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Insomnia Mediates the Longitudinal Relationship between Anxiety and Depressive Symptoms in a Nationally Representative Sample of Adolescents

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Abstract

Background—Anxiety and depression are commonly comorbid with each other, with anxiety often temporally preceding the development of depression. Although increasingly research has begun to investigate the role of sleep problems in depression, no study has examined insomnia as a mediator in the longitudinal relationship between anxiety and subsequent depression.

Methods—The current study utilizes data from Waves I, II, and IV of the National Longitudinal Study of Adolescent to Adult Health, a nationally representative prospective study conducted over a 14-year period (n = 20,745, 50.5% female, M age at Wave I = 16.20). Participants completed portions of the Center for Epidemiologic Studies Depression Scale (CES-D) at Waves I and IV to assess depressive symptoms, a six-item anxiety measure at Wave I, and three items assessing insomnia, sleep quality, and sleep duration at Wave II.

Results—Structural equation modeling indicated that insomnia and unrestful sleep significantly mediated the relationship between anxiety and subsequent depression. The relationship between anxiety and depression was not significantly mediated by sleep duration.

Conclusions—Findings suggest that anxiety may increase risk for the development of later depression through insomnia.

Keywords

Anxiety/Anxiety Disorders; Depression; Sleep Disorders; Child/Adolescent; Mood Disorders

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Conflict of Interest: Y. Irina Li, Lisa R. Starr, and Laura Wray-Lake declare that they have no conflict of interest.

Compliance with Ethical Standards

Ethical Approval: All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed Consent: Informed consent was obtained from all individual participants included in the study.

Introduction

Depression and anxiety disorders are commonly comorbid with each other, with estimates of current and lifetime comorbidity often among the highest of any disorders (Avenevoli, Stolar, Li, Dierker, & Merikangas, 2001; Brown, Campbell, Lehman, Grisham, & Mancill, 2001; Yorbik, Birmaher, Axelson, Williamson, & Ryan, 2004). Between 27% and 77% of individuals with a principal diagnosis of an anxiety disorder meet lifetime criteria for depression (Brown et al., 2001). There is reason to believe that anxiety functions as a direct risk factor for the development of later depression. Anxiety often temporally precedes depression (Kaufman & Charney, 2000; Starr, Hammen, Connolly, & Brennan, 2014) and predicts later depression both longitudinally and within daily patterns (Cole, Peeke, Martin, Truglio, & Seroczynski, 1998; Starr & Davila, 2012b). Most anxiety disorders have average ages of onset in childhood, with rates of anxiety-depression comorbidity substantially increasing during later adolescence and young adulthood (Merikangas et al., 2010; Wittchen, Kessler, Pfister, Höfler, & Lieb, 2000). Although the association between anxiety and depression is likely bidirectional (Jacobson & Newman, 2017), converging evidence suggests that for a substantial portion of comorbid youth, anxiety increases risk for depression under a likely “direct causation” model (Cummings, Caporino, & Kendall, 2014; Mathew, Pettit, Lewinsohn, Seeley, & Roberts, 2011). However, little is known about processes that may underlie this transition. A small emerging literature has begun to pinpoint factors such as interpersonal dysfunction, negative anxiety response styles, rumination, attentional biases, and behavioral avoidance (Jacobson & Newman, 2014; Jacobson & Newman, 2016; Price et al., 2016; Starr et al., 2014; Starr, Stroud, & Li, 2016) as processes that may bridge anxiety with depression during adolescence, but more work is needed to identify other potential mechanisms.

