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The Impact of Relationship Functioning on Cortisol in Married Couples: A Dyadic Exploration of Sleep as a Potential Mediator

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The Impact of Relationship Functioning on Cortisol in Married
Couples: A Dyadic Exploration of Sleep as a
Potential Mediator

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A dissertation submitted to the faculty of
Brigham Young University
in partial fulfillment of the requirements for the degree of
Doctor of Philosophy

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ABSTRACT

The Impact of Relationship Functioning on Cortisol in Married Couples: A Dyadic Exploration of Sleep as a Potential Mediator

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Relationship functioning in couples has been linked to numerous health outcomes. The purpose of this study was to examine the association between 1) marital functioning and sleep dimensions, 2) marital functioning and cortisol, and 3) sleep dimensions and cortisol. The sample consisted of 108 heterosexual, married couples and was part of a larger marital intervention study. As predicted, poor marital functioning was related to negative sleep outcomes. However, these effects were only significant for wives. There was also evidence to suggest that poor marital functioning was associated with increased cortisol levels in husbands. These effects were independent of age and BMI. Contrary to our hypotheses, cortisol was not linked to sleep outcomes and, therefore, not a mediator of effect between marital functioning and cortisol. However, we did find evidence to suggest that stress and depressive symptomology could mediate the association between dyadic adjustment and sleep. Together, these findings provide evidence for how marital functioning can affect both physical and psychological health.

Keywords: marital functioning, dyadic adjustment, therapy, social support, sleep, sleep latency, sleep quality, cortisol, health

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The Impact of Relationship Functioning on Cortisol in Married
Couples: A Dyadic Exploration of Sleep as a
Potential Mediator

We spend roughly one-third of our lives sleeping. Getting adequate sleep is essential for optimal daytime functioning and has implications for both physical and mental health (Banks & Dinges, 2007; Gangwisch et al., 2007). Social relationships also play a large role in our lives and are predictive of health outcomes (Berkman, 1995; Cohen, 2004). A recent review acknowledged the link between social relationships and health, as well as an association between sleep and health, but suggested that these areas have developed independent of one another (Troxel, 2010). Thus, more research is needed to gain a better understanding of the interplay between social relationships, sleep, and health.

The link between social relationships and health outcomes is well established. Social isolation is a well-known predictor of broad-based morbidity and mortality (House, Landis, & Umberson, 1988). The effect of not having adequate social support is comparable to the negative effects of smoking and obesity (Holt-Lunstad, Smith, & Layton, 2010). Furthermore, mechanisms linking social relationships to health outcomes have been explored to some extent. Cohen and Wills (1985) have proposed the *stress-buffering model*, which suggests that perceived access to social support “buffers” individuals from negative effects of stress that could potentially put their health at risk. The *main effects model* argues that social integration promotes healthy behaviors and directly influences physiological processes, regardless of stress (Cohen & Wills, 1985). Although these models are not independent, they provide a framework to explore potential stress-buffering or main effects mediators of social relationships and health.

Cortisol is one physiological outcome that may be influenced by social relationships. The end result of hypothalamic pituitary adrenal (HPA) axis activation in response to a stressor is cortisol secretion and research suggests that chronically elevated levels might impair immune functioning as well as other health outcomes (Gruenewald, Seeman, Ryff, Karlamangla, & Singer, 2006). Many studies have examined cortisol using average daytime levels, or area under the curve (Adam & Kumari, 2009). For instance, loneliness has been linked to higher daytime salivary cortisol levels (Cacioppo et al., 2000). Hostility and cynicism are associated with higher daytime cortisol (Pope & Smith, 1991; Ranjit et al., 2009), which may have important implications when close relationships adversely affect mood or are a source of stress. In experimental conditions where participants engage in laboratory-based challenge tasks, inadequate social support has been linked to increased cortisol response (Seeman, McEwen, Singer, Albert, & Rowe, 1997; Uchino, Cacioppo, & Kiecolt-Glaser, 1996). In more naturalistic settings, individuals that have strained relationships with a spouse, family member, and/or friend exhibit poor cortisol regulation over time (Friedman, Karlamangla, Almeida, & Seeman, 2012). As daytime cortisol has direct implications for health, it is important to consider how close relationships, and stress within those relationships, influence neuroendocrine response.

For many individuals in adulthood, marriage is considered one of the most important close relationships and is of particular interest due to the unique influence on physiological and psychological processes (Holt-Lunstad, Birmingham, & Jones, 2008). For instance, one review concluded that marital happiness affects global happiness more than any other variable, including satisfaction with work and friendships (Kiecolt-Glaser & Newton, 2001). In another study, married individuals had the lowest premature mortality rate when compared to those who were cohabiting, single, divorced, or widowed (Drefahl, 2012). Marital status is also linked to

both depressive symptoms and cardiovascular risk (Burman & Margolin, 1992). Clearly, married couples may enjoy some unique benefits in terms of their health.

Although marriage may be an important source of social connection, the relationship may be a source of stress if an individual is not happily married (Burman & Margolin, 1992; Holt-Lunstad et al., 2008). High-functioning marriages show more favorable health outcomes compared to low-functioning marriages by directly, and possibly indirectly, influencing physiological processes (Kiecolt-Glaser & Newton, 2001). Negative behaviors during marital conflict are associated with elevated cardiovascular activity, increased hormone levels related to stress (i.e. cortisol), and poor immune functioning (Robles & Kiecolt-Glaser, 2003). Perceptions of relationship factors, including quality and emotional responsiveness, are also important (Holt-Lunstad, Uchino, Smith, & Hicks, 2007; Selcuk & Ong, 2013). Thus, perceived aspects of marital functioning should be considered when examining the tie between marriage and health.

In addition to marital status and functioning, sleep is linked to both physical and mental health outcomes. Restricted sleep can cause deficits in attention, working memory, mood, and has been associated with increased risk for diabetes, obesity, heart attack, and stroke (Altman et al., 2012; Banks & Dinges, 2007; Gangwisch et al., 2007; Roth, 2007). Restricted sleep is also tied to the dysregulation of neuroendocrine functioning (Van Cauter et al., 2007). One purpose of HPA activation is to maintain physiological equilibrium and homeostasis. Sleep disruption may disrupt HPA activation and alter the circadian rhythm of cortisol (Backhaus, Junghanns, & Hohagen, 2004; Lasikiewicz, Hendrickx, Talbot, & Dye, 2008). There is also evidence to suggest a bidirectional association between stress, cortisol, and sleep (Garde, Albertsen, Persson, Hansen, & Rugulies, 2011). Hyperarousal of physiological processes throughout the day has been linked to insomnia (Roth, 2007), which might suggest that sleep quality is negatively

affected when low-functioning marriages result in hyperarousal. More research examining sleep as potential mediator between relationship stress and daily neuroendocrine rhythms could offer clarity on these issues.

