex (n)	56 female (50.9%)
Age (y)	32.3 ± 12.35 Range: 18 – 72
Duration of Bed-sharing Relationship (y)	7.6 ± 10.2
Nature of Relationship	
Married (number of couples)	27
De Facto (number of couples)	12
Dating (number of couples)	16
Presence of children under 18 years in household (%)	23.6
Presence of pets in household (%)	31.8
Dyadic Adjustment Scales (total)	123.19 ± 12.96
Communication Patterns	
Constructive Communication	65.17 ± 10.46
Self-Demand / Partner-Withdraw	17.00 ± 8.44
Partner-Demand / Self-Withdraw	16.48 ± 8.25
Insomnia Severity Index	3.90 ± 2.50
STOPBang	1.23 ± 0.95
Sleep Diary	**M ± SD**
Sleep Efficiency (%)	95.04 ± 3.72
Sleep Latency (mins)	13.75 ± 8.88
Total Sleep Time (mins)	459.59 ±49.21
Wake After Sleep Onset (mins)	9.68 ± 13.99
Number of nocturnal awakenings (n)	1.44 ± 0.95
Actigraphy	**M ± SD**
Sleep Efficiency (%)	79.05 ± 5.70
Sleep Latency (mins)	13.55 ± 8.55
Total Sleep Time (mins)	387.23 ± 44.53
Wake After Sleep Onset (mins)	74.69 ± 22.77
Dyadic Sleep Variables	**Individual/Transmission to Individual (M ± SD)**
Concordance	
Sleep (%)	66.79 ± 6.80
Wake (%)	6.84 ± 3.06
Combined (%)	73.63 ± 7.57
Number Transmissions Received (n)	6.00 ± 2.72
Percent Transmissions Received (%)	18.86 ± 7.54
Transmissibility (%)	19.97 ± 6.15
Percent Minutes Resistant (% of bedpartners wake minutes)	52.11 ± 13.62

Concordance

Wake concordance was negatively correlated with age of the individual (p <.001) and percent minutes resistant to transmission (p < .001), and was positively correlated with percent transmissions received (p = .001), number of transmissions received (p < .001) and transmissibility (p < .001; Table 2). Sleep concordance was negatively correlated with transmissibility (p = .024) and percent minutes resistant to transmission (p = .049). Sleep concordance was positively associated with sleep diary-reported SE (p = .009). See Table 2.

Transmission

Number of transmissions received and transmissibility were each negatively correlated with age of the individual (p = .001, p = .008 respectively), and percent minutes resistant to transmission was positively correlated with age (p < .001). See Table 2. There was a negative within-couple correlation between each partner's percent transmissions received (p < .001), indicating wake was frequently transmitted unidirectionally within a couple. Percent transmissions received was positively correlated with number transmissions received (p < .001), transmissibility (p < .001), and percent minutes resistant (p = .005). Number transmissions received and transmissibility were also positively correlated (p < .001). Percent minutes resistant to transmission was positively correlated with STOPBang scores (p = .009). See Table 2.

Table 2.

Within-person bivariate correlations for dyadic sleep measures (concordance, transmission), age, and sleep disorder symptoms (ISI, STOPBang, Diary-based SE). Within-person values are displayed below the diagonal. Between-person (i.e., between members of the same dyad) correlations for each variable are displayed in italics along the diagonal. Results presented are r (p).

	Age	1.	2.	3.	4.	5.	6.	7.	8.	9.
Age	***.96*** ***(<.001)***									
1. Sleep Concordance	-.06 (.53)	***.61*** ***(<.001)***								
2. Wake Concordance	**-.60** **(<.001)**	.04 (.66)	***.97*** ***(<.001)***							
3. Number Transmissions Received	**-.31** **(.001)**	-.16 (.096)	**.55** **(<.001)**	*.25* *(.062)*						
4. Percent Transmissions Received	-.12 (.20)	-.16 (.10)	**.31** **(.001)**	**.80** **(<.001)**	***-.52*** ***(<.001)***					
5. Transmissibility	**-.25 (.008)**	**-22** **(.024)**	**.41** **(<.001)**	**.81** **(<.001)**	**.70** **(<.001)**	*-.22* *(.11)*				
6. Percent Minutes Resistant	**.52** **(<.001)**	**-.19** **(.049)**	**-.68** **(<.001)**	.02 (.81)	**.27** **(.005)**	-.07 (.45)	*.23* *(.095)*			
7. ISI	.02 (.85)	-.04 (.72)	.02 (.82)	.09 (.38)	.01 (.89)	.12 (.21)	-.01 (.88)	*.15* *(.29)*		
8. STOPBang	**.41** **(<.001)**	.13 (.16)	-.17 (.082)	-.08 (.42)	.06 (.54)	-.03 (.74)	**.25** **(.009)**	.09 (.36)	***.37*** ***(.006)***	
9. Sleep Diary SE	**-.22** **(.023)**	**.25** **(.009)**	.11 (.24)	.07 (.50)	.02 (.80)	.09 (.38)	-.09 (.33)	**-.22** **(.021)**	.01 (.95)	*.17* *(.23)*

Significant results are displayed in bold. ISI = Insomnia Severity Index. SE = Sleep Efficiency.

Predictors of Wake Transmission

Percent Transmissions Received. The most parsimonious model predicting percent transmissions received at the level of the individual included variables of the chronotype difference by bedtime difference interaction ($p < .001$; Figure 2a), the bedtime difference by hour across the rest interval interaction ($p < .001$; Figure 2b), the sex by hour across the rest interval interaction ($p = .048$; Figure 2c), and the bedtime difference by sex interaction ($p = .071$). This model did not perform significantly worse than the full model, $X^2(4) = 7.19$, $p = .130$ and thus was adopted as the final model. The chronotype by bedtime difference interaction revealed percent transmissions was highest for individuals who are more evening type and have concordant bedtime with their bedpartners ($\beta = 4.6\%$, $p < .001$). The bedtime difference by hour of the rest interval interaction showed smallest percent transmissions at the start of the rest interval was experienced by those with earlier bedtimes (hour 1, $p < .001$), whilst at the end of the rest interval, those with later bedtimes had smallest percent transmissions (hours 7, $p = .039$; and 8, $p = .021$). The sex by hour of the rest interval interaction showed females had lower percent transmissions than males at hour 5 ($p = .017$) and 8 ($p = .005$).

The most parsimonious model predicting average percent transmissions received at the level of the dyad included variables of bedtime difference ($p < .001$), and the bedtime difference by hour interaction ($p = .003$; Figure 3a). This model did not perform significantly worse than the full model, $X^2(3) = 1.43$, $p = .70$ and thus was adopted as the final model. Dyad-level percent transmissions was found to be higher for dyads with concordant than discordant bedtimes ($\beta = 5.8\%$, $p < .001$). The bedtime difference by hour interaction showed concordant bedtimes had higher dyad-level percent transmissions at hour 1 ($p < .001$), 2 ($p = .012$), 5 ($p = .023$), 7 ($p = .003$), and 8 ($p = .001$).

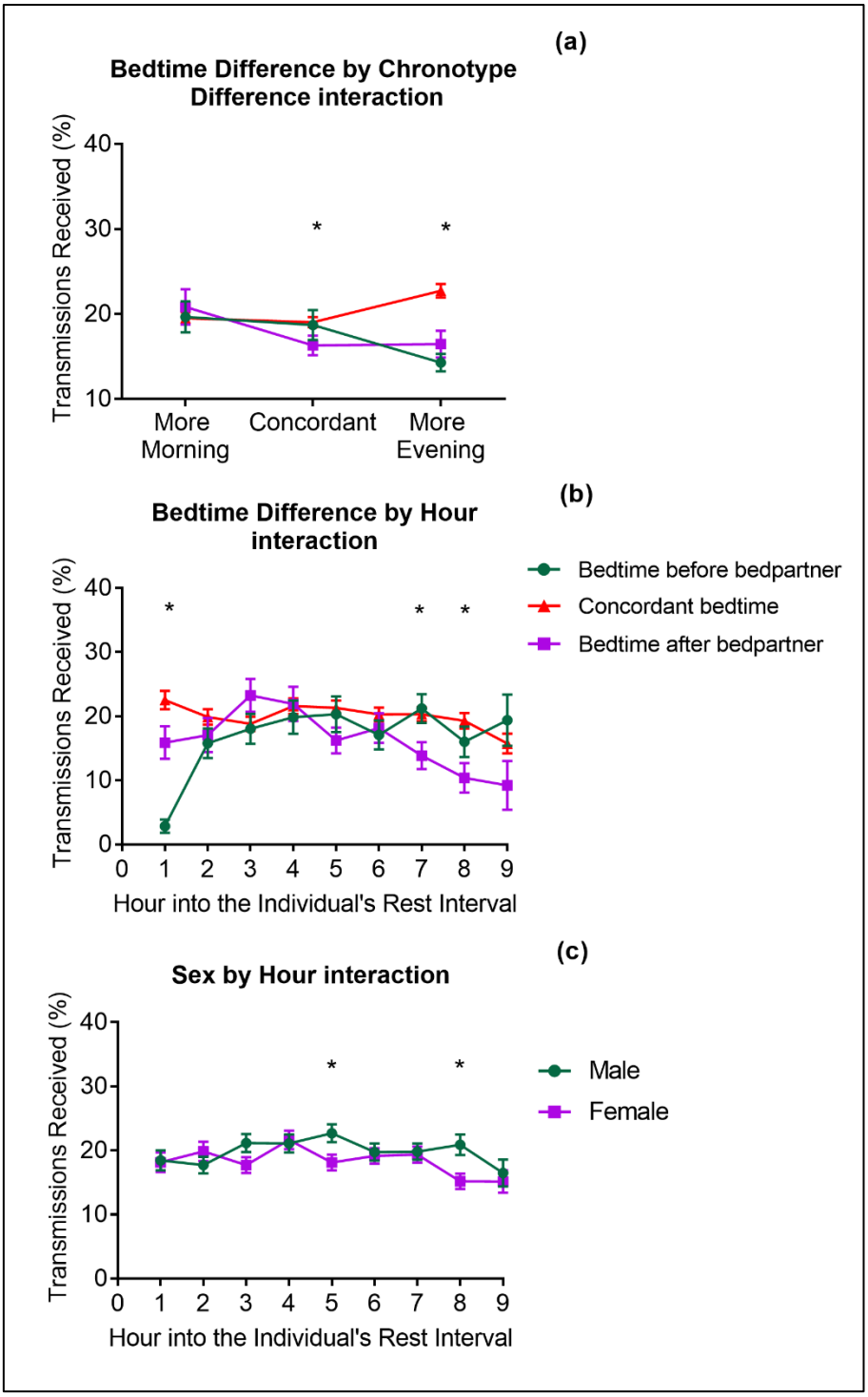

Figure 2. Multi-level modelling significant predictors of percent transmissions received, at the level of the individual, across the rest interval: (a) Bedtime difference by Chronotype difference interaction; (b) Bedtime difference by Hour interaction; (c) Sex by Hour interaction. Post-hoc one-way ANOVA and *t*-tests determined significant differences. Graphs display M ± SEM. * $p < .05$.

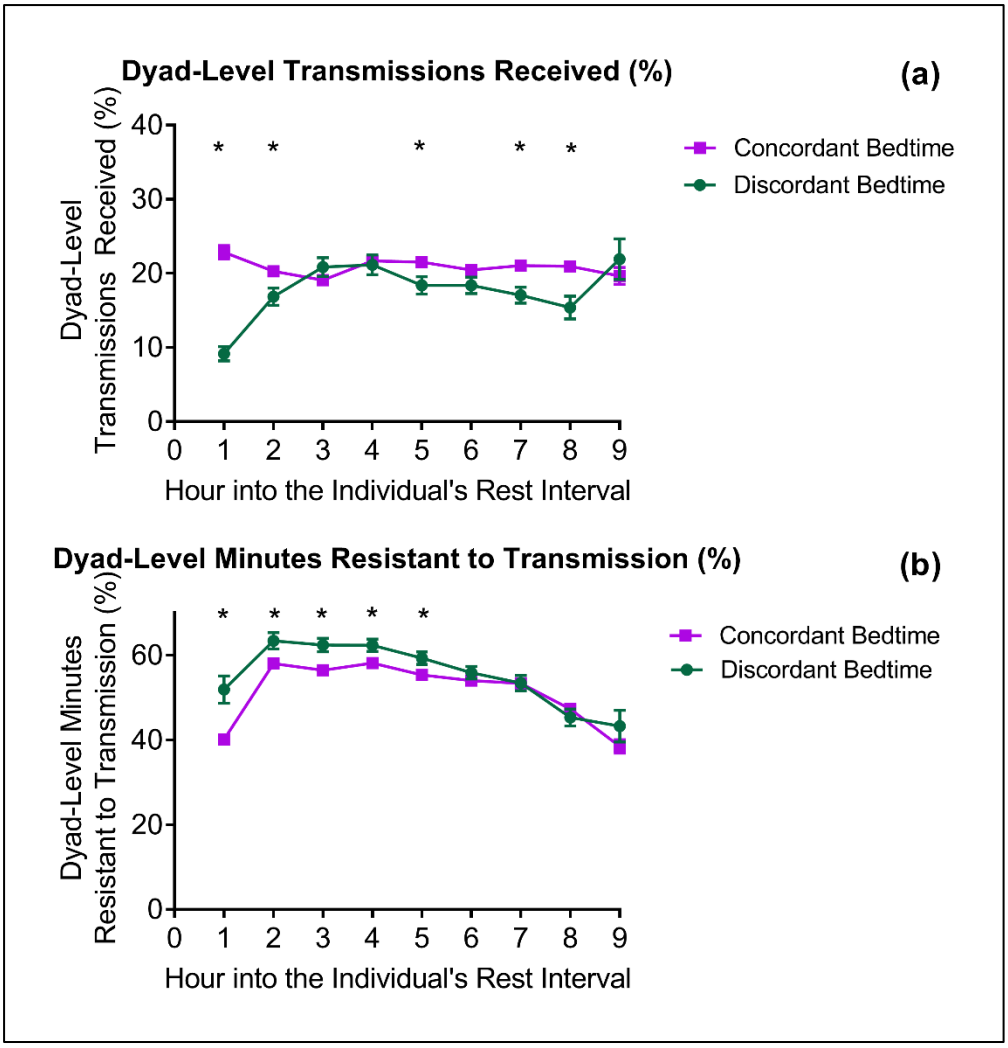

Figure 3. Multi-level modelling significant predictors of dyadic sleep variables across the rest interval, at the level of the couple: Bedtime difference by Hour interaction. (a) Average Dyad-Level Percent Transmissions Received, (b) Average Dyad-Level Percent Minutes Resistant to Transmission. Post-hoc *t*-tests determined significant differences between groups at Hours displayed. Graph displays M ± SEM. *p < .05.

Percent Minutes Resistant to Transmission. The most parsimonious model predicting percent minutes resistant at the level of the individual included variables of chronotype difference (p < .001; Figure 4a), bedtime difference (p < .001), the bedtime difference by hour across the rest interval interaction (p <.001; Figure 4b), and the sex by hour interaction (p < .001; Figure 4c). This did not significantly worsen the full model, $X^2(11)$ = 10.58, p = .48 and thus was adopted as the final model. Percent of minutes resistant to transmission was found to be highest in individuals who were more evening type than their

partner (more evening/shared chronotype: β = 5.5%; more evening/more morning: β = 5.1%). The significant interaction between bedtime difference and hour of the rest interval showed percent minutes resistant was significantly lower for those with concordant bedtimes than those with earlier bedtimes at hours 1 (p = .001), 3, (p = .023), and 5 (p = .017), but those with later bedtimes had lower percent minutes resistant than both other groups at hour 7 (p = .011) and 8 (p =.028). The significant interaction between sex and hour of the rest interval found males experienced lowest percent minutes resistant at the end (hours 8, p = .012; and 9, p = .002) of the rest interval.

The most parsimonious model predicting average percent minutes resistant to transmission at the level of the dyad included variables of bedtime difference (p < .001), and the bedtime difference by hour interaction (p < .001; Figure 3b). This model did not perform significantly worse than the full model, $X^2(3) = 1.02$, p = .80 and thus was adopted as the final model. Dyad-level percent minutes resistant was found to be lower for dyads with concordant than discordant bedtimes (β = 8.7%, p < .001), and the interaction showed this was particularly true at hours 1 (p < .001), 2 (p = .010), 3 (p = .002), 4 (p = .020), and 5 (p = .028).

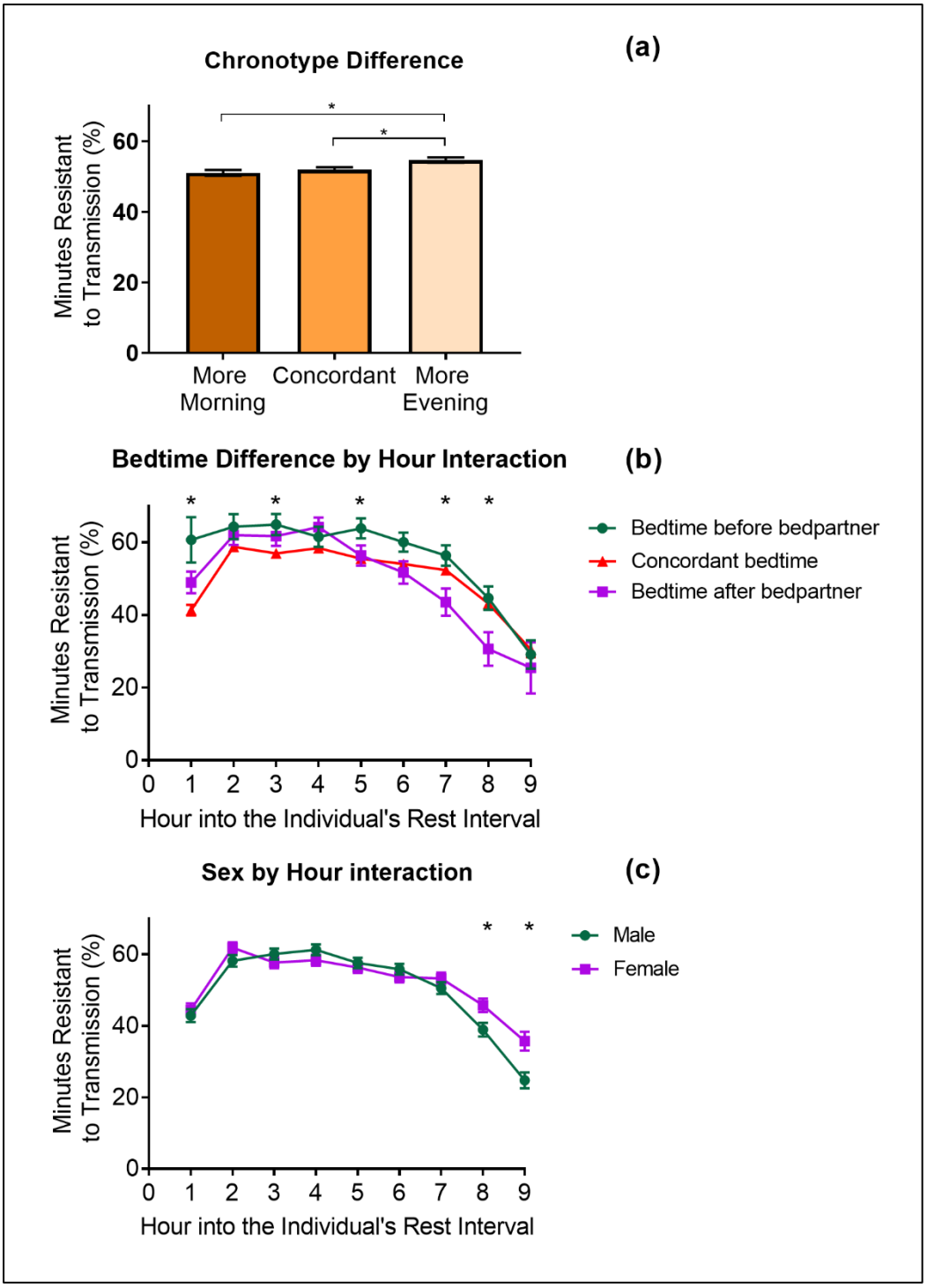

Figure 4. Multi-level modelling significant predictors of Percent Minutes Resistant to Transmission, at the level of the individual, across the rest interval: (a) within-couple Chronotype difference; (b) Bedtime difference by Hour interaction; (c) Sex by Hour interaction. Post-hoc one-way ANOVA and *t*-tests determined significant differences. Graphs display M ± SEM. *p < .05.

Discussion

This study described dyadic sleep and wake concordance, wake transmission (i.e., waking in sequential minutes after a bedpartner has awoken), and factors that predicted vulnerability to transmission in bedsharing couples without sleep disorders. Our results provide evidence that concordance and transmission are relevant to understanding the dyadic sleep process in the healthy population, and occur at levels greater than can be accounted for by chance. Specifically, having a shared bedtime predicted the highest rate of wake transmissions and the lowest percent minutes resistant to transmission, particularly at the beginning of rest intervals. Having a more evening chronotype than a bedpartner, on the other hand, was associated with the highest percent minutes resistant to transmission. Findings demonstrate the complexity of dyadic sleep in couples without sleep disorders.

Our findings add to previously published data outlining the importance of concordance in healthy sleeping populations. The present data gave similar rates of combined nightly sleep/wake concordance (74%) as previous estimates (75%; Gunn et al. (2015)). The data also extend and provide further context for dyadic sleep parameters published within an insomnia population (Walters et al., 2020). It was hypothesised non-disordered sleepers would have lower transmission rates than insomnia sleepers. This was not the observed pattern. The present sample had similar levels of percent transmissions received as individuals with insomnia (18.9% vs 18.7%; Walters et al. (2020)), and slightly lower than partners of individuals with insomnia (23.6%; Walters et al. (2020)). This is interesting, because it has been suggested that sleep disturbance caused by a bedpartner could precipitate or maintain sleep disorders (Rogojanski, Carney, & Monson, 2013). Present data, combined with those published in the earlier insomnia study, suggest there may be factors in addition to frequency of wake transmissions that puts an individual at risk for clinically relevant sleep disruption. For example, it has been argued that it is the perception of wake occurring that contributes to

clinically relevant sleep disruption (Bianchi, Williams, McKinney, & Ellenbogen, 2013). If this were true, it would suggest there are cognitive components related to nocturnal awakenings in individuals with insomnia that may contribute to the initiation or maintenance of insomnia (Harvey, 2002). There may also be other reasons rates did not differ between insomnia and non-sleep-disordered couples. For example, parallels can be drawn between event-related potential (ERP) data and the present research. Within ERP research, attenuation of responses occurs to self-induced noises (Horvath, 2015; Luck, 2014). That is, the brain does not react to self-induced noises the way it reacts to noises produced externally. This same principle may extend to self and bedpartner noise and movement, and provide a hypothesis for why we do not see elevated rates of wake transmission in insomnia couples: an individual may have habituated to the bedpartner, thus not responding to their movement, even when the brain experiences hyperarousal to other sensory stimuli. This hypothesis will need to be specifically tested in future research combining both sleep disordered and non-sleep-disordered participants.

The present findings have implications for the importance many couples place on a shared bedtime (Arber, Hislop, Bote, & Meadows, 2007; Rosenblatt, 2006). Given this common preference, we expected shared bedtime would be optimal for minimizing wake transmission rates and maximizing percent minutes resistant to transmission. Our results did not show this. In fact, we found the exact opposite: discordant bedtimes were optimal for lowest transmissions received and greatest percent minutes resistant to transmission (Figure 2b, 3a-b, 4b). Thus, benefits derived from shared bedtimes may relate more to the social aspects of this shared activity (e.g., moments of emotional or physical connection; Rosenblatt, 2006), rather than benefit conferred to sleep quality. Bedtime order may be even more relevant in sleep disorders, particularly cases where an individual has difficulty recommencing sleep after a transmission has occurred (e.g., sleep maintenance insomnia). It may also be important to

consider the possibility of discrepancies between individual's intended and actual sleep times, as reports may be less accurate if couples use this time to achieve relationship goals, losing track of time. This is something future researchers may wish to monitor on a couple-specific sleep diary.

There were two initially counterintuitive findings, here. First, the greater proportion of an individual's wake episodes that were transmitted to them (higher percent transmission), the greater number of partner wake minutes the individual slept through (greater percent resistance; Table 2, $r = .27$, $p = .005$). This might suggest individuals vulnerable to being awoken by their bedpartner were also able to fall back to sleep quickly. If these awakenings were sufficiently short they did not remember them the next day, it would be consistent with the low subjective WASO observed. Second, chronotype difference predicted percent transmissions received (Figure 2a) differently than it predicted percent minutes resistant to transmission (Figure 4a). The highest percent transmissions received were observed in later-chronotype partners, on nights when couples had concordant bedtimes. Simultaneously, later-chronotype partners spent a greater percent of minutes resisting transmission than other partners. This may be understood by considering the correlation between chronotype and circadian rhythm (Natale, Esposito, Martoni, & Fabbri, 2006). Increased transmissions may occur early in the night for later-chronotype partners, perhaps because the chosen bedtime is more appropriate for the earlier-chronotype partner. In the latter half of the rest interval, the earlier-chronotype partner may experience more WASO, during which time the later-type partner may be in a more optimal sleep phase, and thus demonstrate more minutes resistant to transmission. Overall, however, the different prediction patterns of higher rates of transmissions received in later-chronotype partners, and lower percent minutes resistant in earlier-chronotype partners, supports previous findings suggesting mild benefit of matched chronotype for partners (Larson, Crane, & Smith, 1991; Randler et al., 2014). For this sample, mutual benefit of matched chronotypes represents

a compromise on each variable rather than a superior outcome for both partners. It is worth noting that extreme chronotypes are uncommon in the population (Fischer, Lombardi, Marucci-Wellman, & Roenneberg, 2017) and so significant discordances between couples are also rare.

