Published in final edited form as:

Cogn Emot. 2017 December ; 31(8): 1698–1706. doi:10.1080/02699931.2016.1247035.

Why might poor sleep quality lead to depression?: A role for emotion regulation

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Abstract

Disordered sleep is strongly linked to future depression, but the reasons for this link are not well understood. This study tested one possibility-- that poorer sleep impairs emotion regulation (ER), which over time leads to increased depressive symptoms. Our sample contained individuals with a wide range of depression symptoms (current depression, $N = 54$, remitted depression, $N = 36$, and healthy control, $N = 53$), who were followed clinically over six months and reassessed for changes in depressive symptom levels. As predicted, maladaptive ER mediated both cross-sectional and prospective relationships between poor sleep quality and depression symptoms. In contrast, an alternative mediator, physical activity levels, did not mediate the link between sleep quality and depression symptoms. Maladaptive ER may help explain why sleep difficulties contribute to depression symptoms; implications for interventions are discussed.

Keywords

major depression; emotion regulation; sleep quality; sleep; physical activity

Consequences of poor sleep have been highlighted recently in both public incidents (e.g. Exxon Valdez disaster) and in commentary concerning the substantial social and economic burdens of sleep loss (Kessler et al., 2011). In tandem, the worldwide burden and prevalence of depressive disorders has increased (Moussavi et al., 2007). Disordered sleep is strongly associated with depression (e.g., Goldstein & Walker, 2014), as well as key emotional difficulties that occur therein (e.g. emotion regulation, Baglioni et al., 2010). In fact, although not required for a diagnosis, sleep disturbances are among the symptoms used to diagnose a major depressive disorder (DSM-V; American Psychiatric Association, 2013). Relative to other inpatients, suicidal depressive patients have worse reports of subjective sleep quality, sleep latency, sleep duration, and habitual sleep efficiency (Agargun et al., 1997). A comprehensive review found strong relationships between sleep alterations and depression (Tsuno et al., 2005).

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The authors declare no conflicts of interest.

Recent work also indicates that disordered sleep often precedes depression. Longitudinal studies have found that sleep problems in childhood put children at 4 times the risk for internalizing problems 18 years later (Touchette et al., 2012). Similarly, follow-up studies reveal sleep problems are a primary risk factor for depression in those not currently depressed (Livingston et al., 1993). In multiple reviews, early sleep problems predict later depression – but not the other way around (e.g. early depressive symptoms do not significantly predict later sleep problems, Alvaro et al., 2013). In fact, insomnia symptoms for a period of more than two weeks predict an increase in developing depression within 1–3 years (Riemann & Voderholzer, 2003). Although the relationship between sleep and depression is complex and likely bi-directional (Kahn, Sheppes, & Sadeh, 2013), it is fair to say that sleep problems often precede depression.

One key unsolved question is *why* disordered sleep might precipitate depression. Conceptually, there are several mechanisms to explain why poorer sleep quality predicts increased depression symptoms, including sleep-induced physiological changes (rapid eye movement (REM)-related mechanisms, blunted pituitary ACTH responses; Novati et al., 2008), cognitive deterioration (Manber et al., 2008), or emotional deficits (e.g., emotional information processing; Kahn et al., 2013). Surprisingly, despite the suggestions that dysregulation of emotional reactivity underlies sleep-depression relationships (e.g. Baglioni et al., 2010), we are unaware of studies providing specific empirical tests of emotion-based pathways. Such tests are important for understanding the process by which depressive symptomology emerges, and for presenting potential points for clinical intervention. In this paper, we take a first step, proposing emotion regulation difficulties (Gross, 1998) as one potential pathway between sleep problems and depression symptoms. In the sections below, we define emotion regulation, and review work that has linked problematic emotion regulation to both disordered sleep and to depressive symptoms.

Emotion regulation (ER) refers to a variety of efforts to modify the experience and expression of emotions, including how emotions are monitored and evaluated (Gross, 1998). Researchers have increasingly been interested in examining maladaptive ER, often using “broadband” measures (e.g. difficulties in emotion regulation strategies overall) or scales that assess specific problematic emotion regulation strategies (e.g. worry or rumination).