There is reason to question whether sleep could potentially mediate the effect between marital functioning and cortisol. For instance, social factors, such as loneliness, have been shown to modulate the efficiency of restorative behaviors like sleep (Cacioppo et al., 2002). Specifically in marriage, sleep has been described as an attachment behavior, characterized by feelings of physical and emotional safety (Troxel, 2010), and requiring reduced awareness and vigilance (Dahl, 2002). Accessibility and responsiveness are two key attachment behaviors that have been discussed extensively in the literature (Bowlby, 1978). Thus, high-functioning marriages where a spouse is accessible, responsive, and promotes feelings of safety and security, might allow an individual to down-regulate physiological processes and attain optimal sleep. However, in low functioning marriages where a spouse is less available and/or less responsive, the individual may feel vulnerable or threatened, leading to poor sleep outcomes. Furthermore, interpersonal conflict within couples is associated with depressive symptoms and anxiety, which can potentially affect sleep duration, efficiency, and latency (El-Sheikh, Kelly, & Rauer, 2013). As marital conflict is associated with increased stress and dysregulation of cortisol (Barnett, Steptoe, & Gareis, 2005; Saxbe, Repetti, & Nishina, 2008), it seems reasonable to question whether various dimensions of sleep could play a mediational role in the association between marital functioning and daily cortisol regulation.

When testing the effects of marriage functioning on sleep, it is also important to consider the dyadic nature of both relationship functioning and sleep quality. Much of literature on sleep, relationship functioning, and health outcomes has focused on the individual as the unit of

analysis (Troxel, 2010). Relationship problems and poor sleep often co-occur as a result of major life stressors which are experienced by both the individual and the spouse. Rather than focusing solely on the individual, using appropriate statistical methods to analyze the dyadic nature of relationship functioning and sleep behaviors could shed light on potential mechanisms linking these variables to health outcomes.

It is evident that both relationship and sleep factors have direct implications for health outcomes. What remains unclear is whether dimensions of sleep could potentially mediate the association between relationship functioning and health. Investigating sleep as a possible mechanism could offer insights as to how negative health outcomes could be avoided as a result of relationship stress. Furthermore, understanding mechanisms could have therapeutic relevance for both the psychological and medical community.

Therefore, the first aim of this study was to confirm an association between 1) marital functioning and sleep dimensions, 2) marital functioning and cortisol, and 3) sleep dimensions and cortisol. We hypothesized that individuals who were seeking therapy, had poor dyadic adjustment, and had negative perceptions of spousal support would have longer sleep latency, poorer subjective sleep quality, and lower cortisol levels. Additionally, we expected those with poorer subjective sleep quality and longer sleep latency to have increased daily cortisol levels. The second aim of this study was to test whether dimensions of sleep would mediate the effect between marital functioning cortisol. We predicted that sleep would, at least partially, mediate the effect between marital functioning (marriage therapy seeking, dyadic adjustment, perceived support from spouse) and cortisol (see Figure 1).

Methods

Participants and Procedure

This study analyzed and reported results from an existing data set which was collected by Dr. Julianne Holt-Lunstad. The sample consisted of 108 heterosexual, married couples and was part of a larger marital intervention study. Participants were recruited from the community through flyers, paid advertisement, and a university mental health clinic. 48 couples were actively seeking therapy and had an average dyadic adjustment (DAS) score of 97.08 ($SD = 19.45$). 60 couples were recruited as control couples, were not actively seeking therapy, and had an average dyadic adjustment (DAS) score of 119.13 ($SD = 14.09$). The average length of marriage was 5.38 years ($SD = 7.77$) and the average age was 28.86 ($SD = 9.20$). The ethnic breakdown of participants was as follows: 82% White (Caucasian), 7% Hispanic, 4.5% “Other”, 1.5% African American, 1.5% Native American, 1.5% “Mixed”, 1% Asian, and 1% did not disclose their ethnicity. Importantly, the sample size of this study does not provide optimal power for dyadic analysis, but is generally considered to be acceptable and exceeds recommended standards for non-independent data (Kenny, Kashy, & Cook, 2006).

Interested individuals were excluded from participation if taking medications that influence blood pressure or had a current chronic illness with a cardiovascular component. Individuals were also excluded if pregnant, nursing, within six months postpartum, or planning on becoming pregnant in the next three months. After informed consent was obtained, participants were instructed on how to collect saliva samples over a 24-hour period using salivettes. A packet of questionnaires was used to assess general demographics variables (i.e. age, ethnicity, income, years married), physical health (i.e. health history, sleep), mental health (i.e. depressive symptoms, general stress), and relationship functioning (i.e. adjustment, spousal

support). After 24-hours, participants returned the saliva samples and questionnaires to the research lab.

Measures

Dyadic Adjustment Scale (DAS). To assess marital adjustment, participants completed the DAS (Spanier, 1976). The original four subscales of the DAS are dyadic consensus (13 items assessing the degree to which the couple agrees or disagrees about various issues), dyadic satisfaction (10 items assessing perceived stability of the marriage and how fights are handled), affectional expression (4 items assessing level of agreement on how affection is expressed), and dyadic cohesion (5 items assessing frequency of positive interactions between the couple) (Spanier & Thompson, 1982). For the current analysis, the 32 items that make up the DAS were summed to create a total score ranging from 0 to 151, with higher scores indicating more positive dyadic adjustment (Spanier, 1976). In order to verify our results from using the continuous measure, we also performed a separate analyses using the clinical cutoffs of 107 or greater for distress and 92 or less for non-distressed (Sabourin, Valois, & Lussier, 2005). In these analyses, 23 of the 108 couples were excluded for not meeting the clinical cutoff criteria. However, the results were consistent with the continuous DAS measure and, therefore, not reported

Social Relationships Index (SRI). To assess perceived social support and get a sense of possible attachment behaviors (i.e. availability, responsiveness), participants completed a version of the SRI that specifically targets their spouse. The SRI is a reliable and valid measure used in health studies. Each participant rated how helpful and how upsetting they perceived their spouse to be during times when they needed support, such as advice, understanding, or a favor (1 = not at all, 6 = extremely). Typically, relationships are classified as supportive, ambivalent, aversive, or indifferent. An individual is classified as supportive if they received a rating of 2 or higher on

helpfulness and a rating of 1 on upsetting. Ambivalence is classified by a rating of 2 or higher on both helpfulness and upsetting. Although uncommon in spouse relationships, indifference is classified by 1 on both helpfulness and upsetting. Aversive relationships are classified by a rating of 1 on helpfulness and 2 or higher on upsetting. When following these criteria, only 26 of the 216 individuals in our sample perceived their spouse as supportive. The remaining 96 were classified as ambivalent. To get a better sense of these classifications in spouse relationships, measures of how helpful and how upsetting were mean centered and individuals were placed into one of the four classifications.

