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Delineating the Relationship Between Insomnia, Dysfunctional Sleep Beliefs, Perceived Stress, Anxiety, and Depression

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Abstract
Insomnia is a sleep disorder highly prevalent among university students which can increase the risk for developing anxiety and depression. Vulnerability to dysfunctional sleep beliefs, and cognitive arousal (perceived stress) have been shown to be predisposing factors for insomnia. Although insomnia in university students is associated with deleterious effects, limited research has focused on this at-risk population. The aim of the current study was to further delineate the relationships between insomnia, perceived stress, dysfunctional sleep beliefs, anxiety and depression among a sample of 195 Australian university students (33 males; 162 females; \(M_{age} = 22.37, \ SD = 7.02\)). Mediation and regression analyses were conducted to test findings. Results suggested the prevalence of insomnia among Australian university students was 16.9% respectively. Regression analyses indicated that anxiety, depression and insomnia all predicted outcomes in each other, indicative of a bidirectional relationship between these variables. Dysfunctional sleep beliefs partially mediated the predictive relationships between perceived stress, anxiety, depression, and insomnia. These findings may have implications for incorporating belief-targeted insomnia treatment into cognitive behavioural therapy for anxiety and depressive disorders.

Keywords: Insomnia, perceived stress, anxiety, depression, dysfunctional sleep beliefs

Introduction
As sleep is recognised as playing an important role in many areas of health and psychological well-being, sleep disorders have become a serious public health concern. Insomnia, the most common sleep disorder, is associated with long-term physical and mental exhaustion, decreased concentration, and cognitive deficits (Morin, LeBlanc, Daley, Gregoire, & Merette, 2006). Emerging research has indicated the academic and social
pressures which accompany university life may lead to a greater prevalence of insomnia among university students (18.5%) than in the general population (7.4%) (Jiang et al., 2015). There is limited empirical information about the prevalence of insomnia among Australian university students. Furthermore, insomnia has been causally linked to a host of specific mental health issues, including anxiety and depression (Wright et al., 2011). Limited evidence suggests insomnia is bi-directionally related to anxiety and depression among the general population, however, no research has been conducted among university students (Jansson-Fröjmark & Lindblom, 2008). While depression and anxiety seem to be important predictors of insomnia, further studies suggest cognitive arousal (perceived stress) and dysfunctional sleep cognitions can lead to the development of the disorder (Harvey, 2002).

The aims of this study were to examine the prevalence of insomnia in a sample of Australian university students and to investigate the bidirectional relationship between anxiety, depression, and insomnia, and the mediating role of dysfunctional sleep beliefs in the relationships between perceived stress, anxiety, depression, and insomnia among Australian university students.

**Insomnia, Depression, and Anxiety**

Insomnia is defined as difficulties in initiating and maintaining sleep, frequent early morning awakenings, and sleep dissatisfaction (Morin, 1996). Among university students, insomnia has detrimental effects on academic performance (Kelly, Kelly, & Clanton, 2001); is associated with decreased concentration (Maquet, 2001); and increased depression and anxiety (Garlow et al., 2008). Specifically, Gaultney (2010) found that of students at risk of failing university classes, 22% presented with symptoms of insomnia.

Anxiety and depression are the most common comorbid conditions associated with insomnia (Ford & Kamerow 1989). According to Wolfson and Armitage (2009), approximately 90% of young adults with depression suffer from concomitant insomnia. Furthermore, Moo Estrella, Perez-Benitez, and Arunkowsky-Sandoval (2005) demonstrated depression was significantly related to delayed sleep onset, and disturbed sleep among students. For the most part, sleep studies investigating the relationship between depression and insomnia have almost exclusively utilised insomnia as a predictor of depressed outcomes (i.e., Ohanyon & Roth, 2003) and few studies have predicted insomnia outcomes from depression. The following study will therefore assess the association between depression and insomnia, by utilising depression as a predictor variable.