There is a large discrepancy between subjective (mean diary WASO = 9.7 min) and objective (mean actigraphy WASO = 74.4 min) measures of sleep. Some of this may be accounted for by some sleep misperception and underreporting of brief awakenings. Previous studies have found a greatly elevated WASO in actigraphy than diaries in adolescents (Short, Gradisar, Lack, Wright, & Carskadon, 2012), which we may be observing in this slightly older population as well. Actigraphy in individuals with more motor activity while asleep can underestimate sleep compared with PSG (Johnson et al., 2007). This may be especially true when using actigraphy on high sensitivity mode. This has implications for the interpretation of wake transmission results. The present results may indicate that where there is movement overnight, regardless of whether these are true awakenings or moments of greater motor activity while asleep, this activity is frequently transmitted to a bedpartner. This emphasizes the need for replication of present results.

A note on technical decisions in defining wake transmission. No assessments of transmission with further lag time were made (e.g., of labelling a transmission when there was wake recorded in the bedpartner two, three, or more epochs prior to wake in an individual). This was for the reason of wishing to reduce noise in the data. While it would be interesting to examine whether transmissions could occur with several minutes' lag time between bedpartner wake, this introduces a range of other reasons for wakefulness that are unrelated to the bedpartner, including spontaneous awakenings or due to an unrelated environmental factor. Along this same theoretical rationale, a transmission was not coded when both partners recorded the beginning of a wake bout on the identical minute. This is because there is a high likelihood of sleep being disrupted simultaneously in both partners by an extraneous third

factor (e.g., a car alarm sounding, a child entering the room). Future research using PSG may investigate this in more detail, and may be able to assess wake transmission which results in quiet, unmoving wakefulness.

This study had a number of strengths, including objective sleep recordings, with multiple nights per couple. However, there are also important limitations to note. First, our objective measure of sleep was actigraphy, which can overestimate sleep during periods of quiet wakefulness, which could lead to underestimation of wake transmission. While this was controlled for via use of high sensitivity mode for wake detection (Kushida et al., 2001; Maskevich et al., 2017), actigraphy still lacks the accuracy of gold-standard polysomnography, and may conversely enhance overestimation of wakefulness during periods of motor activity during sleep. Second, while we showed dyadic variables occurred at levels greater than chance, there is the possibility couples with concordant bedtimes are more likely to wake at similar times through the night by virtue of being at similar stages in their sleep cycles. This is an alternate hypothesis which will require further research using polysomnography. Third, our cross-sectional design means we do not know how participants may sleep without a partner, or how dyadic sleep changes with age or relationship length. Fourth, couples who participated in this study all scored highly on measures of relationship satisfaction, thus due to lack of variability, we were unable to control for this factor or examine differences in couples reporting different levels of relationship satisfaction. Results may differ with a broader range of relationship quality. Finally, effect sizes for all analyses were modest, so we had a potential lack of power for testing variables which varied only at the individual or dyad level. There are almost certainly additional biopsychosocial factors that contribute to understanding wake transmission (for example, mental/physical health of each partner, relationship factors, presence and ages of children, length of the sleep opportunity, individual sleep factors such as

total sleep time or sleep architecture, and environment), which future research could benefit from considering.

Conclusions

This study has provided an in-depth analysis of bedpartner sleep in a community sample. There was no clear marker of vulnerability to sleep disruption based on within-couple order of bedtimes or chronotypes, underscoring the importance of considering multiple factors to optimize sleep outcomes for both individuals and couples. These data showed similarities to a similar analysis in an insomnia sample, indicating that wake transmission is a general phenomenon, common to both sleep-disordered and healthy couples, which is currently not routinely considered in research or clinics. Given the importance of both sleep and relationships to health and wellbeing of both self and bedpartner, it is vital to consider their intersection. Future studies should examine outcomes associated with variability in dyadic sleep, and consider whether increased bedpartner influence on sleep is beneficial or harmful to the holistic functioning of an individual.

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Walters, E. M., Phillips, A.J.K., Mellor, A., Hamill, K., Jenkins, M. M., Norton, P., J., Baucom, D. H., & Drummond, S. P. A. (2020). Sleep and Wake are Shared and Transmitted between Individuals with Insomnia and their Bed-Sharing Partners. *Sleep, 43(1):1-12*. doi: 10.1093/sleep/zsz206.

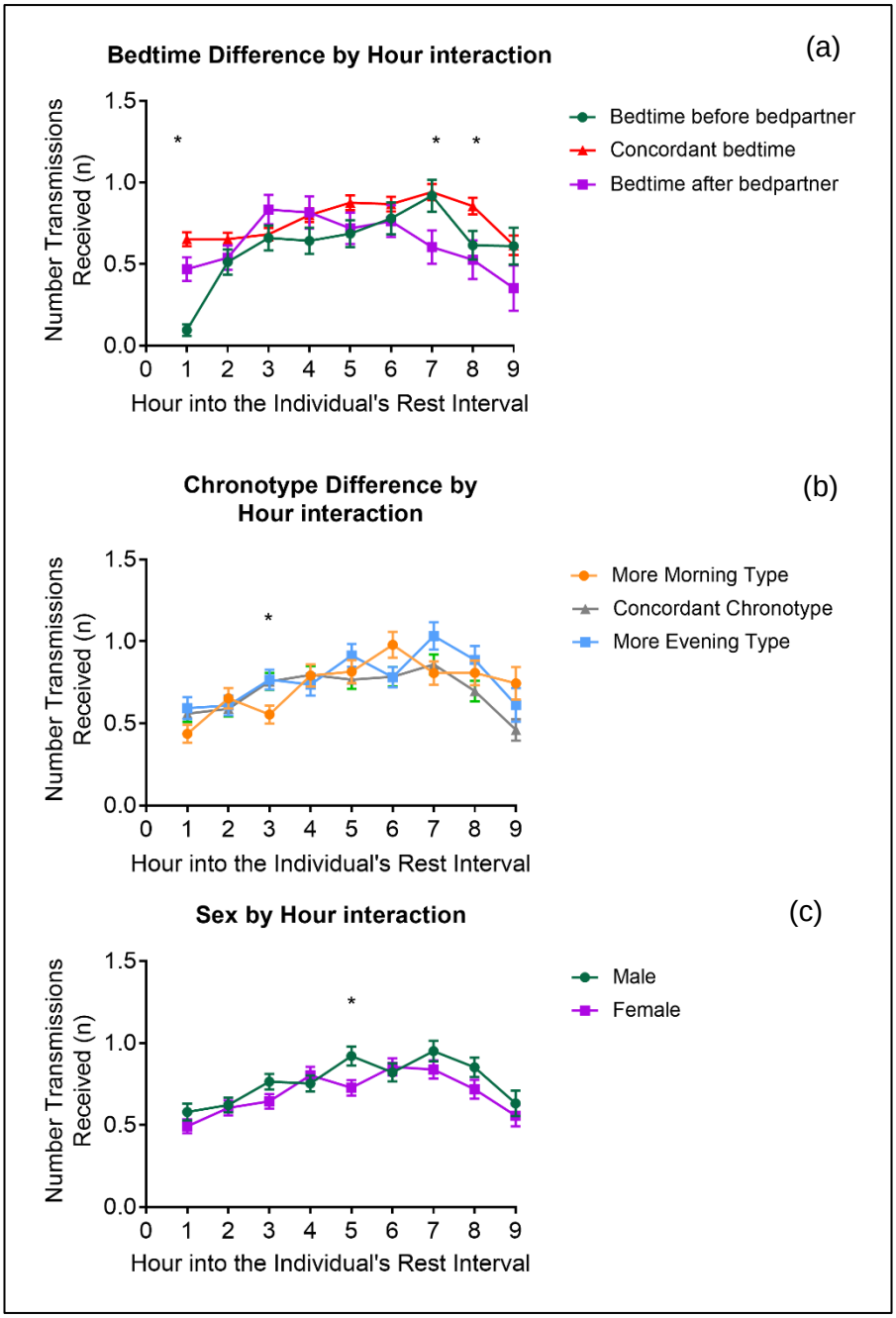
Supplemental Material for Paper 2

Supplemental Table 1.
Predicting number wake transmissions received at the individual level, by within-couple Bedtime difference, Chronotype difference, Sex, and Hour of the rest interval.

Parameters	Transmissions Received β	*df*	[95% CI]	*p*	f^2
Fixed Effects					
Bedtime Difference (Earlier Bedtime)[a]	-0.28	2928.6	[-0.43, -0.13]	<.001	.003
Bedtime Difference (Later Bedtime)[a]	0.05	2973.9	[-0.11, 0.20]	.55	
Bedtime Difference (Earlier Bedtime) by Hour	0.04	3245.9	[-0.01, 0.07]	.002	.003
Bedtime Difference (Later Bedtime) by Hour	-0.02	3256.0	[-0.05, 0.01]	.16	
Bedtime Difference (Concordant Bedtime) by Hour	0.003	3203.3	[-0.01, 0.02]	.68	
More Morning Type [b] by Hour	0.02	1871.9	[0.01, 0.04]	.002	.005
More Evening Type [b] by Hour	0.02	2158.9	[0.01, 0.03]	.006	
Sex (male)[c] by Hour	0.02	2229.5	[0.01, 0.03]	.001	.005
Random Effects					
Intercept (Dyad)	0.04		[0.02, 0.06]	<.001	
Repeated Measures AR1 diagonal	0.87		[0.84, 0.90]	<.001	
AR1 rho	0.06		[0.04, 0.09]	<.001	
-2 Log Likelihood	15940.9				
AIC	15966.7				
No. of Observations	5891				
No. of Participants	55				

*p <.05, **p <.01, ***p<.001. Covariate entered: Age. [a] = Reference group: Concordant bedtime (within 30 mins), [b] = Reference group: Concordant chronotype classification, [c] = Reference group: Female

Effect size reference:
Lorah, J. (2018). Effect size measures for multilevel models: Definition, interpretation, and TIMSS example. *Large-Scale Assessments in Education*, *6*(1), 8.



Supplemental Figure 1. Multi-level modelling significant predictors of number of wake transmissions received across the rest interval: (a) Bedtime difference by Hour interaction, (b) Chronotype difference by Hour interaction, and (c) Sex by Hour interaction. Post-hoc one-way ANOVA and *t*-tests determined significant differences. Graphs display M ± SEM. * $p < .05$.

Supplemental Table 2.

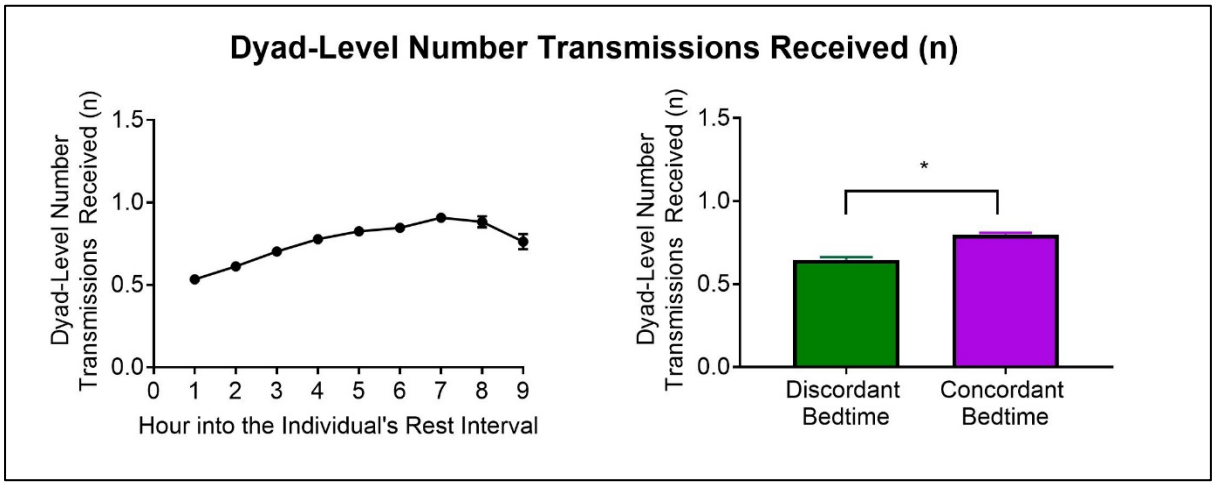
Multi-level model effects predicting average Number Transmissions Received, Percent Transmissions Received, Transmissibility, and Percent Minutes Resistant to Transmission at the level of the dyad, by dyadic Bedtime difference and Hour of the rest interval.

Variable	Number Transmissions Received+			Percent Transmissions Received+			Transmissibility#			Percent Minutes Resistant to Transmission		
Parameters	β	*df*	95% CI	β	*df*	95% CI	β	*df*	95% CI	β	*df*	95% CI
Fixed Effects												
Bedtime Difference (Discordant)[a]	-0.08	3611.6	[-0.13, -0.03]	-0.06	5030.3	[-0.08, -0.04]	N/A			0.09	5175.3	[0.06, 0.12]
p	.001			<.001						<.001		
f^2	.002			.002						.009		
Hour	.04	5567.1	[0.03, 0.05]	N/A			N/A			N/A		
p	<.001											
f^2	.02											
Bedtime Difference (Discordant) [a] by Hour	N/A			0.006	5421.9	[0.002, 0.009]	N/A			-0.02	5160.1	[-0.02, -0.01]
p				.001						<.001		
f^2				.004						.006		
Bedtime Difference (Concordant) by Hour	N/A			-0.001	5403.3	[-0.003, 0.001]	N/A			-0.00	5154.2	[-0.01, 0.00]
p				.37						.13		
Random Effects												
Intercept (Dyad)	N/A		N/A	N/A			0.00		[0.000,	0.01		[0.006, 0.014]
p							<.001		0.002]	<.001		
Intercept (Night)	N/A		N/A	N/A			0.00		[0.000,	0.00		[0.000, 0.001]
p							.81		0.018]	.59		
Intercept + Night (Dyad)							N/A			N/A		
UN(1,1)	0.07		[0.04, 0.11]	0.002		[0.000, 0.003]						
p	<.001			.004								
UN (2,1)	-0.01		[-0.01, -0.00]	-0.000		[-0.001, -0.00]						
p	.005			.031								
UN (2,2)	0.002		[0.001, 0.004]	0.000		[0.0000, 0.0002]						
p	.001			.006								
-2 Log Likelihood	11085			-			-3799.9			-1863.9		
	.3			4370.1								
AIC	11101			-			-3791.9			-1847.9		
	.3			4352.1								
No. of Observations	5648			5458			5106			5210		
No. of Participants	55			55			55			55		

*p <.05, **p <.01, ***p<.001. Covariate entered: Age. [a] = Reference group: Concordant bedtime (within 30 mins). N/A= Parameter not included in final model.

#The full transmissibility model did not improve predictive ability above that of the intercepts-only model. Therefore intercepts-only model results displayed.

+ These models did not display adequate fit with night of sleep monitoring entered as a nesting level; thus night was entered as a random effect.



Supplemental Figure 2. Multi-level modelling significant predictors of dyadic sleep variables across the rest interval: Hour, and Bedtime difference, predicting average Dyad-Level Number Transmissions Received. Graph displays M ± SEM. *p < .05.

Supplemental Table 3.
Predicting percent wake transmissions received at the level of the individual, by dyadic Bedtime difference, Chronotype difference, Sex, and Hour of the rest interval.

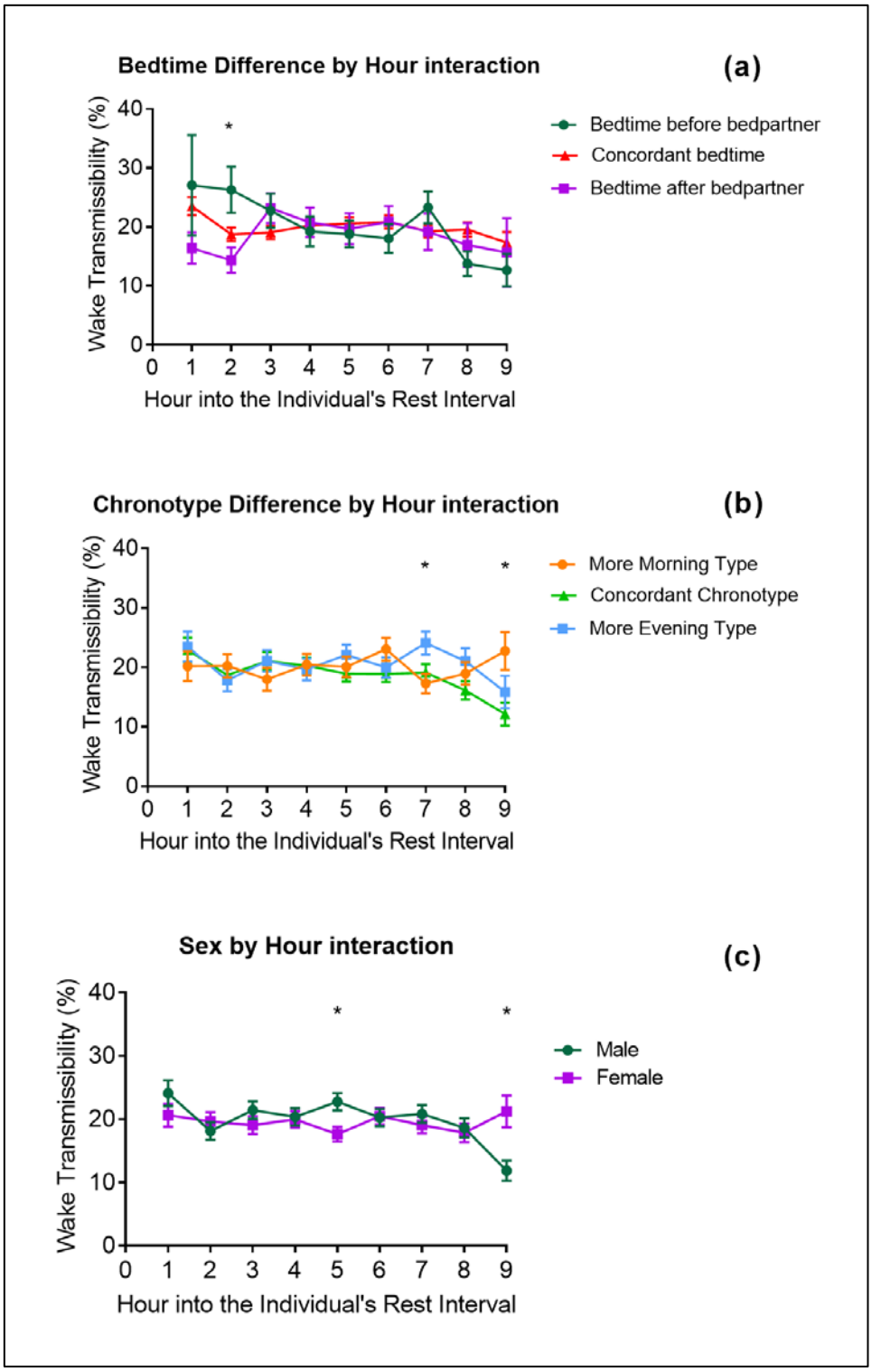
Parameters	Percent Transmissions Received β	*df*	[95% CI]	*p*	f^2 *for each predictor*
Fixed Effects					
More Evening Type [b] by Earlier Bedtime	-0.11	2197.9	[-0.18, -0.05]	<.001	.04
More Evening Type [b] by Later Bedtime	0.01	2293.2	[-0.05, 0.07]	.75	
More Evening Type [b] by Concordant Bedtime	0.05	1153.2	[0.02, 0.07]	<.001	
More Morning Type [b] by Earlier Bedtime	-0.07	2083.1	[-0.13, -0.02]	.011	
More Morning Type [b] by Later Bedtime	0.04	2314.2	[-0.02, 0.10]	.18	
More Morning Type [b] by Concordant Bedtime	0.01	617.7	[-0.01, 0.04]	.29	
Bedtime Difference (Earlier Bedtime) by Sex (male) [c]	0.04	2017.1	[0.01, 0.09]	.13	.01
Bedtime Difference (Later Bedtime) by Sex (male) [c]	-0.02	2074.0	[-0.06, 0.03]	.41	
Bedtime Difference (Concordant Bedtime) by Sex (male) [c]	-0.02	3036.5	[-0.05, 0.01]	.21	
Bedtime Difference (Earlier Bedtime) by Hour	0.01	3331.4	[0.00, 0.02]	.004	.01
Bedtime Difference (Later Bedtime) by Hour	-0.01	3308.1	[-0.02, -0.004]	.005	
Bedtime Difference (Concordant Bedtime) by Hour	-0.007	3659.0	[-0.01, -0.002]	.003	
Sex (male)[c] by Hour	0.006	3684.8	[0.00, 0.01]	.048	.005
Random Effects					
Intercept (Dyad)	0.001		[0.0003, 0.001]	.015	
Repeated Measures AR1	0.06		[0.06, 0.07]	<.001	
diagonal	0.07		[0.05, 0.10]	<.001	
AR1 rho					
-2 Log Likelihood	483.5				
AIC	523.5				
No. of Observations	5798				
No. of Participants	55				

*p <.05, **p <.01, ***p<.001. Covariate entered: Age. [a] = Reference group: Concordant bedtime (within 30 mins), [b] = Reference group: Concordant chronotype classification, [c] = Reference group: Female

Supplemental Table 4.
Predicting transmissibility at the individual level, by dyadic Bedtime difference, Chronotype difference, Sex, and Hour of the rest interval.

Parameters	Transmissibility β	*df*	[95% CI]	*p*	f^2 *for each predictor*
Fixed Effects					
Bedtime Difference (Earlier Bedtime)[a]	0.07	2702.1	[0.02, 0.12]	.007	<.001
Bedtime Difference (Later Bedtime)[a]	-0.03	2730.1	[-0.08, 0.01]	.14	
Sex [c]	0.04	2994.7	[0.01, 0.07]	.014	<.001
Bedtime Difference (Earlier Bedtime) by Hour	-0.01	2966.4	[-0.02, -0.004]	.004	<.001
Bedtime Difference (Later Bedtime) by Hour	0.005	3031.5	[-0.003, 0.01]	.19	
Bedtime Difference (Concordant Bedtime) by Hour	-0.000	3099.6	[-0.005, 0.004]	.92	
More Morning Type [b] by Hour	.004	804.8	[0.000, 0.007]	.033	.02
More Evening Type [b] by Hour	.005	1292.0	[0.002, 0.009]	.003	
Sex [c] by Hour	-0.006	3391.9	[-0.01, 0.00	.039	<.001
Random Effects					
Intercept (Dyad)	0.001		[0.000, 0.001]	.012	
Repeated Measures AR1 diagonal	0.07		[0.065, 0.070]	<.001	
AR1 rho	0.05		[0.02, 0.08]	.001	
-2 Log Likelihood	819.0				
AIC	847.0				
No. of Observations	5634				
No. of Participants	55				

*p <.05, **p <.01, ***p<.001. Covariate entered: Age. [a] = Reference group: Concordant bedtime (within 30 mins), [b] = Reference group: Concordant chronotype classification, [c] = Reference group: Female



Supplemental Figure 3. Multi-level modelling significant predictors of transmissibility across the rest interval: (a) Bedtime difference by Hour interaction; (b) Chronotype Difference by Hour Interaction; (c) Sex by Hour interaction. Post-hoc one-way ANOVA determined significant differences. Graphs display M ± SEM. * $p < .05$.

Supplemental Table 5.
Predicting Percent Minutes Resistant to Wake Transmission of an individual, by dyadic Chronotype difference, Bedtime difference, Sex, and Hour of the rest interval.

Parameters	Resistance β	*df*	[95% CI]	*p*	f^2 *for each predictor*
Fixed Effects					
More Morning Type [b]	0.003	1296.5	[-0.03, 0.03]	.82	.02
More Evening Type [b]	0.05	1427.4	[0.03, 0.08]	<.001	
Bedtime Difference (Earlier Bedtime)	0.17	2652.8	[0.11, 0.23]	<.001	.007
Bedtime Difference (Later Bedtime)	0.04	3192.1	[-0.02, 0.09]	.18	
Bedtime Difference (Earlier Bedtime) by Hour	-0.03	3119.6	[-0.04, -0.02]	<.001	.004
Bedtime Difference (Later Bedtime) by Hour	-0.02	3192.1	[-0.03, -0.01]	<.001	
Bedtime Difference (Concordant Bedtime) by Hour	-0.01	3456.2	[-0.01, -0.00]	.002	
Sex (male)[c] by Hour	-0.007	1869.3	[-0.01, -0.00]	<.001	.005
Random Effects					
Intercept (Dyad)	0.01		[0.005, 0.012]	<.001	
Repeated Measures AR1 diagonal	0.08		[0.07, 0.08]	<.001	
AR1 rho	0.22		[0.19, 0.24]	<.001	
-2 Log Likelihood	1692.9				
AIC	1718.9				
No. of Observations	5699				
No. of Participants	55				

*p <.05, **p <.01, ***p<.001. Covariate entered: Age. [a] = Reference group: Concordant bedtime (within 30 mins), [b] = Reference group: Concordant chronotype classification, [c] = Reference group: Female

(a) Sample Syntax for Cross-Classified Models:

The following is an example of the intercepts-only model for the cross-classified model. This is the model used to analyse resistance to transmission:

```
MIXED Resistance_to_transmission BY MEQdifference Bedtimedifference Sex WITH Night Hour Age
/CRITERIA=CIN(95) MXITER(100) MXSTEP(10) SCORING(1) SINGULAR(0.000000000001)
HCONVERGE(0,
 ABSOLUTE) LCONVERGE(0, ABSOLUTE) PCONVERGE(0.000001, ABSOLUTE)
/FIXED= | SSTYPE(3)
/METHOD=ML
/PRINT= SOLUTION TESTCOV
/RANDOM= INTERCEPT | SUBJECT(DyadID) COVTYPE(CS)
/REPEATED= NIGHT*HOUR | SUBJECT(DyadID*PersonID) COVTYPE(AR1).
```

Key:

Resistance_to_transmission is measured at Hour level, and is different for each individual
Night has 7 levels (night 1-7 of data collection)
Hour has 9 levels (hour 1-9 of the rest interval)
DyadID is a unique code for each dyad (55)
PersonID is a within-dyad dummy code coding members of dyads into person 1 and 2, made for the purpose of being able to identify the repeated measures variable.