Strong evidence suggests that sleep problems may play a critical role in both depression and anxiety (Baglioni et al., 2011; Benca, Obermeyer, Thisted, & Gillin, 1992; Dahl & Harvey, 2007). Sleep disturbances are present in high rates in individuals with anxiety and depressive disorders in both adults and children (Johnson, Chilcoat, & Breslau, 2000; van Mill, Hoogendijk, Vogelzangs, van Dyck, & Penninx, 2010). Although research has more recently begun to examine sleep in the context of both anxiety and depression, none have specifically examined the role of insomnia in the temporal relationship between the two. Research on the directionality of sleep and anxiety has been somewhat mixed, although evidence suggests that anxiety may precede sleep problems. Data from a large-scale longitudinal study suggest that generalized anxiety may predict increases in sleep problems over time (Shanahan, Copeland, Angold, Bondy, & Costello, 2014). Consistent with a view of insomnia as an intervening link in the temporal relationship between anxiety and depression, Ohayon and Roth (2003) found that while insomnia primarily preceded a diagnosis of mood disorder or appeared concurrently, in the case of anxiety disorders, insomnia appeared mostly either following anxiety or concurrently rather than before. Another study found similar results, with anxiety disorders preceding insomnia in the majority of cases among adolescents with comorbid disorders; further, although prior anxiety disorder was associated with increased risk of insomnia, prior insomnia was not significantly associated with anxiety onset (Johnson, Roth, & Breslau, 2006). Anxious individuals have been found to engage in worry

about sources of their anxiety as well as about sleep, which may contribute to sleep difficulties (Kelly, 2002; McCann & Stewin, 1988). Generalized anxiety is associated with disrupted sleep, including longer sleep latency and decreased percentage of deep sleep (Alfano, Reynolds, Scott, Dahl, & Mellman, 2013; Fuller, Waters, Binks, & Anderson, 1997). Anxious individuals are more likely to engage in repetitive, ruminative thinking regarding anxiety symptoms, which may be particularly disruptive to sleep (Starr & Davila, 2012a).

Although sleep disturbance is a criterion for depression, insomnia is also increasingly being conceptualized as a mechanism in depression etiology (Baglioni et al., 2011; Buysse, Germain, Hall, Monk, & Nofzinger, 2012). A large body of literature has consistently supported insomnia and sleep problems as risk factors for later depression in both children and adults (Baglioni et al., 2011; Gregory, Rijsdijk, Lau, Dahl, & Eley, 2009; Roane & Taylor, 2008). In a systematic review, Alvaro and colleagues (2013) found evidence for bidirectionality of sleep disturbances with depression, although insomnia appeared to be a stronger and more consistent predictor of subsequent depression than depression is of subsequent insomnia. A meta-analysis found that sleep disturbance significantly predicted development of depression in adolescents, providing further support for sleep disturbance as a risk factor (Lovato & Gradisar, 2014). Individuals with major depression and insomnia symptoms report greater risk of depression recurrence and higher rates of comorbid anxiety than those without insomnia (Soehner & Harvey, 2012). Several theories have been proposed to explain why insomnia may lead to increased risk for depression, including disrupted emotion regulation and impaired reward processing (Harvey, Murray, Chandler, & Soehner, 2011; Luking, Pagliaccio, Luby, & Barch, 2016).

Thus, insomnia appears to play a role in the transition from anxiety to depression. However, limitations of existing studies include employment of cross-sectional and retrospective designs that limit both interpretability as well as strength of conclusions. Longitudinal studies may be especially important in increasing understanding of these processes. Most anxiety disorders typically develop by late childhood and early adolescence, whereas depressive symptoms increase in frequency during adolescence into adulthood (Nolen-Hoeksema & Girgus, 1994; Wittchen et al., 2000). Sleep patterns also alter dramatically during adolescence, driven by physiological as well as environmental changes (Dahl & Harvey, 2007; Dahl & Lewin, 2002). Thus, an examination of longitudinal associations between anxiety and depression may be particularly useful in understanding how this transition occurs.