There is also a strongly defined relationship between anxiety and insomnia (Palagini et al., 2016). Monti and Monti (2004) purported anxiety is more integral in the development of insomnia than depression among
students because it is more prevalent in this population. Shamsuddin et al. (2013) found the prevalence of anxiety was significantly higher than the prevalence of depression among Malaysian university students, 34% versus 27.5% respectively. Additionally, Alfano, Pina, and Zerr (2010) suggested anxiety forms one of the most robust associations between primary emotional disturbance and insomnia. Despite this, few studies have investigated if anxiety predicts insomnia outcomes, particularly among university students. This paucity in the literature was addressed by utilising anxiety as a predictor variable in the current study.

Recent empirical shifts in the literature to investigate the possible bidirectional relationship between depression, anxiety, and insomnia have been observed (Johnson, Roth, & Breslau, 2006). Findings by Jansson-Fröjmark and Lindblom (2008) suggest the relationship between depression, anxiety, and insomnia among adults was bidirectional when longitudinally investigated. The current study aims to develop the work of Jansson-Fröjmark and Lindblom (2008) by utilising a cross-sectional design, whereby results cannot be compromised by attrition.

**Perceived Stress (Cognitive Arousal)**

In addition to anxiety and depression, cognitive arousal or perceived stress is a predictive factor associated with the development of insomnia (Bastien, Vallieres, Morin, 2001). Perceived stress is one of the most common predictors of insomnia, precipitating acute insomnia episodes, and perpetuating chronic forms of the disorder (Bastien et al., 2001). Morin and Espie (2012) suggested cognitive arousal is associated with a basal threshold; whereby individuals are more likely to experience insomnia when cognitive arousal exceeds this threshold. Presumably, individuals prone to worry operate at higher levels of cognitive arousal, which subsequently predisposes them to insomnia. The basic premise of this concept is higher levels of perceived stress (cognitive arousal) lead to greater emotional reactivity and subsequent insomnia (Adrien, 2002; Morin & Espie, 2012; Van Reeth et al., 2000).

Jarrin, Chen, Ivers, and Morin (2014) examined the relationship between perceived stress, and insomnia in a sample of adults ($N = 1449$) and found individuals who experienced a temporarily stressful condition were more likely to experience situational and subsequent insomnia than participants who did not perceive the situation as stressful. While this study established an association between perceived stress and insomnia in an adult population. Further research is warranted to examine the factors that may influence the relationship between perceived stress and insomnia. Therefore, the current study aimed to examine the predictive relationship between
perceived stress, dysfunctional sleep beliefs, and insomnia among university students.

**Dysfunctional Sleep Beliefs**

Cognitive and social theories have suggested negative rumination prior to sleeping, and high levels of perceived stress, may further contribute to symptoms of insomnia (Calkins, Hearon, Capozzoli, & Otto, 2013). Harvey (2002) proposed a cognitive model that suggests insomnia occurs in association with worry and excessive rumination (Calkins et al., 2013). Empirical evidence supports that cognitive processes represent a core element contributing to and perpetuating symptoms of insomnia (Morin & Epsie, 2012). Dysfunctional sleep beliefs, pre-sleep worry, and attentional bias towards sleep initiation significantly interfere with the circadian process (Epsie, 2002). Harvey (2002) argues individuals with sleep difficulties engage in persistent, uncontrolled negative rumination prior to sleeping. Additionally, Van Egeren, Haynes, Franzen, and Hamilton (1983) found in comparison to good sleepers, insomniacs engaged in significantly more negative thinking prior to nocturnal sleep initiation.

Dysfunctional sleep beliefs are defined as negatively toned beliefs, which represent an individual’s erroneous cognitive attempt to avoid the feared consequences associated with severe sleep loss (Harvey, 2002; Morin, 1996). Theadom and Cropley (2008) investigated the relationship between perceived stress and insomnia, in relation to dysfunctional sleep beliefs, among individuals with fibromyalgia ($n = 83$) and healthy controls ($n = 83$). Results indicated relative to healthy controls, fibromyalgia patients had significantly higher levels of perceived stress, endorsed higher levels of dysfunctional sleep beliefs, and had greater disturbed sleep. Findings support the notion that dysfunctional sleep beliefs may further contribute to the development of possible insomnia.