Bolger, N., & Laurenceau, J. P. (2013). *Intensive longitudinal methods: An introduction to diary and experience sampling research*. Guilford Press.

Leyland, A. H. (2004). A review of multilevel modelling in SPSS. Retrieved from University of Bristol Centre for Multilevel Modelling website: http://www.bristol.ac.uk/media-library/sites/cmm/migrated/documents/reviewspss.pdf

(b) Sample Syntax for Dyad-Level Model Percent Minutes Resistant to Transmission, and Transmissibility

```
MIXED Dyadic_resistance_to_transmission BY MEQdifference_twolevels Bedtimedifference_twolevels
WITH HOUR Age
/CRITERIA=CIN(95) MXITER(100) MXSTEP(10) SCORING(1) SINGULAR(0.000000000001)
HCONVERGE(0,
 ABSOLUTE) LCONVERGE(0, ABSOLUTE) PCONVERGE(0.000001, ABSOLUTE)
/FIXED= | SSTYPE(3)
/METHOD=ML
/PRINT= SOLUTION TESTCOV
/RANDOM INTERCEPT | SUBJECT(Night) COVTYPE(UN)
/RANDOM INTERCEPT | SUBJECT(DyadID) COVTYPE(UN).
```

Dyadic_resistance_to_transmission is measured at the Hour level, and is different for each dyad.

(c) Sample Syntax for Dyad-Level Model Percent Transmissions Received and Number Transmissions Received.

```
MIXED Dyadic_percent_wake_transmissions BY MEQdifference_twolevels Bedtimedifference_twolevels
WITH HOUR Age
/CRITERIA=CIN(95) MXITER(100) MXSTEP(10) SCORING(1) SINGULAR(0.000000000001)
HCONVERGE(0,
 ABSOLUTE) LCONVERGE(0, ABSOLUTE) PCONVERGE(0.000001, ABSOLUTE)
/FIXED= | SSTYPE(3)
/METHOD=ML
/PRINT= SOLUTION TESTCOV
/RANDOM INTERCEPT Night | SUBJECT(DyadID) COVTYPE(UN).
```

Tabachnick, B. G., & Fidell, L. S. (2013). *Using multivariate statistics* (Vol. 6). Boston, MA: Pearson.

Supplemental Figure 4. Syntax used for multi-level models.

CHAPTER 4

Sleep and Wake are Shared and Transmitted between Individuals with Insomnia and their Bed-Sharing Partners

Chapter 4 is a manuscript accepted for publication in *Sleep*.

Walters, E.M., Phillips, A.J.K., Mellor, A., Hamill, K., Jenkins, M.M., Norton, P.J., Baucom, D.H., & Drummond, S.P.A. (2020). Sleep and wake are shared and transmitted between individuals with insomnia and their bed-sharing partners. *Sleep,* 43(1):1-12. doi: 10.1093/sleep/zsz206.

Foreword to Chapter 4

Paper 2 described and characterised dyadic sleep processes of sleep/wake concordance and wake transmission in couples without evidence of sleep disorders. This chapter now replicates this study in a treatment-seeking population of 52 bedsharing couples aged 18-82 years where one partner experiences insomnia.

Paper 3 was published before Paper 2, so does not reference findings of the previous paper, and does not include reference to one of the variables described in Paper 2 (transmissibility). As this was the first manuscript accepted for publication, it was important to keep the results simple and coherent, without overwhelming the non-specialist reader: a mission reiterated to us through the peer review process. These two papers are intended to be taken together as a coherent piece.

This paper has been accepted for publication in *Sleep*, the official publication of the Sleep Research Society (SRS). It is presented here in its corrected proof format.

Declaration

Declaration of the Candidate: In the case of Chapter 4, the nature and extent of my contribution to the work was the following:

Nature of Contribution	Extent of Contribution
Formulation of research questions and experimental design, data collection, analysis, and writing the manuscript	60%

The following co-authors contributed to the work:

Name	Nature of Contribution
Andrew JK Phillips	Input into data analysis, consultation in formulation of experimental design, and critical review of manuscript
Alix Mellor	Data collection and critical review of manuscript
Kellie Hamill	Data collection and critical review of manuscript
Melissa M Jenkins	Critical review of manuscript
Peter J Norton	Critical review of manuscript
Donald H Baucom	Critical review of manuscript
Sean PA Drummond	Consultation in formulation of research questions and experimental design, discussion of ideas expressed in manuscript, and critical review of manuscript.

The undersigned hereby certify that the above declaration correctly reflects the nature and extent of the candidate and co-authors' contributions to this work.

Candidate's Signature:

Primary Supervisor's Signature:

SLEEPJ, 2019, 1–12

doi: 10.1093/sleep/zsz206
Advance Access Publication Date: XX XX XXXX
Original Article

ORIGINAL ARTICLE

Sleep and wake are shared and transmitted between individuals with insomnia and their bed-sharing partners

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Abstract

Patients with insomnia frequently report disturbing, or being disturbed by, their bedpartner. We aimed to (1) characterize how individuals with insomnia and their bedpartners influence each other's sleep and (2) identify characteristics predicting vulnerability to wake transmission. Fifty-two couples (aged 19–82 years), where one individual was diagnosed with insomnia, participated. Sleep/wake patterns were monitored via actigraphy and sleep diaries for seven nights. Minute-by-minute sleep and wake concordance (simultaneous sleep/wake epochs), number of wake transmissions received (awakenings immediately preceded by wakefulness in the bedpartner), percent wake transmissions received (percentage of total awakenings that were transmitted), and percent of bedpartner's wake minutes resistant to transmission (ability to sleep through bedpartner wakefulness) were calculated. Mixed-effects modeling assessed within-couple bedtime and chronotype differences as predictors of dyadic sleep. We described rates of sleep concordance ($M_{Patient}$ = 63.8%, $M_{Partner}$ = 65.6%), wake concordance ($M_{Patient}$ = 6.6%, $M_{Partner}$ = 6.6%), total transmissions received ($M_{Patient}$ = 5.5, $M_{Partner}$ = 6.9 per night), percent transmissions received ($M_{Patient}$ = 18.5%, $M_{Partner}$ = 23.4% of total awakenings), and percent minutes resistant ($M_{Patient}$ = 56.4%, $M_{Partner}$ = 58.6% of bedpartner's wake time). Partners received wake transmissions at 1.25 times the rate of patients. Percent transmissions received was increased in couples with concordant bedtimes and individuals with later chronotype than their bedpartner. Patterns of chronotype and bedtime order predicting percent minutes resistant to transmission differed across the length of the rest interval. Transmission provides a novel characterization of how bedpartners influence sleep and provide insight into mechanisms of insomnia generation and maintenance. Understanding modifiable risk factors may provide ways to personalize insomnia treatments.

Clinical Trial: Researching Effective Sleep Treatments (Project REST), ANZCTR Registration: ACTRN12616000586415

Statement of Significance

Recent research suggest that sleep problems in an individual can precipitate sleep problems in their bedpartner, but ways to systematically assess this process do not exist. Here, we quantify a novel metric in bed-sharing couples comprising one individual with insomnia and their partner: transmission of wake between bedpartners (awakenings occurring immediately after wake in the bedpartner). Partners of insomnia patients receive more frequent wake transmissions than those with insomnia, indicating that having a bedpartner with insomnia is disruptive to sleep. Individual sleep timing and chronotype characteristics predict the degree of bedpartner disruption. Understanding modifiable risk factors related to dyadic sleep has implications for preventing the development of insomnia and improving insomnia therapies.

Key words: couples; insomnia; concordance; wake transmission; relationships; sleep disorders; dyad

Submitted: 5 March, 2019; **Revised:** 6 June, 2019

Insomnia disorder affects 10%–15% of the global adult population [1–5] and is associated with significant economic and personal burden. However, the interpersonal dynamics of insomnia are not yet fully understood. This is, in part, likely due to the fact that sleep has traditionally been studied as an individual behavior, despite 59%–63% of adults reporting sleeping with a romantic partner [6, 7]. When two people share a bed, one individual's sleep problems may disrupt the other's sleep [8–10]. Despite this, the role of the bedpartner is frequently overlooked in the context of insomnia. Where bedpartners have been considered, patients strongly endorse their partners as being an important factor for insomnia predisposition, precipitation, and perpetuation [11] and for engaging in treatment [12]. Examining sleep at the level of the dyad could more specifically elucidate the role bedpartners play in insomnia disorder.

Bedpartners have described their sleep being influenced in ways including: altering timing of the sleep period, waking one another with noise or movement, and influencing the sleep environment [11, 13–15]. A handful of previous studies have quantified interactions between healthy bedpartners in terms of sleep/wake concordance [13, 16, 17]. Concordance assesses the extent to which couples are in the same sleep/wake state on a minute-by-minute basis. Couples who are bedpartners are found to be more concordant in actigraphically determined sleep/wake state than randomly matched pseudo-couples [13, 16], and concordance of sleep stages is higher when couples sleep in the same bed versus apart [17]. Preliminary research has found an individual's physical movements or wake bouts overnight frequently precede awakenings in a bedpartner [18]. Given this finding and considering adults have multiple awakenings per night [19, 20], it is plausible that awakenings could transmit between bedpartners (i.e., one individual's awakening could cause their bedpartner to awaken) [18]. While this process is frequently described in clinics in the context of insomnia and sleep apnea [11, 21], research has not yet quantified it objectively within insomnia.

Chronotype and circadian timing may help further explain how bedpartners impact one another's sleep. An individual's preference for earlier or later sleep timing, which is in part driven by the circadian system [22], may differ from that of their bedpartner. Sleeping at a misaligned circadian time leads to poorer quality sleep [23, 24] which may result in a greater likelihood of having wake transmitted by the bedpartner. Despite differences in chronotype and/or circadian timing, many couples choose to share the same bedtime [25]. This can be for social reasons or to minimize perceived sleep disruptions caused by a bedpartner coming to bed at a later time [11, 26]. The impacts of these behaviors on sleep and insomnia severity have, however, not been quantified.

The current study used wrist actigraphy in couples where one bedpartner experienced insomnia disorder to measure concordance and transmission of sleep and wake throughout the night. Study aims were: (1) Characterize minute-by-minute sleep and wake concordance in couples where one bedpartner experiences insomnia disorder. We hypothesized that sleep and wake concordance would be lower in couples with more severe insomnia. (2) Define measures of wake transmission and report their rates in an insomnia population for the first time. We hypothesized wake transmission rates would occur predominantly in the direction from patient to partner, but patients would demonstrate a lower percent of bedpartner's wake minutes resistant to wake transmission. (3) Determine whether bedpartners' chronotypes and bedtimes influence wake transmission. We hypothesized that having a different chronotype or bedtime from one's bedpartner would increase likelihood for wake transmissions to occur and decrease the amount of time resistant to transmission. We expected that this effect would be greater for individuals with insomnia compared to their partners and that effects would be seen predominantly at the beginning and end of the sleep period.

Materials and Methods

Participants

This study recruited cohabiting couples with one member seeking treatment for insomnia. In total, this study included 52 couples (48 different-sex couples and 4 same-sex couples) where at least one member experienced clinically diagnosed insomnia disorder. Participants were recruited by print media, radio, online advertisements, and via sleep clinics in Melbourne from psychologist or General Practitioner (GP) referrals. Couples were enrolled as part of a larger clinical trial (Project REST; Australian New Zealand Clinical Trials Registry Registration: ACTRN12616000586415), which involved partner-assisted behavioral interventions for insomnia [27]. Ethical approval was obtained from Monash University Human Research Ethics Committee.

Couples were included if they were: (1) 18+ years old, (2) stable bedpartners for more than 1 month, (3) fluent in English, and (4) had no evidence of ongoing domestic violence in the relationship. The individual experiencing insomnia ("patient") met the criteria for insomnia disorder according to the Duke Structured Interview for Sleep Disorders (DSISD) [28]. Exclusion criteria included: (1) having any other unmanaged sleep disorders, assessed via DSISD and a diagnostic polysomnogram; (2) receiving any behavioral treatment for insomnia in the past month; (3) unmanaged psychosis or history of manic episodes; (4) substance/alcohol use disorder in the past 6 months; (5) engagement in shift work within the past 6 months; (6) transmeridian travel of more than one time zone per week since last travel before starting treatment; (7) current pregnancy past the first trimester; and (8) children under the age of 1 year. Exclusion criteria for the nonpatient partner ("partner") included (1) unmanaged psychosis or history of manic episodes and (2) current substance/alcohol use disorder. Partners did not complete diagnostic assessment for insomnia. If either party was ineligible, the couple was not enrolled. Participant involvement for the current protocol included an assessment session with the researchers, a night of polysomnography (PSG) for the patient to rule out presence of other sleep disorders, and seven nights of at-home sleep monitoring within 1 month of the assessment session (0–34 day afterward; M = 9.5, SD = 9.5 days). All data for this manuscript came from the baseline assessment and the first week of treatment.

Measures

The Insomnia Severity Index (ISI) comprises seven items assessing the severity of insomnia in the past week [29, 30]. The STOPBang is an eight-item self-report scale measuring an individual's risk of experiencing symptoms of Obstructive Sleep Apnea (OSA) [31]. If any patients were assessed as "high risk"

for OSA, the couple was excluded from the protocol. High risk was defined as having either an overall score of ≥5 or at least two endorsements on the STOP questions and having body mass index (BMI) >35 kg/m^2 [32]. The Morningness–Eveningness Questionnaire, reduced form (MEQr) [33], was used to assess individuals' propensity to be either morning or evening oriented, therefore assessing chronotype [34].

All participants completed a daily sleep diary for seven nights [35]. This diary included reported bedtime, sleep latency (SL), number and duration of awakenings (wake after sleep onset; WASO), wake time, early morning awakening (minutes between the final awakening and time the individual got out of bed), and total time in bed. Participants concurrently wore an actigraph to measure objective sleep. Respironics Actiwatch Spectrum Pro and Actiware software (Respironics, Bend, OR) were used to collect and analyze actigraphy data in 60 s epochs. Rest intervals were manually set each night by the investigators; diary-reported bed and wake times were used as guidelines, although rest intervals could be extended up to 60 min on either side to account for sleep outside self-reported time in bed. This procedure is similar to methods used in other studies [36, 37]. Sleep detection settings on the Actiwatch were set to high sensitivity (20 activity counts per epoch as the wake threshold) [38].

Data analysis

Calculation of variables

Dyadic sleep variables were sleep and wake concordance and wake transmission within couples (Figure 1). *Concordance* was calculated as the percentage of actigraphy epochs within an individual's rest interval where both bedpartners were in the same sleep or wake state (i.e., epochs both in same state/total rest interval epochs). This is similar to methodological approaches used by Gunn et al. [16]. Sleep and wake were separated, so, for each individual, a score was calculated for wake concordance (wake epochs/total rest interval epochs) and sleep concordance (sleep epochs/total rest interval epochs).

Wake transmission was calculated by determining occurrences throughout the night where an individual experienced an actigraphically determined awakening (i.e., transition from sleep to wake), when their bedpartner was awake the minute immediately preceding the beginning of the individual's wake bout. Three transmission variables were calculated: number transmissions received, percent transmissions received, and the percent of a bedpartner's wake minutes an individual was resistant to transmission. *Number transmissions received* was the total number of wake transmissions in a given time interval. *Percent transmissions received* was calculated as the percentage of an individual's awakenings in a given time interval that were classified as wake transmission (i.e., total transmitted wakes/total wakes). *Percent minutes resistant to transmission* was defined as the percentage of minutes an individual was asleep during their bedpartners' wake minutes. Additionally, the mean of the two bedpartner scores was taken to create measures of average dyad-level number transmissions received and percent transmissions received. Percent transmissions received and percent minutes resistant were the primary outcomes for this manuscript. Number transmissions received was calculated to more comprehensively describe the sample.

Two additional explanatory variables were computed. (1) Chronotype difference was determined based on the category (definitely morning, moderately morning, neither type, moderately evening, or definitely evening-type) [33] into which each bedpartner fell. Individuals were then coded as being in the "same chronotype category as bedpartner," "more morning-type than bedpartner," or "more evening-type than bedpartner." (2) Bedtime difference was calculated using actigraphically derived rest intervals on a nightly basis for each individual, with the night categorized as either: individual in bed first (by >30 min), concordant bedtime (within a 30 min window), or individual in bed later (by >30 min than bedpartner.

Missing data

Nights where couples did not share a bed were excluded. These nights (n = 20 out of 364) were determined based on notations in the sleep diary or clinical notes. Two additional couples indicated they slept apart two nights during the week, but which nights these were could not be determined. For these two couples, all nights were included in the analysis. Analyses were also run excluding all nights for these couples; since key outcomes did not change, these couples were included. Additionally, there were n = 19 nights where actigraphy data were not available due to participant nonadherence or technical issues. This resulted in n = 325 actigraphy nights available for analysis. For sleep diaries, there were n = 32 nights not available due to participant nonadherence. This resulted in n = 312 sleep diary nights available for analysis.

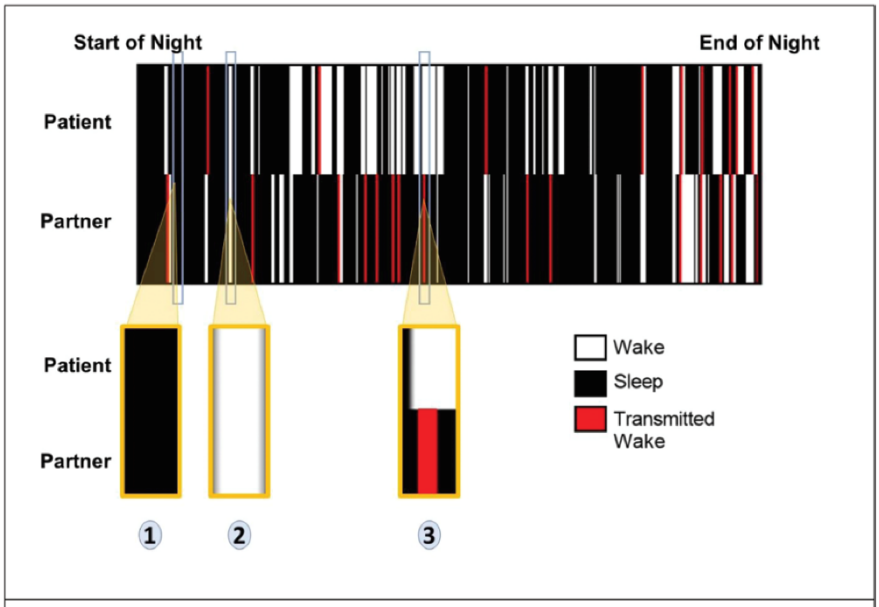
All data were checked for normality through visual inspection of histograms and statistical diagnostics. Four univariate outliers, defined as z-scores over a threshold of ±3.29, were Winsorized: one univariate outlier each on partner ISI and patient percent transmissions received and two for patient number transmissions received.

Statistics

The Sleep Regularity Index (SRI) provided a measure of night-to-night sleep variability. The SRI measures the percentage probability of any two time points 24 h apart being in the same sleep/wake state, across a series of days, with higher scores denoting greater regularity (for detailed description, see Phillips et al. [39]. To assess differences between patients and partners, t-tests were conducted for all individual and dyadic sleep variables. Two variables (sleep diary SL and WASO) had nonnormal distributions and, therefore, nonparametric Mann–Whitney U-tests were conducted.

Pearson's correlations were used to assess relationships between dyadic sleep variables and both age and sleep disorder symptoms. As ISI and STOPBang scores were not significantly correlated with dyadic sleep variables, these were not included as predictors in subsequent analyses. Age was significantly associated with several dyadic sleep variables for patients and partners and, thus, was included as a covariate for subsequent multilevel models. Bivariate correlations and t-tests were conducted where appropriate to determine the association of age with other predictors. A complete bivariate correlation table is displayed in Supplementary Table S1.

To determine whether concordance, number transmissions received, percent transmissions received, and percent minutes resistant to transmission rates observed were higher than those



Actogram of a couple's sleep on a single night, annotated to demonstrate dyadic sleep phenomena across the night. Each epoch is graphed across the night from left to right. (1) **Sleep Concordance**. The percentage of epochs during an individual's rest interval both partners are coded as asleep. (2) **Wake Concordance**. The percentage of epochs during an individual's rest interval both partners are coded as awake. (3) **Wake Transmission**. An individual's wake bout is immediately preceded by wake of a bedpartner. For each night, 3 variables are calculated: **Number Transmissions Received**, defined as the total number of occasions an individual receives a wake transmission from their bed partner; **Percent Transmissions Received**, the percent of all wake bouts coded as being transmitted from the bed partner, providing a measure of vulnerability to transmission; **Percent of Minutes Resistant to Transmission**, the percentage of time an individual is asleep during a bedpartners' wake minutes.

Graph represents a night with 62.5% sleep concordance for Patient, 58.3% sleep concordance for Partner, 8.3% wake concordance for Patient, 7.7% wake concordance for Partner. Patient received 9 transmissions (23.1% of their total awakenings), and was resistant to transmissions for 49.9% of the partner's overnight wake minutes. Partner received 15 transmissions (38.5% of their total awakenings), and was resistant to transmissions for 70.4% of the patient's overnight wake minutes.

Figure 1. Graphical representation of dyadic sleep parameters.

of randomly matched pairs of patients and partners, individuals within the sample were used to obtain a bootstrap distribution of 100 000 randomly paired nights. This was completed by randomly sampling nights with replacement using MATLAB R2018b (The MathWorks, Inc., Natick, MA). Concordance, number transmissions received, percent transmissions received, and percent minutes resistant levels for true couples were then analyzed using one-sample t-tests to determine significant differences from this pseudo-couple distribution.

Multilevel mixed-effects modeling predicted primary outcome variables percent transmissions received and percent minutes resistant to transmission. As we conceptualized part of this paper's contribution to the literature as being to determine which variables are most relevant to understanding bedpartner sleep, we also ran these models with number transmissions received as the outcome variable; this analysis can be found in Supplementary Tables S4 and S5 and Supplementary Figures S1 and S2. To identify nightly predictors of percent transmissions received and percent minutes resistant to transmission, multilevel mixed-effects modeling was conducted. The model structure was selected due to the nested structure of the data set, allowing analysis to incorporate differences by hour, night, individual, and dyad. First-level units were hours into the rest interval, up to a maximum of 9 h due to small sample size beyond 9 h, resulting in 5423 units total for percent transmissions received data and 5334 units total for percent wake minutes resistance to transmission. Second-level units were nights of data, with a maximum of seven nights per individual, resulting in 325 units total. Third-level units were individuals, resulting in 104 units total. Fourth-level units were the 52 dyads. Only dyads with both individuals' data available were selected for analysis. The models also included age, chronotype difference, bedtime difference, and all two-way interactions among age, chronotype difference, bedtime difference, and hour of the rest interval. Model

parameters were fit using a full information maximum likelihood estimation method with SPSS v.24. Akaike's Information Criterion (AIC) was used to determine the most parsimonious models. For percent bedpartner's wake minutes resistant to transmission, where a bedpartner did not wake during a given hourly interval and, therefore, an individual could not demonstrate resistance (or lack thereof) to transmission, no value was calculated.

These same models were run with dyad-level average number of transmissions received, percent transmissions received, and percent minutes resistant to transmission in order to provide couple-level understanding of whether overall concordant or discordant bedtimes and chronotypes related to higher percent transmissions received and percent minutes resistant to transmission. To create dyad-level variables, average values for the two individual bedpartner scores were computed. Bedtime difference and chronotype difference were recoded into "discordant" and "concordant" categories.

Positive skewness was observed in the distribution of wake transmission. However, model residuals were approximately normally distributed according to visual inspection of histograms, Q–Q plots, and statistical diagnostics. Additionally, repeating the modeling with square root-transformed outcomes did not change the main findings, so the raw values were used for reporting results (transformed results displayed in Supplementary Tables S2 and S3). For the individual percent transmissions received model, intraclass correlations for night and hour were very low, indicating there may not be value in including night or hour as random units. Therefore, this model was run with Dyad entered as a random unit, and night and hour entered as fixed effects. For the percent minutes resistant models, intraclass correlations for night were low. Therefore, this model was run with dyad and hour entered as random units and night entered as a fixed effect. Parameter estimate tables for all models are presented in the Supplementary Tables S4–S7).

Results

Sample characteristics

Table 1 presents sample demographics, as well as individual and dyadic sleep variables. All diary variables except total sleep time (TST) and early morning awakenings showed poorer self-reported sleep in patients, relative to partners. In contrast, actigraphy variables indicated higher WASO and lower sleep regularity in patients compared to partners but no significant differences in other sleep variables. Age did not differ between men or women nor between patients and partners ($t = 1.67$, $p = .10$ and $t = .05$, $p = .96$, respectively). Age was unrelated to ISI score ($r = .05$, $p = .61$). As expected, age was highly correlated ($r = .81$; $p < .001$) with relationship length. Age was positively associated with STOPBang scores ($r = .53$; $p < .001$).