In the current study, we investigated the relationship between anxiety and depressive symptoms, focusing on insomnia as a potential mediator in this association, utilizing a nationally representative longitudinal sample of adolescents followed for over a decade. As symptoms of insomnia can include both difficulty initiating or maintaining sleep and poor sleep quality (unrestful sleep) (American Psychiatric Association, 2013), we examined both in the present study. Additionally, as evidence suggests that sleep quality and duration may have differential associations with psychological outcomes (Pilcher, Ginter, & Sadowsky, 1997), we also examined sleep duration separately. We hypothesized that the relationship

between anxiety and subsequent depressive symptoms would be mediated by sleep problems (insomnia, unrestful sleep, and decreased sleep duration).

Material and Methods

Participants

Data came from the National Longitudinal Study of Adolescent to Adult Health (Add Health), a nationally representative prospective study (Harris et al., 2009). The study used a clustered sampling design based on a stratified sample of 80 high schools and 52 paired middle schools. Clusters were sampled with unequal probability of selection. This study used data from in-home questionnaires, which were collected in four waves. As we were interested in examining the long-term effect of anxiety on depressive symptoms in adulthood, the current study uses Waves I (W1), II (W2), and IV (W4) of Add Health's restricted-access dataset: W1 was collected during 1994-1995; W2 occurred during 1995-1996; W4 was collected during 2007-2008. Of the full sample of 20,745 adolescents who participated in W1, 28.9% (n = 6009) were missing in W2 and 24.3% (n = 5046) participants were non-responders in W4. Table 1 displays demographic information of the sample at baseline.

Measures

Anxiety Symptoms—Anxiety was assessed at W1 with six items constituting a physiological symptom-based measure, as in prior research (see Jacobson & Newman, 2014; Jacobson & Newman, 2016; Noel, Groenewald, Beals-Erickson, Gebert, & Palermo, 2016). Table 1 lists study items. Internal consistency for the items in the current sample was α = .61. Internal consistency is comparable to previous studies that have grouped the same items together into a single anxiety factor (Jacobson & Newman, 2016; Noel et al., 2016).

Depressive Symptoms—Depressive symptoms at W1 and W4 were measured using portions of the adapted Center for Epidemiologic Studies Depression Scale (CES-D; Radloff, 1977), a brief self-report questionnaire designed to measure depressive symptoms in the general population. As unequal numbers of items from the CES-D were administered across waves, in the current study we used only items that were administered in both W1 and W4. We also excluded items that conceptually overlap with sleep ["*You felt that you were too tired to do things*"] and anxiety ["*You were bothered by things that usually don't bother you*"]. Previous examinations of the reduced scale supported a single factor structure with good internal consistency (Catrett & Gaultney, 2009; Jacobson & Newman, 2014). Additionally, combining the reduced scale with anxiety items yielded two distinct depression and anxiety factors (Jacobson & Newman, 2014). Internal consistency in this sample was good for W1 and W4 (α = .87 and .84, respectively).

Sleep Variables—Sleep variables were measured at W2 with one item each assessing difficulty with sleep initiation or maintenance (insomnia) and unrestful sleep. An additional item assessed typical total sleep time (TST) [Table 2]. Previous studies with the Add Health dataset have used both the single insomnia item and TST item to operationalize insomnia

and sleep duration, respectively (see Asarnow, McGlinchey, & Harvey, 2014; Roane & Taylor, 2008; Wong, Robertson, & Dyson, 2015).

Covariates—As the anxiety scale is comprised of physiological symptom-based items that could feasibly be indicative of physical health problems, to partial out the effects of this potential confound, a single item assessing self-perceived general health at W1 was also included as a covariate. W1 depressive symptoms, gender (1 = male, 2 = female), age, and socioeconomic status (SES) were also entered as controls. SES was a composite of mother and father education and family income.

