Abstract

The goal of the current study was to examine whether anxiety acts as a moderator in the association between sleep and inflammation. Poor sleep patterns have been linked to disruptions in inflammation factors (Irwin, 2019). This study utilized the Midlife in the United States (MIDUS) Biomarker data (n=1255) to test the moderating role of anxiety in the sleep and inflammation relationship. Findings showed that anxiety symptoms exacerbated the negative effects that sleep disturbances have on the dysregulation of inflammation factors. This study has implications for reducing the level of stress experienced to lessen the impact of sleep disturbances.

Introduction

- Twenty five percent of the adult population in the United States report insomnia concerns (Irwin, 2016).
- Poor sleep patterns are linked to disruptions in inflammation factors, specifically increases in the levels of expressed C-Reactive Protein and Interleukin-6 (Irwin, 2016).
- Other studies have found increases in C-Reactive Protein and Interleukin-6 with additional habitual sleep time (Patel et al., 2009).
- Individuals with anxiety disorders commonly report longer sleep onset latency and decreased time spent asleep and efficiency (Papadimitriou & Linkowski, 2005).
- Higher levels of anxiety have been linked to increased levels of inflammation factors in healthy adults (Pitavsos et al., 2006).
- Increased levels of inflammation factors is linked to adverse health consequences, such as heart attacks and diabetes (Patel et al., 2009).
- Anxiety will moderate the link between sleep and inflammation factors, such that at higher levels of anxiety, sleep disturbances will predict to higher levels of inflammation.

Hypothesis

Moderation Model

Method

Participants: The sample included 1255 individuals from the MIDUS II biomarker study.

Measures:
- Sleep
  - Pittsburg Sleep Quality Index Factors (subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbance, use of sleep medications, daytime dysfunction, and a global sleep scale (Buysse et al., 1988)
  - ActiGraph watch data (Number of sleep and wake bouts, onset latency, sleep efficiency, sleep hours, snooze time, wake after sleep onset, wake time, and average duration of sleep and wake bouts)
- Inflammation
  - Blood Creatinine, serum Interleukin-6, serum soluble interleukin-6 receptor, blood fibrinogen, blood C-Reactive Protein, serum soluble E-Selectin, and serum soluble intercellular adhesion molecule-1
- Anxiety
  - General Distress-Anxious (α=.82), Positive Affect (α=.93), and Anxious Arousal (α=.81) derived from Mood and Symptom Questionnaire (Clark & Watson, 1991)
  - Spielberger Trait Anxiety Inventory (α=.91) (Spielberger, 1983)
  - Social Anxiety Scale (α=.80) (Fresco et al., 2001)

Analyses: Moderated regression analysis was conducted using the PROCESS macro in SPSS (Hayes, 2009).

Results

1326 moderations of the relationships between sleep variables and inflammation factors by anxiety variables were tested. 213 moderations were found to be statistically significant.

As identified in Figure 1, social anxiety was a significant moderator in the relationship between the global sleep scale and ICAM-1 (B= -3.21, p=.039, LLCI= -17, ULCI= 6.26). At moderate and high levels of anxiety, higher levels of sleep disturbances were associated with elevated levels of ICAM-1.

Figure 2 shows a moderation effect of social anxiety in the relationship between sleep disturbances and E-Selectin (B= 3.97, p=.043, LLCI= -12, ULCI= 6.26). There is positive relationship between sleep disturbances and E-Selectin when anxiety is high; however, there is no relationship when anxiety is moderate and low.

As indicated in Figure 3, trait anxiety was a significant moderator of the relationship between sleep onset latency and E-Selectin (B= -0.092, p=.001, LLCI= -0.04, ULCI= 0.015). At high levels of trait anxiety (but not low and moderate levels), higher onset latency predicted higher E-Selectin levels.

As shown in Figure 4, anxious distress symptoms were a significant moderator of the relationship between sleep efficiency and ICAM-1 (B= -0.001, p=.035, LLCI= -0.17, ULCI= -0.001). The relationship between decreased sleep efficiency and higher levels of ICAM-1 was significant at moderate and high levels of anxious distress, but not at low levels.

Discussion

- Purpose →
  - Examining whether reported anxiety moderates the relationship between sleep and inflammation factors.
- Findings →
  - Anxiety exacerbated the link between sleep components and inflammation factors across varied measures of the sleep and inflammation. 
  - Experiencing higher levels of anxiety strengthened the connections between dysregulated sleep and inflammation factors.
- Implications →
  - Decreasing the levels of stress individuals experience could help to decrease their levels of inflammation factors and potential for negative health outcomes.
- Limitations →
  - Participants were obtained through convenience sampling, so the results may not be generalizable.
  - Self-report measures were used for anxiety which are subjective.
- Future research →
  - Should examine physiological indicators of anxiety and attempt to establish causal links between these variables.