Clinical trials have also demonstrated that dysfunctional sleep beliefs are responsive to treatment, suggesting these may serve as a mediator in reducing insomnia, as well as improving sleep dysfunction (Morin, Blais, & Savard, 2002). To further investigate the mediating effects of dysfunctional sleep beliefs, the following study examined the mediating relationship between perceived stress, dysfunctional sleep beliefs, and insomnia among a non-clinical sample of university students.

**Current Study**

The aims of the current study were to determine the prevalence of insomnia among university students, as well as examining both the bidirectional relationship between anxiety, depression, and insomnia and the mediating role of dysfunctional sleep beliefs in the predictive relationships
between perceived stress, anxiety, depression, and insomnia among Australian university students. Based on aforementioned insomnia research, it was predicted that: (1) the prevalence of insomnia for the current sample would be similar to that of 18.5%, as reported in previous university samples; (2) the prevalence of anxiety would be higher than the prevalence of depression in the current sample (e.g., Shamsuddin et al., 2013); (3) a bidirectional relationship would exist between anxiety, depression, and insomnia; (4) dysfunctional sleep beliefs would significantly mediate the relationships between anxiety and insomnia, (5) depression and insomnia, and (6) perceived stress and insomnia.

Method
Participants
A convenience sample consisting of 195 University students aged between 18 and 57 years ($M_{age} = 22.37$, $SD = 7.02$) comprised the sample. There were 33 males (16.9%); and 162 females (83.1%). Participants were recruited at Bond University. All participants were required to be at least 18 years of age.

Materials
Insomnia Severity Index (ISI; Morin, 1996). The ISI is a 7-item self-report measure that assesses the nature, impact, and severity of insomnia; Bastien et al., 2001). A total score (i.e., 0 to 28) is calculated by summing item responses, and are categorised into severity ranges: non-clinical insomnia (0-7); subthreshold insomnia (8-14); clinical insomnia – moderate (15-21); and clinical insomnia- severe (22-28). The internal consistency of the ISI is adequate in both clinical and non-clinical populations ($\alpha = .91$; Morin, Belleville, Bélanger, & Ivers, 2011), and in the current sample ($\alpha = .84$).

Depression Anxiety and Stress Scale- 21 (DASS-21; Lovibond & Lovibond, 1995). The DASS-21 is a 21-item self-report measure assessing the presence (i.e., over the past week) of primary emotional disturbances across three subscales: depression, anxiety, and stress. While the depression and anxiety subscales have been widely validated, the operationalisation of the stress construct has been contended (e.g., Clara, Cox, & Enns, 2001). Therefore, the current study utilised the Perceived Stress Scale-Short Form (PSS-10; Cohen, Kamarck, & Mermelstein, 1983) as a specific measure of cognitive arousal (perceived stress). Total scores are calculated by summing subscale responses. The internal consistency of the DASS-21 has been deemed adequate in previous literature ($\alpha = .97$; Sinclair et al., 2012), and in the current sample (e.g., anxiety, $\alpha = .85$, and depression, $\alpha = .90$).

Perceived Stress Scale-Short Form (PSS-10; Cohen et al., 1983).
The PSS-10 is a 10-item self-report measure designed to assess the degree to which persons perceive life events as stressful relative to their ability to cope. Items are designed to assess respondent’s feelings and cognitions in the past month towards a stressful situation. A total score (ranging from 0 to 40) is calculated by summing all ten items, whereby higher scores are related to higher levels of perceived stress. The PSS-10 has demonstrated excellent internal consistency both in previous literature (\(\alpha = .90;\) Karam et al., 2012) and in the current study (\(\alpha = .83\)).