Statistical Analysis

We conducted structural equation modeling in M*plus 7.4*. Prior to each analysis, the model's assumptions were tested. The data met statistical assumptions for multivariate normality, linearity, and non-multicollinearity. Table 3 displays Pearson's product moment correlations, means, and standard deviations for study variables. We first conducted a confirmatory factor analysis (CFA) to test the model fit of the anxiety and depression latent variables. A structural model was then examined that included the three sleep items as mediators, insomnia, unrestful sleep, and TST. The model included paths from anxiety at W1 predicting insomnia, unrestful sleep, and TST at W2, to depressive symptoms at W4 as the outcome variable. Anxiety was modeled as both a direct and indirect predictor of depressive symptoms through insomnia, unrestful sleep, and TST. W1 Depression was included as a predictor of W4 depression to control for the influence of prior depression. Gender, SES, age, and health were also included as controls. Robust maximum likelihood (MLR) estimation method was used as it provides standard errors robust to unmodeled nonnormality of data (Muthén & Muthén, 2007). Full-information maximum-likelihood (FIML) estimation was used to estimate missing values (Arbuckle, Marcoulides, & Schumacker, 1996).

Practical indices of goodness of fit were evaluated in place of the chi-square statistic, as chi-square values are highly influenced by large sample sizes. Model fit was evaluated based on the following criteria (Kline, 2015): Tucker-Lewis Index (TLI) ≥ .90, with > .95 preferred, Root Mean Squared Error of Approximation (RMSEA) and Standardized Root Mean Square Residual (SRMR) < .05. If model fit was less than acceptable, modification indices were examined for correlated error terms that were conceptually justified and would enhance model fit.

Results

A CFA was conducted to examine the factor structure of the W1 depression and anxiety indicators. Results showed adequate fit to the data (χ^2 (102) = 5147.26, p < .001, TLI = .917, RMSEA = .049, SRMR = .037), with all factor loadings significant at p < .001. After examination of modification indices, the residuals of select items on the CES-D were correlated to improve model fit.

We then tested the structural model, examining whether insomnia, unrestful sleep, and TST at W2 significantly mediated the relationship between W1 anxiety and W4 depressive

symptoms, controlling for W1 depression, general health, gender, age, and SES [Figure 1]. The resulting model showed adequate fit to the data: $\chi^2(350) = 8905.44$, $p < .001$, TLI = .90, RMSEA = .03, SRMR = .03. Supporting our hypothesis, the indirect effect of W1 anxiety to W4 depressive symptoms through insomnia at W2 was significant and positive (indirect effect = .020, *SE* = .006, $p < .001$, 95% CI [.009, .031]). Unrestful sleep also significantly mediated the relationship between W1 anxiety and W4 depressive symptoms (indirect effect = .019, *SE* = .004, $p < .001$, 95% CI [.010, .027]). The residual direct effect showed that anxiety was not significant in predicting later depressive symptoms ($\beta = .004$, *SE* = .022, $p = .844$), suggesting that insomnia and unrestful sleep significantly mediated the relationship between anxiety and subsequent depressive symptoms. W1 anxiety predicted TST at W2, but TST did not predict depressive symptoms at W4. TST was not significant in mediating the relationship between anxiety and depressive symptoms (indirect effect = .00, *SE= .002,* $p = .790$, 95% CI [-.004, .003]).

As both insomnia and hypersomnia are associated with depression (Liu et al., 2007), we also examined potential non-linear effects of TST. We conducted a separate model, entering both linear and quadratic terms for TST. The resulting model showed adequate fit to the data ($\chi^2(325) = 8329.15$, $p < .001$, TLI = .90, RMSEA = .03, SRMR = .04). Neither the linear term (indirect effect = .001, *SE* = .001, $p = .273$, 95% CI [-.001, .004]) nor quadratic term (indirect effect = .002, SE = .001, $p = .059$, 95% CI [.000, .005]) was significant in mediating the anxiety—depressive symptoms relationship.