Concordance

Patients had significantly lower sleep concordance than partners (small effect size) and did not significantly differ by wake concordance (Table 1; correlations between patient and partner displayed in Figure 2). Sleep and wake concordance were both positively correlated between patients and partners ($r = .76$ and $r = .98$, respectively; Figure 2). Wake concordance was negatively correlated with age for both patients and partners, whereas sleep concordance was not associated with age. Sleep and wake concordance were not significantly associated with sleep disorder symptoms (in patients: sleep concordance/ISI $r = -.08$, $p = .58$, wake concordance/ISI $r = .22$, $p = .12$; in partners: sleep concordance/ISI $r = -.10$, $p = .48$, wake concordance/ISI $r = .20$, $p = .16$).

Patient and partner mean sleep and wake concordance were significantly higher than scores for randomly paired pseudo-couples (all p values $< .001$: pseudo-couple patient sleep concordance $M = 54.5\%$, $SD = 1.25\%$; pseudo-couple patient wake concordance $M = 2.7\%$, $SD = 2.0$ %; pseudo-couple partner sleep concordance $M = 59.3\%$, $SD = 1.2\%$; pseudo-couple partner wake concordance $M = 3.0\%$, $SD = 2.1\%$), indicating that concordance between bedpartners occurred at levels greater than chance.

Transmission

Patients and partners were significantly different by wake transmission measures (i.e., receiving wake transmissions from the bedpartner). Wake transmissions from patients to partners occurred at 1.25 times the rate of wake transmission from partners to patients, both for number transmissions received ($p = .001$, $d = 0.54$) and percent transmissions received ($p = .021$, $d = 0.52$; Table 1). However, patients and partners did not differ in the percent of minutes they spent resisting wake transmission from a bedpartner (correlation displayed in Figure 2; there was no relationship between patient and partner percent wake minutes resistant). Number transmissions received were positively correlated between patients and partners ($r = .29$, $p = .041$), while percent transmissions received was negatively correlated between patients and partners ($r = -.27$, $p = .049$; Figure 2). Females had 1.32 times higher percent transmissions received than males, $p = .002$ (i.e., a larger proportion of females' wake episodes were preceded by wake in the males than vice versa).

Number transmissions received were negatively correlated with age for both patients and partners and, for partners, percent minutes resistant to transmission was positively correlated with age. Number transmissions received, percent transmissions received, and percent minutes resistant were not significantly associated with sleep disorder symptoms (ISI and STOPBang).

Number transmissions received were significantly different than for randomly paired pseudo-couples for both patients ($p = .02$; pseudo-couple $M = 4.4$, $SD = 2.9$) and partners ($p < .001$; pseudo-couple $M = 5.1$, $SD = 3.4$), as were percent transmissions received rates (patients $p < .001$; pseudo-couple $M = 13.8\%$, $SD = 8.4\%$; partners $p < .001$; pseudo-couple $M = 17.2\%$, $SD = 10.2\%$) and percent minutes resistant to transmission (patients $p < .001$, pseudo-couple patient $M = 68.1\%$, $SD = 16.3\%$; partners $p = .042$, pseudo-couple partner $M = 62.1\%$, $SD = 17.5\%$), indicating that awakenings classified as transmissions occurred at rates greater than chance, and individuals were awake during their bed partner's wake episodes for a higher percentage than would occur by chance.

Predictors of transmission

Percent transmissions received

The most parsimonious model predicting an individual's percent transmissions received included variables of role ($p < .001$), bedtime difference ($p < .001$), chronotype difference ($p < .001$),

Table 1. Description of sample: demographics, individual, and dyadic sleep variables, $n = 52$ couples

Variable	Patients (M ± SD)	Partners (M ± SD)	p	Effect sizes
Sex (*n*)	35 female (67.3% female)	21 female (40.38% female)	.006	V = 0.27
Age	51.59 ± 14.05 years Range: 19–80 years	51.41 ± 14.41 years Range: 20–82 years	.85	0.01
Duration of bed-sharing relationship	22.3 ± 16.7 years			
Nature of relationship	37			
Married (*n*)	12			
De facto (*n*)	3			
Dating (*n*)				
Presence of children <18 years in household	23.5%			
Presence of pets in household	23.5%			
ISI	16.40 ± 3.45	4.69 ± 4.84	<.001	2.79
No symptoms (*n*)	0	39		
Subthreshold symptoms (*n*)	20	10		
Clinical insomnia (moderate; *n*)	29	2		
Clinical insomnia (severe; *n*)	3	1		
STOPBang	2.21 ± 1.16	2.21 ± 1.61	>.99	<0.001
Sleep diary				
Sleep efficiency (%)	71.13 ± 14.76	86.85 ± 7.67	.021	1.34
SL[U] (min)	47.71 ± 46.02	18.66 ± 13.19	<.001	0.36
TST (min)	369.49 ± 85.37	442.85 ± 67.29	.082	0.95
WASO[U] (min)	53.07 ± 42.45	22.26 ± 21.50	<.001	0.45
Terminal awakening (min)	41.38 ± 25.74	24.94 ± 15.98	.073	0.77
Actigraphy				
Sleep efficiency (%)	76.89 ± 7.85	79.24 ± 5.98	.077	0.34
SL (min)	13.76 ± 11.71	13.83 ± 10.56	.96	0.01
TST (min)	393.85 ± 53.32	391.58 ± 50.23	.78	0.04
WASO (min)	82.61 ± 25.68	71.07 ± 22.61	.016	0.48
SRI	69.93 ± 1.19	73.14 ± 6.33	.014	0.71
Dyadic sleep variables	Patient/transmission to patient (M ± SD)	Partner/transmission to partner (M ± SD)		
Concordance				
Sleep (%)	63.44 ± 9.63	65.43 ± 8.80	.028	0.22
Wake (%)	6.58 ± 2.96	6.64 ± 2.89	.46	0.02
Number transmissions received (*n*)	5.35 ± 2.65	6.93 ± 3.21	.001	0.54
Percent transmissions received (%)	18.66 ± 8.72	23.55 ± 10.19	.021	0.52
Percent minutes resistant to transmission (%)	56.42 ± 13.81	58.62 ± 12.02	.35	0.17

[U]Mann–Whitney U-test completed. Effect sizes displayed are Cohen's *d* for t-tests, *r* for Mann–Whitney U-tests.

and the bedtime difference by hour interaction ($p < .001$). This model did not perform significantly worse than the full model, $X^2(11) = 9.55$, $p = .57$, and, thus, was adopted as the final model. Percent transmissions received was greater for partners than for patients ($\beta = 6.0\%$, $p < .001$) and for individuals who were more evening-type than their bedpartner ($\beta = 4.3\%$, $p = .007$). The bedtime difference by hour interaction showed percent transmissions received was greatest in the first 2 h of the rest interval for individuals who have concordant bedtime with their bedpartners, relative to other bedtime differences and hours of the night ($p < .001$; see Figure 3).

The most parsimonious model predicting average dyad-level percent transmissions received (i.e., the average of both partner's percent transmissions received) included variables of bedtime difference ($p < .001$), chronotype difference ($p = .002$), and the chronotype difference by hour interaction ($p < .001$). This model did not perform significantly worse than the full model, $X^2(2) = 4.64$, $p = .098$, and, thus, was adopted as the final model. Dyad percent transmissions received was found to be higher for dyads with concordant than discordant bedtimes ($\beta = -2.9\%$, $p < .001$) and for dyads with discordant than concordant chronotypes ($\beta = 4.4\%$, $p = .002$). The chronotype difference by hour interaction showed dyadic percent transmissions received rates diverged so that discordant chronotypes had higher dyadic percent transmissions received at hour 1 ($p < .001$) and hour 4 ($p = .007$), whereas there was no difference between groups at other hours of the rest interval (see Figure 4).

Percent of bedpartner's wake minutes resistant to transmission

The most parsimonious model predicting an individual's percent minutes resistant included variables of role ($p < .001$), the chronotype difference by bedtime difference interaction ($p < .001$), the bedtime difference by hour interaction ($p < .001$), and the chronotype difference by hour interaction ($p = .003$; see Figure 5). This did not significantly worsen the full model, $X^2(3) = 2.40$, $p = .49$, and, thus, was adopted as the final model. Percent minutes resistant to transmission was found to be lower for patients than partners ($\beta = -4.0\%$, $p < .001$). The chronotype difference by bedtime difference interaction showed resistance was not dependent on bedtime difference when an individual

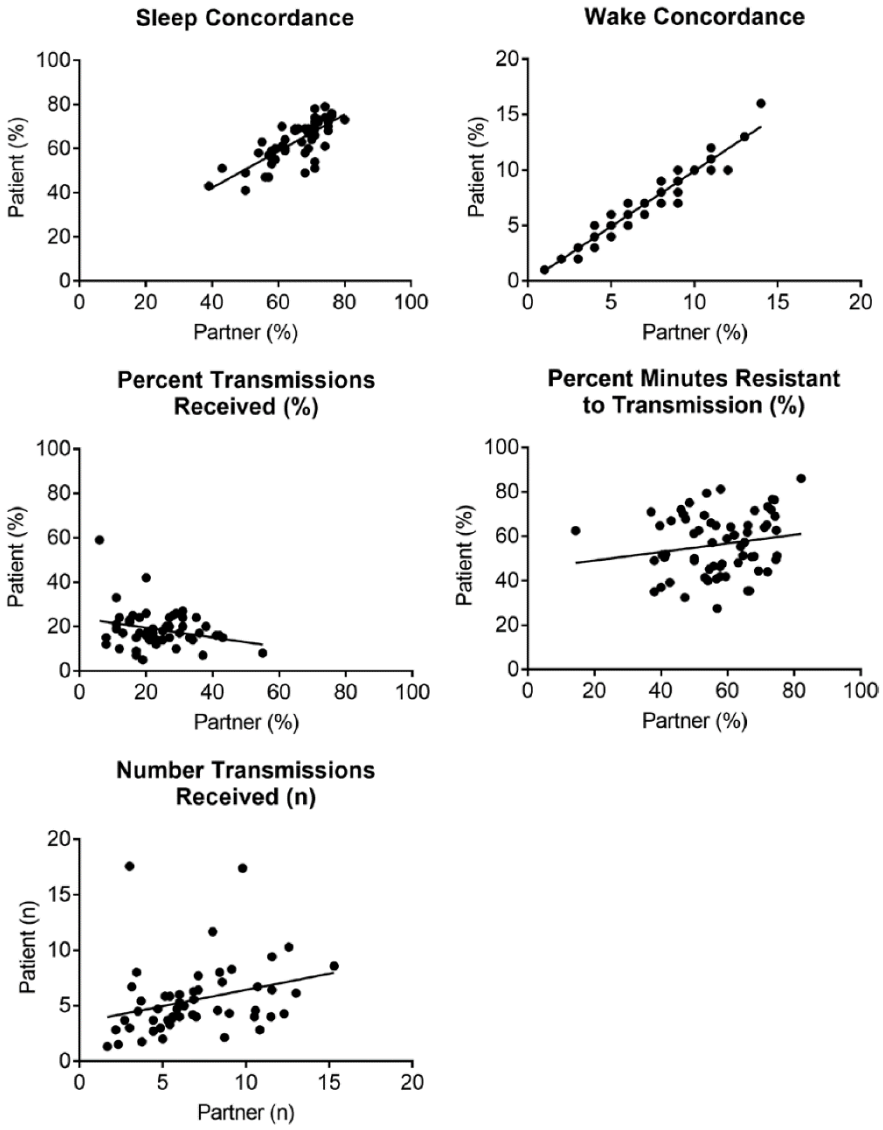


Figure 2. Between-person (patient vs. partner) bivariate correlations of dyadic sleep variables. Sleep concordance $r = .76$, $p < .001$; wake concordance $r = .98$, $p < .001$; percent transmissions received $r = -.27$, $p = .049$; percent minutes resistant to transmission $r = .18$, $p = .21$; number transmissions received $r = .29$, $p = .041$.

had a later chronotype than their bedpartner; however, when an individual had the same chronotype, then resistance to transmission was highest in those who went to bed before their bedpartners ($p = .025$) and, when an individual had earlier chronotype, resistance was highest when bedtime was shared ($p = .015$). The chronotype difference by hour interaction showed those who had an earlier chronotype than their partner experienced the lowest percent minutes resistant to transmission, relative to other chronotypes, at hours 1 ($p = .017$), 3 ($p = .027$), and 4 ($p = .001$), and resistance to transmission was highest in those who had later chronotype than their partner at hours 8 ($p = .006$) and 9 ($p = .007$). The bedtime difference by hour interaction showed that percent minutes resistant to transmission was lowest for individuals who have concordant bedtime with their bedpartners at hour 1 ($p < .001$) and, at the end of the rest interval, individuals with later bedtime than their partners had lower percent minutes resistance to transmission (hour 7: $p = .025$, hour 8: $p = .049$).

The most parsimonious model predicting average dyad-level percent minutes resistant (i.e., the average of both partner's percent minutes resistant to transmission) included variables of bedtime difference ($p < .001$), chronotype difference ($p = .030$), the bedtime difference by hour interaction ($p = .012$), and the chronotype difference by hour interaction ($p < .001$). This did not significantly worsen the full model, $X^2(4) = 8.40$, $p = .08$, and, thus, was adopted as the final model. Dyadic percent minutes resistant was found to be lower for those couples with concordant than discordant bedtimes ($\beta = -7.6\%$, $p < .001$) and for dyads with discordant than concordant chronotypes ($\beta = -6.7\%$, $p = .03$. The bedtime difference by hour interaction showed dyadic percent

minutes resistance rates differed so that concordant bedtimes had lower resistance at hour 1 ($p < .001$) and hour 9 ($p < .001$) relative to discordant bedtime couples. The chronotype difference by hour interaction showed dyadic percent minutes resistance rates diverged so that discordant chronotypes had lower resistance at hour 1 ($p = .004$) and hour 4 ($p < .001$), whereas there was no difference between groups at other hours of the rest interval (see Figure 4b).

Discussion

This paper reports the first examination of dyadic sleep and wake concordance (time spent in shared sleep/wake) and wake transmission (how frequently wake bouts were immediately preceded by a bedpartner's wake) within a sample of cohabiting couples where one member was seeking treatment for insomnia. We found that partners of individuals with insomnia receive more frequent wake transmissions than those with insomnia, indicating that having a bedpartner with insomnia is disruptive to sleep. There were no significant associations between any dyadic sleep variables and insomnia severity. Percent wake transmissions received and percent wake minutes resistant to transmission were predicted by within-couple differences in chronotype and bedtime, with concordant bedtimes conferring highest risk of wake transmissions, especially at the beginning of the night.

Our findings indicate that there are two potential bedpartner disruption processes occurring in insomnia. First, disruption from a partner could perpetuate or increase wake transmissions and, thus, insomnia symptoms for some individuals. Second, disruption caused by the patient with insomnia to their bedpartner leads to interrupted sleep for the otherwise healthy sleeping partner as seen previously in OSA [21, 40]. These bidirectional interactions could potentially generate a feedback loop, in which the increased frequency of partner awakenings increases the likelihood of wake transmissions back to the patient with insomnia. We note, however, that the observed frequencies of wake transmissions received (5.5 and 6.9 times per night for patients and partners, respectively) is considerably less than the frequency of awakenings seen in OSA, where partners are at least partially aroused by their bedpartner's snores a median of 9 times/h [8]. Thus, disruption to a bedpartner caused by someone with insomnia may be considerably less than that caused by someone with OSA.

The physiological bases of insomnia are still being determined. The fact that individuals with insomnia tend to have more frequent nocturnal awakenings in the home could be due to a greater sensitivity to environmental stimuli or it could be due to an endogenous process. While we found evidence that patients with insomnia were disrupted by their partners (5.4 times/night, representing 18.7% of their total wake bouts), this was lower than the disruptions partners received from patients (6.9 times/night, representing 23.6% of their total wake bouts). Although patients had lower resistance to transmission than partners when controlling for other factors, effect sizes were small (56.4% vs. 58.6%) and, when other factors were not controlled, there were no significant differences in nightly resistance between patients and partners. Overall, these findings suggest sources of wake bouts in insomnia disorder may be mostly endogenous (i.e., an internal generation of excessive awakening overnight) as opposed to abnormal responsivity to the environment at least as represented by a bedpartner.

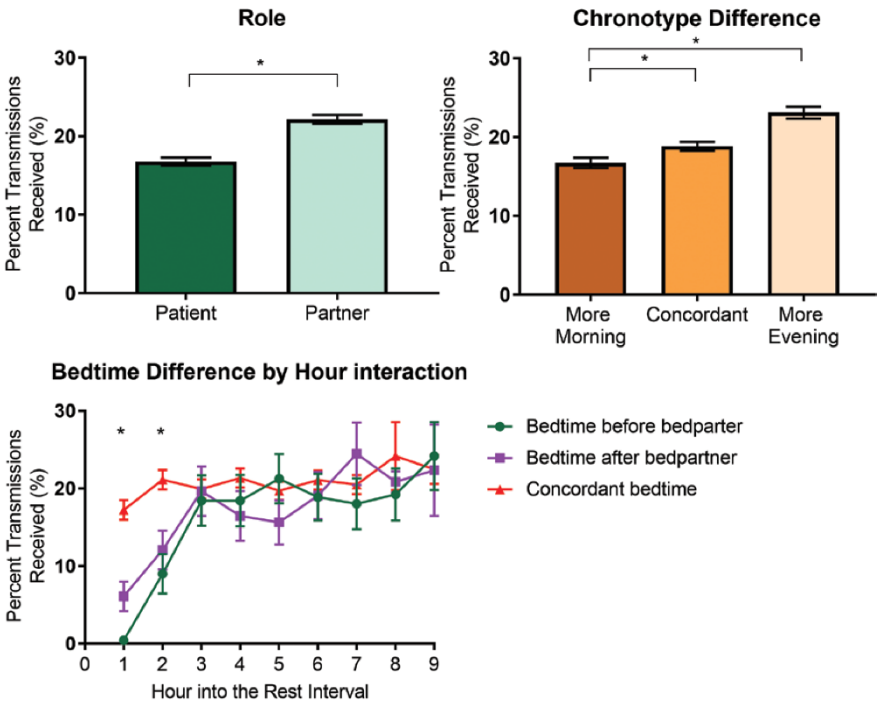


Figure 3. Significant predictors of percent transmissions received at the individual level: within-couple chronotype difference, role within couple, and within-couple bedtime difference by hour of the rest interval interaction. Graphs display M ± SEM. *$p < .05$.

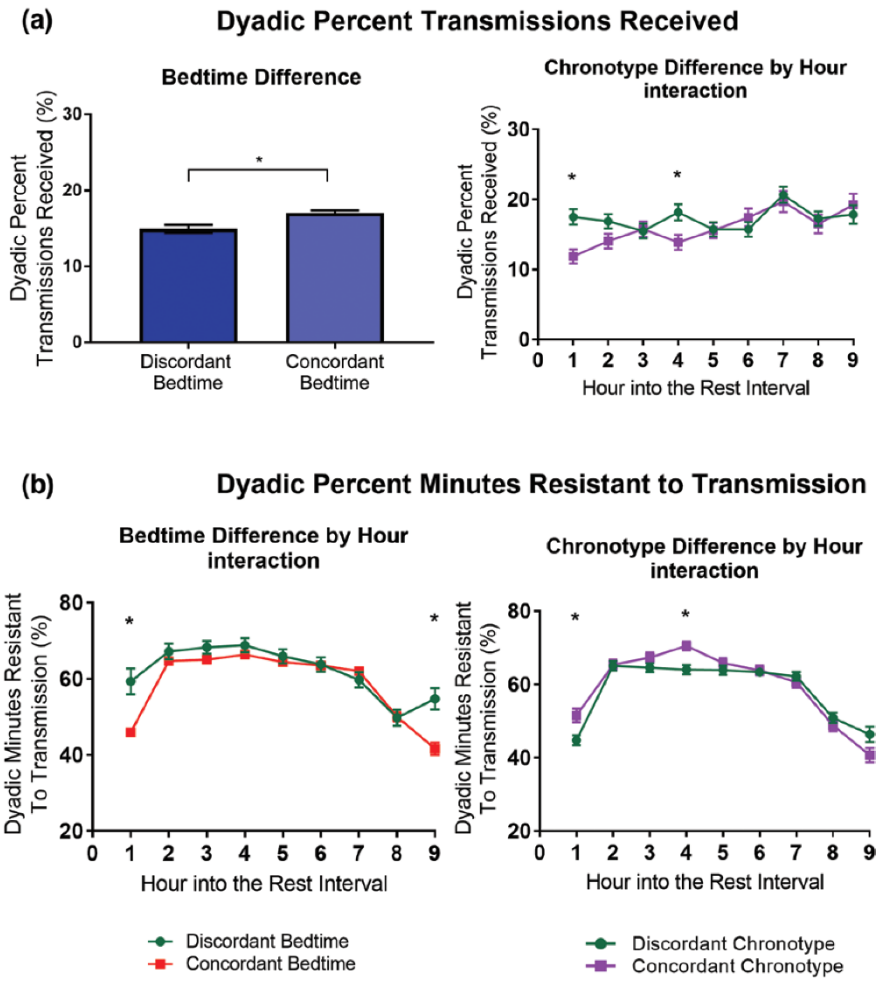


Figure 4. Multilevel modeling significant predictors of dyadic sleep variables across the rest interval: (a) bedtime difference main effect and chronotype difference by hour interaction predicted average dyadic percent transmissions received, (b) bedtime difference by hour interaction and chronotype difference by hour interaction predicted average dyadic percent minutes resistant to transmission. Post hoc t-tests determined significant differences between groups at hours displayed. Graphs display M ± SEM. *$p < .05$.

We found a mismatch in the sleep parameters of partners between actigraphy and sleep diaries. Actigraphically defined sleep parameters fell outside accepted healthy guidelines (e.g., sleep efficiency and WASO; Table 1), whereas sleep parameters derived from diaries fell inside the healthy range. This subjective–objective mismatch is consistent with the current consensus within the dyadic sleep literature: when bed-sharing, individuals tend to report improved subjective sleep quality but poorer objective sleep quality [13, 41]. These data may, therefore, provide evidence that dyadic sleep disruption from patients (i.e., higher number and percent transmissions received in partners) could be driving lower than expected partner objective sleep.

The present research makes novel contributions to the existing literature on sleep/wake concordance, defining this construct differently from previous papers [16, 42]. Previously, combined sleep/wake concordance has been considered the most relevant unit of analysis. Our study considers sleep concordance and wake concordance separately (analogous to sensitivity and specificity in classification testing). Insomnia is a disorder characterized by nocturnal wake; therefore, it is important to understand specifically how these periods of wakefulness overlap and/or interact with those of the bedpartner. Because most of the rest period consists of sleep, the combined sleep/wake concordance is relatively insensitive to awakenings. This reflects the pattern observed in the present data. Where previous studies have identified that total concordance decreases as age and relationship length increase [13], the present data show, more specifically, a negative correlation between concordance of wake with both age and relationship length, while concordance of sleep epochs is uncorrelated with either age or relationship length. At the same time, percent minutes resistant to wake transmission was higher in older partners. Whilst the present data are correlational in nature, they argue for the possibility that lower wake concordance in older adults is driven by the partner becoming more resistant to the awakenings of their partner, possibly due to age-related changes such as hearing loss. Since age and relationship length were highly correlated, this could alternatively be due to

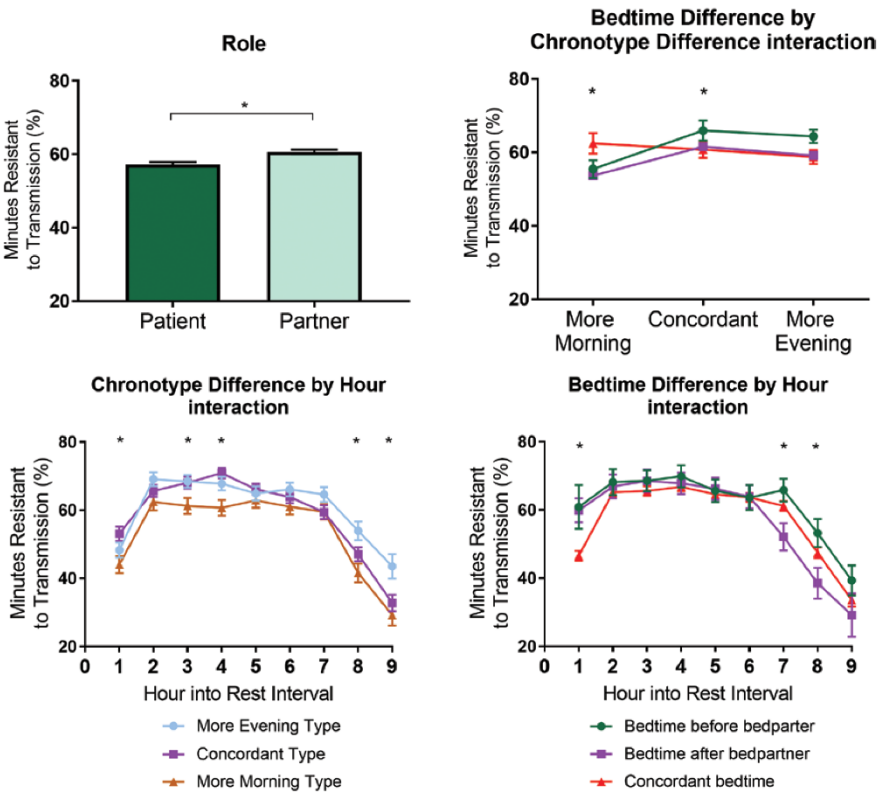


Figure 5. Significant predictors of percent minutes resistant to transmission at the individual level: role within the couple; within-couple bedtime difference by within-couple chronotype difference interaction, within-couple chronotype by hour interaction, and within-couple bedtime difference by hour interaction. Graphs display M ± SEM. *$p < .05$.

a habituation effect over time. Longitudinal studies are needed to test these alternative hypotheses and allow for more causal inferences to be drawn.