**Dysfunctional Beliefs and Attitudes about Sleep Questionnaire (DBAS-16; Morin, Vallieres, & Ivers, 2007).** The DBAS-16 is a 16-item self-report measure which assesses cognitive distortions related to insomnia (e.g., unrealistic sleep beliefs and expectations). A total score is calculated by summing all 16 items then dividing the total by 16. Total scores above four are reflective of unrealistic sleep expectations. The internal consistency of the measure has been deemed as adequate in both community (\(\alpha = .79;\) Fu, Ou, & Lu, 2014), and clinical samples (\(\alpha = .77\)), as well as in the current sample, (\(\alpha = .89\)).

**Procedure**

Prior to participant recruitment, approval for data collection was obtained through the Bond University Human Research Ethics Committee (BUHREC). Prior to survey completion, participants were required to provide their electronic informed consent as consistent with the explanatory statement by clicking “next”. Completion took approximately 35 minutes.

**Results**

**Descriptive Statistics**

The prevalence rates of clinical levels of insomnia, depression, and anxiety can be seen in Table 1. A single sample \(t\)-test indicated anxiety was significantly higher (\(t(32) = 5.81, p < .001\)) than depression (\(t(32) = 4.28, p < .001\)) in students with insomnia, supporting hypothesis one and two.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Depression</th>
<th>Anxiety</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>%</td>
<td>n</td>
<td>%</td>
</tr>
<tr>
<td>Insomnia</td>
<td>6.2</td>
<td>12</td>
<td>8.7</td>
</tr>
</tbody>
</table>

**Preliminary Analyses**

To determine whether predictor variables and criterion variables were related prior to entry in the regression equation, simple bivariate correlations were performed on all variables. Table 2 presents uncentered means, standard deviations, and intercorrelations among variables. Perceived stress, anxiety, depression, and dysfunctional sleep beliefs were significantly positively correlated with insomnia, such that higher scores on the predictor
measures (PSS-10, DASS-21 and DBAS-16) were related to higher scores on the criterion measure (e.g., ISI; Morin, 1996). Correlations were also performed on demographic variable age, and a one-way ANOVA was conducted on categorical variable gender to assess for the presence of covariates. While age did not co-vary with any variables, gender was found to significantly co-vary with perceived stress, $F(1, 194) = 4.97, p = .027$.

Thus, to control for this covariate, gender was entered in the subsequent mediation analysis that utilised perceived stress as a predictor variable.

Table 2

Summary of Intercorrelations, Uncentered Means, and Standard Deviations for PS, Anxiety, Depression, DSB, and Insomnia (N = 195).

<table>
<thead>
<tr>
<th>Variable</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>M</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>1) PS</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>17.35</td>
<td>6.38</td>
</tr>
<tr>
<td>2) Anxiety</td>
<td>.51***</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>2.98</td>
<td>3.57</td>
</tr>
<tr>
<td>3) Depression</td>
<td>.60***</td>
<td>.62***</td>
<td></td>
<td></td>
<td></td>
<td>3.60</td>
<td>4.04</td>
</tr>
<tr>
<td>4) DSB</td>
<td>.38***</td>
<td>.36***</td>
<td>.40***</td>
<td></td>
<td></td>
<td>68.14</td>
<td>28.00</td>
</tr>
<tr>
<td>5) Insomnia</td>
<td>.43***</td>
<td>.38***</td>
<td>.41***</td>
<td>.41***</td>
<td></td>
<td>9.37</td>
<td>5.27</td>
</tr>
</tbody>
</table>

*Note:***$p < .001$; PS = Perceived Stress; DSB = Dysfunctional Sleep Beliefs.

Bidirectional Analyses

Four simple regressions were conducted to examine the bidirectional relationship between anxiety, depression, and insomnia. Results indicated that anxiety significantly predicted insomnia, $F(1, 194) = 44.02, p < .001$, with higher levels of anxiety relating to higher levels of insomnia, $\beta = 0.10$, $p < .001$. Additionally, insomnia also significantly predicted anxiety, $F(1, 194) = 44.02, p < .001$, with higher levels of insomnia relating to higher levels of anxiety, $\beta = 0.04, p < .001$, indicative of a bidirectional relationship between these variables. Furthermore, depression significantly predicted insomnia, $F(1, 194) = 36.92, p < .001$, with higher levels of depression relating to higher levels of insomnia, $\beta = 0.09, p < .001$. Similarly, insomnia significantly predicted depression, $F(1, 194) = 36.99, p < .001$, with higher levels of insomnia relating to higher levels of depression, $\beta = 0.40, p < .001$, further indicating a bidirectional relationship between these variables.