Discussion

Using a nationally representative sample, we sought to examine the role of insomnia and TST in mediating the relationship between anxiety in adolescence and depression in adulthood. Consistent with our hypothesis, results provided evidence in support of insomnia as a mediator in the relationship between anxiety and subsequent depressive symptoms. To our knowledge, this is the first study to examine a mediational role of insomnia in the sequential comorbidity of anxiety and later depression. Worrying and catastrophic thinking characteristic of anxiety disorders may contribute to sleep related problems, such as delays in sleep onset (Harvey, 2000; Kelly, 2002). Individuals who are more anxious may be more sensitive to normal disruptions in sleep. The three-factor model of insomnia suggest that over time as sleep problems persist, anxiety over insomnia itself becomes a perpetuating factor (Spielman, Caruso, & Glovinsky, 1987); anxiety may thus increase risk for both emergence and maintenance of insomnia.

In turn, individuals with sleep problems are at greater risk for developing subsequent depression (Baglioni et al., 2011; Roane & Taylor, 2008). Depression has been hypothesized to be a disorder of impaired emotion regulation (Joormann & Gotlib, 2010), with maladaptive emotion regulation more likely to result in increased mood instability and vulnerability to depressed mood (Ehring, Tuschen-Caffier, Schnülle, Fischer, & Gross, 2010; Erk et al., 2010). Sleep plays a crucial role in emotion regulation, with sleep deprivation and restriction associated with subjective reports of increased negative emotionality and affective instability (Dinges et al., 1997; Horne, 1985). Additionally, sleep loss blunts positive benefits associated with rewarding experiences (Zohar, Tzischinsky, Epstein, & Lavie,

2005). Sleep also plays a role in maintaining regulation of physiological reactivity to stress (Buckley & Schatzberg, 2005; Meerlo, Sgoifo, & Suchecki, 2008). Elevated cortisol levels have been found in individuals after partial sleep deprivation, suggesting that disturbances in sleep may increase physiological stress reactivity (Meerlo et al., 2008), potentially impacting individuals' abilities to successfully cope with stress. It appears that insomnia may decrease adaptive responding to stress and increase emotional dysregulation.

Poor sleep may also negatively influence cognitive processes such as attention, impulse control, and memory. Children experiencing sleep loss or disruptions report difficulties with focused attention and modulating impulses (Dahl, 1996; Dahl, 1999; Wolfson & Carskadon, 1998). Sleep also plays an important role in emotional memory encoding and consolidation (Hu, Stylos-Allan, & Walker, 2006). Although sleep deprivation impairs memory formation generally, encoding of negative memory appears to be more resistant to sleep loss (Yoo, Gujar, Hu, Jolesz, & Walker, 2007), potentially contributing to the decreased recall specificity of positive autobiographical memories compared to negative memories seen in depression (van Vreeswijk & de Wilde, 2004). It is possible that for individuals with anxiety, insomnia further contributes to the predominance of memory encoding and recall for negative events, contributing to greater depressive symptoms over time.

In addition to self-reported insomnia, we also found support for the role of subjective sleep quality as a mediator in the anxiety-depression relationship. Studies using neuroimaging techniques have shown that subjective reports of sleep quality are significantly related to amygdala reactivity as well as to reports of negative affect and stress, suggesting that self-perceived sleep quality may be an indicator of emotional processing efficacy during sleep (Goldstein & Walker, 2014; Prather, Bogdan, & Hariri, 2013). Indeed, subjective poor sleep quality has been found to be associated with increased risk for depression as well as emotion dyregulation (Franzen, Buysse, Rabinovitz, Pollock, & Lotrich, 2010; Mauss, Troy, & LeBourgeois, 2013; Park, Meltzer-Brody, & Stickgold, 2013), independent of disrupted sleep initiation and maintenance. As subjective sleep quality appears to reflect sleep depth and continuity (Argyropoulos et al., 2003), our findings suggest a unique role of perceived sleep quality in the relationship between anxiety and depression.