Wake transmission is important to understand as it has therapeutic implications. Bedpartners are not traditionally included within insomnia assessment or treatment, and bedpartner considerations are not an inherent or assumed part of manualized Cognitive Behavioural Therapy for Insomnia (CBT-I). This is in contrast to OSA, where a bedpartner may be included within the assessment. Indeed, for OSA, treating an individual can be considered a sleep treatment for the bedpartner [43]. It remains to be seen whether this is also the case within insomnia populations. Our results found that concordant bedtime corresponds to the highest percent transmissions received within the first 2 h of an individual's rest interval, while having an earlier bedtime than a bedpartner offered the greatest protection. If our data suggesting the benefit of discordant bedtimes in insomnia couples are replicated in longitudinal studies or studies actively manipulating bedtime concordance, then it would have important implications for the implementation of insomnia treatments, such as CBT-I. For example, an effective CBT-I component is sleep restriction or sleep efficiency therapy, which often results in a delayed bedtime [29]. Our findings suggest this may be associated with a decrease in patients' vulnerability to receiving wake transmissions or an increase in minutes resistant to transmissions. For individuals who find bed restriction so challenging as not to be sustainable, recommending a discordant bedtime may provide an alternative sleep strategy to lower risk of external sleep disruptions. Additionally, higher percent transmissions received by partners suggests that they may benefit from targeted sleep recommendations for themselves to reduce bedpartner transmissions received (e.g., use of appropriate sleep aids such as earplugs, manipulating bedtimes to reduce possible transmissions, and sleeping in different rooms). Through enhanced knowledge of insomnia's effects on others, we can improve the assessment and treatment of this highly prevalent sleep disorder.

While this study addresses an important gap in our understanding, there are important limitations. First, the sample was comprised of community-based treatment-seeking couples, where the partner was not screened for sleep disorders. A small number of partners also experienced a variety of sleep difficulties, including insomnia symptoms and sleep-disordered breathing. This may be expected: a bedpartner's sleep influences an individual's sleep, leading to clustering of disordered sleep within couples, similar to the way health status clusters within couples [44–46]. On the other hand, the present design, therefore, has strong generalizability to clinical contexts. Second, our measurements do not allow us to identify moments where both bedpartners were woken by a common external factor, such as street noise. Countering this concern though, where an external factor did trigger wake in both bedpartners, this would typically result in wake beginning at approximately the same time for both individuals (i.e., within the same 1 min bin); therefore,

wake transmission would not be coded. Future studies will benefit from examining results of concurrent PSG in controlled settings to understand in greater depth how bedpartners influence one another's sleep in the context of sleep disorders and, even more generally, in healthy sleeping populations. As this report details novel sleep characteristics, it is currently unknown whether we are observing a typical population or whether this pathological population is characteristically different in terms of dyadic influence.

Transmission of wake provides a novel way to characterize dyadic sleep in the context of insomnia. In this sample, the relationship between patient and partner is shown to be more bidirectional than observed in other sleep disorders, such as OSA [8]. It may be important to, therefore, understand how current findings compare with healthy sleeping populations to understand the extent to which pathological factors specific to insomnia interfere with typical presentations of dyadic sleep. More broadly, our findings support the need to consider bedsharing status in both clinical and research contexts to accurately understand sleep of an individual.

Supplementary Material

Supplementary material is available at *SLEEP* online.

Supplementary Table S1: Within-person and between-person bivariate correlations for dyadic sleep measures (concordance, transmission), age, and sleep disorder symptoms (Insomnia Severity Index [ISI], STOPBang). Within-person values for patients are shaded gray and displayed above the diagonal. Within-person values for partners are shaded white and displayed below the diagonal. Between-person (i.e., Patient/Partner) correlations for each variable are shaded yellow and displayed in italics along the diagonal. Results presented are *r* (*p*).

Supplementary Table S2: Predicting square root of Percent Transmissions Received, by Dyadic Chronotype Difference, Bedtime Difference, Role, and Hour of the rest interval.

Supplementary Table S3: Predicting square root of Number Transmissions Received, by Bedtime Difference, Role, the Chronotype Difference by Bedtime Difference interaction, and the Bedtime Difference by Hour of the rest interval interaction.

Supplementary Table S4: Predicting Number Transmissions Received at the individual level, by Role, Bedtime Difference, Chronotype Difference, and Hour of the rest interval.

Supplemental Table S5: Multi-level model effects predicting average Number Transmissions Received, Percent Transmissions Received, and Percent Minutes Resistant to Transmission at the level of the dyad, by dyadic Bedtime difference and Hour of the rest interval.

Supplementary Table S6: Predicting Percent Transmissions Received at the individual level, by Role, Bedtime Difference, Chronotype Difference, and Hour of the rest interval.

Supplementary Table S7: Predicting Percent Minutes Resistant to Transmission at the individual level, by Dyadic Chronotype difference, Bedtime Difference, Role, and Hour of the rest interval.

Supplementary Figure S1: Significant predictors of Number Transmissions Received at the individual level: Role within couple, within-couple Chronotype Difference by Bedtime Difference interaction, and within-couple Bedtime Difference by Hour of the rest interval interaction. Graph displays M ± SEM. * p <.05, ** p <.01, *** p <.001.

Supplementary Figure S2: Significant predictors of Number Transmissions Received at the dyad level: within-couple Bedtime Difference by Hour of the rest interval interaction. Graph displays M ± SEM.

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Supplemental Material for Paper 3

Supplementary Table S1.
Within-person and between-person bivariate correlations for dyadic sleep measures (concordance, transmission), age, and sleep disorder symptoms (Insomnia Severity Index [ISI], STOPBang). Within-person values for patients are shaded grey and displayed above the diagonal. Within-person values for partners are shaded white and displayed below the diagonal. Between-person (i.e., Patient/Partner) correlations for each variable are shaded yellow and displayed in italics along the diagonal. Results presented are r (p).

	Age	Sleep Concordance	Wake Concordance	Number Transmissions Received	Percent Transmissions Received	Percent minutes resistant	ISI	STOPBang
Age	***.91*** ***(<.001)***	.11 (.44)	**-.35** **(.011)**	**-.31** **(.025)**	-.22 (.12)	.16 (.26)	.12 (.41)	**.51** **(<.001)**
Sleep Concordance	.06 (.67)	***.76*** ***(<.001)***	-.22 (.11)	-.26 (.065)	**-.31** **(.026)**	.11 (.44)	-.08 (.58)	.03 (.85)
Wake Concordance	**-.30** **(.034)**	**-.39** **(.005)**	***.98*** ***(<.001)***	**.59** **(<.001)**	**.40** **(.003)**	**-.64** **(<.001)**	.22 (.12)	-.15 (.30)
Number Transmissions Received	**-.32** **(.021)**	**-.43** **(.002)**	**.70** **(<.001)**	***.29*** ***(.041)***	**.87** **(<.001)**	-.04 (.77)	.24 (.082)	-.17 (.23)
Percent Transmissions Received	-.11 (.45)	**-.48** **(<.001)**	**.50** **(<.001)**	**.85** **(<.001)**	***-.27*** ***(.049)***	.18 (.20)	.18 (.21)	-.12 (.41)
Percent minutes resistant	**.38** **(.005)**	.09 (.55)	**-.53** **(<.001)**	-.07 (.65)	.12 (.38)	*.18* *(.21)*	-.02 (.88)	.09 (.51)
ISI	.03 (.83)	-.10 (.48)	.20 (.16)	-.08 (.56)	-.09 (.53)	-.17 (.24)	*.22* *(.11)*	.22 (.11)
STOPBang	**.52** **(<.001)**	-.04 (.79)	-.04 (.79)	-.24 (.09)	-.18 (.20)	-.09 (.53)	**.31** **(.027)**	*.12* *(.39)*

Significant results are displayed in bold.

Supplementary Table S2.
Predicting square root of Percent Transmissions Received, by Dyadic Chronotype Difference, Bedtime Difference, Role, and Hour of the rest interval.

Parameters	Percent Transmissions Received B	95% CI	*p*	*r2*
Fixed Effects				
Role (Partner)[a]	.08	[0.06 , 0.09]	<.001	.01
Bedtime Difference (Earlier Bedtime)[b]	-0.18	[-0.24, -0.12]	<.001	.01
Bedtime Difference (Later Bedtime)[b]	-0.14	[-0.20, -0.08]	<.001	.003
Chronotype Difference (More Evening Type)[c]	0.06	[0.02, 0.10]	.006	.12
Chronotype Difference (More Morning Type)[c]	-0.03	[-0.07, 0.01]	.20	.03
Bedtime Difference (Earlier Bedtime) [b] by Hour	0.03	[0.02, 0.04]	<.001	.01
Bedtime Difference (Later Bedtime) [b] by Hour	0.02	[0.01, 0.03]	<.001	.003
Bedtime Difference (Concordant Bedtime) [b] by Hour	0.01	[0.00, 0.01]	.015	.001
Random Effects				OR
Intercept (Dyad)	0.003	[0.002, 0.006]	<.001	1.003
Residual	0.11	[0.10, 0.11]	<.001	1.12
-2 Log Likelihood	3287.52			
AIC	3311.52			
No. of Observations	5423			
No. of Participants	52			

*p <.05, **p <.01, ***p<.001. Covariate entered: Age. [a] = Reference group: Patient, [b] = Reference group: Concordant bedtime (within 30 mins), [c] = Reference group: Concordant chronotype classification.

Supplementary Table S3.
Predicting square root of Number Transmissions Received, by Bedtime Difference, Role, the Chronotype Difference by Bedtime Difference interaction, and the Bedtime Difference by Hour of the rest interval interaction.

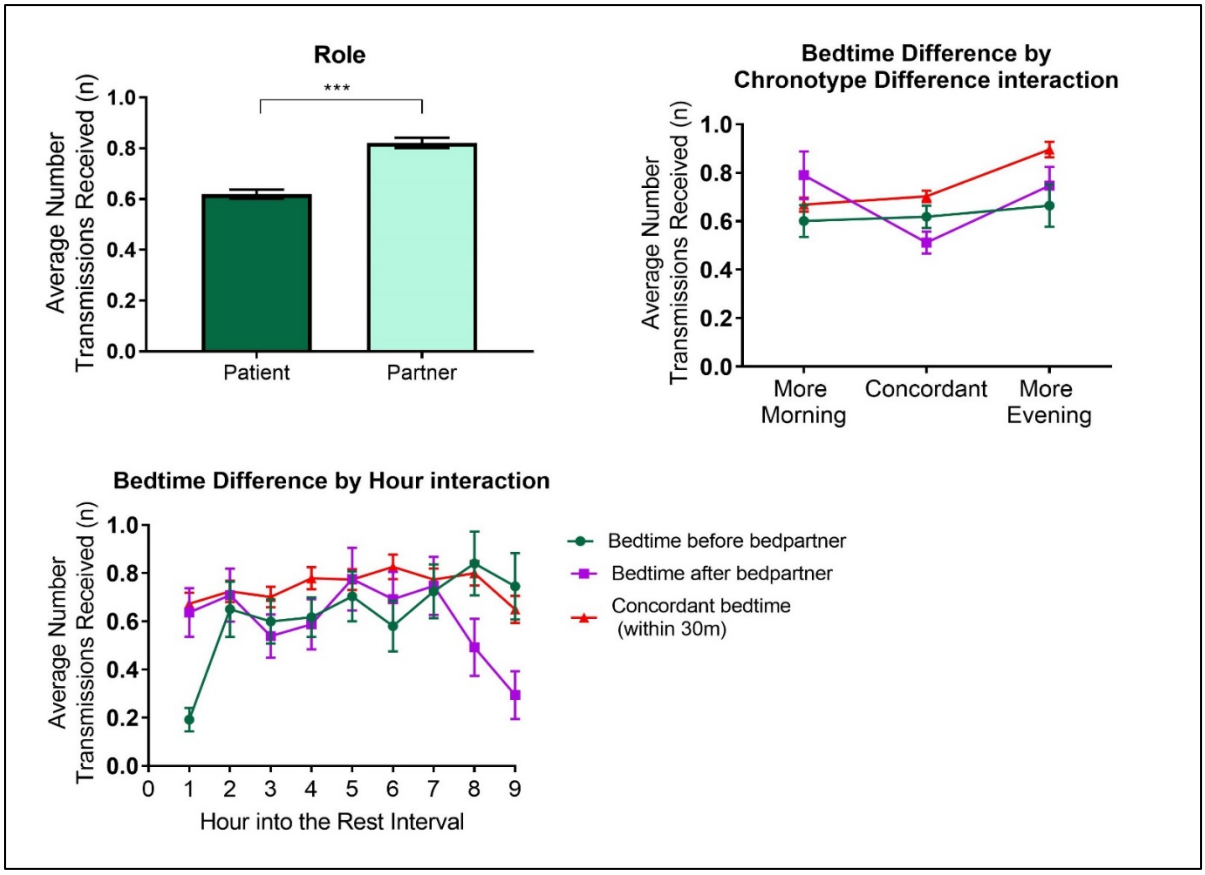
Parameters	Number Transmissions Received B	95% CI	*p*	*r2*
Fixed Effects				
Role (Partner)[a]	.15	[0.12 , 0.18]	<.001	.01
Bedtime Difference (Earlier Bedtime)[b]	-0.21	[-0.33, -0.08]	.001	.002
Bedtime Difference (Later Bedtime)[b]	-0.03	[-0.16, 0.09]	.61	<.001
More Evening Type by Earlier Bedtime	0.06	[-0.09, 0.21]	.41	.001
More Evening Type by Later Bedtime	0.12	[-0.02, 0.27]	.10	.01
More Evening Type by Concordant Bedtime	0.14	[0.04, 0.23]	.005	.11
More Morning Type by Earlier Bedtime	0.01	[-0.12, 0.16]	.84	<.001
More Morning Type by Later Bedtime	0.13	[-0.02, 0.29]	.089	.01
More Morning Type by Concordant Bedtime	-0.04	[-0.13, 0.05]	.40	.01
Bedtime Difference (Earlier Bedtime) [b] by Hour	0.03	[0.01, 0.05]	.001	.002
Bedtime Difference (Later Bedtime) [b] by Hour	-0.01	[-0.03, 0.01]	.18	<.001
Bedtime Difference (Concordant Bedtime) [b] by Hour	0.004	[-0.00, 0.01]	.32	<.001
Random Effects				
Intercept (Dyad)	0.02	[0.01, 0.03]	<.001	
Residual	0.37	[0.36, 0.39]	<.001	
-2 Log Likelihood	10119.20			
AIC	10151.20			
No. of Observations	5423			
No. of Participants	52			

*p <.05, **p <.01, ***p<.001. Covariate entered: Age. [a] = Reference group: Patient, [b] = Reference group: Concordant bedtime (within 30 mins), [c] = Reference group: Concordant chronotype classification.

Supplementary Table S4.
Predicting Number Transmissions Received at the individual level, by Role, Bedtime Difference, Chronotype Difference, and Hour of the rest interval.

Parameters	Number Transmissions Received B	95% CI	*p*	*r2*
Fixed Effects				
Role (Partner)[a]	0.22	[0.16 , 0.27]	<.001	.01
Bedtime Difference (Earlier Bedtime)[b]	-0.32	[-0.51, -0.13]	.001	.002
Bedtime Difference (Later Bedtime)[b]	-0.06	[-0.25, 0.14]	.56	<.001
More Evening Type by Earlier Bedtime	0.12	[-0.11, 0.36]	.31	.003
More Evening Type by Later Bedtime	0.20	[-0.02, 0.43]	.080	.01
More Evening Type by Concordant Bedtime	0.20	[0.05, 0.35]	.010	.10
More Morning Type by Earlier Bedtime	-0.07	[-0.16, 0.29]	.55	.001
More Morning Type by Later Bedtime	.19	[-0.05, 0.43]	.12	.01
More Morning Type by Concordant Bedtime	-.07	[-0.22, 0.08]	.37	.01
Bedtime Difference (Earlier Bedtime) [b] by Hour	0.05	[0.02, 0.08]	.001	.002
Bedtime Difference (Later Bedtime) [b] by Hour	-0.01	[-0.04, 0.02]	.42	<.001
Bedtime Difference (Concordant Bedtime) [b] by Hour	0.01	[-0.00, 0.02]	.16	<.001
Random Effects				
Intercept (Dyad)	0.06	[0.04, 0.09]	<.001	
Residual	0.90	[0.87, 0.94]	<.001	
-2 Log Likelihood	14924.07			
AIC	14956.07			
No. of Observations	5423			
No. of Participants	52			

*p <.05, **p <.01, ***p<.001. Covariate entered: Age. [a] = Reference group: Patient, [b] = Reference group: Concordant bedtime (within 30 mins), [c] = Reference group: Concordant chronotype classification.



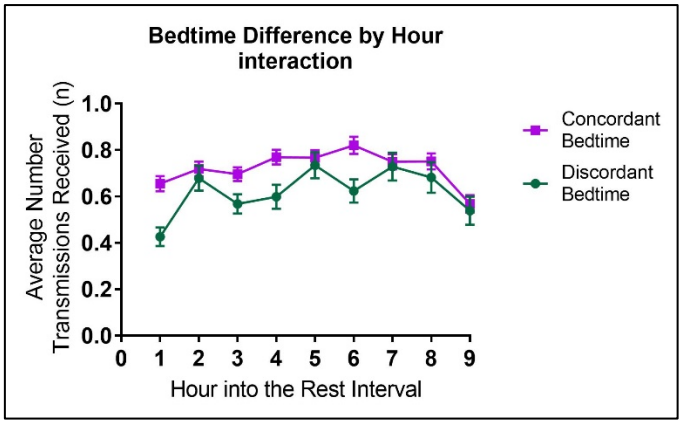
Supplementary Figure S1. Significant predictors of Number Transmissions Received at the individual level: Role within couple, within-couple Chronotype Difference by Bedtime Difference interaction, and within-couple Bedtime Difference by Hour of the rest interval interaction. Graph displays M ± SEM. * p <.05, ** p <.01, *** p <.001.

Supplemental Table S5.

Multi-level model effects predicting average Number Transmissions Received, Percent Transmissions Received, and Percent Minutes Resistant to Transmission at the level of the dyad, by dyadic Bedtime difference and Hour of the rest interval.

Variable	Number Transmissions Received		Percent Transmissions Received		Percent Minutes Resistant	
Parameters	β	95% CI	β	95% CI	β	95% CI
Fixed Effects						
Bedtime Difference (Discordant)[a]	-0.15	[-0.24, -0.06]	-0.03	[-0.04, -0.02]	0.08	[0.04, 0.11]
p	.002		.002		<.001	
r^2	.002		.05		.003	
Chronotype Difference (Discordant)[b]	0.10	[-0.04, 0.24]	0.04	[0.02, 0.07]	-0.07	[-0.12, -0.01]
p	.17		.002		.030	
r^2	.04		.005		.07	
Discordant Bedtime by Hour	0.01	[0.00, 0.03]	N/A		-0.02	[-0.05, 0.00]
p	.036				.039	
r^2	.001				.34	
Concordant Bedtime by Hour	0.001	[-0.01, 0.01]	N/A		-0.02	[-0.04, 0.01]
p	.66				.14	
r^2	<.001				.22	
Discordant Chronotype by Hour	N/A		0.001	[-0.001. 0.004]	0.01	[0.01, 0.02]
p			.30		<.001	
r^2			<.001		.003	
Concordant Chronotype by Hour	N/A		0.01	[0.004, 0.01]	N/A	
p			<.001			
r^2			.005			
Random Effects						
Intercept (Hour)	N/A		N/A		0.006	[0.00, 0.01]
p					.037	
Intercept (Dyad)	0.06	[0.04, 0.09]	0.001	[0.001, 0.002]	0.01	[0.006, 0.014]
p	<.001		<.001		<.001	
Residual	0.45	[0.43, 0.46]	0.04	[0.03, 0.04]	0.04	[0.04, 0.05]
p	<.001		<.001		<.001	
-2 Log Likelihood	11645.89		-2436.08		-1214.91	
AIC	11661.89		-2420.08		-1194.91	
No. of Observations	5423		5423		4998	
No. of Participants	52		52		52	

*p <.05, **p <.01, ***p<.001. Covariate entered: Age. [a] = Reference group: Concordant bedtime (within 30 mins). [b]= Reference group: Concordance Chronotype. N/A= Parameter not included in final model.



Supplementary Figure S2. Significant predictors of Number Transmissions Received at the dyad level: within-couple Bedtime Difference by Hour of the rest interval interaction. Graph displays M ± SEM.

Supplementary Table S6**.**
Predicting Percent Transmissions Received at the individual level, by Role, Bedtime Difference, Chronotype Difference, and Hour of the rest interval.

Parameters	Percent Transmissions Received B	95% CI	*p*	*r2*
Fixed Effects				
Role (Partner)[a]	0.06	[0.05 , 0.07]	<.001	.01
Bedtime Difference (Earlier Bedtime)[b]	-0.12	[-0.17, -0.08]	<.001	.004
Bedtime Difference (Later Bedtime)[b]	-0.11	[-0.16, 0.06]	<.001	.003
Chronotype Difference (More Evening Type)[c]	0.04	[0.01, 0.07]	.007	.11
Chronotype Difference (More Morning Type)[c]	-0.03	[-0.06, 0.00]	.053	.06
Bedtime Difference (Earlier Bedtime) [b] by Hour	0.02	[0.01, 0.02]	<.001	.003
Bedtime Difference (Later Bedtime) [b] by Hour	0.02	[0.01, 0.02]	.001	.002
Bedtime Difference (Concordant Bedtime) [b] by Hour	0.00	[0.00,0.01]	.060	<.001
Random Effects				
Intercept (Dyad)	0.002	[0.001, 0.003]	<.001	
Residual	0.07	[0.07, 0.08]	<.001	
-2 Log Likelihood	1211.38			
AIC	1235.38			
No. of Observations	5423			
No. of Participants	52			

*p <.05, **p <.01, ***p<.001. Covariate entered: Age. [a] = Reference group: Patient, [b] = Reference group: Concordant bedtime (within 30 mins), [c] = Reference group: Concordant chronotype classification.

Supplementary Table S7.

Predicting Percent Minutes Resistant to Transmission at the individual level, by Dyadic Chronotype difference, Bedtime Difference, Role, and Hour of the rest interval.

Parameters	Percent Minutes Resistant to Transmission B	95% CI	*p*	*r2*
Fixed Effects				
Role (Partner)[a]	0.04	[0.02, 0.06]	<.001	.005
More Evening Type by Earlier Bedtime [d]	0.03	[-0.07, 0.13]	.56	<.001
More Evening Type by Later Bedtime [d]	0.06	[-0.03, 0.15]	.22	.004
More Evening Type by Concordant Bedtime [d]	-0.05	[-0.12, 0.02]	.15	.02
More Morning Type by Earlier Bedtime [d]	-0.03	[-0.13, 0.06]	.51	.001
More Morning Type by Later Bedtime [d]	0.11	[0.01, 0.21]	.027	.01
More Morning Type by Concordant Bedtime [d]	-0.10	[-0.17, -0.03]	.004	.08
Concordant Type by Earlier Bedtime [d]	0.06	[-0.01, 0.12]	.084	<.001
Concordant Type by Later Bedtime [d]	0.10	[0.04, 0.16]	.001	.002
Bedtime Difference (Earlier Bedtime) by Hour [b]	-0.03	[-0.05, 0.00]	.071	.25
Bedtime Difference (Later Bedtime) by Hour [b]	-0.05	[-0.08, -0.02]	.003	.53
More Evening Type by Hour [c]	0.01	[0.01, 0.02]	.001	.002
More Morning Type by Hour [c]	0.01	[0.00, 0.01]	.087	<.001
Random Effects				
Intercept (Hour)	0.01	[0.00, 0.02]	.037	
Intercept (Dyad)	0.01	[0.006, 0.014]	<.001	
Residual	0.08	[0.078, 0.085]	<.001	
-2 Log Likelihood	1926.72			
AIC	1964.72			
No. of Observations	5334			
No. of Participants	52			

*p <.05, **p <.01, ***p<.001. Covariate entered: Age. [a] = Reference group: Patient, [b] = Reference group: Concordant bedtime (within 30 mins), [c] = Reference group: Concordant chronotype classification. [d]=Reference Group: Concordant Type by Concordant Bedtime (within 30 mins).

CHAPTER 5

Anxiety predicts dyadic sleep characteristics in couples experiencing insomnia but not in couples without sleep disorders

Chapter 5 is a manuscript currently under review at the *Journal of Affective Disorders.*

Walters, E.M., Phillips, A.J.K., Hamill, K., Norton, P.J., & Drummond, S.P.A. (under review). Anxiety predicts dyadic sleep characteristics in couples experiencing insomnia but not in couples without sleep disorders.

Foreword to Chapter 5

Results of the previous two papers highlight the importance of wake transmission to understanding sleep of a bedsharing adult. Demographic and sleep-related factors were found to predict vulnerability to wake transmission: specifically, bedtime order, role in the couple, and chronotype. However, these are unlikely be the only factors predicting dyadic sleep and so prompted the authors to examine the dyadic nature of sleep as it relates to health and well-being.