Disordered Sleep and Emotion Regulation Ability

Disordered sleep has been shown to disturb many aspects of emotional experience (for a review, see Baglioni et al., 2010), including ER capacity (i.e., Mauss et al., 2013). Recent reviews highlight the unique impact of poor sleep on ER, particularly on regulating negative emotions (Palmer & Alfano, 2016). Disordered sleep has commonly been associated with altered neurological functioning (e.g. Yoo et al., 2007; decreased emotional expressiveness, Minkel, 2011) and difficulty in regulating behavior (e.g., poor impulse control, Peach & Gaultney, 2013); therefore, it logically follows that poor sleep would impair emotion regulation. Impaired sleep quality and shorter sleep duration are both associated with poorer emotion regulation reported by individuals and observers (Baum et al., 2014; Vriend et al., 2013). Most research has focused on the consequences of acute sleep loss (i.e. artificially shortened night sleep) on subsequent emotional functioning (Pilcher & Huffcut, 1996). For

instance, a shortened night sleep among young adolescents has been associated with worse ER; this has been found when sleep loss is measured by actigraphy, (objective measurements of sleep-related somatic activity; Vriend et al., 2013), experimental deprivation (Baum et al., 2014), or self-reports (Tavernier & Willoughby, 2014). People who report poor sleep for the past week exhibit worse emotion-regulation ability in the laboratory (Mauss et al., 2013).

Emotion Regulation and Depression

Likewise, there is evidence that maladaptive ER is associated with a diagnosis of depression. Depressed and depression-vulnerable groups report elevated use of maladaptive ER strategies (Gilbert & Gruber, 2014; Kovacs, Joormann, & Gotlib, 2008). Initial data indicate that ER deficits may be a risk and maintaining factor for MDD (Berking et al., 2014), since poorer self-reported ER predicts later increases in depressive symptoms.

Theoretically, within a process mediation model, it is plausible that problems in ER would predict worse functioning over time, including increases in depressive symptoms. Maladaptive ER has been shown to mediate processes related to individual adjustment, such as the relationship between sleep problems and detrimental social ties in university students (Tavernier & Willoughby, 2014) or between insomnia and negative mood (Xia & Zhou, 2010). Maladaptive ER has specifically predicted increased depressive symptoms at a five-year follow-up (Berking et al., 2014), and, conversely, endorsement of successful ER strategies predicts recovery from depression (Arditte & Joormann, 2011). Additionally, treatments that purport to increase ER skills are associated with decreased depression symptom severity – whereas the opposite is not true (decreased depressive symptoms do not always predict better ER skills; Radkovsky et al., 2014). Similarly, successful application of ER skills predicts lower depressive symptom severity, even when controlling for the effects of initial depression symptoms (Berking et al., 2014).

The Present Study

Although ER has strong ties to both sleep and depression, impaired ER has not been examined as a mediator of links between sleep problems and depression. Our primary goal was to examine whether problematic ER mediates the relationship between impaired sleep quality and elevations in depressive symptoms, modeled both concurrently and prospectively. In light of the evidence that disordered sleep impacts ER and that ER impacts depressive symptoms, our primary hypothesis was that that emotion regulation would mediate the relationship between impaired sleep and later depressive symptoms.

Our secondary hypothesis examined the specificity of ER as a mediator by considering physical activity in alternative mediating models. The logic for considering physical activity is that physical activity level has similar relationships to the other variables, as does ER. That is, poor sleep quality is likewise associated with diminished levels of physical activity (e.g. Calhoun et al., 2011). Moreover, low physical activity is likewise related to elevations in depression symptoms (e.g. McKercher et al., 2013; Uebelacker et al., 2013). Thus, it is conceivable that the relationship between poor sleep and depression can be (partially) understood through the lens of diminished physical energy and reduced daily activity level

rather than worse emotion regulation. To address the issue of specificity of ER, we included physical activity level in a series of alternative mediator models.

To ensure we had an adequate variability and range in sleep, depression, and emotional problems, our sample included groups of individuals diagnosed with current major depression, remitted depression, and healthy controls (e.g. Allen et al., 2014). To represent maladaptive emotion regulation, we used derived a factor score from commonly used ER scales (worry: Penn State Worry Questionnaire, Meyer et al., 1990; rumination: Response Style Questionnaire, Butler & Nolen-Hoeksema, 1994; Difficulties in Emotion Regulation Scale, Gratz & Roemer, 2004). We assessed sleep quality using the Pittsburgh Sleep Quality Index (PSQI; Buysse, 1989) and depression symptoms six months later with the Beck Depression Inventory II (BDI-II; Beck et al., 1996).