We did not find evidence for the role of sleep duration in mediating the relationship between anxiety and later depression. This is not entirely surprising as previous research has often failed to find effects of shorter sleep duration, suggesting that self-perceived sleep problems may be more strongly associated with negative outcomes than sleep duration (Asarnow et al., 2014; Pilcher et al., 1997). Individuals suffering from insomnia commonly both under- and over-estimate their own sleep duration, with some evidence suggesting that sleep misperception may occur to a greater degree in individuals with depression (Fernandez-Mendoza et al., 2011; Tsuchiyama, Nagayama, Kudo, Kojima, & Yamada, 2003). Estimates of sleep duration may be influenced by other factors such as mood and impaired cognitive function (Bastien et al., 2003). Further, estimates of one's typical sleep amount (as in the present study) may be more likely to be influenced by retrospective bias and perception of one's own sleep difficulties, particularly as sleep problems persist. In contrast, objective shortened sleep duration is associated with greater severity of insomnia as well as risk for depression and poorer treatment outcomes (Fernandez-Mendoza et al., 2015; Troxel et al.,

2012; Vgontzas, Fernandez-Mendoza, Liao, & Bixler, 2013). Thus, future studies should examine objective sleep duration in anxiety and depression comorbidity.

Our findings should be considered in context of several limitations. First, despite the heterogeneity of sleep related problems, insomnia and sleep quality were assessed using a single self-report item. Although previous studies using Add Health data have similarly used the same item to measure insomnia (e.g., Jacobson & Newman, 2016; Wong & Brower, 2012), future studies should examine the effect of other forms of sleep difficulties on anxiety and depression using objective measures. Additionally, anxiety and depression symptoms were assessed via self-report. In particular, anxiety was assessed using reported physiological symptoms, which excludes cognitive components of anxiety that may contribute to sleep disruptions. Physiological symptoms may also be indicative of other problems associated with depression, although this is somewhat mitigated in this study by the inclusion of a general health item. Further research should explore potential differential relationships between insomnia and various forms of anxiety. However, despite these limitations in measurement, our findings were robust given the use of a large nationally representative sample.

Although we did not examine whether sleep problems mediate the bidirectional relationships between depression and later anxiety, it is possible that insomnia is a potential mechanism through which this process occurs. Depression has been shown to increase risk for insomnia in adults and adolescents (Roberts & Duong, 2013; Sivertsen et al., 2012). In turn, insomnia and sleep disturbances could feasibly increase anxiety (Gillin, 1998; Jansson-Fröjmark & Lindblom, 2008). Adolescents with sleep disruptions experience more self-reported anxiety as well as greater likelihood of engaging in catastrophizing and rate their worries as more threatening (Talbot, McGlinchey, Kaplan, Dahl, & Harvey, 2010). Although we were not able to examine insomnia as a mediator of the bidirectional relationship due to lack of anxiety data at Wave IV, future studies should seek to clarify and disentangle these potentially bidirectional relationships.

Conclusions

The current study has theoretical and clinical implications for the study and treatment of comorbid anxiety and depression. First, it adds to emerging research on the role of anxiety as a risk factor for later depression, and highlights insomnia as a potential process through which this temporal relationship unfolds. We found significant associations over a decade later, suggesting a continued impact of anxiety on depression that extends from adolescence into adulthood. In addition, our findings build on existing studies on sleep as a transdiagnostic factor for anxiety and depression (Harvey et al., 2011), and further suggest that treatments targeting anxiety may benefit from incorporation of interventions for insomnia and sleep related problems, in order to ameliorate risk for the development of subsequent depression and future impairment.