Sleep is known to be strongly linked with mental health. As described in Paper 1, bidirectional relationships between the two exist. The dyadic component of this relationship is less well understood. Whether mental health symptoms (anxiety and depression) predict one's own or a bedpartner's sleep quality is not well understood in the literature. Whether or not these symptoms predict dyadic sleep characteristics remains, to the authors' knowledge, unstudied.

Paper 4 thus addresses the question: do mental health symptoms predict an individual or their bedpartner's sleep? This question is examined using data from both of the previously described samples: couples without sleep disorders, and couples where one partner experiences insomnia.

This manuscript has been submitted to the *Journal of Affective Disorders.* It has been formatted in compliance with journal requirements. The only way it has been altered is to change the in-text references to APA style and in accordance with thesis examiner suggestions.

Declaration

Declaration of the Candidate: In the case of Chapter 5, the nature and extent of my contribution to the work was the following:

Nature of Contribution	Extent of Contribution
Formulation of research questions and experimental design, data collection, analysis, and writing the manuscript	60%

The following co-authors contributed to the work:

Name	Nature of Contribution
Andrew JK Phillips	Input into data analysis, consultation in formulation of experimental design, and critical review of manuscript
Kellie Hamill	Data collection and critical review of manuscript
Peter J Norton	Critical review of manuscript
Sean PA Drummond	Consultation in formulation of research questions and experimental design, discussion of ideas expressed in manuscript, and critical review of manuscript.

The undersigned hereby certify that the above declaration correctly reflects the nature and extent of the candidate and co-authors' contributions to this work.

Candidate's Signature:

Primary Supervisor's Signature:

Chapter 5: Anxiety predicts dyadic sleep characteristics in couples experiencing insomnia but not in couples without sleep disorders (Paper 4)

Anxiety Predicts Dyadic Sleep Characteristics in Couples Experiencing Insomnia but not in Couples without Sleep Disorders

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Abstract

Background: Anxiety and depression are commonly comorbid with sleep problems. Despite growing acknowledgement that bedpartners are important determinants of sleep quality, few studies have explored mental health as a risk factor for disrupted sleep of the bedpartner. We examined whether anxiety or depression symptoms predicted an individual's sleep or their bedpartner's sleep, in couples where one partner experienced insomnia and in couples without sleep disorders.

Methods: Fifty-two bed-sharing couples where one individual had insomnia ("Patient"), and 55 non-sleep-disordered couples completed the Beck Anxiety Inventory, Patient Health Questionnaire-9, and Insomnia Severity Index (ISI). Sleep was monitored for seven nights. Actor-Partner Interdependence Models assessed whether anxiety or depression symptoms predicted individual or dyadic sleep (wake transmission).

Results: Greater anxiety symptoms predicted increased vulnerability to being woken by their bedpartner, as well as increased frequency of waking their bedpartner up during the night in Patients with insomnia, but not in non-sleep-disordered couples. Neither anxiety nor depression symptoms predicted an individual's or their bedpartner's sleep efficiency in either subsample. However, ISI was positively predicted by own anxiety and depression symptoms for Patients with insomnia and in non-sleep-disordered couples.

Limitations: The non-sleep-disordered subsample experienced only mild symptoms of anxiety and depression, potentially reducing predictive power.

Conclusions: Anxiety may help reveal social determinants of sleep in couples experiencing insomnia. These data underscore the importance of considering sleep, the bedpartner, and affective symptoms in mental health and sleep assessments.

Key words: couples, anxiety, insomnia, wake transmission, sleep disorders, dyad

Clinical Trial: Researching Effective Sleep Treatments (Project REST), ANZCTR Registration: ACTRN12616000586415

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Highlights

- Insomnia Patients with anxiety symptoms frequently wake immediately after bedpartner
- No relationship was observed for insomnia Patients with elevated depression symptoms
- Assessments of self/bedpartner sleep should be considered in Patients with anxiety

Introduction

The relationship between mental health and sleep is well-documented (Alvaro, Roberts, & Harris, 2013; Spoormaker & Van Den Bout, 2005; Taylor, Lichstein, Durrence, Reidel, & Bush, 2005). Insomnia and sleep disturbance are diagnostic criteria for Major Depressive Disorder (MDD) and Generalized Anxiety Disorder (GAD), among many other psychiatric disorders (American Psychiatric Association, 2013). Affective symptoms and sleep difficulties likely have bidirectional relationships (Alvaro et al., 2013; Ford & Kamerow, 1989; LeBlanc et al., 2009), resulting in increased risk of affective disorders within insomnia populations (Breslau, Roth, Rosenthal, & Andreski, 1996; Sivertsen et al., 2012) and increased sleep symptoms within affective disorder populations (Goldstein et al., 2013; Monti & Monti, 2000; Spira et al., 2008).

Anxiety can contribute to difficulties with both sleep onset and maintenance, resulting in lowered sleep efficiency and total sleep time, and more time awake during the night (i.e., increased wake after sleep onset [WASO], more awakenings, and longer sleep latency [SL]) (Gould, Spira, et al., 2018; Monti & Monti, 2000; Spira et al., 2008). Even subclinical levels of trait anxiety are associated with increased self-reported WASO (Spira et al., 2008), worse global sleep quality (Gould, Karna, et al., 2018), and increased daytime sleepiness (Gould, Karna, et al., 2018). Within depressive disorders, both hypersomnia and insomnia symptoms are common (Kaplan & Harvey, 2009; LeBlanc et al., 2009; Sivertsen et al., 2012). Even subclinical depressive symptoms predict worse global sleep quality (Gould, Karna, et al., 2018).

There are many contributors to poor sleep (Hale, Emanuele, & James, 2015; Ohayon, 2005; Stamatakis, Kaplan, & Roberts, 2007). One potentially important but largely overlooked factor is the presence of a bedpartner. Most current knowledge of factors that determine sleep quality comes from research on individuals. We (Walters et al., 2020) and others (Drews et al.,

2017; Gunn, Buysse, Hasler, Begley, & Troxel, 2015; Meadows, Venn, Hislop, Stanley, & Arber, 2005; Pankhurst & Horne, 1994; Spiegelhalder et al., 2015) have shown that the bedpartner is an important determinant of sleep quality. Bedsharing adults tend to: (1) follow similar sleep schedules (Gunn et al., 2015; Hasler & Troxel, 2010; Pankhurst & Horne, 1994); (2) be awake or asleep at the same times during the night (Gunn et al., 2015; Gunn et al., 2017); and (3) transmit wake between each other (i.e., one bedpartner wakes up immediately after the other awakens during the night; Walters et al., 2020).

Considering the relationship between sleep and health in the context of bedpartners is important, since most adults have bedpartners (De Vaus, 2004; National Sleep Foundation, 2012). Although there is evidence that sleep and affective states, separately, are linked within couples (Hill, Griffiths, & House, 2015; Joiner Jr & Katz, 1999), and evidence that sleep is linked with affective symptoms within the individual, few studies have considered the sleep-affect connection in couples. A handful of studies have suggested greater depressive or anxiety symptoms in an individual may be associated with poorer sleep outcomes in the bedpartner (Chan et al., 2017; Moturu, Khayal, Aharony, Pan, & Pentland, 2011; Revenson, Marín-Chollom, Rundle, Wisnivesky, & Neugut, 2015; Strawbridge, Shema, & Roberts, 2004). However, one limitation of these studies is that sleep was assessed with limited questions such as average self-reported sleep duration (Moturu et al., 2011; Revenson et al., 2015) or global sleep quality (Chan et al., 2017; Strawbridge et al., 2004). To our knowledge, no study has assessed sleep using detailed, objective sleep assessment tools to more fully characterize the association between an individual's affective symptoms and their partner's sleep.

This study aimed to examine whether an individual's anxiety or depression symptoms predicted their own, or their bedpartner's, individual and dyadic (i.e., wake transmission between bedpartners) sleep parameters. Analysis was performed in two separate samples of couples: one sample without evidence of sleep disorders and one sample where at least one

partner experienced insomnia. Previous research has demonstrated the same dyadic variables are present in both samples (Walters et al., in press, Walters et al., 2020). Therefore, analysing both samples here allows, for the first time, a comparison between the two on both of the following hypotheses. It was hypothesized, in each sample: (1) both anxiety and depression symptoms would predict poorer sleep quality and greater wake transmission in the individual; and (2) both anxiety and depression symptoms in one partner would predict poorer sleep quality and greater wake transmissions received by their bedpartner.

Methods

Participants

The combined sample comprised 107 cohabiting couples. A subsample of 52 couples (48 different-sex; 4 same-sex) had at least one partner who experienced insomnia. These couples participated in a larger clinical trial (Project REST; Australian New Zealand Clinical Trials Registry: ACTRN12616000586415; Mellor et al., 2019) investigating partner-assisted behavioral interventions for insomnia. The other subsample of 55 couples (54 different-sex; 1 same-sex couple) did not show evidence of sleep disorders in either partner and were recruited concurrently with couples completing Project REST. Couples were recruited from Melbourne, Australia, responding to physical, online, and radio advertisements seeking couples to either participate in an insomnia intervention, or to help understand bedsharing couples' sleep. Both protocols had ethical approval from Monash University Human Research Ethics Committee.

Inclusion criteria for both bedpartners within the insomnia couples were being 18+ years old, being stable bedpartners for 1+ month, and no evidence of ongoing intimate partner violence in the relationship. The individual experiencing insomnia ("Insomnia Patient") met criteria for insomnia disorder according to the Duke Structured Interview for Sleep Disorders (DSISD-IV-TR) (Edinger et al. 2004), with the duration criterion modified to match DSM-5. Exclusion criteria included (a) any other unmanaged sleep disorders; (b) receiving behavioral intervention for insomnia within the past month; (c) unmanaged psychosis or history of manic episodes; (d) substance use disorder within the past 6 months; (e) shift work in past 6 months; (f) had returned home less than one week per time zone traveled in cases of recent transmeridian travel; (g) current pregnancy past first trimester; and (h) children <1 year old living in the house, due to significant sleep disruption that occurs for many parents in the first year. Exclusion criteria for the non-patient partner ("Insomnia Partner") were (a) unmanaged psychosis or history of manic episodes; and (b) current substance use disorder. Partners did not

undergo diagnostic assessment for insomnia. Insomnia couples had shared a bed for a mean of 22.3 years (*SD* = 16.7 years), and most (76.5%) did not currently live with children under the age of 18. The majority (37 couples) were married, 12 were de facto, and 3 were dating.

The non-sleep disordered couples were eligible if neither partner (a) displayed evidence of sleep disorders according to the DSISD; (b) used any medication to assist sleep; (c) had worked night shift work in the past 3 months; or (d) had returned home less than 3 days per time zone traveled in cases of recent transmeridian travel. Non-sleep-disordered couples had shared a bed for a mean of 7.6 years (range 1 month – 46 years), and most (76.4%) did not currently live with children under the age of 18. The majority (27 couples) were married, 12 were de facto, and 16 were dating.

Procedure

Potential participants expressed interest in both studies via email and phone; if deemed eligible after initial screening they were scheduled for an appointment. After providing informed consent, they were checked against the inclusion/exclusion criteria and completed self-report measures. Individuals then monitored their sleep/wake patterns for seven nights via sleep diary and actigraphy, concluding monitoring within two weeks (non-sleep-disordered couples) or within one month (insomnia couples) of baseline assessment.

Measures

The Insomnia Severity Index (ISI) measures insomnia symptoms over the past week (Bastien, Vallieres, & Morin, 2001; Morin, 1993). It is a 7-item self-report, where participants respond to items on a 5-point Likert scale. For the non-sleep-disordered subsample, scores ≥10 were exclusionary (Morin, Belleville, Belanger, & Ivers, 2011). ISI was not an inclusion/exclusion criterion for the Insomnia Patient or Insomnia Partner. For all participants, ISI was also used to measure insomnia symptom severity.

The STOPBang measures risk of Obstructive Sleep Apnea (OSA) symptoms (Chung et al., 2008). For Insomnia Patients and for both partners within the non-sleep-disordered subsample, scores indicating high risk were exclusionary. “High risk” was defined as either of the following two conditions: (1) overall score of 5 or above, or (2) two or more endorsements on the STOP questions, and Body Mass Index greater than 35 kg/m^2 (Chung, Yang, Brown, & Liao, 2014). This measure was not used for screening of the Insomnia Partner; data were gathered for descriptive purposes only.

The Patient Health Questionnaire 9 (PHQ-9) measures severity and frequency of depression symptoms (Kroenke & Spitzer, 2002). The PHQ-9 is a 9-item questionnaire, where each item is scored on a 4-point Likert scale ranging from “not at all” to “nearly every day,” with total scores ranging from 0 to 27. Internal consistency reliability was acceptable, sample $\alpha = .78$.

The Beck Anxiety Inventory (BAI) assesses severity of anxiety symptoms with 21 items on a 4-point Likert scale, ranging from “not at all” to “severely – it bothered me a lot” (Beck, Epstein, Brown, & Steer, 1988), For both scales, higher scores indicated greater symptom severity. Reliability was good, sample $\alpha = .85$.

The Quality of Life Enjoyment Satisfaction Questionnaire (Q-LES-Q-SF) is a 16-item self-report measure of an individual’s perceived quality of life, where individuals rate items on a 5-point Likert scale (Endicott, Nee, Harrison, & Blumenthal, 1993). Scores are expressed as a percentage. Reliability for this sample was good, $\alpha = .90$.

Sleep Diary. All participants completed a prospective sleep diary for seven nights. This included the variables: reported bedtime, sleep latency (SL), number and duration of awakenings, wake time, and rise time. Sleep efficiency (SE; percentage of the rest interval spent asleep) was then calculated.

Actigraphy. Concurrently, participants had objective sleep/wake patterns measured by actigraphy. Respironics Actiwatch Spectrum Pro and Actiware software (Respironics, Bend, OR, USA) were used to collect and analyze data in 60-second epochs. The end of the rest interval was defined differently in the two subsamples when calculating individual-level parameters (i.e., sleep diary and actigraphic SE). Within insomnia couples, the end of the rest interval was defined as the time the individual got out of bed. Within the good sleeper subsample, we used an adapted rest interval as recommended by Reed and Sacco (2016), defining the rest interval ending at the time the individual reported waking for the final time. These formulae allowed us to analyze a period most representative of a time each individual attempted to sleep. Specifically, since many individuals with insomnia lie in bed trying to fall back asleep before finally getting out of bed, the terminal awakening is not considered a reliable measure of the time the individual stopped trying to sleep in insomnia. Thus, the time the individual got out of bed is considered the more accurate point to end the rest interval for calculation of individual-level sleep measures. In contrast, within the non-sleep-disordered subsample, there were several individuals who reported intentionally spending time in bed awake after their terminal awakening but not trying to fall back asleep (e.g., reading, watching television), making the final wake time a more accurate estimate of when an individual stopped attempting to sleep for purposes of calculating individual-level sleep measures (Reed & Sacco, 2016). Nonetheless, this same time spent in bed awake not trying to sleep may very well create awakenings in a bedpartner who is trying to sleep. Thus, for the purposes of calculating dyadic sleep variables, the end of the rest interval was defined as the time the individual got out of bed in both subsamples.

Rest intervals were manually set using sleep diary entries, although rest intervals could be shortened (non-sleep-disordered couples only) or extended (both subsamples) up to 60 minutes on either side to account for times when obvious actigraphic signs of wakefulness

(non-sleep-disordered couples only) or sleep (both subsamples) occurred at either end of the window (Goldman et al., 2007; Straus, Drummond, Nappi, Jenkins, & Norman, 2015; Walters et al., 2020). Scoring the beginning of the rest interval slightly differently in insomnia compared to good sleepers is common within the literature (Ganesan et al., 2019; Goldman et al., 2007; Straus et al., 2015; Walters et al., 2020). This is because patients with insomnia often take a long time to fall asleep or wake long before they finish attempting to sleep. These periods are theoretically important to capture. All actigraphy data were double-scored by two independent researchers. Sleep detection settings were set to high sensitivity (i.e., a wake threshold of 20 activity counts per epoch).

Data Analysis

Calculation of Dyadic Sleep Variables. The development of these variables is described in detail in Walters et al. (2020). Definitions and calculations for each variable are described below.

Sleep and **wake concordance** were calculated as the percentage of all epochs throughout an individual's rest interval that were spent in the same sleep or wake state as the bedpartner (e.g., number of epochs both asleep [or awake] / total number of rest interval epochs). This is similar to methods used by Gunn et al. (2015).

Wake transmission was defined to have occurred during an individual's awakenings only when their bedpartner was awake the 60-second epoch immediately preceding their awakening. That is, if person A is awake during the preceding epoch and person B wakes up in the current epoch, this is defined as A transmitting wake to B and B receiving a wake transmission from A. Four variables describing wake transmission were calculated as per Walters et al. (2020). (i) The total number of wake transmissions occurring in an individual's rest interval (**number transmissions received).** (ii) The percentage of an individual's total awakenings coded as wake transmissions from their bedpartner (**percent transmissions**

received). (iii) The percentage of all the bedpartner's awakenings that corresponded to wake transmissions for the individual (**transmissibility**). (iv) **Percent minutes resistant** to wake transmission provided an assessment of how long an individual resisted transmissions occurring, calculated as the percentage of bedpartner wake minutes an individual slept through, within their overlapping rest intervals.

Data Cleaning. There were maximum seven nights' sleep data analyzed per couple. In both subsamples, nights where couples reported they did not share a bed were excluded (35 out of 759; 4.6%). Within the insomnia subsample, two couples indicated they slept apart on two unspecified nights. As these nights could not be identified, all nights were included within the analysis, rather than dropping those two couples entirely. Within both subsamples, there were a small number of nights where actigraphy data were unavailable due to technical issues or participant non-adherence (Insomnia subsample: 19 nights; Non-sleep-disordered subsample: 6 nights). This resulted in a total of 364 actigraphy nights across 55 couples for the non-sleep-disordered subsample and 325 actigraphy nights across 52 couples for the insomnia subsample. For sleep diaries, there were two nights (across 2 couples) within the non-sleep-disordered subsample and 32 nights (across 8 couples) in the insomnia subsample unavailable due to participant non-adherence. All data were checked for statistical assumptions (Walters, Phillips, Boardman, Norton, & Drummond, in press; Walters et al., 2020). To meet the assumption of normality, four univariate outliers defined at z-scores over a threshold of ±3.29 were Winsorised to the next most extreme score +1 (Tabachnick & Fidell, 2013): within the insomnia sample, one outlier on patient percent transmissions received and two for patient number transmissions received; within the non-sleep-disordered subsample, one outlier on transmissibility.

Statistical Approach. Sample descriptive statistics were analyzed with SPSS v.24. Kruskal-Wallis tests assessed differences among Insomnia Patients, Insomnia Partners, and non-sleep-disordered individuals for all individual and dyadic sleep variables.

Actor-partner interdependence model (APIM; Kashy & Kenny, 2000) analyses were conducted using MPLUS v8.1 (Muthén & Muthén, 1998-2007) as a framework to analyze the links between each partner's mental health and sleep. APIM is a path analysis model allowing examination of dyadic processes, providing a test of (a) whether an individual's mental health predicts their own sleep variable (i.e., actor effect); and (b) whether an individual's mental health predicts their bedpartner's sleep variable (i.e., partner effect). Depression and Anxiety were run in separate models. APIMs were created by first grand mean centering depression and anxiety scores for actors and partners. The phantom variable method was used, estimating a new parameter *K*, which shows the underlying pattern of dyadic influence (Kenny & Ledermann, 2010).

Within the insomnia subsample, as we *a priori* hypothesized differences between roles (Insomnia Patient vs. Insomnia Partner), dyads were treated as distinguishable (Fitzpatrick, Gareau, Lafontaine, & Gaudreau, 2016). Within the non-sleep-disordered subsample, as there were no systematic role differences hypothesized, dyads were treated as indistinguishable, meaning only one actor and partner effect was created for each dyad (Kenny, Kashy, & Cook, 2006).

In the insomnia subsample, model fit statistics revealed Depression and Anxiety models fit well when predicting percent transmissions received, transmissibility, SE, and ISI score, and the Anxiety models predicting number transmissions received and percent minutes resistant. However, Depression models predicting number transmissions received and percent minutes resistant, did not converge or violated model fit assumptions (Fitzpatrick et al., 2016; Tabachnick & Fidell, 2013), and therefore could not be interpreted.

In the non-sleep-disordered subsample, model fit statistics revealed that Anxiety models fit well predicting number transmissions received, percent transmissions received, transmissibility, percent minutes resistant to transmission, and ISI. Depression models fit well predicting number transmissions received, percent transmissions received, transmissibility, percent minutes resistant, ISI, and SE. All other models violated model fit assumptions (Fitzpatrick et al., 2016; Hooper, Coughlan, & Mullen, 2008; Tabachnick & Fidell, 2013), and therefore could not be interpreted.

Results

Sample characteristics and subgroup differences are displayed in Table 1.

Table 1.
Sample Characteristics

	Insomnia Patients (n = 52)	Insomnia Partners (n = 52)	No Sleep Disorders (n = 110)	p
Variable	M ± SD or n	M ± SD or n	M ± SD or n	
Age (years)	50.96±14.62[C] Range: 19 – 80	50.81±14.92[C] Range: 20 – 82	32.30±12.35[BA] Range: 18 – 72	**<.001**
Sex (n)#	35 females (67.3% females)	21 females (40.4% females)	56 females (50.9% females)	**.021**
Beck Anxiety Inventory	8.50±6.77[BC]	4.65±6.21[A]	3.56±3.63[A]	**<.001**
Patient Health Questionnaire-9	7.31±4.66[BC]	2.29±2.46[A]	1.93±2.06[A]	**<.001**
Quality of Life Enjoyment Satisfaction	66.65±13.78[BC]	76.82±9.79[A]	77.93±12.04[A]	**<.001**
ISI	16.40±3.45[BC]	4.69±4.84[A]	3.90±2.50[A]	**<.001**
Sleep Diary Sleep Efficiency (%)	71.69±14.59[BC]	86.85±7.67[AC]	95.04±3.72[BA]	**<.001**
Actigraphy Sleep Efficiency (%)	76.89±7.85	79.24±5.98	79.05±5.70	.27
Dyadic Sleep Variables				
Concordance				
Sleep (%)	63.44±9.63	65.43±8.80	66.20±6.97	.32
Wake (%)	6.58±2.96	6.64±2.89	7.52±3.35	.12
Number Transmissions Received (n)	5.35±2.65[BC]	6.93±3.21[A]	6.41±2.89[A]	**.011**
Percent Transmissions Received (%)	18.66±8.72[B]	23.55±10.19[AC]	19.20±7.79[B]	**.007**
Transmissibility (%)	18.03±7.38[BC]	23.55±7.90[AC]	20.20±6.29[BA]	**<.001**
Percent Minutes Resistant (% of bedpartner's wake minutes)	56.42±13.81[C]	58.62±12.02[C]	51.03±15.11[BA]	**.002**

χ^2 test completed. [A] significant difference from Insomnia Clients; [B] significant difference from Insomnia Partners; [C] significant difference from No Sleep Disorders.

Anxiety Symptoms as Predictor of Dyadic Sleep Parameters

Greater Insomnia Patient anxiety levels positively predicted both greater number of wake transmissions they received (p = .006), and greater number of transmissions their bedpartner received (p = .02; Table 2; Figure 1). Insomnia Partner anxiety levels did not predict either own or bedpartner (Patient) number transmissions received. Effects were present within-person (i.e., actor effects) and between-person (i.e., partner effects) for both Insomnia Patients (k = 0.35, 95% CI [-0.76, 1.50]) and Insomnia Partners (k = 1.30, 95%CI [0.16, 11.01]).

Table 2.
Actor-Partner effects for couples seeking treatment for insomnia. Dyadic Sleep Variables are derived from actigraphically determined sleep. Individual sleep parameters are derived from sleep diary entries.