The current study addressed three questions: (1) Does maladaptive ER mediate the concurrent relationship between poor sleep quality and increased depression severity? (2) Does maladaptive ER mediate the prospective relationship between poor sleep quality and increased depression severity (six months later)? (3) Are these mediating relationships specific to maladaptive ER or would they also hold for an alternative mediator, physical activity level?

Method

Participants

We report how we determined our sample size, all data exclusions (if any), all manipulations, and all measures in the study. Specifically, the sample was collected originally for a study of autonomic nervous system functioning in depression and sample sizes reflected the requirements of the parent study (see Salomon et al., 2013 for details of recruitment and screening procedures); the current sample size, however, was also adequate to assess mediation effects using bootstrapping (Koopman et al., 2014). Community participants were recruited and visited the laboratory for clinical and psychophysiological assessments, as well as completing self-report scales. Our sample contained individuals with a wide range of depression symptoms, specifically 143 community-recruited participants who met criteria for MDD ($N = 54$), remitted MDD ($N = 36$), or healthy controls ($N = 53$) as assessed with the Structured Clinical Interview for DSM-IV (SCID; First et al., 2002). Subjects were excluded for reported diagnosed cardiovascular disease, use of medication with known cardiovascular side effects, history of a head injury, hearing impairment, bipolar disorder diagnosis, substance abuse, or history of primary psychotic symptoms. Other psychiatric comorbidity was permitted and as might be expected was relatively common in the sample. Notably, 74% of the depressed group had a history of anxiety disorder.

Six months after initial testing, data was collected on depression symptoms and emotion regulation in 95 participants. Attrition analyses revealed no differences between study completers and non-completers on key variables (including sleep quality, depressive symptoms, and emotion regulation scales; all p 's $> .4$). Although our primary mediation analyses collapsed the sample across diagnostic group, models were tested for moderated-

mediation (with depression status) and did not differ on mediation analyses. All procedures were approved by the relevant ethics committees and subjects consented.

Procedure

Participants completed measures in the lab at both Time 1 and Time 2. At Time 1, participants received a clinical interview, reported on sleep quality, emotion regulation, and depressive symptoms. At Time 2, six months later, participants were re-interviewed and were assessed for emotion regulation and depressive symptom severity.

Measures

Beck Depression Inventory (BDI-II).—The Beck Depression Inventory is a 21-item self-report measure that assesses depression severity. We used a 20-item version because of IRB (Institutional Review Board) concerns (the suicidality item was not included). The BDI-II has previously shown good psychometric properties (Beck et al., 1996), and Cronbach's alphas for this sample were adequate at both time points ($\alpha = .77$ at Time 1 and $\alpha = .95$ at Time 2).

Pittsburgh Sleep Quality Index (PSQI).—The PSQI is a 19-item self-report questionnaire, which assesses sleep quality over the last month. Items are rated on 0–3 scales (with 3 indicating worse functioning). Although the PSQI can be used to derive different sleep component scores (subjective sleep-quality, sleep latency, sleep duration, sleep efficiency, sleep disturbances, medicine to sleep, and daytime dysfunction), it is typical to combine these components into an overall sleep quality index (Buysse, 1989; $\alpha = .84$ within this sample).

Maladaptive Emotion Regulation (ER).—Three commonly used measures were used to represent the domain of maladaptive emotion regulation: the *Penn State Worry Questionnaire (PSWQ)*; designed to assess pathological worry, with questions concerning excessive worry, duration, and uncontrollability, Meyer et al., 1990), the *Response Style Questionnaire (RSQ)*, which measures overall tendencies to respond to low mood with rumination, Butler & Nolen-Hoeksema, 1994), and the *Difficulties in Emotion Regulation Scale (DERS)*; a broadband ER scale that covers the awareness, understanding, and acceptance of emotions, Gratz & Roemer, 2004). On each of these scales, higher scores indicate more maladaptive emotional functioning.

Not unexpectedly, emotion regulation scales were correlated $> .7$. As a data reduction strategy, we performed exploratory factor analysis (EFA) using an orthogonal rotation to potentially simplify hypothesis testing. In our EFA, we limited the number of factors to those with eigenvalues > 1 . Analyses supported a single factor solution, with all items loading on that factor; the single factor accounted for 41.56% of the variance, with the next potential factor accounting for only 6.80% of the variance. The alpha for the combined scale was $\alpha = .98$ at Time 1 and $\alpha = .97$ at Time 2. Therefore, we used the emotion regulation factor score, which we refer to below as maladaptive ER, in our hypothesis testing.