Pittsburgh Sleep Quality Inventory (PSQI). An abbreviated version of the PSQI was used to assess sleep. The PSQI is well-validated and widely used self-report scale. Participants are asked to respond to questions about their sleep during the past month. The full scale examines seven dimensions including sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleeping medications, and daytime dysfunction (Buysse et al., 1989). In an effort to minimize the number of survey questions, an abbreviated version of the scale was used and only select items were collected from participants in this study. These items included sleep latency (i.e. minutes taken to fall asleep) and subjective sleep quality.

Cortisol. Cortisol was assessed via saliva sampling and calculating area under the curve (AUC). This measure has been used in multiple studies to provide an average assessment of daily cortisol regulation (Adam & Kumari, 2009). Consistent with standard salivary sampling procedures, we sampled at standardized times to account for diurnal effects. Samples were obtained at approximately 7AM, 12 noon, 5 PM, 10 PM, and upon waking (i.e. before the participants got out of bed). Samples were obtained by using a standard sampling product (Salivette, Sarstedt, Inc., Newton, North Carolina). Participants were instructed to suck or chew

on a cotton roll until it was saturated (very soggy – usually about 2 to 3 minutes, 1 minute minimum). The cotton roll was then placed inside a retainer and then in the centrifuge tube. To minimize any potential contamination, we gave the participants the following instructions. Participants were told not to eat a major meal within 60 minutes before sample collection, to avoid alcohol for 24 hours before sample collection, and to avoid dairy products 30 minutes before sample collection. We also informed them to be careful about acidic or high-sugar foods; ideally, they should rinse their mouths thoroughly with water 10 minutes before giving a sample to minimize the potential for saliva contamination. To avoid potential blood contamination, we also recommended that they not brush their teeth within 3 hours before sample collection. Saliva samples were stored in a freezer (-20°C) until shipped for assay. Salivary cortisol was measured with a commercial immunoassay with chemiluminescence detection (CLIA, IBL-Hamburg, Germany).

Analytic Strategy

We performed preliminary analyses to examine demographic information, missing data, and assumptions of normality, using SPSS (Software Version 21). Using Little's missing completely at random (MCAR) test, data was not found to be missing at random. Specifically, data were missing in 4 cases for sleep latency, 1 case for sleep quality, and 9 cases for cortisol. Thus, listwise deletion was used for these cases and full information maximum likelihood (FIML) estimation was used for the remaining sample. There were 6 extreme outliers in our cortisol measures. These were fenced at 3 standard deviations of the mean.

Next, we examined variables that had potential to bias the interpretation of our results. Similar to previous studies involving sleep and cortisol outcomes (Dmitrieva, Almeida, Dmitrieva, Loken, & Pieper, 2013; Ohayon, Carskadon, Guilleminault, & Vitiello, 2004; Wirtz,

Ehlert, Emini, & Suter, 2008; Yang, Matthews, & Chen, 2014), age and body mass index (BMI) were significantly associated with multiple dependent variables and were, therefore, included as covariates in all the subsequent analyses. We also examined the effects of general stress (using the Perceived Stress Scale), depressive symptoms (using the Center for Epidemiologic Studies Depression Scale as a continuous variable and the clinical cutoffs for depressive symptoms as a categorical variable), whether or not the couple had children, number of years married, income, and perceived socioeconomic status (SES) on our outcome measures (see Table 6). Data were then organized by couple, rather than individual, and converted to a format compatible for analysis in Mplus (Version 7.0, Muthen & Muthen, 2012).

Actor-partner interdependence models (APIM) were used to account for the dyadic and non-independent nature of the data (Kenny et al., 2006). Before testing each model, the omnibus test of distinguishability (I-SAT) was used to ensure that the dyads were both theoretically and empirically distinguishable. This test imposes equality constraints on the means, variances, and covariances of the manifest variables for both members of the dyad. If χ^2 was significant when these constraints were imposed, the distinguishable version of the APIM was used. Otherwise, the indistinguishable version was used and equality constraints were imposed on predictor means, variances, covariances, and loadings (Kenny et al., 2006). Results of each I-SAT can be seen in Tables 2 through 4. Unless otherwise noted, all models met the criteria for the distinguishable version of the APIM.

Multiple models were used to test the effects of marital functioning on sleep outcomes (see Figure 2 and Tables 1-4). Each dependent measure was separately regressed on each independent predictor and covariates in an APIM model. For our independent predictors of marital functioning, we used the study condition (a categorical variable of therapy seeking vs.

not therapy seeking), the dyadic adjustment scale (as a continuous measure and a categorical variable of distressed vs. not distressed), and perceived quality of spouse support (using categorical variables derived from the SRI). Sleep measures consisted of sleep latency (i.e. minutes taken to fall asleep) and subjective sleep quality. We also used multiple models to test the effects of each operationalized aspect of marital functioning on area under the curve (AUC_G) daily cortisol outcomes (see Figure 3 and Tables 1-4). Prior to testing sleep as a mediator of relationship functioning and cortisol, we also examined the effects of sleep on cortisol (see Figure 4 and Table 5).

With structural equation modeling, multiple indices are typically used to assess model fit: Chi-square test (a significant chi-square indicates lack of satisfactory model fit), Comparative Fit Index ($CFI > 0.95$ indicates good fit); Tucker-Lewis Index ($TLI > 0.95$ indicates good fit); and root mean squared error of approximation (RMSEA between 0.05 and 0.08 indicates adequate model fit). Fit indices are reported for each APIM test, as well as mediation models.

Results

Marital Functioning and Sleep

In the first set of models, we examined whether couples seeking therapy differed from those not seeking therapy for sleep latency and quality (see Table 1). The model testing sleep latency as a dependent variable provided a poor fit to the data, $\chi^2(4) = 5.30, p = .25; CFI = .90; TLI = .73; RMSEA = .06$. With this limitation noted, the results are presented but any theoretical interpretations will be withheld. Compared to those not seeking therapy, wives seeking therapy had a longer sleep latency while controlling for age and BMI, $\beta = .28, p = .003, 95\% CI [.10, .46]$. Therapy seeking was not associated with sleep latency in husbands and no partner effects were tested in this model, as the condition (i.e. therapy vs. no therapy) was the

same for both the husband and wife in each dyad. The model testing sleep quality as a dependent variable also provided a poor fit to the data, $\chi^2(4) = 9.86, p = .04$; CFI = .76; TLI = .34; RMSEA = .12. Therapy seeking was not associated with sleep quality in husbands or wives and no partner effects were tested in this model.

Next, we examined the effects of dyadic adjustment (as a continuous measure) on sleep latency and quality (see Table 2). The model testing sleep latency as a dependent variable provided a good fit to the data, $\chi^2(4) = 2.13, p = .71$; CFI = 1.00; TLI = 1.22; RMSEA = .00. For wives, poor dyadic adjustment was associated with longer sleep latency, $\beta = -.67, p < .001$, 95% CI [-.95, -.40], while controlling for age and BMI. Dyadic adjustment was not associated with sleep latency in husbands and no partner effects were observed in this model. The model testing sleep quality as a dependent variable provided a good fit to the data, $\chi^2(4) = 4.38, p = .36$; CFI = .99; TLI = .97; RMSEA = .03. Poor dyadic adjustment was associated with poor sleep quality in wives, $\beta = -.51, p < .001$, 95% CI [-.77, -.17]. Dyadic adjustment was not linked to sleep quality in husbands and no partner effects were observed for husbands or wives in this model.

We then examined the influence of ambivalent perceptions of spousal support (using categorical variables derived from the SRI) on sleep outcomes (see Table 3). The model testing sleep latency as a dependent variable provided a good fit to the data, $\chi^2(4) = 1.45, p = .83$; CFI = 1.00; TLI = 1.00; RMSEA = .00. Ambivalent perceptions of spousal support had no actor or partner effects on sleep latency for husbands or wives. The model testing sleep quality as a dependent variable provided a good fit to the data, $\chi^2(4) = 1.74, p = .78$; CFI = 1.00; TLI = 1.00; RMSEA = .00. While controlling for age and BMI, wives who perceived their husbands as ambivalent had poor sleep quality, $\beta = .28, p = .05$, 95% CI [.001, .55], compared to wives who

viewed their husbands and a positive source of support. No actor effects for sleep quality were observed for husbands. However, a partner effect was observed such that when wives perceived their husbands as ambivalent, husbands had poor subjective sleep quality $\beta = .27, p = .05$, 95% CI [-.002, .55], while controlling for age and BMI. There were no other partner effects observed in this model.

Lastly, we examined the influence of aversive perceptions of spousal support (using categorical variables derived from the SRI) on sleep outcomes (see Table 4). The model testing sleep latency as a dependent variable provided a good fit to the data, $\chi^2(4) = 4.07, p = .40$; CFI = .99; TLI = .97; RMSEA = .02. No actor effects were observed for sleep latency in husbands or wives. However, a partner effect was observed, such that when husbands perceived their wives as aversive, wives had longer sleep latency, $\beta = .30, p = .04$, 95% CI [.01, .59], while controlling for age and BMI. The model testing sleep quality as a dependent variable provided a good fit to the data, $\chi^2(4) = 4.41, p = .35$; CFI = .98; TLI = .94; RMSEA = .04. No actor effects were observed for sleep quality in husbands or wives. However, a partner effect was observed, such that when husbands perceived their wives as aversive, wives had poor sleep quality, $\beta = .29, p = .03$, 95% CI [.03, .55], while controlling for age and BMI.

Marital Functioning and Cortisol

In the first set of models, we examined whether couples seeking therapy differed from those not seeking therapy on cortisol (see Table 1). This model provided a good fit to the data, $\chi^2(4) = 1.43, p = .84$; CFI = 1.00; TLI = 1.58; RMSEA = .00. Therapy seeking had no significant effects on cortisol for husbands or wives.

Next, we examined the effects of dyadic adjustment (as a continuous variable) on cortisol (see Table 2). This model provided a good fit to the data, $\chi^2(4) = 1.36, p = .85$; CFI = 1.00; TLI

= 1.46; RMSEA = .00. Poor dyadic adjustment was associated with higher AUC_G in husbands (β = -.31, p = .05, 95% CI [-.62, .006], while controlling for age and BMI. No actor effects were observed for wives and no partner effects were found for husbands or wives.

We then examined the effects of perceived ambivalent spousal support (using categorical variables derived from the SRI) on cortisol (see Table 3). This model provided a poor fit to the data, $\chi^2(4) = 4.88$, $p = .30$; CFI = .93; TLI = .76; RMSEA = .07. With this limitation noted, the results are presented but any theoretical interpretations will be withheld. No actor effects were observed for husbands or wives. A partner effect unexpectedly revealed that when wives perceive ambivalence in their husbands, the husbands have a lower AUC_G, $\beta = -.29$, $p = .03$, 95% CI [-.55, -.07], while controlling for age and BMI. No other partner effects were found.

Lastly, we examined the effects of perceived aversive spousal support (using categorical variables derived from the SRI) on cortisol (see Table 3). This model provided a good fit to the data, $\chi^2(4) = 2.54$, $p = .63$; CFI = 1.00; TLI = 1.73; RMSEA = .00. No actor or partner effects were observed in this model for husbands or wives.

Sleep and Cortisol

Prior to mediational analysis, we examined the effects of our sleep measures on cortisol outcomes (see Figure 4 Table 5). The model testing sleep latency as a predictor of cortisol provided good fit to the data, $\chi^2(4) = 1.75$, $p = .94$; CFI = 1.00; TLI = 2.09; RMSEA = .00. Sleep latency was not associated with AUC_G for husbands or wives, $p > .20$. The model testing sleep quality as a predictor of cortisol also provided a good fit to the data, $\chi^2(4) = 2.32$, $p = .89$; CFI = 1.00; TLI = 1.78; RMSEA = .00. Sleep quality was not associated with AUC_G for husbands or wives, $p > .20$. Because sleep and cortisol were not linked, there was no justification to test sleep as a mediator of relationship functioning and cortisol.

Follow-Up Exploratory Analyses Examining Potential Mediators and Moderators

Although there is no evidence in this study to suggest that sleep is a mediator of marital functioning and cortisol, marital functioning was significantly tied to both sleep and cortisol outcomes. As part of an exploratory follow-up analysis, we examined potential mediators and moderators of these effects (see Table 6). While controlling for age and BMI, participants who had longer sleep latency also had significantly higher perceptions of general stress (measured as a continuous variable using the PSS, $\beta = .19$, $p = .008$, 95% CI [.01, .03], higher depressive symptomology (measured as a continuous variable using the CESD, $\beta = .37$, $p < .001$, 95% CI [.02, .04], and were more likely to have clinically significant depressive symptomology, $\beta = .21$, $p = .004$, 95% CI [.11, .56]. Similarly, while controlling for age and BMI, participants with poorer sleep quality also had significantly higher perceptions of general stress, $\beta = .36$, $p < .001$, 95% CI [.02, .04], higher depressive symptomology, $\beta = .41$, $p < .001$, 95% CI [.02, .04], and were more likely to have clinically significant depressive symptomology, $\beta = .29$, $p < .001$, 95% CI [.24, .64]. Thus, we examined stress and depressive symptomology as potential moderators (using a statistical interaction) and mediators (using the bootstrapping method) of the association between marital functioning and sleep outcomes. All potential mediation and moderation effects were examined given our previously tested hypotheses. No significant moderation effects were found. Only the significant mediation effects are reported.

We examined stress as a potential mediator of the effect between marital functioning and sleep outcomes. As previously mentioned, poor dyadic adjustment was associated with poor sleep quality in wives, $\beta = -.51$, $p < .001$, 95% CI [-.77, -.17]. Poor dyadic adjustment was also associated with more stress, $\beta = -.63$, $p < .001$, 95% CI [-.75, -.51], and more stress was associated with poor sleep quality, $\beta = -.39$, $p < .001$, 95% CI [.19, .59], in wives. When testing

for mediation using the bootstrapping method, the model provided adequate fit to the data, $\chi^2(2) = 2.06, p = .36$; CFI = .99; TLI = .99; RMSEA = .02. The direct effect was no longer significant, $\beta = -.18, p = .17$, 95% CI [-.02, .002], and the indirect effect was significant, $\beta = -.24, p = .001$, 95% CI [-.01, -.004], which suggests that perceived stress mediated approximately 65% of the effect between dyadic adjustment and sleep quality in wives, while controlling for age and BMI.

We also examined depressive symptomology as a potential mediator of the effect between marital functioning and sleep latency in wives. As mentioned, poor dyadic adjustment was associated with longer sleep latency, $\beta = -.67, p < .001$, 95% CI [-.95, -.40]. Poor dyadic adjustment was also associated with higher CESD scores, $\beta = -.70, p = .001$, 95% CI [-.80, -.59], and higher CESD scores were associated with longer sleep latency, $\beta = -.59, p = .001$, 95% CI [.35, .82]. When testing for mediation using the bootstrapping method, the model provided poor fit to the data, $\chi^2(2) = 12.67, p = .002$; CFI = .90; TLI = .65; RMSEA = .23. With this limitation noted, the results are presented but any theoretical interpretations will be withheld. The direct effect was no longer significant, $\beta = -.05, p = .71$, 95% CI [-.32, .22], and the indirect effect was significant, $\beta = -.41, p < .001$, 95% CI [-.60, -.21], which suggests that depressive symptomology (as operationalized by CESD scores) mediated 93% of the effect between dyadic adjustment and sleep latency in wives, while controlling for age and BMI.

We further examined depressive symptomology as a mediator of the effect between marital functioning and sleep quality in wives. As mentioned, poor dyadic adjustment was associated with longer sleep latency, $\beta = -.67, p < .001$, 95% CI [-.95, -.40]. Poor dyadic adjustment was also associated with higher CESD scores, $\beta = -.70, p = .001$, 95% CI [-.81, -.60], and higher CESD scores were associated with poor sleep quality, $\beta = -.55, p = .001$, 95% CI [.33, .77]. When testing for mediation using the bootstrapping method, the model provided poor fit to

the data, $\chi^2(2) = 12.15, p = .002$; CFI = .91; TLI = .69; RMSEA = .27. With this limitation noted, the results are presented but any theoretical interpretations will be withheld. The direct effect was no longer significant, $\beta = .01, p = .96, 95\% \text{ CI } [-.28, .29]$, and the indirect effect was significant, $\beta = -.39, p < .001, 95\% \text{ CI } [-.58, -.20]$, which suggests that depressive symptomology mediated approximately 99% of the effect between dyadic adjustment and sleep quality in wives, while controlling for age and BMI.

Discussion

The purpose of this study was to examine the association between 1) marital functioning and sleep dimensions, 2) marital functioning and cortisol, and 3) sleep dimensions and cortisol. As predicted, poor marital functioning was related to negative sleep outcomes. There was also evidence to suggest that poor marital functioning was associated with increased cortisol levels in husbands. Contrary to our hypotheses, cortisol was not linked to sleep outcomes and, therefore, not a mediator of effect between marital functioning and cortisol. However, we did find evidence to suggest that stress and depressive symptomology mediate the association between dyadic adjustment and sleep.

Specifically, marital distress as measured dyadic adjustment was associated with longer sleep latency in wives. These findings supplement prior research that has demonstrated a link between interpersonal conflict and longer sleep latency (El-Sheikh et al., 2013). Marital distress may lead individuals to feel less physically or emotionally safe, making it less likely to down-regulate physiological processes and attain optimal sleep. Longer sleep latency may also be the result of added worry and rumination regarding the marital relationship. Rumination involves repetitively thinking about the causes, consequences, and symptoms of current negative emotions (Nolen-Hoeksema, 1991), whereas worry involves thinking about the possible negative outcomes

of future events (Nitschke, Heller, Imig, McDonald, & Miller, 2001). Worry, rumination, and other negative emotions have been linked to insomnia (Mitchell, Mogg, & Bradley, 2012) and, thus, may explain why marital distress and therapy seeking were linked to longer sleep latency.