Mediation Analyses

Anxiety. To test hypothesis four, a mediation analysis following Baron and Kenny’s (1986) steps was conducted to establish if the relationship between anxiety and insomnia was mediated by dysfunctional sleep beliefs. A standard multiple regression predicting dysfunctional sleep beliefs from anxiety was conducted to test pathway $a$. Results indicated the model was significant, $F(1, 194) = 29.69, p < .001$, whereby anxiety significantly accounted for 13.3% of the variance in dysfunctional sleep beliefs, $R^2_{change} = .13, p < .001$. Thus, anxiety was a significant positive predictor of dysfunctional sleep beliefs, whereby higher levels of anxiety
were related to higher levels of dysfunctional sleep beliefs, $\beta = .37$, $p < .001$.

To examine pathways $c$, $b$, and, $c'$, a hierarchical multiple regression analysis was conducted. Anxiety was entered at Step 1 and dysfunctional sleep beliefs was entered at Step 2 of the regression model. Results revealed at Step 1, the model was significant, $F(1, 194) = 44.01$, $p < .001$, whereby anxiety significantly accounted for 18.6% of the variance in insomnia, $R^2_{\text{change}} = .19$, $p < .001$. Therefore, higher levels of anxiety were related to higher levels of insomnia severity, $\beta = .43$, $p < .001$. At Step 2, the model remained significant, $F(2, 194) = 32.66$, $p < .001$, whereby dysfunctional sleep beliefs significantly accounted for 6.8% of the variance in insomnia, $R^2_{\text{change}} = .07$, $F_{\text{change}} (1, 192) = 17.53$, $p < .001$. Higher levels of dysfunctional sleep beliefs were related to higher levels of insomnia, $\beta = .28$, $p < .001$. Although the introduction of dysfunctional sleep beliefs decreased the contribution of anxiety in the prediction of insomnia, the $\beta$ coefficient remained significant, indicative of partial mediation. A Sobel Test (Sobel, 1982) indicated the decrease in the $\beta$ coefficient was statistically significant, $z = 5.41$, $p < .001$, further supporting partial mediation occurred. Figure 1 displays the direct and mediated pathways of the aforementioned model.

a) Direct Path

```
Anxiety -----> Dysfunctional Sleep Beliefs -----> Insomnia
```

$b = .28**$

```
Anxiety -----> Insomnia
```

$c = .43***$

```
d = .37***
```

Figure 1. Direct and indirect pathways between anxiety, dysfunctional sleep beliefs, and insomnia. Note: ***p < .001.

**Depression**

To test hypothesis five, a mediation analysis following Baron and Kenny’s (1986) steps was conducted to establish if the relationship between depression and insomnia was mediated by dysfunctional sleep beliefs. A standard multiple regression analysis predicting dysfunctional sleep beliefs from depression was conducted to test pathway $a$. Results indicated the model was significant, $F(1, 194) = 28.70$, $p < .001$, whereby depression significantly accounted for 12.9% of the variance in dysfunctional sleep beliefs, $R^2_{\text{change}} = .13$, $p < .001$. Thus, depression was a significant positive
predictor of dysfunctional sleep beliefs, whereby higher levels of depression were related to higher levels of dysfunctional sleep beliefs, $\beta = .36, p < .001$.