Paffenbarger Physical Activity Scale.—This is a widely used self-report scale devised to assess physical activity based on daily activities of life. Questions include type and duration of sport or recreational activities, which are converted to measures of caloric expenditure (Simpson, 2011). A total score was computed via summing caloric expenditure for all activities, using standard scoring.

Hypothesis Testing

To ascertain whether emotion regulation problems mediate the relationship between sleep quality and depressive symptoms, three overarching mediation models were utilized. We first modeled the variables concurrently (depression symptoms at Time 1 as the outcome) and second modeled the variables prospectively (emotion regulation as the mediator at Time 2 and depression symptoms at Time 2 as the outcome). The third modeled the variables prospectively using maladaptive ER at Time 2, controlling for maladaptive ER and depression at Time 1. Accounting for these variables at Time 1 provided a more stringent test of the prospective model. In all models, the dependent variable was depression and the predictor was sleep quality (PSQI; Buysse, 1989). To test specificity to maladaptive ER, we tested parallel mediation models using physical activity level in place of maladaptive ER.

The indirect effect for all models was tested using maximum likelihood bootstrapping with 10,000 bootstrap samples at the 95th percent confidence interval. Bootstrapping tests whether the mediator carries the influence of the predictor on the outcome – the indirect effect (Mallinckrodt et al., 2006). A particular advantage of the process, bootstrapping involves repeatedly and randomly resampling from the data, with a recommended 10,000 iterations minimum (Preacher & Hayes, 2004). PROCESS bootstrap methods were used for each mediation model; in bootstrapping, the convention for statistical significance is when lower and upper confidence intervals do not contain zero.

Results

Preliminary Analyses

A total of 143 participants' data for Time 1 was entered into the Hayes Process Model for Mediation in SPSS (36 remitted, 54 depressed, and 53 healthy controls). At Time 2, 95 participants' data was entered for bootstrapping; when missing data was accounted for, total sample size for Time 2 was 64. Participants showed adequate variability in depression severity and sleep quality at both Time 1 and Time 2. Participants reported on sleep quality ($M = 7.09$, $SD = 4.27$, $N = 117$) at Time 1 and ($M = 5.14$, $SD = 3.00$, $N = 64$) at Time 2, depression severity ($M = 14.23$, $SD = 14.75$, $N = 139$ at Time 1 and $M = 10.55$, $SD = 11.24$, $N = 95$ at Time 2), and maladaptive ER ($M = 177.91$, $SD = 56.57$, $N = 128$ at Time 1 and $M = 172.96$, $SD = 49.04$, $N = 73$ at Time 2). Additionally, participants reported on their total energy expenditure ($M = 975.48$, $SD = 1601.26$, $N = 120$) at Time 1. Given the potential for diagnostic group as a moderator, models were checked for moderated-mediation, but analyses for diagnostic group as a moderating variable were nonsignificant. Furthermore, diagnostic groups did not differ on demographic variables (age, sex, socioeconomic status; p 's $> .250$). Inclusion of demographic variables as covariate mediators did not change model

significance in any case; thus, we did not consider demographic variables further. There was no missing data on key variables.

Before testing mediation models, we first assessed whether the main study variables (sleep, depression, and maladaptive ER) were correlated as expected (see Table 1 for full correlational matrix). Specifically, sleep quality was correlated with maladaptive ER ($r = .56, p < .001$). In turn, maladaptive ER was correlated with depression severity ($r = .85, p < .001$). In the overall longitudinal model, sleep quality alone predicted depression severity ($B = 1.65, SE = .18, p < .001, R^2 = .48$).

Does ER mediate the concurrent relationship between poor sleep quality and depression?—

The first set of models were tested at Time 1 for all variables, with sleep quality as a predictor and depression symptom severity as the outcome and maladaptive ER as a potential mediator (see Table 2). The direct effect of sleep quality on depression was significant (Effect = .73, $SE = .18, p < .001, R^2 = .77$ for overall model). Additionally, the path between maladaptive ER and depression was also significant (Effect = .18, $SE = .01, p < .001$), as was the relationship between sleep quality and maladaptive ER (Effect = 7.39, $SE = 1.08, p < .001, R^2 = .32$).

The indirect effect, indicating the mediation of maladaptive ER, was significant and can be labeled a large effect (see Table 2). The maladaptive ER mediated the indirect effect, accounting for 47% of the relationship between poor sleep quality and depression at Time 1. Thus, as hypothesized, maladaptive ER partially mediated the link between poor sleep and depression symptoms at Time 1, and accounted for substantial variance therein.