	Actor Effect				**Partner Effect**			
	Patient		Partner		Patient		Partner	
	B	*p*	*B*	*p*	*B*	*p*	*B*	*p*
Predictor: Anxiety								
Number Transmissions Received	0.15	**.006**	0.12	.072	0.05	.44	0.15	**.024**
Percent Transmissions Received	0.004	**.013**	0.002	.28	0.001	.58	0.001	.52
Transmissibility	0.002	.14	0.002	.11	0.001	.76	0.003	.097
Percent Minutes Resistant	-0.22	.42	-0.31	.14	-0.04	.92	-0.41	.068
Insomnia Severity Index	0.26	**<.001**	0.21	.18	0.05	.51	0.11	.22
Sleep Efficiency	-0.001	.74	-0.001	.55	0.001	.74	-0.003	.099
Predictor: Depression								
Number Transmissions Received*								
Percent Transmissions Received	0.002	.28	-0.002	.71	0.004	.39	0.004	.22
Transmissibility	0.002	.23	-0.005	.22	0.005	.17	0.003	.13
Percent Minutes Resistant*								
Insomnia Severity Index	0.26	**.017**	0.76	.084	-0.10	.69	0.13	.51
Sleep Efficiency	-0.005	.29	0.00	.98	0.009	.28	-0.004	.19

*Model did not converge/demonstrate good fit

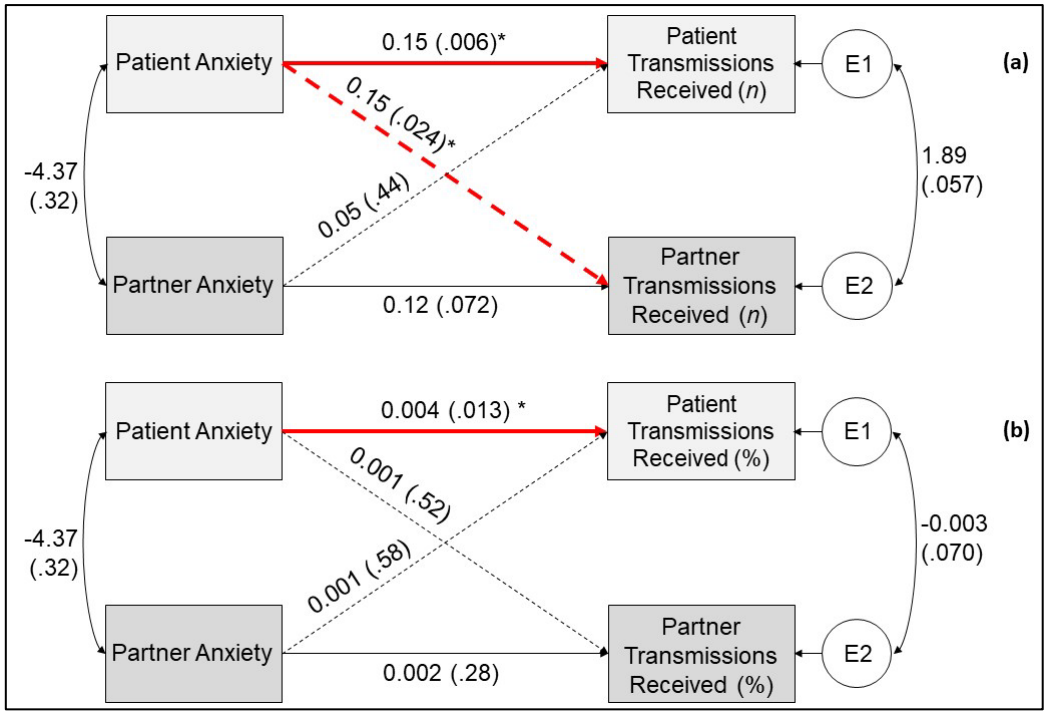

Figure 1. Within a sample where the "Patient" experiences insomnia: Significant dyadic sleep variable models, predicted by anxiety: (a) Number Transmissions Received; (b) Percent Transmissions Received. Identical models run within the non-sleep-disordered subsample were not significant.

Greater Insomnia Patient anxiety levels predicted greater percent transmissions they received (p = .01). Insomnia Partner anxiety levels did not predict either own or bedpartner percent transmissions received. Effects were present within-person for Insomnia Patients (k = 0.19, 95%CI [-0.88, 0.91]). There was no between-person effect.

Anxiety levels in the Insomnia Patient or Partner did not significantly predict any other dyadic sleep measure. For non-sleep-disordered couples, anxiety did not significantly predict any dyadic sleep variable.

Depression Symptoms as Predictor of Dyadic Sleep Parameters

Depression did not significantly predict any dyadic sleep variables in either the insomnia subsample or non-sleep disordered subsample.

Anxiety Symptoms as Predictor of Individual Sleep Parameters

Greater Insomnia Patient anxiety levels predicted greater Patient insomnia severity (p <.001). Insomnia Partner anxiety levels did not predict own or Patient insomnia severity. Effects were present within-person and between-person for both Insomnia Patients (k = 1.01, 95%CI [0.55, 1.59]) and Insomnia Partners (k = 0.99, 95%CI [0.99, 1.00]. Anxiety levels in the Insomnia Patient or Partner did not significantly predict sleep efficiency.

For non-sleep-disordered couples, the only individual sleep characteristic that was significantly predicted by anxiety in self or bedpartner was insomnia symptom severity (Table 3). Anxiety level positively predicted one's own insomnia severity (p = .004). Anxiety level did not significantly predict any sleep characteristic in the bedpartner.

Depression Symptoms as Predictor of Individual Sleep Parameters

For insomnia couples, higher Insomnia Patient depression severity predicted higher Patient insomnia severity score (p = .02). Insomnia Partner depression severity did not predict own or bedpartner insomnia severity. Effects were present within-person and between-person for both Insomnia Patients (k = 0.97, 95%CI [0.96, 1.00] and Insomnia Partner (k = 1.07, 95%CI [0.95, 1.23]), although did not meet statistical significance for all effects (Table 2).

Depression symptoms in the Insomnia Patient or Partner did not significantly predict sleep diary-derived sleep efficiency.

For non-sleep-disordered couples, the only sleep characteristic that was significantly predicted by depression symptoms in self or bedpartner was insomnia symptom severity (Table 3). Greater depression symptoms predicted increased own insomnia severity (p = .01). Depression symptoms did not significantly predict sleep efficiency in the bedpartner.

Table 3.

Actor-Partner effects for good sleepers. Dyadic Sleep Variables are derived from actigraphically determined sleep. Individual sleep parameters are derived from sleep diary entries.

	Actor Effect		Partner Effect	
	B	*p*	B	*p*
Predictor: Anxiety				
Number Transmissions Received	0.08	.34	0.11	.32
Percent Transmissions Received	0.00	.84	0.00	.91
Transmissibility	0.000	.94	0.001	.71
Percent Minutes Resistant	0.002	.60	-0.002	.52
Insomnia Severity Index	0.22	**.004**	-0.05	.48
Sleep Efficiency*				
Predictor: Depression				
Number Transmissions Received	0.02	.91	0.20	.14
Percent Transmissions Received	-0.002	.59	0.002	.58
Transmissibility	-0.002	.52	0.004	.22
Percent Minutes Resistant	0.004	.70	0.001	.84
Insomnia Severity Index	0.39	**.014**	0.17	.14
Sleep Efficiency	0.000	.93	-0.001	.38

*Model did not converge/demonstrate good fit

Discussion

The primary aim of this study was to understand how symptoms of depression and anxiety could predict dyadic and individual sleep parameters within members of couples, both with and without insomnia. Overall, anxiety predicted more aspects of sleep than did depression, dyadic sleep variables were more strongly linked to mental health symptoms than were individual sleep measures, and couples with insomnia showed many more significant relationships than did couples without sleep disorders. Specifically, for Insomnia Patients, higher anxiety symptoms predicted higher number and percent of wake transmissions received. For Insomnia Partners, higher anxiety symptoms in their bedpartner (i.e., the Patient) predicted higher numbers of wake transmissions they received. In contrast, contrary to expectations, neither subsample's mental health symptoms systematically correlated with their individual sleep efficiency. These findings suggest anxiety symptoms play a more important role than depression symptoms in determining the transmission of wake when an individual or their bedpartner experiences insomnia.

This study adds to the small but growing literature exploring sleep in a dyadic context, by exploring the association between mental health and sleep in couples. APIMs demonstrated higher anxiety levels were associated with higher wake transmission levels in insomnia couples (Table 2; Figure 1). However, models of depression did not significantly predict any dyadic sleep parameters. The reason why anxiety appears more important to understanding bedpartner sleep than depression in this population may be a result of the differing underlying pathology of the two disorders. Anxiety disorders are typically characterized by elevated levels of both cognitive and somatic arousal (Etkin & Wager, 2007; Goldstein et al., 2013; Lau, Edelstein, & Larkin, 2001), which can manifest as lower wake thresholds, leading to greater likelihood of sleep disturbance in response to external stimuli (Monti & Monti, 2000; Staner, 2003). For example, in situations where an individual is bedsharing with a partner, this low wake threshold

could put them at risk for increased receipt of wake transmissions. In contrast, depressive disorders are more heterogeneous in nature: symptoms can include either psychomotor retardation or agitation (American Psychiatric Association, 2013; Bennabi, Vandel, Papaxanthis, Pozzo, & Haffen, 2013; Hasler, Drevets, Manji, & Charney, 2004). In situations where the individual is bedsharing, symptoms of inhibition or psychomotor slowing may not confer the same risk of lowered wake threshold; therefore, this heterogeneous symptom profile may not robustly predict vulnerability to wake transmissions from the bedpartner. Hyperarousal may thus play a role in the differences observed between anxiety and depression in relation to transmissions.

We observed important role differences of anxiety when examining dyadic measures in the insomnia subsample. For Insomnia Patients, the strongest predictor of vulnerability to receiving wake transmissions from the bedpartner was the Patient's own anxiety levels. However, for Insomnia Partners, the strongest predictor of the vulnerability to receiving wake transmissions was not their own, but their bedpartner's (i.e., the Insomnia Patient's) anxiety level. The lack of actor effect for Insomnia Partners (who, while they were not screened for sleep disorders, on average reported ISI scores in normal range and higher subjective sleep efficiency than Insomnia Patients) mirrored the lack of effects observed in couples without sleep disorders (Tables 2, 3). Sensitivity to wake transmissions associated with anxiety therefore may be different depending on whether an individual has insomnia. In general, results suggest that for individuals with insomnia, it is their own anxiety levels that confer vulnerability to be awoken for both members of the couple. We hypothesize Insomnia Patients who are more anxious are more disruptive during their wake periods in bed, plausibly due to greater levels of restlessness or movement (e.g., tossing and turning, leaving the bed). For example, the anxious Insomnia Patient may become more quickly frustrated during periods of wakefulness, leading to greater levels of activity. However, there are currently no validated

ways to assess restlessness and movement throughout the night using actigraphy. While a small number of previous studies have reported nocturnal activity count statistics (Meltzer et al., 2019; Zheng et al., 2015), these methods have not been validated, nor are they recommended by current practice guidelines (Smith et al., 2018a, 2018b). Therefore, we elected not to examine them here. This currently untested hypothesis may explain why we see a partner effect in the Insomnia Partners and no effect for the non-sleep-disordered couples (as they do not have an anxious insomnia patient in bed with them). These results also support data previously published by our group (Walters et al., 2020) in which awakenings in insomnia were found to be more endogenously driven, whereas Insomnia Partner awakenings are more likely to be precipitated by wake in the Insomnia Patient.

Despite the established link between depression and insomnia (Bei et al., 2018; Bei, Ong, Rajaratnam, & Manber, 2015; Franzen & Buysse, 2008; McCall, Reboussin, & Cohen, 2000; Novick et al., 2005; Thase, 2006; Thase et al., 1997), present modeling found depression symptoms did not predict dyadic sleep in either a positive or negative direction in couples with insomnia, nor in couples without sleep disorders. Models showed positive effects of Insomnia Patient's depression symptoms on their own insomnia severity. This is consistent with prior literature in depression, observing strong relationships between depression and early morning awakening in particular (Perlis et al., 1997). In general, the findings here indicate depression may not be critically important to understanding dyadic sleep, despite the established relationship between depression and individual sleep characteristics.

A question of interest is whether the observed significant relationships are stronger in couples with insomnia than those without sleep disorders. Within the present sample, this was unable to be evaluated quantitatively due to a lack of distinguishable partner roles in the non-sleep-disordered sample. This resulted in different methodology for estimating model parameters in each subsample. Nonetheless, hypotheses as to whether meaningful differences

exist can be generated by a qualitative appraisal of the data. In those without sleep disorders, anxiety and depression symptoms each positively predicted insomnia severity, of similar magnitude to the same relationships observed in Insomnia Patients. Mental health symptoms did not significantly predict any other dyadic or individual sleep measures in those without sleep disorders. This difference suggests that presence of mental health symptomology is most relevant for dyadic sleep when an underlying predisposition to sleep disruption is already present. Additionally, subsample demographics were not equivalent. Couples without sleep disorders had milder mental health symptoms than Insomnia Patients did, and were on average, younger and in shorter relationships than insomnia couples (Table 1). Age and/or relationship length are related to dyadic sleep/wake concordance (Gunn et al., 2015; Pankhurst & Horne, 1994), meaning age/relationship length could potentially contribute to these differences. However, to provide a more quantitative analysis of reasons for observed differences, further research is required.

There are some notable limitations present within this study. First, there was a limited range of anxiety and depression symptoms within the non-sleep-disordered subsample. Participant selection criteria explicitly focused on sleep symptoms. Within our non-sleep-disordered subsample, participants were excluded in cases where it was suspected sleep problems were driven primarily by mental health concerns. Within the insomnia subsample, sleep problems driven by mental health concerns were included only if they met diagnostic criteria for insomnia. This may have reduced variability in health and well-being of the non-sleep-disordered subsample. It is possible more psychopathologically diverse samples would provide differing results. Given the high comorbidity of insomnia with both anxiety and depression, it may be challenging to achieve significant diversity in mental health symptoms while also excluding insomnia. Similarly, the insomnia partner was not assessed for insomnia (other than via ISI), or other sleep disorders. This may influence the interpretation of these

models, particularly in cases where both partners experienced insomnia symptoms or where a partner's OSA regularly disrupted the patient. Second, the two participants from the insomnia sample who reported sleeping apart on two unidentified nights did not provide details on whether disturbance from the bedpartner formed part of their decision to sleep apart. Future studies will find it valuable to monitor decision-making processes regarding whether or not to bedshare on a specific night. Third, it is possible insomnia severity influences dyadic or individual sleep parameters. To address this issue, we repeated our main analyses with ISI score as predictor (*Supplementary Tables 1 and 2)* as opposed to outcome (Table 2). We observed no significant relationship between ISI and any dyadic sleep variable. This strengthens the interpretation that vulnerability to dyadic wake transmissions increases when an individual has both insomnia (of any severity) and elevated anxiety symptoms. Fourth, it is important to acknowledge limitations in these analyses: they were correlational in design, and did not control for additional variables such as age (there were large age ranges, and samples differed in terms of mean age). For this reason, it is not possible to determine whether the cause of wake transmissions was indeed the bedpartner. Finally, there were minor differences in the way actigraphy data were cleaned between subsamples that may have had a small effect on the rest interval analysed. Cleaning differences were indicated by the literature for each population (Ganesan et al., 2019; Goldman et al., 2007; Straus et al., 2015; Walters et al., 2020). This, together with the different analyses used for each subsample, means the models are not directly comparable between groups. Despite these limitations, this work represents an important starting point for the field. The next step is to develop longitudinal and causal study designs with matched samples, for example using a combination of at-home polysomnography, audio recordings, and annotated sleep diaries to garner more specific information.

This study has direct clinical implications. For example, addressing elevated anxiety symptoms in insomnia patients may be an additional target for insomnia interventions may

result in enhanced therapeutic outcomes, including potential bedpartner benefit. This, as yet, remains untested. Ultimately, these data show for an individual who does not have insomnia, neither anxiety nor depression symptom severity appear to influence likelihood of being woken by a bedpartner, nor influence the likelihood of transmitting or receiving wake. Conversely, if an individual has both insomnia and elevated anxiety symptoms, they are more likely to wake their bedpartner and are more vulnerable to being woken by their bedpartner. These findings add to our knowledge of the intersection between psychopathology and sleep, in one of the most formative and encompassing relationships experienced by many adults: that of the bedpartner.

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Supplemental Material for Paper 4

Supplementary Table 1.

Actor-Partner effects of Insomnia Severity for couples seeking treatment for insomnia. Dyadic Sleep Variables are derived from actigraphically determined sleep. Individual sleep parameters are derived from sleep diary entries.

	Actor Effect				**Partner Effect**				**Pattern of Influence**
	Patient		Partner		Patient		Partner		
	B	*p*	*B*	*p*	*B*	*p*	*B*	*p*	
Number Transmissions Received	.17	.084	-.09	.28	.06	.52	.22	.13	
Percent Transmissions Received	.002	.58	-.002	.36	.004	.18	.003	.49	
Transmissibility*									
Percent Minutes Resistant	-.25	.68	-.40	.28	.53	.22	-.06	.91	
Sleep Efficiency	-.004	.43	-.007	**.003**	.003	.54	-.010	**<.001**	Couple pattern Partner *k* = 0.72, 95%CI [0.19, 2.41]

*Model did not converge or display adequate fit.

Supplementary Table 2.

Actor-Partner effects of Insomnia Severity for couples without evidence of sleep disorders. Dyadic Sleep Variables are derived from actigraphically determined sleep. Individual sleep parameters are derived from sleep diary entries.

	Actor Effect		Partner Effect	
	B	*p*	*B*	*p*
Number Transmissions Received	0.08	.41	0.06	.55
Percent Transmissions Received	0.000	.94	0.001	.80
Transmissibility	0.002	.29	0.001	.64
Percent Minutes Resistant*				
Sleep Efficiency	-0.003	**.005**	-0.002	.11

*Model did not converge or display adequate fit.

CHAPTER 6

Integrative Discussion

Chapter 6: Integrative Discussion

Overview of Aims and Hypotheses

The importance of sleep for health and well-being is well-documented. While various factors that may affect an individual's sleep have been described in the literature, research into interpersonal aspects of sleep, particularly with a bedpartner, is in its infancy. Despite acknowledgement that for many adults sleep is shared with a bedpartner, the role of the bedpartner in affecting sleep quality remains relatively unexplored. Thus, this thesis aimed to respond to this identified gap by investigating dyadic sleep, that is, sleep in adult bedsharing couples, in 1) good sleeping couples, and 2) couples seeking treatment for insomnia.

Specifically, this thesis aimed to:

(1) Outline the current state of the literature on dyadic sleep, and why factors affecting sleep are important to understand by considering the close relationship between sleep and health;

(2) Characterise how bedpartners influence each other's sleep, in couples with and without insomnia;

(3) Identify sleep-related and demographic factors predicting vulnerability to wake transmission, in couples with and without insomnia;

(4) Examine whether anxiety or depression symptoms predicted an individual or bedpartner's sleep, in couples with and without insomnia.

In order to meet these aims, a book chapter (Paper 1), literature review, and three empirical studies (Papers 2-4) were completed. This final chapter will integrate the key findings of the thesis, discuss theoretical, methodological, and clinical implications of the findings, evaluate the strengths and limitations of the research, as well as provide recommendations for future research.

Overview of Findings

Paper 2 investigated couples without evidence of sleep disorders, characterising dyadic sleep features of wake concordance and wake transmission, and identifying whether characteristics (individual sex, concordant/discordant bedtime, concordant/discordant chronotype) could predict levels of wake transmission. Couples completed validated self-report assessments of chronotype, then monitored sleep via sleep diary and actigraphy for seven nights. This allowed in-depth analysis of dyadic sleep across multiple nights, and specifically at effects relative to hour into the rest interval.

First, specific features of dyadic sleep were conceptualised, and the rate at which they occurred in a non-sleep disordered population were described. This included: epoch-by-epoch sleep and wake concordance (i.e., minutes both partners were both coded as being in the same sleep or wake state, M = 66.8% and M = 6.8% respectively), total transmissions received (number of awakenings immediately preceded by a bedpartner's wakefulness; M = 6.0 times per night), percent transmissions received (percentage of an individual's total awakenings which were transmitted by the partner; M = 18.9% of all awakenings), transmissibility (percentage of all bedpartner awakenings transmitted to an individual; M = 20.0%), and percent minutes resistant to transmission (ability to sleep through bedpartner wake; M = 52.1%). These characteristics of dyadic sleep were found to occur at rates higher than would be expected by chance.

Second, data demonstrated having a shared bedtime predicted the highest rate of wake transmissions and the lowest percent minutes resistant to transmission. Differences were observed only at the beginning of rest intervals. Third, bedpartners with later chronotypes than their partners had higher percent minutes resistant to transmission (compared with those who had same or earlier chronotypes than their partners). However, later chronotype bedpartners also received the most wake transmissions, specifically on nights when they had approximately

shared bedtime (compared with same/earlier chronotype partners and other bedtime configurations).

These findings suggest there are additional processes underpinning dyadic sleep compared with those typically acknowledged in the literature, and demonstrate the complexity of dyadic sleep in couples without sleep disorders.

Paper 3 aimed to conduct parallel analyses to Paper 2, with participants this time drawn from couples with one partner seeking treatment for insomnia. Aims and protocol were the same as Paper 2. Partners received wake transmissions at 1.25 times the rate of patients, indicating having a bedpartner with insomnia is disruptive to sleep. Percent wake transmissions received and percent wake minutes resistant to transmission were predicted by within-couple differences in chronotype and bedtime. Shared bedtimes and having a more evening chronotype than a bedpartner conferred highest risk of wake transmissions regardless of role status (i.e., whether the individual was a patient or partner), especially at the beginning of the night. These findings provide insight into one social mechanism of insomnia generation and maintenance, and the level of risk experienced by the bedpartner.

Paper 4 investigated mental health symptoms of both samples who comprised Paper 2 and 3 in more depth. Anxiety symptoms were found to be more important for understanding dyadic sleep characteristics than were depression symptoms. Greater anxiety symptoms in patients with insomnia predicted increased total wake transmissions received and percent transmissions received; as well as frequency of wake transmissions transmitted to the bedpartner (i.e., partner total transmissions received). Anxiety symptoms in the subsample of couples without sleep disorders did not predict dyadic sleep characteristics. Neither anxiety nor depression symptoms predicted a consistent pattern of individual-level sleep diary parameters, however insomnia severity was positively predicted by own anxiety and depression symptoms in both couples with and without insomnia. This research indicates anxiety may help

understand psychosocial determinants of sleep in couples experiencing insomnia, particularly those cases with elevated anxiety symptoms.

Discussion of Findings

Throughout the thesis, the following themes within the research emerged. Each is considered in context of the current literature.

Conceptualising the Dyadic Nature of Sleep.

This study built on prior studies of dyadic sleep by developing novel metrics for characterising and quantifying bedpartner influence on sleep. Specifically, the concept of wake transmission was defined, and four key variables describing elements of transmission were developed: number of transmissions received, percent of wakes that were transmissions received, percent of a bedpartner's wakes which were transmitted to an individual (transmissibility), and the percent of minutes an individual was able to resist transmission from a bedpartner. These metrics describe elements of dyadic sleep which have previously not been quantified in detail.

Previously, the most accepted measure for assessing dyadic sleep was the process of sleep/wake concordance. This has been published in several studies by Gunn and colleagues (e.g., Gunn et al., 2015; Gunn et al., 2017) and was replicated within the present thesis. The measure of concordance has several advantages, which are also held by wake transmission: (1) it assesses an aspect of sleep truly novel to dyadic sleep rather than individual sleep; (2) it is simple to assess and quantify. The present study's concordance results were consistent with prior literature, demonstrating rates of approximately 74-75% in non-sleep-disordered couples.

Despite similar findings, it is worth noting the differences between concordance definitions used here compared with Gunn et al. (2015). Firstly, the time period defined as the rest interval is not standardised in the literature. Gunn et al. (2015) elected to use the maximal dyadic window, i.e., beginning the moment the first partner of the couple attempted sleep, and

ending when the last member of the couple got out of bed. They reported this was intended "to capture the broadest range of couples' sleep and sleep behaviours" by describing a single concordance statistic for each night (Gunn et al., 2015). In contrast, the present study defined the analytic period as the individual's rest interval, creating one concordance statistic per person, per night. This was chosen in order to consider functional outcomes for an individual and standardise the rest interval selected to compute wake transmission variables. Given most of an individual's rest interval overlapped with that of their bedpartner, each method has its own advantages. It will be interesting to note which alternative, if any, becomes the standard within the literature. Secondly, while Gunn et al. (2015) reported a single sleep/wake concordance statistic, we propose the usefulness of reporting sleep and wake components of concordance separately. This may be important when assessing contexts such as insomnia or other sleep disorders, where there are questions about the different importance and function of wake periods, versus sleep. When combining sleep and wake into one measure, the measure is also sensitive to sleep quality. As most of the night is concordant sleep rather than wake, the measure biases towards sleep quality. If one partner were to have more disrupted sleep, this would be reflected in the concordance calculated for both members of the couple. Future studies may wish to control for sleep quality while attempting to uncover the specific role of concordance within other relationships. A suggestion for future research is to directly compare both methods for deriving concordance.

A final consideration when discussing concordance is to consider its theoretical underpinnings. The purpose of concordance has been posited as characterising coregulation within couples. This is important when viewing couples in light of their physiological linkage (Butler, 2011; Timmons et al., 2015) and the extent to which they may coregulate their body systems. However, this falls short of describing the influence bedpartners may have on one

another (i.e., how coregulation may occur). Wake transmission provides a mechanistic hypothesis for the "how".

This thesis introduces the concept of wake transmission and is the first to quantify its role and frequency in couples with and without insomnia. By mapping wake transmission and nominating variables which can be used to describe bedpartner influence (i.e., total number of transmissions received, percent transmissions received, percent minutes resistant to transmission), this paper has furthered the literature on dyadic sleep. This thesis is not the first time sleep in dyads has been considered. Two previous studies have attempted to characterise dyadic sleep variables, with similarities and differences from the present thesis. Preliminary research has demonstrated an individual's physical movements overnight can precede awakenings in a bedpartner (Meadows, Venn, Hislop, Stanley, & Arber, 2005; Pankhurst & Horne, 1994). To the author's knowledge, studies to date have reported only the percent of wakes preceded by bedpartner movement: no studies to date have considered multiple aspects of these transmissions (e.g., the similarities and differences among total number of occasions, percent wakes, and how long one was able to "sleep through" a bedpartner's nocturnal awakening). Interestingly, taking into account different definitions, previous movement-based analyses have found approximately 30-40% of all awakenings are preceded by wake or movement in the bedpartner (Meadows et al., 2005; Pankhurst & Horne, 1994). This is substantially higher than the present research which found 18-25% of all awakenings were preceded by a wake bout in the bedpartner, which may be due to differences within the samples (e.g., different ages, relationship lengths, and bed sizes; all of which are reported differently or not at all in each of the above studies, preventing direct comparison with the present research), or methodological differences (e.g., differences in devices used, as well as wake/movement threshold and epoch length settings used for actigraphy devices).