To examine pathways $c$, $b$, and, $c'$, a hierarchical multiple regression analysis was conducted. Depression was entered at Step 1, and dysfunctional sleep beliefs at Step 2. Results revealed at Step 1, the model was significant, $F(1, 194) = 36.99, p < .001$, whereby depression significantly accounted for 16.1% of the variance in insomnia, $R^2_{\text{change}} = .16, p < .001$. Therefore, higher levels of depression were related to higher levels of insomnia severity, $\beta = .40, p < .001$. At Step 2, the model remained significant, $F(2, 194) = 29.68, p < .001$, whereby dysfunctional sleep beliefs significantly accounted for 7.5% of the variance in insomnia, $R^2_{\text{change}} = .08, F_{\text{change}} (1, 192) = 18.93, p < .001$. Therefore, higher levels of dysfunctional sleep beliefs were related to higher levels of insomnia, $\beta = .29, p < .001$. Although the introduction of dysfunctional sleep beliefs decreased the contribution of anxiety in the prediction of insomnia the $\beta$ coefficient remained significant, indicative of partial mediation. A Sobel Test (Sobel, 1982) indicated the decrease in the $\beta$ coefficient was statistically significant, $z = 3.32, p < .001$, further supporting the occurrence of partial mediation. Figure 2 displays the direct and mediated pathways of the aforementioned model.

a) Direct Path

| Depression | $c = .40^{***}$ | Insomnia |

b) Mediated Path

| Depression | $a = .36^{***}$ | Dysfunctional Sleep Beliefs | $b = .29^{***}$ |

| Dysfunctional Sleep Beliefs | $c' = .30^{***}$ | Insomnia |

*Figure 2. Direct and indirect pathways between depression, dysfunctional sleep beliefs, and insomnia. Note: ***$p < .001$.*

**Perceived Stress**

To test hypothesis six, a mediation analysis following Baron and Kenny’s (1986) steps was conducted to determine if the relationship between perceived stress and insomnia could be mediated by dysfunctional sleep beliefs. A standard multiple regression analysis predicting dysfunctional sleep beliefs from perceived stress and gender was conducted to test pathway $a$. Gender was entered with perceived stress at Step 1 of the regression to control for its effects. Results indicated the model was significant, $F (2, 194) = 17.06, p < .001$, whereby gender and perceived stress together accounted for 15.1% of the variance in dysfunctional sleep beliefs, $R^2_{\text{change}} = .15, p < .001$. 

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Thus, perceived stress was a significant positive predictor of dysfunctional sleep beliefs such that higher levels of perceived stress were related to higher levels of dysfunctional sleep beliefs, $\beta = .38, p < .001$.

To examine pathways $c$, $b$, and, $c'$, a hierarchical multiple regression analysis was conducted. Both gender and perceived stress were entered at Step 1, and dysfunctional sleep beliefs was entered at Step 2. Results revealed at Step 1 the model was significant, $F(2, 194) = 22.34, p < .001$, whereby gender and perceived stress conjointly contributed 18.9% of the variance in insomnia $R^2_{\text{change}} = .19, p < .001$. Therefore, higher levels of perceived stress were related to higher levels of insomnia severity, $\beta = .43, p < .001$. At Step 2 the model remained significant, $F(3, 194) = 21.45, p < .001$, whereby dysfunctional sleep beliefs significantly accounted for 6.3% of the variance in insomnia, $R^2_{\text{change}} = .06, F_{\text{change}} (1, 191) = 16.16, p < .001$. Therefore, higher levels of dysfunctional sleep beliefs were related to higher levels of insomnia, $\beta = .27, p < .001$. Although the introduction of dysfunctional sleep beliefs decreased the contribution of perceived stress in the prediction of insomnia, the $\beta$ coefficient remained significant, indicative of partial mediation. A Sobel Test (Sobel, 1982) revealed the decrease in the $\beta$ coefficient was statistically significant, $z = 3.22, p < .001$, further supporting partial mediation occurred. Figure 3 displays the direct and mediated pathways of the aforementioned model.