Does ER mediate the prospective relationship between poor sleep quality and depression?—

Two prospective mediation models were tested. Both models tested sleep quality at Time 1 as a predictor, Time 2 maladaptive ER as the mediator, and depression symptom severity at Time 2 (6 months later) as the outcome. The second prospective model was identical, except it added control for Time 1 maladaptive ER and depression.

The direct effect of the prospective relationship between sleep quality and depressive symptoms was significant (Effect = .89, $SE = .24, p < .001, R^2 = .65$ for overall model). Additionally, the path between maladaptive ER at Time 2 and depression was also significant (Effect = .13, $SE = .02, p < .001$), as was the relationship between sleep quality and maladaptive ER at Time 2 (Effect = 6.72, $SE = .95, p < .001, R^2 = .42$).

Time 2 maladaptive ER was a mediator in first prospective model, significantly mediating the indirect effect between initial sleep quality and later depressive symptoms. Maladaptive ER accounted for 23% of the variance across time (kappa of .23), essentially replicating the pattern found in cross-sectional results, with maladaptive ER partially accounting for the indirect effect in the sleep quality-prospective depression relationship.

Results were similar (maladaptive ER continued to mediate the sleep-depression relationship) in our more prospective mediation model that controlled for Time 1 both maladaptive ER and depression (see Table 2).

Was maladaptive ER specific in mediating the relationships between poor sleep quality and depression?—To test for specificity, we replaced maladaptive ER with an alternative mediator, physical activity. As above, the first model tested was at Time 1 for all variables, with sleep quality as a predictor and depression symptom severity as the outcome. Physical activity did not significantly mediate the indirect effect of the relationship between sleep quality and depressive symptoms at Time 1 (confidence intervals included zero). Because mediation was not significant at Time 1, we did not examine mediation at Time 2.

Similarly, when we re-run our models including physical activity alongside maladaptive ER, maladaptive ER remained significant (results from above relatively unchanged) and physical activity did not mediate the indirect effect between initial sleep quality and depressive symptoms (confidence intervals included zero). Results are included in Table 2. In sum, the combined model found that maladaptive ER continued to significantly mediate the indirect effect, whereas the physical activity was nonsignificant.

Discussion

Understanding how poor sleep might contribute to depression is important given the high social and economic burdens of sleep loss (Kessler et al., 2011) and the alarming increases in depression prevalence (Moussavi et al., 2007). Surprisingly, we know little about the pathways by which poor sleep begets depression symptoms. This study was a first step in this direction, examining whether sleep problems might be associated with depression because of disturbance to emotion regulatory mechanisms (Gruber & Cassoff, 2014).

Consistent with our main hypothesis, problems in emotion regulation partially accounted for concurrent and prospective relationships between sleep quality and depressive symptoms. Maladaptive ER accounted for substantial variance in the sleep-depression relationship. Moreover, maladaptive ER exhibited specificity as a mediator. Another plausible candidate, physical activity levels, did not mediate the sleep-depression relationship in alternative models. Maladaptive ER remained a significant mediator when physical activity was included in the model. Overall, then, our major results were consistent with the idea that ER difficulties may be implicated in the process by which disordered sleep leads to depression. Although this study provides initial evidence with a longitudinal design, it should be acknowledged that the mediator and dependent variables were measured at the same time point, precluding definitive statements about the temporal relationship between sleep problems and emotion regulation.

These results invite speculation concerning the various intervening steps in the chain between sleep problems to depression. Future research should address possible causal steps in each part of this chain, such as, how do sleep difficulties translate into emotion regulation problems? Experimentally, sleep-deprived participants have shown reduced functional connectivity in systems associated with cognitive control of emotional response (Yoo et al., 2007; but see Minkel et al., 2012 for additional discussion). Furthermore, disordered sleep impairs executive function systems, which have direct consequences for the ability to regulate emotions (for a review, see Gruber & Cassoff, 2014).

These findings also invite a consideration of clinical implications. Given the fact of existing interventions to improve sleep and/or maladaptive ER, our results suggest the possibility that such interventions could also prevent the development of depression symptoms. Whether erosion of ER is a byproduct of sleep or occurs simultaneously, targeting ER may be a promising area for intervention as a means to interrupt the pathway by which both problems contribute to increased depressive symptomology. One critical question in this work will be try to isolate whether these effects are specific to depression versus anxiety (or other psychiatric comorbidity), which was not possible in the current project because of the high levels of comorbidity in our sample.