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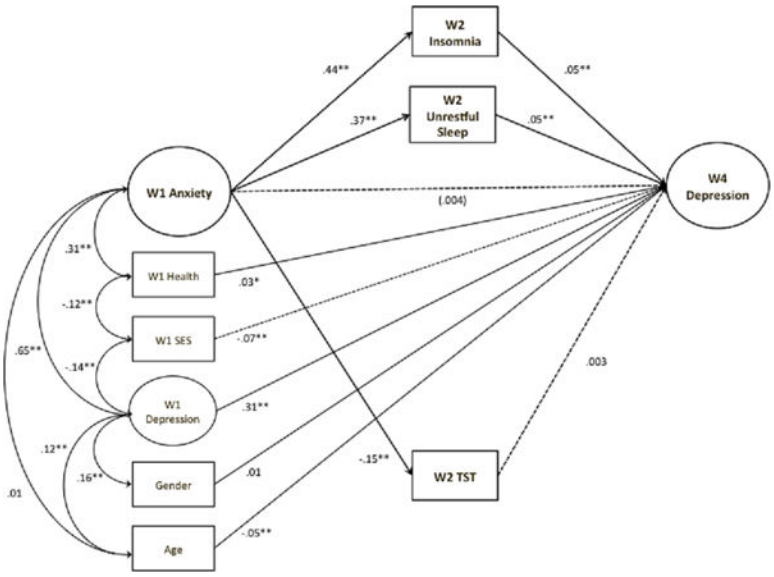

Figure 1.

Structural equation modeling examining insomnia, unrestful sleep, and TST as mediators of the relationship between anxiety and later depression. Unstandardized coefficients are presented. $\chi^2(350) = 8905.44$, $p < .001$, TLI = .90, RMSEA = .03, SRMR = .03.

*p < .01, **p < .001

Note. Manifest indicators and error terms are excluded from the model to enhance ease of interpretation.

Table 1

Demographics Information at Wave I

Female	50.5%
Male	49.5%
Ethnicity	
Caucasian	62%
African American	23%
American Indian	4%
Asian/Pacific Islander	8%
Other	9%
	M (SD)
Age	16.2 (1.7)
SES	.06 (.8)

Table 2

Relevant study variables

Measure	Time Frame	Item	Scale	Waves
CES-D	Past week	You were happy.	0 (*never*) - 3 (*most or all of the time*)	1, 2, 4
		You could not shake the blues.		1, 2, 4
		You enjoyed life.		1, 2, 4
		You felt depressed.		1, 2, 4
		You felt that you were just as good as other people.		1, 2, 4
		You felt sad.		1, 2, 4
		You had trouble keeping your mind on what you were doing.		1, 2, 4
		You felt that people disliked you.		1, 2, 4
Anxiety	Past 12 months	Feeling hot all over suddenly, for no reason	0 (*never*) - 4 (*every day*)	1, 2
		Cold sweats		1, 2
		Chest pains		1, 2
		Fearfulness		1, 2
		A stomach ache or an upset stomach		1, 2
		Trouble relaxing		1, 2
Sleep	Past 12 months	Trouble falling asleep or staying asleep	0 (*never*) - 4 (*every day*)	1, 2, 4[a]
		Waking up feeling tired		1, 2
		How many hours of sleep do you usually get?		1, 2
General Health	Past 12 months	In general, how is your health?	1 (*excellent*) – 5 (*poor*)	1

[a]Time frame for the item administered in Wave IV is *past four weeks*

Table 3

Pearson's product moment correlations, means, and standard deviations for relevant study variables.

	1	2	3	4	5	6	7	M	SD
1. W1 Depressive Symptoms								12.61	8.35
2. W1 Anxiety Symptoms	.47*							2.78	2.11
3. W2 Insomnia	.27*	.28*						0.97	0.96
4. W2 TST	−.11*	−.06*	−.14*					7.58	1.41
5. W2 Unrestful Sleep	.23*	.22*	.29*	−.12*				1.49	1.11
6. W4 Depressive Symptoms	.38*	.19*	.17*	−.05*	.16*			5.73	5.37
7. W4 Insomnia	.14*	.11*	.18*	−.05*	.10*	.35*		1.15	1.34

* $p < .01$