An important consideration is the complementary roles played by the concepts described above: concordance and transmission. Combining wake transmission data with concordance data may be a step towards interpreting the mechanism by which couples' sleep and wake coregulates. Having a minimal ability to resist wake transmissions from a partner, particularly a bedpartner who is restless or wakes frequently (and thus provides greater opportunity for wake transmissions to occur), may result in elevated concordance. This combination could thus contribute to development of acute and/or chronic insomnia. These new metrics for describing dyadic sleep might even help us to understand an additional predisposing factor for insomnia in the context of the 4-factor model (Perlis et al., 2010) – i.e., a potential trait towards vulnerability to receiving wake transmission - or might help understand the role of a bedpartner in the transition from acute to chronic insomnia (which is poorly understood at present). However, given the preliminary nature of this research, there may be additional processes underlying dyadic sleep that future research may uncover.

Factors Affecting Dyadic Sleep.

We did not observe vast differences in dyadic sleep variables between good sleepers and those with insomnia. Indeed, it was remarkable how similar these populations appeared in relation to sleep-related behaviours and characteristics within Papers 2 and 3.

The first key factor which predicted dyadic sleep was role status of an individual's bedpartner, rather than of the individual themselves. Specifically, in terms of percent wake transmissions received, insomnia patients and individuals in couples without sleep disorders - both of whom were sharing a bed with an individual not seeking treatment for a sleep disorder - had similar rates of transmissions (18.6% and 19.2% respectively); while insomnia partners had significantly elevated rates of transmissions (23.5%). That is, almost 1 in 4 awakenings in someone sharing a bed with a partner who had chronic insomnia was transmitted by that insomnia patient, whereas when someone's bedpartner did not have insomnia, just under 1 in

5 of their awakenings were transmitted to them. This comparison is presented most clearly in Table 1 of Paper 3. It highlights the vulnerability that lies with bedpartners of sleep-disordered individuals and echoes complaints made by partners of individuals with OSA (Henry & Rosenthal, 2013; Luyster et al., 2016; McArdle et al., 2001). However, it was interesting there was less resistance to transmissions in the non-clinical sample (asleep for 51% of bedpartner's wake minutes) than in couples with insomnia (asleep for 56-58% of bedpartner's wake minutes). This may be due to there being a smaller number of wake minutes in total, or due to the fact that the non-clinical sample were, on average, younger and in relatively newer relationships than the insomnia sample, both of which were associated with higher rates of sleep/wake concordance in prior literature (Gunn et al., 2015; Pankhurst & Horne, 1994).

A second factor which helps explain the rate of wake transmissions was within-couple bedtime order. Consistent in both the non-clinical and insomnia sample, shared bedtimes were associated with the highest rates of percent transmissions at the start of an individual's rest interval. This somewhat conflicts with prior literature, which indicates couples prefer to go to bed at the same time (Revenson et al., 2015). It suggests couples' preference for a shared bedtime may be for reasons other than improved sleep quality: for example, this time together may serve an important function unrelated to sleep, such as maintenance of relationship quality or comfort (Rosenblatt, 2006). The findings appear to be driven by two factors. Firstly, concordant bedtimes offer a greater opportunity for transmissions to occur in the initial hours of the rest interval (it is impossible for a transmission to occur if the bedpartner is not yet in bed). Discordant bedtimes offer the individual in bed earlier an uninterrupted sleep opportunity. Secondly, if a bedpartner begins their rest interval at a different time, they may be in different stages of sleep. For example, the individual in bed first may be in a deep stage of sleep when the partner begins their own rest interval, protecting the former against wake transmissions. That later bedpartner would also have less disturbance from a deep-sleeping earlier partner.

Consistent with expectations, having the same chronotype as one's bedpartner was mildly advantageous for low wake transmissions both in couples with and without insomnia. This supports prior literature which has emphasised the advantages of concordant chronotypes within couples (Larson et al., 1991; Randler & Kretz, 2011). However, our findings of the advantage of a shared chronotype was actually representative of a compromise: particularly in couples with insomnia, those who were more morning-type received fewest transmissions while those who were more evening-type received most transmissions. Therefore, for the benefit of the couple overall, shared chronotype had the best outcomes. While chronotype is generally considered a trait factor, it can reflect a preference for time of day (Roenneberg, 2015), which could change over time. Thus, it could likely be changed over time, based on changing one's sleep schedule, light exposure, times of activity, and exposure to other *zeitgebers*. This may provide some insight into how a "compromise" chronotype could develop, perhaps even unintentionally, simply from a couple co-regulating activities. Previous studies which have investigated the dyadic importance of chronotype order have tended to separate couples into those with same vs. different chronotypes (Larson et al., 1991). As this thesis has shown, shared can represent a middle ground. As such, future research should consider the differential impact of specifically having the earlier or later preference.

In terms of mental health findings; anxiety levels predicted vulnerability to wake transmissions such that we need to consider the intersection between anxiety symptoms and presence of insomnia. Anxiety predicted wake transmissions only when an individual had both insomnia and elevated anxiety symptoms. Our results replicated and extended findings of the anxiety/sleep relationship (Chan et al., 2017; Revenson et al., 2015; Strawbridge et al., 2004). However, our results also differed from part of those by Chan et al. (2017), as we did not find depressive symptoms predicted bedpartner sleep or levels of wake transmissions. This may be due to different methods for assessing sleep (i.e., previous studies assessed sleep using limited

questions such as average self-reported sleep duration, or global sleep quality, in which sleep symptoms may also be symptoms of depression). A significant strength of this study was that we modelled how anxiety and depression predicted both individual and dyadic sleep variables. By doing this, we were able to provide preliminary suggestions why prior literature has not reached consensus. While individual/overall sleep quality could not be predicted by subclinical anxiety and depression symptoms, dyadic sleep variables could. This informs future research into both individual and dyadic sleep patterns, and underscores the value of dyadic sleep measures. Since more individual-level sleep parameters were not predicted by mental health symptoms, we would have missed potentially important sleep/mental health interactions if we did not examine dyadic sleep.

Given the remarkable similarities in findings related to sleep-related behaviours and characteristics observed in Papers 1 and 2, the difference between the two samples in Paper 3 is notable. Lack of findings in non-clinical samples suggest one of two things: (1) the vulnerability of insomnia is required in order to observe the effect of mental health symptoms on top of that; or (2) our non-clinical sample did not experience severe enough mental health symptoms to observe the effect on bedpartners. Given the close relationships between depression/anxiety and an individual's sleep, this is not surprising, as individuals with more elevated symptoms may have been excluded from the sample due to the effect these had on their sleep.

Combining this finding with the sleep-related predictors described in Papers 1 and 2, it may be that when an individual has insomnia and elevated anxiety symptoms, they may elect to alter their sleep habits; for example, to go to bed at the same time as their bedpartner to seek reassurance, and the attachment-related security described in the introduction to this thesis. This may increase their vulnerability even further. Such analyses were outside the scope of this thesis but would be valuable lines of questioning for future research.

Results may preliminarily suggest greater levels of concordance and transmission are not necessarily detrimental. Sleep linkage may serve an additional function for the relationship (Gunn et al., 2015; Gunn et al., 2017). Of course, greater concordance and transmission may serve different functions depending on the individual or partner's reaction to being woken up: for some, a source of frustration (Luyster et al., 2016); for others, a neutral or a pleasant reaction. An example of this final reaction would be if an individual does not realise the bedpartner is the source of wake, but instead notices their partner interacts with a hug or other expression of affection (Rosenblatt, 2006).

Methodological Implications

The use of multiple measures and development of specific metrics to assess sleep in couples are significant strengths of this thesis. The data presented here supports the prior findings that couples' sleep is related, but the novel methods herein have shown this relationship is likely more complex than previously reported. This provides valuable information to researchers and clinicians about the potential impact of a bedpartner.

Subjective vs. Objective Measures of Dyadic Sleep.

A combination of subjective and objective measures of sleep were assessed within these studies. This revealed that when assessing couples, the impact of the bedpartner was not observable in subjective measures. In order to record the impact of the bedpartner, objective measures are required, such as actigraphy (used in Papers 2-4) or PSG (piloted previously by Drews et al. (2017) and Monroe (1969), but yet to be systematically assessed in a well-powered study). There are currently no validated ways to accurately assess bedpartner impact via subjective report. Thus, the best recommendation to researchers and clinicians based on this work would be to use a combination of subjective and objective measures if assessing dyads in order to understand their differential effects. Where subjective measures are used, individuals should be asked to specifically report any perceived disturbance related to the bedpartner.

Overall, results highlight the importance of not relying on subjective reports of bedpartner sleep disturbance as an accurate measure of the extent to which wake transmissions occur. Results suggest where an individual does subjectively report they are aware of bedpartner disruption, sleep disruption is likely of a substantial level and so further assessment is warranted. Given the multidimensional nature of sleep, researchers and clinicians should choose carefully, driven by the working formulation, which style of sleep assessment would be most appropriate.

Design Features for Novel Metrics.

A significant strength of this thesis is the design of novel metrics, and suggestions we can make for future research in this field. During development we utilised the guiding principle to describe variables which were as simple, as interpretable, and as useful as possible. In particular, the definition of wake transmission was simple and intuitive, thereby increasing likelihood that future research can utilise these metrics, attempt to replicate these findings, and expand the literature using this methodology.

Using both raw total "occurrences per night" as well as expressing transmission as a percentage (percent wake transmissions received; percent minutes resistant to transmission), we normalised the occurrences so that interpretation was less biased by an individual's sleep quality. Additionally, by presenting both raw and percentage transmission variables, it became apparent each may have assessed slightly different information. For example, raw "occurrences per night" as well as "percent of all wakes by Partner A which were transmitted to Partner B" (transmissibility), provide insight into the *transmitter*: how frequently did they transmit wake to their partner? Conversely, "percent of all Partner B's wakes which were coded as being transmitted from Partner A", appears to provide insight into the *receiver*, and may be a more functional variable. It may give insight into questions such as: what effect does wake transmission have on the receiver? How likely is Partner B to perceive that a factor waking

them up is their bedpartner? Of course, currently no data relating to partner awareness of their bedpartner's awakenings or daytime functioning afterwards were collected. While these interpretations at this time are only tentative, future research should continue to consider the different role differences (i.e., transmitter vs. receiver) each variable encompasses.

Future studies, whether they use the present definitions and calculations or refine them further, should continue holding these guiding principles in mind. While previous studies have suggested incorporating raw activity counts as a measure of active wake to assist in determining when wake transmission occurs (Meadows et al., 2005; Pankhurst & Horne, 1994), there are currently no validated ways to assess restlessness and movement throughout the night using actigraphy. Considering movement in the present and surrounding epochs are accounted for within the actigraphic algorithm for determining sleep or wake status, we elected to use only this validated algorithm rather than using methods not currently recommended by practice guidelines (Smith et al., 2018a, 2018b) to emphasise the importance of simplicity and applicability of the metrics. Of course, the actigraphy algorithm is not perfect: validation studies show high sensitivity (for sleep epochs assessed as sleep) but relatively low specificity (for wake epochs assessed as wake; de Souza et al., 2003; Hyde et al., 2007). Thus, there are likely many PSG wake epochs missed by actigraphy, which may have affected the present recording of wake transmissions. Future PSG studies may find different results, particularly as periods of quiet wakefulness are more likely to be picked up.

Considering Bedpartner Status in Research Studies.

To date, it is rare to consider the bedpartner when assessing sleep. We propose here that it may be important to do so. Present data, together with previous findings, suggest there may be differences between sleep quality and sleep continuity, as well as behavioural choices such as sleep opportunity and sleep schedule when bedsharing compared with sleeping alone (Dittami et al., 2007; Rogojanski et al., 2013; Rosenblatt, 2006; Spiegelhalder et al., 2015). It

is thus surprising and a little concerning that research rarely asks for, or reports, bedsharing status. Our research along with others (Gunn et al., 2015; Pankhurst & Horne, 1994) suggest the effect of a bedpartner is associated with age and/or relationship length, with younger individuals more closely linked with their bedpartner. For the purpose of research, this is important to consider in cases such as many experimental studies which recruit young, healthy university students - who may be in short-term bedsharing relationships and thus be the most vulnerable to sleep disturbance by that bedpartner.

Theoretical and Clinical Implications

The results of this thesis have important implications for theory and practice. Firstly, theories related to couples and sleep have largely been conceptualised in terms of attachment and coregulation (Carmichael & Reis, 2005; Troxel, Cyranowski, et al., 2007). This thesis provides evidence that the mechanism underpinning sleep coregulation may be movement disrupting the person next to you. Of course, an individual's sensitivity to being woken by that individual may be explained by coregulation of physiological mechanisms. Both coregulation of sleep and at least some physiological parameters may, in turn, be influenced by social factors such as choices around sleep timing and activities around the sleep onset and sleep offset periods. Future research will be required to disentangle the direction(s) of these relationships.

Secondly, within clinics, we recommend bedsharing status be assessed routinely, much as are other sleep behaviour and environmental factors. Sleep does not occur within a vacuum and the psychosocial context should be considered. Currently, where clinicians do ask questions about a bedpartner's impact, they do so without guidelines or recommendations guiding this line of questioning. The lack of routine assessment is evidenced by a lack of validated questionnaires from which clinicians can draw. However, questions can be guided by the recommendations related to a clinical interview with a patient's partner outlined by Rogojanski et al. (2013). This interview, however, focuses on a bedpartner's knowledge of the

patient's sleep difficulties through questions such as "How severe do you perceive your partner's sleep difficulties to be?" and "How routine is your partner's daytime and sleep schedule?". Adding a question such as "How do your partner's sleep difficulties impact on your own sleep?" or "Does your partner report being disrupted by your own sleeping habits?" would allow clinical insight into the bedpartner aspect of the sleep experience. It is hoped this thesis and the publications arising from it promote interest in the field and validated assessment tools assessing common bedpartner complaints can be developed.

This thesis provides important data which can be provided to individuals with sleep difficulties, particularly where anxiety symptoms are also elevated. Specifically, these data can help normalise the experience of sleep coregulation and wake transmission. For patients who are concerned about disturbing their bedpartner, reassurance can be offered that transmissions are not commonly remembered the next day, as well as reassuring both partners the vast majority of their bedpartner's awakenings are not transmitted to them. In fact, insomnia patients can be reassured they are not more likely than non-sleep-disordered sleepers to be awoken by their bedpartner. Where anxious thoughts or attempts to actively avoid waking the partner up act to perpetuate or exacerbate sleep difficulties, normalising the dyadic aspects of sleep may be an important way to target cognitive components of insomnia. Nonetheless, considering the interrelationship observed between anxiety and dyadic sleep, this thesis emphasises the importance of considering sleep in all anxiety assessments and anxiety in all sleep assessments. This will further inform the likely extent of bedpartner impact and inform strategies for symptom reduction.

It is worth noting that in future, there may be promise in assessing dyadic sleep using novel, commercial-grade assessment tools (e.g., watches, headbands, mattresses). However, due to the scarcity of published data, we are unable to speculate further on their usefulness. If data regarding their validity and reliability are published, they may show promise as an

affordable alternative for concurrently monitoring two people. With the rise in popularity of commercial-grade wearable sleep monitoring devices there is potential for couples to monitor their own sleep and make consumer comparisons between themselves. It goes beyond the scope of this thesis or its results to comment on the validity or utility of these potential comparisons, however clinicians should inform themselves of the available literature to negotiate these conversations while not providing unhelpful misinformation.

This thesis has identified the bedpartner of an individual with insomnia is somewhat compromised (i.e., they experience more frequent wake transmissions per night, and a larger percentage of all their awakenings are preceded by wake in their bedpartner). Having identified this group as a compromised population, strategies can now be implemented to assist with their sleep as well as the patients'. This may take the form of suggesting "simple" behavioural fixes such as wearing earplugs or an eye mask; having separate bedtimes or at least a shared couple conversation about each partner's desired sleep and wake times, and whether it is important to them to go to bed or rise at the same time. Facilitating these conversations may serve to improve sleep and well-being of both members of the couple. Sharing information about the relative likelihood of receiving wake transmissions between patient and partner (i.e., that that partner is more likely to be awoken by the patient than vice versa) may also enhance partner support for the aims of behavioural interventions such as CBTI. It also taps into something which remains unanswered: taking a holistic view of the person or couple, is it beneficial or harmful to have greater sleep/wake concordance and greater wake transmissions? Whilst on a superficial level, it is easy to assume more interrupted sleep leads to poorer daytime and physiological outcomes, dyadic sleep interruption is not associated with typical daytime complaints of interrupted sleep (Monroe, 1969). This was supported by the present data which showed no significant correlations between ISI scores (which measure daytime complaints of sleep disturbance) and dyadic sleep measures (non-sleep-disordered sample results shown in

Paper 2, Table 2; insomnia sample data not shown). Therefore, it is short-sighted to assume shared wakeful moments over the night may not serve some important function for relationship quality or attachment, and this should be considered further in future research.

Strengths of the Thesis

The three empirical studies included within this thesis explored novel concepts, a) characterising how bedpartners influence each other's sleep, in couples with and without insomnia, and b) identifying a range of factors that predict vulnerability to bedpartner-driven sleep disruption. By doing so in naturalistic sleep settings which allowed for strong ecological validity due to presence of typical contextual factors, and including both cross-sectional and longitudinal research methods with large datasets, all studies provide important steps in better understanding the dyadic nature of sleep.

First and foremost, a strength of this body of research is the multiple populations examined. By replicating this study in a population where neither bedpartner experienced insomnia (Paper 2), and a population where one partner actively sought treatment for insomnia (Paper 3), comparisons can be drawn between the two groups. It is important to note dyadic sleep is an area of research which has only really received attention in the past two decades (Troxel, 2010). As such, previous bodies of research have focused on a single population, predominantly, good sleeping couples (Gunn et al., 2015; Pankhurst & Horne, 1994; Spiegelhalder et al., 2015). The clinical implications of dyadic sleep and extent of wake transmission occurring may be somewhat different in good sleeping couples as compared with couples experiencing insomnia. While data presented in this thesis emphasised similarities between good sleepers and those with insomnia in Papers 2 and 3, Paper 4 suggested a more unidirectional influence in the context of comorbid insomnia and anxiety. There is theoretical and clinical suggestion that sleep disruption may similarly be more unidirectional within OSA (Henry & Rosenthal, 2013; Luyster et al., 2016). Meanwhile, patients commonly report to

clinicians that they are either frequently disrupted by a bedpartner, or conversely, that their partner “sleeps like a log” and is never woken throughout the night. Frequently this is a source of frustration and can act to perpetuate cognitive contributors to insomnia symptoms. Having normative data available to clinicians about the frequency and normality of wake transmissions, both in insomnia and good sleeping populations, may be useful to share with clientele to provide context and validation of their own experience.

Another significant strength of this research was the multiple different methodologies by which sleep was characterised. This is particularly important in light of our contrasting findings between subjective and objective measurements. We determined that the influence of a bedpartner in terms of wake transmissions was observed in objective data but rarely recorded in subjective diaries. Meanwhile, if a clinician asks about the influence of a bedpartner, they typically ask about an individual’s perceptions of their bedpartner’s sleep, i.e., their subjective experience of it. In the present data we observed a comparatively low number of subjective diary-recorded awakenings together with a comparatively high number of objective actigraphy-recorded wake transmissions. This suggests in most cases where a bedpartner does transmit wake to an individual, the individual does not remember this occurring enough to record it as an awakening in the diary by morning. Regardless, it does affect objective sleep continuity. The effect of the bedpartner (particularly one who snores) can therefore be likened to the effect of individuals living under the flight path: despite many individuals’ reports of their sleep being uninterrupted, arousals are observed as planes fly over. This metaphor may be helpful for explaining to patients in clinics the importance of considering their bedpartner.

Additionally, we used validated assessment tools to develop novel metrics: sleep diaries are widely used, and ours was based upon the consensus sleep diary; while actigraphy was scored using the Cole-Kripke algorithm (Cole, Kripke, Gruen, Mullaney, & Gillin, 1992). Specifically, this study used algorithmically determined sleep/wake epochs to determine the

threshold of wake for a wake transmission to occur. In doing so, the measures designed here are also simple and easily replicable by researchers in future studies.

Limitations and Suggestions for Future Research

The shortcomings of this research are acknowledged. We did not have the opportunity to experimentally manipulate bedpartner status to be able to introduce causality into the impact a bedpartner has on sleep. The determination of sequential awakenings using actigraphy provides correlational data that cannot determine whether one partner is really causing their bedpartner to wake up; as additional factors may be at play. However, the present study design already placed quite a heavy burden on participants, and additionally, there are feasibility issues to address in order to answer this question. In order to monitor sleep of couples both together and separately, whilst maintaining the present study design strength of monitoring in naturalistic settings for ecological validity, we would need to manipulate the sleep environment for at least one partner, i.e., sleeping in a different bed, or a different room. The change in location is likely to affect the sleep quality of that individual (Agnew, Webb, & Williams, 1966). Of course, the necessity of a second location for concurrent recording to take place introduces a selection bias for couples participating: only couples with a spare bed in their house could therefore participate. It will certainly be interesting for future research to experimentally manipulate bedpartner status and investigate the impact on sleep characteristics and introduce examination of causality, with a design accommodating the feasibility issues listed above.

A second limitation to consider is mental health was assessed via use of self-report questionnaires; and in our sample without sleep disorders, there was a restricted range, with low levels of mental health symptoms present. Furthermore, this was a cross-sectional design, so the design did not allow explicit testing of the direction of these relationships. We have provided suggestions of the effect of anxiety on one's own and one's bedpartner's sleep,

however this relationship may well be bi-directional. However, the study design did have feasibility benefits. It is far less demanding on both participants and researchers than longitudinal studies and provides important information about the associations between variables. This study is certainly the first step in designing and prompting longitudinal and experimental studies into bedsharing. Future research that recruits a wider variety of mental health symptomology would be beneficial, in particular, studies including clinical samples of anxiety and mood disorders.

A third limitation to consider when comparing results between the two samples (insomnia couples vs. those without sleep disorders) are the differences between the samples: the couples without sleep disorders were, on average, significantly younger than the insomnia couples, and screening methods and actigraphy cleaning guidelines differed between the samples. Additionally, there are a range of other factors we did not assess which could affect sleep and an individual's potential vulnerability to being awoken by their partner. It is hoped future studies will be able to learn from the limitations in the present methodology when designing future studies, considering social factors such as history of childhood adverse experiences, environmental factors such as size of bed, biological factors such as sleep architecture, and gathering subjective reports of the impact of the bedpartner on sleep and reasons for not sharing a bed on any specific night.

It is finally worth noting that a common limitation cited within sleep literature is that actigraphy as the objective sleep measure lacks the accuracy of gold-standard PSG. However, in the context of dyadic sleep, use of actigraphy may actually be a strength. Wake scored using actigraphy involves movement, and is therefore active wake. Active wake may be more meaningful wakefulness in context of bedpartner sleep. It could be argued that an individual waking and not moving (e.g., lying quietly) is not likely to disturb a partner. In contrast, an individual waking and tossing and turning is more likely to make an impact. This brings yet

more meaning to findings for Paper 4: the difference in models between anxiety and depression may be due to greater movement and agitation associated with anxiety symptoms compared with depression (American Psychiatric Association, 2013; Bennabi, Vandel, Papaxanthis, Pozzo, & Haffen, 2013; Goldstein et al., 2013; Hasler, Drevets, Manji, & Charney, 2004). Therefore, there was a greater ability for actigraphs to detect wake in these individuals with greater movement. Larger studies investigating dyadic sleep using PSG are certainly the logical next step within the field of dyadic sleep research, in particular helping to identify sleep staging risk factors for wake transmission or resistance, and providing more detailed findings, especially for those PSG awakenings which are not scored as such by actigraphy, which may potentially provide further details about times an insomnia patient is awake and unmoving (e.g., "stewing" in frustration but not moving enough to be scored actigraphically as wake). Nonetheless it is important to consider the different functional information gathered within PSG compared with actigraphy, as well as noting the importance for future studies to differentiate intended sleep and wake times from time spent in bed to ensure the most meaningful rest interval can be analysed.

Despite these limitations, this research provides an important step for the field of dyadic sleep research. This study examined and characterised (1) good sleepers and (2) insomnia disorder. Examination of other sleep disorders characterised by noise or movement e.g., Restless Legs Syndrome, Periodic Limb Movement Disorder, and more extensive research into OSA, will be important to more fully understand the social impact these disorders have on bedpartners, in both untreated and treated forms. Future research may also benefit from extending the research presented in Papers 3 and 4, investigating in more detail cases where both partners in a couple experience a sleep disorder. While the present research did not rigorously assess sleep disorder status of the bedpartner in our insomnia sample, we had several couples who both reported clinically relevant sleep disturbance. It will certainly be of interest

to examine these cases in more depth and it is somewhat of a loss for this research that these cases were not systematically recorded, and were not in high numbers. Understanding a range of dyadic sleep experiences may be clinically advantageous, helping encourage more patients to seek treatment if they know improving their own health will also improve their partners'.

Overarching Conclusions

In summary, this thesis offers valuable and novel contributions to the study of sleep in couples. Results describe and characterise the phenomenon of wake transmission between bedpartners in couples without sleep disorders, and couples where one partner experiences untreated insomnia. Results provide evidence couples affect one another's sleep, and existing full-night measures of sleep do not fully describe this unique element. Further, vulnerability to transmission can be predicted by a variety of demographic and sleep characteristics. These are exciting findings that have implications both methodologically as well as clinically. This thesis sets the stage for future research into the area of dyadic sleep. It is hoped future studies will design experimental and longitudinal studies to further explore the impact of a bedpartner on sleep and holistic functioning of individuals in a variety of health contexts.

CHAPTER 7

References

Chapter 7: References

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