As an initial study in this area, our study also had some limitations. First, our design largely utilized self-report measures of the key constructs. Although the PSQI is the best accepted measure of poor sleep quality, future research should examine whether these relationships hold using other indicators of sleep (e.g. actigraphy, Vriend et al., 2013). Additionally, poor sleep quality generally signals disruption in circadian rhythm, we cannot speak directly to circadian rhythms because we did not measure them independent of our index of sleep quality. Similarly, given sometimes contextually specific nature of emotion regulatory problems (Aldao, 2013), it would be useful in future work to acquire laboratory proxies of maladaptive ER in addition to trait-level measures.

Second, as noted above, our design does not allow definitive statements about temporal precedence. Our preferred interpretation of the results is that sleep problems precede emotion-related problems. Although this interpretation is guided by the literature, there is also evidence for a bi-directional relationship (Goldstein & Walker, 2014). Moreover, our mediator and outcome variable occurred simultaneously, which, again, precludes definitive statements about the temporal relationship between emotion regulation and depression. However, studies employing both deprivation (e.g. Baum et al., 2014) and self-report of sleep (e.g. Mauss et al., 2013) have found that prior disordered sleep leads to emotion-regulation deficits and abundant data demonstrates that difficulties in emotion regulation are stable predictors of depressive episodes (e.g. Berking et al., 2014). Regardless, future research with a clearer test of temporal precedence is needed. Thus, while our prospective analyses were significant, even after controlling for demographic characteristics and Time 1 ER/depression, it is important not to overstate the strength of our prospective findings.

Relatedly, although a 6-month time frame is meaningful, longer follow up period would be useful. More generally, a key question for future research is whether these mediational relationships hold for longer versus shorter periods of time (and as well as different developmental periods). Past research has documented that the relationship between disordered sleep evolving into depression is robust at an 18-year follow-up (Touchette et al., 2012); this raises the possibility that ER scales may mediate this relationship over longer spans of time.

Work that examines *why* sleep problems yield depression has lagged considerably behind our epidemiological observations that it does. This study provides a first step towards this goal. We hope this work will be a springboard both to better the mechanisms of the sleep-depression relationship and to improve interventions to mitigate the harms of poor sleep.

ACKNOWLEDGEMENTS:

Jonathan Rottenberg was supported by the National Institutes of Health [MH077669–02].

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Table 1.

Pearson product-moment correlations between key variables.

Scale	PSQI	BDI1	ER1	ER2	PAS	PSQI2	BDI2
PSQI	1	.65**	.56**	.65**	-.20*	.56**	.67**
BDI1	.65**	1	.85**	.74**	-.25*	.42**	.79**
ER1	.56**	.85**	1	.80**	-.19*	.28*	.64**
ER2	.65**	.74**	.80**	1	-.16	.51**	.76**
PAS	-.20*	-.25*	-.19*	-.16	1	-.17	-.26*
PSQI2	.56**	.42**	.28*	.51**	-.17	1	.54**
BDI2	.67**	.79**	.64**	.76**	-.26*	.54**	1

* = < .05,

** = < .001

¹PSQI = Pittsburgh Sleep Quality Index; Time 1 BDI = Time 1 Depression Severity; ER1 = Maladaptive Emotion Regulation Time 1; ER2 = Maladaptive Emotion Regulation Time 2; PAS = Physical Activity Scale; Time 2 BDI = Time 2 Depression Severity; PSQI2 = Time 2 Pittsburgh Sleep Quality Index; PAS2 = Time 2 Physical Activity Scale

Table 2.

Bootstrapping mediation: indirect effects between sleep quality and depression severity.

	Time 1				Time 2				Time 2 (with controls)			
	<i>Effect</i>	<i>SE</i>	<i>CI (lower)</i>	<i>CI (upper)</i>	<i>Effect</i>	<i>SE</i>	<i>CI (lower)</i>	<i>CI (upper)</i>	<i>Effect</i>	<i>SE</i>	<i>CI (lower)</i>	<i>CI (upper)</i>
ER	1.30*	.21	.94	1.76	.87*	.24	.49	1.43	.28*	.16	.04	.67
PA	.04	.03	.005	.12								

* Indicates significance (in bootstrapping, the convention for statistical significance is when lower and upper confidence intervals do not contain zero)

ER = Emotion Regulation; PA = Physical Activity