Rumination has also been associated with both stress and depressive symptoms (Nolen-Hoeksema, 1991). In our study, perceived stress mediated the effect between dyadic adjustment and sleep quality in wives. While depressive symptomology appeared to influence sleep outcomes, the mediation models provided poor fit to the data. There is some evidence to suggest that sleep disturbances precede depressive symptoms (Chang, Ford, Mead, Cooper-Patrick, & Klag, 1997). Marital and sleep problems are likely both causes and results of psychological distress, such as depressive symptoms or stress (Troxel, 2010). Longitudinal studies that assess sleep, depressive symptoms, and marital functioning may offer clarity on these issues. The mediational effects of this study align with the existing body of literature and theoretical frameworks suggesting psychological factors may mediate the effects between marital functioning and sleep outcomes (Troxel, 2010; Troxel, Robles, Hall, & Buysse, 2007).

Poor dyadic adjustment and perceived spousal ambivalence were also associated with poor subjective sleep quality in wives. These findings may add to the current literature suggesting that insecure attachments are correlated with poorer subjective sleep quality (Carmichael & Reis, 2005). According to attachment theory, accessibility and responsiveness are key components of perceived security, anxiety, or distress (Bowlby, 1978). Couples that do not perceive their spouse as positive source of support may likewise not feel the spouse is accessible or responsive to them when needed. Attachment was not directly assessed in this study so future research could clarify if there is an association between other attachment behaviors and perceived social support.

While our study tested actor and partner effects for husbands and wives, we cannot conclude gender differences from these tests, nor were there hypothesized gender differences. Other studies that have used the APIM to examine the influence of psychosocial factors on sleep outcomes have found effects for both husbands and wives (El-Sheikh et al., 2013). Why marital functioning was linked to sleep outcomes in wives but not husbands, and cortisol in husbands but not wives, remains somewhat unclear. Some research has suggested that husbands may benefit more than wives from the marital relationship (Wanic & Kulik, 2011). For example, one study found that in dissatisfied marriages, wives reported more mental and physical health problems than their husbands; in satisfied marriages, husbands' and wives' health was equivalent (Levenson, Carstensen, & Gottman, 1993). Future research could explore the inconsistencies that exist in the literature and possibly clarify whether gender differences exist.

Specifically with perceive social support, partner effects were also observed in our study. The interpretation of these partner effects remains somewhat unclear and we would like to see the findings replicated in other studies before making any theoretical explanations.

There was some evidence to suggest a link between marital functioning cortisol. Poor dyadic adjustment was associated with higher AUC_G in husbands. This could potentially indicate that poor marital adjustment resulted in heightened activation of the hypothalamic pituitary adrenal (HPA) axis, and supports existing research linking relational strain to poor cortisol regulation (Friedman et al., 2012).

Unexpectedly, sleep latency and quality were not associated with cortisol levels for husbands or wives. As such, there was no justification to test our hypothesis that sleep might potentially mediate the effect between relationship functioning and cortisol. Our initial prediction was based on studies that have linked restricted sleep to deregulated neuroendocrine

functioning (Van Cauter et al., 2007). Thus, it remains unclear why our sleep measures were not associated with cortisol. One possible explanation could be that the sleep assessment used in this study contained questions pertaining to sleep in the context of the past month, whereas cortisol was measured over one 24-hour period. Cortisol assessed over one day may not accurately portray a normal cortisol response for an individual. Some research has indicated that there are individual differences in the extent to which people's diurnal cycles are consistent (Smyth et al., 1997). Thus, future research should utilize sleep quality and cortisol measures that reflect the same time frame to better test their association.

There are a number of limitations worth consideration. First, sleep functioning was only assessed via self-report and thus our sleep questionnaire may not provide a completely valid or reliable assessment of objective sleep functioning. Self-report measures of sleep quality have been linked to mental (Zhang, Ma, Li, Zhang, & Yu, 2011), and physical health outcomes (Jennings, Muldoon, Hall, Buysse, & Manuck, 2007), but future studies examining marital functioning and sleep outcomes could benefit from using objective measures like sleep actigraphy. Secondly, as previously discussed, cortisol was only taken over a 24-hour period and may not reflect a typical response for the participant. Assessment of cortisol across multiple days may prove informative in future research. Third, our sample consisted primarily of young, Caucasian couples, some of which were seeking therapy in a low-income community clinic. One study found that happily married women report fewer sleep disturbances, but the effect was stronger for Caucasian women than for other ethnicities, including African American women (Troxel, Buysse, Hall, & Matthews, 2009). Additionally, sleep patterns change with age, including sleep latency which has been shown to increase with age (Ohayon et al., 2004). Thus, the extent to which of findings generalize across the lifespan, socioeconomic status, or among

various ethnic groups is unclear. Finally, as this was a cross-sectional analysis, the direction of the effects cannot be verified. While there is research to support the direction of our hypotheses and findings, there are likely bidirectional effects linking marital functioning and sleep (Troxel, 2010; Troxel et al., 2007). Future studies could benefit from examining these measures at multiple time points.

Despite these limitations, this study adds to the existing literature linking marital functioning to sleep and cortisol outcomes. Multiple measures of potential marital functioning were included in this study. For example, to our knowledge, no other studies have examined perceived social support from a spouse as a predictor of both sleep and cortisol outcomes, and assessed sleep and cortisol outcomes in the context of couples seeking marital therapy. This study is also one of few that account for the dyadic nature of relational and sleep measures. Furthermore, the large sample size and vast amount of data collected make this study unique.

In summary, we have shown that poor marital functioning is related to negative sleep outcomes, an effect that is mediated by stress and potentially depression. We have also demonstrated an association between poor marital functioning to increased daily cortisol levels. Together, these findings demonstrate how marital functioning can affect both physical and psychological health. The results of this study also have clinical implications and raise the question of whether sleep disruption and cortisol regulation can be optimized by therapeutic interventions aimed at improving marital functioning. Future studies could examine whether such therapeutic interventions improve both sleep and cortisol outcomes. Research could also clarify whether gender differences exist and examine other aspects of sleep functioning that were not addressed in this study, such as sleep duration, efficiency, disturbances, and daytime dysfunction.

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Appendix A: Tables

Table 1

Main Effects for Therapy

		Sleep Latency		Sleep Quality		AUC _G Cortisol	
		Model 1	Model 2	Model 1	Model 2	Model 1	Model 2
Main Effects	Therapy → Husband DV	-.10	-.11	-.01	-.02	.13	.13
	Therapy → Wife DV	.26**	.28**	.20*	.13	.06	.07

Note. Standardized beta coefficients for Model 1 (main effects only, excluding all covariates) and Model 2 (controlling for age and BMI), *** $p < .001$, ** $p < .01$, * $p < .05$, † $p < .10$.

Table 2

Results for Omnibus Tests of Distinguishability (I-SAT) and Main Effects for Dyadic Adjustment

		Sleep Latency		Sleep Quality		AUC _G Cortisol	
I-SAT	χ^2 Value (df)	16.29 (6)		20.80 (6)		19.31 (6)	
	<i>p</i> -value	<i>p</i> = .01		<i>p</i> < .01		<i>p</i> < .01	
		Model 1	Model 2	Model 1	Model 2	Model 1	Model 2
Actor Effects	Husband DAS → Husband DV	-.25	-.26 [†]	-.00	.02	-.24	-.31 [*]
	Wife DAS → Wife DV	-.60 ^{***}	-.67 ^{***}	-.53 ^{***}	-.51 ^{***}	-.18	-.27 [†]
Partner Effects	Husband DAS → Wife DV	.27 [†]	.22	.07	.05	.26	.18
	Wife DAS → Husband DV	.30 [†]	.24	-.07	-.11	.16	.24

Note. Standardized beta coefficients for Model 1 (main effects only, excluding all covariates) and Model 2 (controlling for age and BMI), ****p* < .001, ***p* < .01, * *p* < .05, † *p* < .10.

Table 3

Results for Omnibus Tests of Distinguishability (I-SAT) and Main Effects for Ambivalence compared to Supportive (Derived from SRI and Treated as Categorical)

		Sleep Latency		Sleep Quality		AUC _G Cortisol	
I-SAT	χ^2 Value (df)	38.89 (6)		26.98 (6)		25.75 (6)	
	<i>p</i> -value	<i>p</i> < .001		<i>p</i> < .001		<i>p</i> < .001	
		Model 1	Model 2	Model 1	Model 2	Model 1	Model 2
Actor Effects	Husband Amb. → Husband DV	-.12	-.11	-.20	.05	-.07	-.16
	Wife Amb. → Wife DV	.19	.22	.27*	.28*	.01	-.01
Partner Effects	Husband Amb. → Wife DV	.16	.14	.05	.05	.06	.06
	Wife Amb. → Husband DV	-.27†	-.26†	.23†	.27*	-.25†	-.29*

Note. Standardized beta coefficients for Model 1 (main effects only, excluding all covariates) and Model 2 (controlling for age and BMI), ****p* < .001, ***p* < .01, * *p* < .05, † *p* < .10.

Table 4

Results for Omnibus Tests of Distinguishability (I-SAT) and Main Effects for Aversive compared to Supportive (Derived from SRI and Treated as Categorical)

		Sleep Latency		Sleep Quality		AUC _G Cortisol	
I-SAT	χ^2 Value (df)	14.82 (6)		20.12 (6)		16.96 (6)	
	<i>p</i> -value	<i>p</i> = .02		<i>p</i> = .003		<i>p</i> = .009	
		Model 1	Model 2	Model 1	Model 2	Model 1	Model 2
Actor Effects	Husband Aver. → Husband DV	-.23	-.15	-.09	.01	-.06	-.16
	Wife Aver. → Wife DV	.04	.04	.17	.08	.13	.24
Partner Effects	Husband Aver. → Wife DV	.28 [†]	.30 [*]	.25 [†]	.29 [*]	-.19	-.18
	Wife Aver. → Husband DV	.04	.07	.13	.15	.03	.05

Note. Standardized beta coefficients for Model 1 (main effects only, excluding all covariates) and Model 2 (controlling for age and BMI), ****p* < .001, ***p* < .01, * *p* < .05, † *p* < .10.

Table 5

Main Effects for Sleep on AUC_G Cortisol

		Sleep Latency		Sleep Quality	
		Model 1	Model 2	Model 1	Model 2
Main Effects	Husband IV → Husband AUC _G	-.01	.04	-.07	-.01
	Wife IV → Wife AUC _G	.10	.10	.02	.09

Note. Standardized beta coefficients for Model 1 (main effects only, excluding all covariates) and Model 2 (controlling for age and BMI), ***p < .001, **p < .01, * p < .05, † p < .10.

Table 6

Effects of Other Possible Variables on Sleep and Cortisol

	Sleep Latency	Sleep Quality	AUC _G Cortisol
PSS	.19**	.36***	.03
CESD	.37***	.41***	.07
Depressed	.21**	.29***	.05
Children	.09	.01	-.00
Years Married	.10	.16	-.21
Income	.08	.01	.08
Perceived SES	-.09	-.03	.06

Note. Standardized beta coefficients (controlling for age, sex, and BMI), ***p < .001, **p < .01, * p < .05, † p < .10

Appendix B: Figures

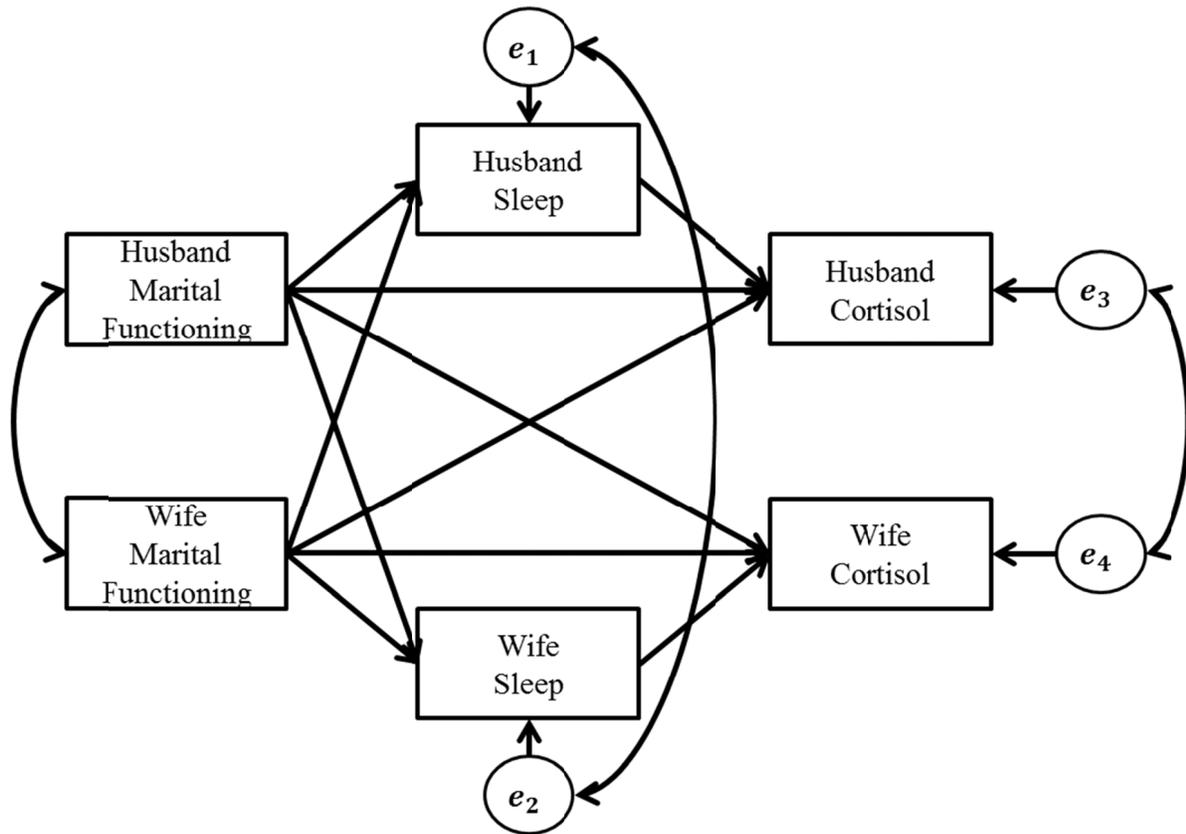


Figure 1. Theoretical actor-partner interdependence model, proposing an association between marital functioning and cortisol levels, with sleep as a potential mediator.

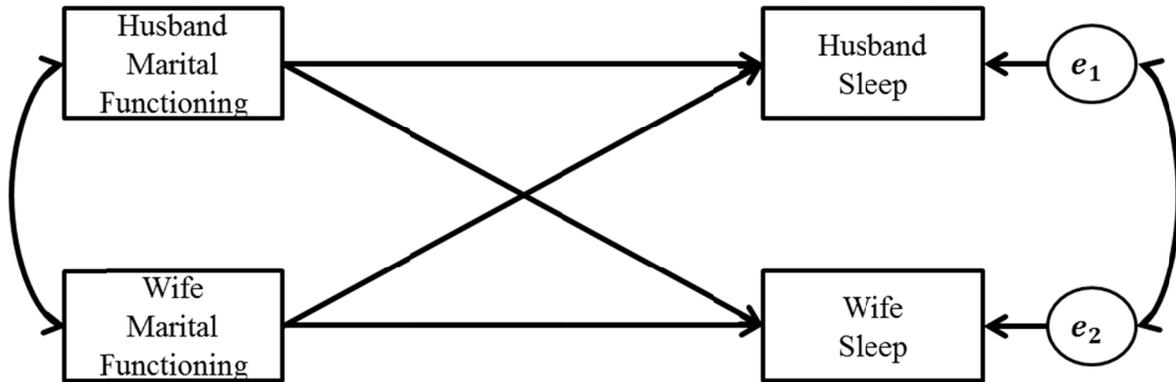


Figure 2. Theoretical actor-partner interdependence model proposing an association between marital functioning and sleep.

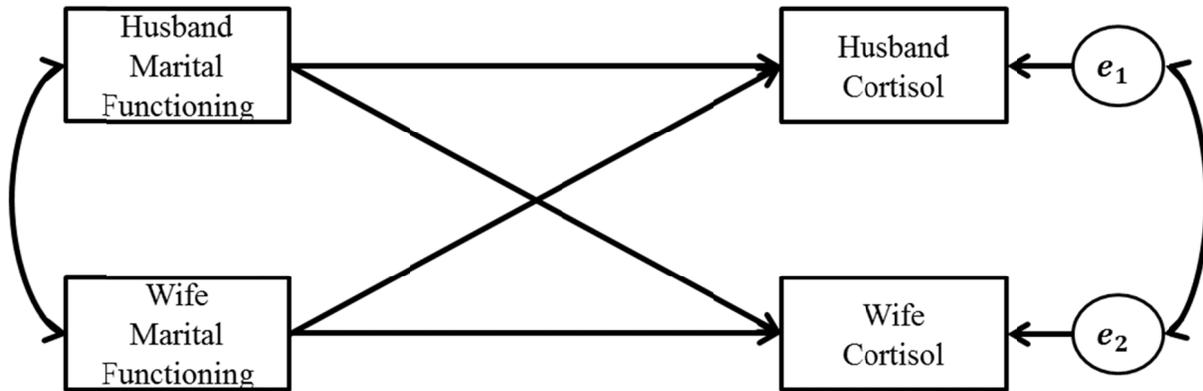


Figure 3. Theoretical actor-partner interdependence model proposing an association between marital functioning and cortisol levels.

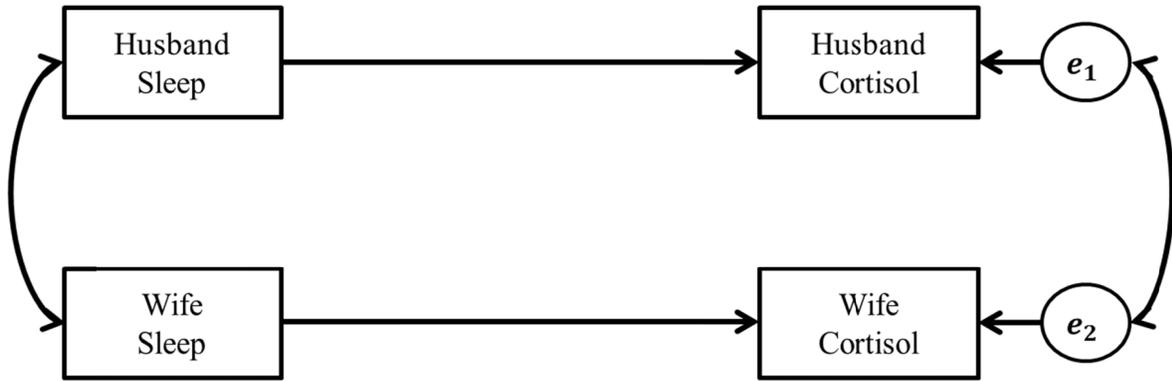


Figure 4. Theoretical model proposing an association between sleep and cortisol levels.