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{figure3.png}
\caption{Direct and indirect pathways between perceived stress, dysfunctional sleep beliefs, and insomnia. Note: ***$p < .001$.}
\end{figure}

**Discussion and conclusion**

The present study aimed to establish the prevalence of insomnia, and the prevalence of anxiety and depression in individuals with insomnia, in an Australian university student population. Additionally, the bidirectional relationship between anxiety, depression, and insomnia, and the mediating role of dysfunctional sleep beliefs in the predictive relationships between perceived stress, anxiety, depression, and insomnia among students was examined.
Support was provided for hypothesis one, as the prevalence rate of insomnia among students sampled was 16.9%, similar to previous literature stating insomnia in university students is typically higher than the general population (Jiang et al., 2015). Support was also provided for hypothesis two, as the prevalence for anxiety was higher than for depression among students, 6.2% versus 8.7% respectively. Findings are consistent with previous literature suggesting that rates of anxiety are typically higher than rates of depression among university students (Shamsuddin et al., 2013). Results also support the notion that anxiety may play a more integral role in the development of insomnia than depression (Monti & Monti, 2004), and that the most robust associations between emotional functioning and insomnia involve anxiety (Alfano et al., 2010).

The third hypothesis predicting a bidirectional relationship between depression, anxiety, and insomnia was supported, as significant intercorrelations existed between variables. Results are consistent with previous longitudinal research that established a bidirectional association between anxiety, depression, and insomnia over a year (Jansson-Fröjmark & Lindblom, 2008). The clinical implications of this finding are significant, as treatment of mood disorders may mitigate students risk of future insomnia and vice versa (Ohanyon & Roth, 2003).

The final three hypotheses were partially supported, as all results yielded partial mediations. The amount of variance accounted for by dysfunctional sleep beliefs in the predictive relationships between depression, anxiety, perceived stress and insomnia was not enough to reduce the statistical contribution of the predictor variables to non-significance. Therefore, aspects of the relationship between perceived stress, anxiety, depression, and insomnia were not accounted for by dysfunctional sleep beliefs. As such, a cognitive model utilising dysfunctional sleep beliefs cannot be considered in isolation as a mediating variable for these relationships. Biological theories of insomnia postulate that neuroendocrine dysfunction (hypothalamic-pituitary-adrenal axis abnormality) and functional decreases in serotonergic neurotransmission influence the development of stress, depression, anxiety, and insomnia (Adrien, 2002; Van Reeth et al., 2000). Therefore, the partial mediations obtained in the current study may reflect a widespread and complex relationship between focal variables.

The findings of the current study should be interpreted with consideration of some limitations. Given the correlational nature of the design, causal inferences cannot be made about the predictive and mediating factors of insomnia investigated in this research (Espie, 2002). In future research, an experimental design should be employed to enhance both the causal nature and reliability of the results. The utilisation of an objective
sleep measure (i.e., polysomnography) may also enhance the reliability of the results by decreasing error associated with retrospective accounts of sleep disturbance (Jansson-Fröjmark & Lindblom, 2008). Future studies may also benefit from utilising a prospective design to enhance the reliability of accurate responding (Ohanyon & Roth, 2003).

The current study investigated some of the potential psychological mechanisms that influence the development of insomnia. The high prevalence of insomnia, depression, and anxiety in the current sample suggest university students are a particularly susceptible population. This research demonstrates that insomnia and comorbid conditions have a bidirectional effect. In addition, dysfunctional sleep beliefs significantly mediated all three relationships between perceived stress, anxiety, depression, and insomnia. Both findings have implications for treatment courses and outcomes. Specifically, the current study provides support for incorporating belief-targeted insomnia treatment into cognitive behavioural therapy for anxiety and depressive disorders.

Given the negative ramifications of insomnia on university students, implementing evidence based education and treatment programs targeted at reducing perceived stress, depression, and anxiety, as well as decreasing negative rumination prior to sleeping, would be beneficial to students (Harvey, 2002). Furthermore, given the societal costs associated with poor academic performance often related to symptoms of insomnia, such programs may assist students in achieving their optimal academic success, while decreasing the risk of developing potential comorbid psychiatric conditions in later adulthood (Ohanyon & Roth, 2003).

References:


