Don't Sleep on It: Less Sleep Reduces Risk for Depressive Symptoms in Cognitively Vulnerable Undergraduates

Gerald J. Haeffel
University of Notre Dame

The current research tested a new theory of depression that integrates work on sleep and cognition. In general, good sleep is essential for physical and mental health. However, we theorize that sleep can actually increase risk for depressive symptoms in cognitively vulnerable individuals. This is because the negative cognitions generated by these individuals are strengthened and consolidated each night during sleep. Three studies were conducted to test this theory. Studies 1 (n = 134) and 2 (n = 47) used prospective designs and showed that undergraduates with high, but not low, levels of cognitive vulnerability were most likely to exhibit increases in depressive symptoms when sleeping well as operationalized by self-reported quality and objectively measured duration (via actigraphy). Study 3 (n = 40) used an experimental design and provides the first causal evidence that it may be possible to prevent future depressive symptoms in cognitively at-risk undergraduates by restricting their sleep during times of high perceived stress.

Keywords: cognitive vulnerability, depression, sleep

Scientists have made great strides during the last 40 years in identifying the factors that create risk and resilience to depression. Cognitive factors have featured prominently in this work. According to the cognitive theories of depression (e.g., Abramson et al., 1989; Beck, 1976; Nolen-Hoeksema & Morrow, 1991), some people have a *cognitive vulnerability* that puts them at heightened risk for developing future depression. Specifically, people are vulnerable to depression when faced with stressful life events[1] because they have a propensity to brood, infer negative self-characteristics, and infer negative future consequences (Abramson et al., 1989; Beck, 1976; Nolen-Hoeksema & Morrow, 1991).

A significant body of research supports the cognitive vulnerability hypothesis (see reviews by Haeffel et al., 2008; Nolen-Hoeksema et al., 2008). Longitudinal studies show that individual differences in how people interpret stressful life events (i.e., their level of cognitive vulnerability) determine risk for developing future depression. Some of the strongest empirical support for the cognitive theories comes from the Cognitive Vulnerability to Depression (CVD) Project (Abramson et al., 1999; Alloy, Abramson, Whitehouse, et al., 2006). This project used a behavioral high-risk design with a never depressed sample and found that participants with high levels of cognitive vulnerability were approximately 7 times more likely than those with low levels of cognitive vulnerability to develop major depressive disorder (Alloy, Abramson, Walshaw, & Neeren, 2006; Alloy, Abramson, Whitehouse, et al., 2006). This research, along with a growing number of short-term prospective studies (see Haeffel et al., 2008 for review), establishes temporal precedence and suggests that cognitive vulnerability may be a causal contributor to depression.

A strength of the cognitive model of depression is the ease with which it can be translated to prevention and treatment interventions. According to the cognitive theories, depression can be prevented and treated if cognitive vulnerability is decreased. Thus, a central goal of many depression interventions is to reduce an individual's cognitive vulnerability, a process referred to as "cognitive restructuring." People are taught to monitor their mood, identify the accompanying automatic negative thoughts, evaluate the veracity of the thoughts, and then generate more adaptive/realistic thoughts (Beck, 1976). This is typically accomplished through a number of strategies including psychoeducation, thought experiments, and homework (e.g., thought-record worksheets). Research shows that cognitive interventions (e.g., Cognitive Behavioral Therapy; CBT) are as effective as any other intervention available (including medication) for preventing and treating depression (Hollon et al., 2002). Thus, cognitive interventions are often considered a gold-standard intervention for both the prevention and treatment of depression (Hollon et al., 2014).

This article was published Online First September 22, 2016.

Thank you to Michael P. Eardley, Shawn Heiss, and Wei Lu for their help with data collection and their thoughtful comments about this research.

Correspondence concerning this article should be addressed to Gerald J. Haeffel, Department of Psychology, 118 Haggar Hall, University of Notre Dame, Notre Dame, IN 46656. E-mail: ghaeffel@nd.edu

[1] Cognitive vulnerability models of depression (like the hopelessness theory and ruminative response style theory) tend to be agnostic to the type of stress that is most important for conferring risk for depression. The stress can be acute, chronic, perceived, objectively occurring, self-generated, or independently generated. The critical theoretical consideration is the meaning the individual attaches to that the stress (e.g., implications for one's self-worth and future), whatever kind it may be.

Journal of Personality and Social Psychology, 2017, Vol. 113, No. 6, 925–938

http://dx.doi.org/10.1037/pspp0000119

However, a closer look at the literature reveals that cognitive interventions are not a panacea. In past studies, the effect sizes for cognitive interventions were only the small to moderate range (Lynch, Laws, & McKenna, 2010). In treatment studies, about half of patients respond to cognitive interventions with only one third of patients meeting criteria for full remission (Hollon et al., 2002). Similarly, in prevention studies, the positive outcomes tend to be small in size and fade over time (e.g., Garber, 2008). These results highlight the critical need to improve interventions for depression, as even this gold-standard intervention does not help most people.

We theorize (Haeffel, 2010; Haeffel, Rozek, Hames, & Technow, 2012) that cognitive interventions for depression are not effective for a majority of people because the skills taught by these interventions are too difficult to use when they are needed most (e.g., during stress). Patients are taught a deliberate and effortful process by which they must identify, evaluate, and ultimately inhibit a negative style of thinking to adopt a more adaptive style of thinking. This process of supplanting a highly engrained pattern of thinking with a new pattern of thinking (in the same domain) is difficult. This is because the existing knowledge can inhibit the learning (and subsequent use) of the new knowledge (e.g., McNeil & Alibali, 2005). After cognitive therapy, a cognitively vulnerable person will have competing cognitive reactions to a stressful event—their well-established negative interpretation and their newly learned adaptive interpretation. For the newly learned interpretation to "win" the competition, the person must have enough cognitive resources to inhibit their automatic negative interpretation (Shiffrin & Schneider, 1977). Thus, for cognitive therapy to be effective, the cognitively vulnerable individual must have enough cognitive resources to inhibit their entrenched negative cognitive style and use a more adaptive style. Unfortunately, cognitive resources are not readily available for vulnerable individuals. Research shows that individuals with high levels of cognitive vulnerability show reduced cognitive capacity, cognitive inflexibility, difficulty with task-switching, and deficits in concentration, attention, and memory (e.g., Lyubomirsky et al., 1999; Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008; Ray et al., 2005; Watkins & Brown, 2002). This work supports recent studies showing that cognitive restructuring might not work for cognitively vulnerable individuals (e.g., Daches, Mor, & Hertel, 2015; Haeffel, 2010) and may even have the potential to backfire (e.g., Baert et al., 2010; Haeffel et al., 2012).

We contend that cognitive interventions should place greater emphasis on mitigating the effects of cognitive vulnerability than on trying to change the vulnerability itself. Instead of teaching cognitively demanding skills aimed at changing a well-established cognitive style, we hypothesize that the risk for depression can by reduced by preventing the consolidation of negative cognitions generated by these individuals. Research (Haeffel, 2011) shows that cognitive interpretations about stress tend to change over time. People's initial cognitive reactions to life stress tends not to be a strong predictor of enduring depressive reactions (Metalsky et al., 1982, 1987; 1993). Rather, it is how these initial cognitive interpretations change over time that confers risks for future depression (Haeffel, 2011). For those with high levels of cognitive vulnerability, the initial cognitive interpretation of stress becomes more negative. However, for those with low levels of cognitive vulnerability the cognitive interpretations of stress become more adaptive (less negative) over time. It is these cognitions that are generated later in time (approximately three days later in the case of an acute stressor) that are most predictive of enduring depressive symptoms (Metalsky et al., 1982, 1987, 1993; Haeffel, 2011). The purpose of the current research was to test a novel strategy for preventing depression by stopping the cognitions of vulnerable individuals from becoming more negative over time. Specifically, we theorize that restricting sleep can prevent the consolidation and strengthening of negative cognitions for individuals with cognitive vulnerability, and thus, make them more resilient to depression.

Sleep is critically involved in cognitive processes, particularly memory (e.g., Rasch & Born, 2013; Wagner et al., 2001). Sleep prior to learning appears to be critical in forming new memory associations (e.g., Walker, 2009). And, once the new information is encoded, sleep is fundamental to the consolidation of that information in memory. Supporting the role of sleep in memory, imaging studies demonstrate that the same neural circuitry (e.g., amygdala, prefrontal cortex, hippocampus) that is activated during memory tasks is reactivated during subsequent sleep (e.g., Maquet, 2001; Peigneux et al., 2004). The consolidation of memory during sleep appears to be exceptionally strong for affective content. Indeed, recent studies suggest that sleep preferentially benefits the consolidation of emotional information (see Walker & van der Helm, 2009 for review). For example, seminal work by Kensinger et al., (2007) and Payne et al., (2008) in humans, has found that sleep strengthens memory for emotional objects in a scene relative to the neutral background. The consolidation of emotional memory seems most likely to occur during REM sleep (see Wagner et al., 2001). For example, Payne et al., (2012) found that memory for emotional objects was positively correlated with measures of REM sleep duration. Taken together, this work suggests that sleep may play an important role in human affective processing via the selective enhancement and consolidation of emotional information off-line.

Research shows that sleep is more likely to strengthen the memory of negative emotional information than positive or neutral information (Deliens, Gilson, & Peigneux, 2014; Walker & van der Helm, 2009). This means that the negative cognitions generated by cognitively vulnerable individuals in response to stress should be strengthened and consolidated each night during good sleep, placing these individuals at heightened risk for depression. However, when such individuals have restricted or disrupted sleep, the consolidation of their negative cognitions should be weakened. We theorize that good sleep is *necessary* for negative thinking patterns to create risk for depression (see Figure 1). Without good sleep, negative cognitions about a stressor weaken in memory, leading to fewer negative cognitions and depressive symptoms. This means that it should be possible to prevent depression in cognitively at-risk individuals by restricting their sleep during times of stress.

At first blush, the idea of less sleep being beneficial appears counter intuitive. Sleep is critical for healthy mental functioning (Irwin, 2015; Peterson & Benca, 2006; Vgontzas et al., 2004) and less sleep should not promote emotional well-being. Indeed, sleep disturbance is associated with almost every form of psychopathology (Benca et al., 1992). Given this strong connection, Harvey and colleagues (2011) argue that sleep disturbance should be considered a transdiagnostic cause of mental illness. According to Harvey and colleagues, sleep impacts psychological and biological systems that cut across current diagnostic categories. For example, sleep disturbance is associated with emotion dysregulation, a defining characteristic of most forms of psychopathology. Sleep

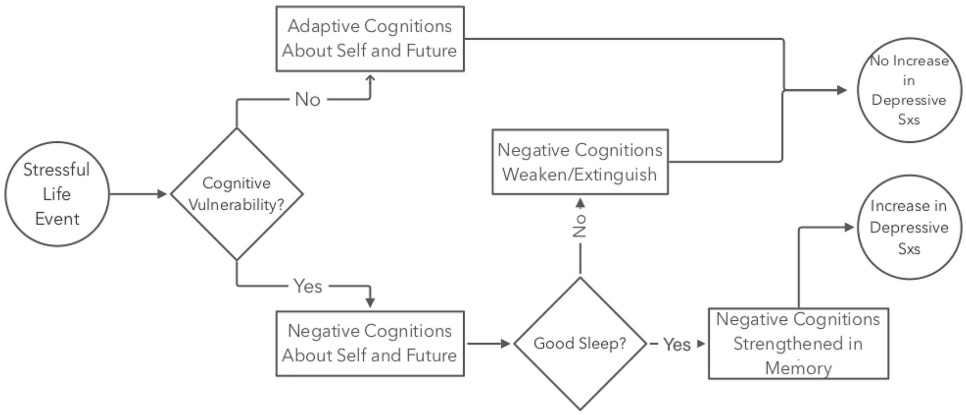

Figure 1. Flowchart to illustrate the cognitive vulnerability and sleep theory of depression. The negative cognitions generated by cognitively vulnerable individuals in response to stress are strengthened and consolidated each night during good sleep, placing these individuals at heightened risk for depression. However, when such individuals have poor sleep, the consolidation of their negative cognitions should be weakened, placing them at low risk for depression.

problems increase negative mood and decrease positive emotional responses (e.g., Dinges et al., 1997). Sleep and psychopathology also seem to share a common set of neurobiological constructs. Both sleep and mental illness are associated with amygdala activation as well as serotonergic and dopaminergic functioning (e.g., Harvey et al., 2011; Yoo, Gujar, Hu, Jolesz, & Walker, 2007; Yoo, Hu, Gujar, Jolesz, & Walker, 2007). In summary, there is a strong body of empirical evidence showing that sleep problems are associated emotional dysfunction and multiple forms of mental illness, including depression.

The link between sleep disruption and depression is undeniable. However, recent research in the area of anxiety and fear provides support for the plausibility of the current theory that less sleep can actually reduce risk for future depressive symptoms. Scientists such as Germain (2013), Poe (e.g., Poe et al., 2010) and others (e.g., Harvey et al., 2011; Kumar & Jha, 2012; Kuriyama et al., 2010; Pace-Schott, Germain, & Milad, 2013; Wagner et al., 2006) have found that sleep can produce dual outcomes. Sleep is associated with good outcomes such as the extinction of anxiety and fear responses. However, recent work shows that sleep also can promote the generalization of anxiety and fear responses. Thus, sleep has the potential to be both fear reducing and fear inducing. For example, Kuriyama and colleagues (2010) found that, in humans, sleep was necessary for generalized and physiological responses to negative stimuli 3 and 10 days after viewing them. Menz and colleagues (2013) replicated and extended this work using a Pavlovian fear condition task, and showed that fear consolidation was positively correlated with time spent in REM sleep. Along these same lines, Graves and colleagues (2003) showed that sleep deprivation 0 to 5 hours after training selectively impairs consolidation of fear conditioning in nonhuman animals (see also Kumar & Jha, 2012). In light of this work, some researchers hypothesize that sleep deprivation may be an adaptive biological-based coping mechanism for dealing with stress (e.g., Wagner et al., 2006). Sleep deprivation is associated with the release of corticotropin-releasing hormone, which inhibits the consolidation of new memories. Research shows that cortisol administration shortly after a traumatic event has a prophylactic effect for the occurrence of anxiety and mood symptoms (Schelling et al., 2004). These results support the idea that, that in some cases, less sleep can lead to better affective outcomes (in this case, less fear and anxiety). The present research will be the first to examine if the prophylactic effects of sleep restriction on fear and anxiety translate to the area of cognitive vulnerability and depression.

We theorize that less sleep can mitigate the depressogenic effects of cognitive vulnerability. There is currently no direct support for this new theory of depression, but there are a number of findings in the depression literature that corroborate its basic premise. For example, research shows that a majority of patients totally deprived of a full night's sleep experience a rapid reduction in depressive symptoms (although symptoms reemerge after sleeping again; Dallaspezia et al., 2015; Schulte, 1959). Sleep restriction has also been shown to trigger highly euphoric states such as mania (e.g., Plante & Winkelman, 2008; Wehr et al., 1987). Finally, sleep restriction has been shown to reduce REM sleep (Banks & Dinges, 2007; Carskadon & Dement, 1981), which is associated with emotional memories. Reduced REM is also considered a possible mechanism for the efficacy of antidepressant medications (Sandor & Shapiro, 1994). These findings, taken together with basic research on sleep and memory as well as work in the area of fear generalization, support the current theory. It is important to underscore that less sleep should only be beneficial for those with high levels of cognitive vulnerability because these are the individuals most likely to generate negative cognitions during times of stress. Less sleep after stress should lead to the weakening of the negative cognitions generated by vulnerable individuals whereas more sleep should selectively strengthen them and, in turn, increase depressive symptoms.

Three studies were conducted to test the theory. Study 1 used a prospective longitudinal design with two time points to provide a preliminary test of the theory. We predicted that that for individuals with high, but not low levels of cognitive vulnerability, poor quality sleep would lead to less depression than

good quality sleep. The purpose of Study 2 was to replicate and extend the findings of Study 1 by using an objective measure of sleep (actigraphy) and by assessing sleep's effect on event-specific negative cognitions. Finally, in Study 3, an experimental design was used to determine the causal influence of sleep on future depressive symptoms in those with high levels of cognitive vulnerability.

Study 1

Method

Overview. According to the theory, individuals with high levels of cognitive vulnerability are at greatest risk for future depressive symptoms during times of stress if they also experience good sleep quality. Put differently, individuals with high cognitive vulnerability should not be at greater risk for depressive symptoms than those with low levels of cognitive vulnerability under conditions of poor sleep. This is because the negative cognitions generated by cognitively vulnerable individuals should be disrupted by poor sleep, placing them at decreased risk for depressive symptoms. The hypothesis was tested using a 4-week prospective longitudinal design. Analyses tested the unique and interactive effects of sleep, cognitive vulnerability, and stress on risk for future depressive symptoms controlling for baseline levels of depressive symptoms.

Participants. Participants were 134 unselected undergraduates from a medium size private university in the midwest. The ethnicity of the sample was 76% Caucasian, 10% Hispanic, 9% Asian, 4% African American, and 1% Other. Participants were recruited online and given extra credit points for their participation. A total of 132 (65 women, 67 men) participants (mean age = 19.07, standard deviation = 1.36) completed both the T1 and T2 assessments and thus were included in the analyses.

Power analysis. Based on prior research, we expected a small effect size for the three-way interaction of cognitive vulnerability, stress, and sleep. Three-way interaction effects tend to be small, particularly when conducting conservative statistical tests that control for initial levels of the dependent variable (in this case, depressive symptoms). That said, McClelland and Judd (1993) argued that moderator effects even explaining 1% of the variance of the outcome should still be considered important (Hankin et al., 2004). The sample size of 132 was used for the statistical power analyses (Zhang & Yuan, 2015) and an 8 predictor variable equation was used as a baseline. The recommended effect sizes used for this assessment using multiple regression were as follows: small ($R^2 = .02$), medium ($R^2 = .13$), and large ($R^2 = .26$). The alpha level used for this analysis was $p < .05$. The analysis revealed the statistical power for this study was .89 for detecting a small effect. Thus, there was more than adequate power (i.e., power * .80) at the small effect size level.

Measures.

Cognitive vulnerability. The Cognitive Style Questionnaire (CSQ; Haeffel et al., 2008) was used to assess cognitive vulnerability (as featured in the hopelessness theory of depression). The CSQ is a self-report questionnaire that presents participants with 12 hypothetical negative events (6 achievement and 6 interpersonal). Participants imagine the events happening to themselves and then make ratings on the three vulnerability dimensions featured in the hopelessness theory of depression—stability and globality, probable consequences of each event, and the self-worth implications of each event. An individual's CSQ score is their average rating across these three dimensions (stability and globality, consequences, and self-worth characteristics) for the 12 hypothetical negative life events. This composite score can range from 1 to 7, with higher scores reflecting greater levels of cognitive vulnerability to depression. The CSQ has good internal consistency, reliability, and validity (see Haeffel et al., 2008 for review). Coefficient alpha for the CSQ in the current study was .90.

Depressive symptoms. The Beck Depression Inventory (BDI; Beck et al., 1979) was used to assess depressive symptoms. The BDI is a 21 item self-report questionnaire. Scores are created by summing the items (range 0–63) with higher scores indicating greater levels of depressive symptoms. The BDI has demonstrated strong reliability and validity (Beck et al., 1988). Coefficient alpha for the BDI was .84 at Time 1 and .93 at Time 2.

Sleep. The Pittsburgh Sleep Quality Index (PSQI; Buysse et al., 1989) is a widely used measure of sleep quality. The PSQI has 19 self-report items that assess sleep quality and disturbance during the "past month." The PSQI has demonstrated strong reliability and validity in both normal and clinical samples (Backhaus et al., 2002; Buysse et al., 1989). The subjective sleep quality score (component 1 on the PSQI; scores range from 1 to 4; 1 = *very good*, 2 = *fairly good*, 3 = *fairly bad*, 4 = *very bad*) and sleep duration score (component 3; self-reported hours slept each night) were used in this study.

Stressful life events. The Acute Life Events Questionnaire (ALEQ; Haeffel et al., 2007) was used to assess levels of life stress. The ALEQ is a self-report questionnaire that presents participants with 30 naturally occurring acute stressful life events. Items assess a broad range of life events (spanning both achievement and interpersonal domains) important to college students. A total score (0–30) is created is created by summing the items participants endorsed as having occurred during the previous 2 weeks. Thus, higher scores indicate the occurrence of more negative events. The ALEQ has demonstrated strong reliability and validity (Haeffel, 2010). Coefficient alpha for the ALEQ in the current study was .79 at Time 1 and .90 at Time 2.

Procedure. The study consisted of two time points over a four-week interval. The four-week time frame was chosen because it has been used in a number of previous longitudinal studies testing cognitive theories of depression (e.g., Haeffel et al., 2007; Metalsky et al., 1993; Potthoff et al., 1995). At Time 1, participants were administered self-report measures of cognitive vulnerability (CSQ), sleep (PSQI), depressive symptoms (BDI), and life stress (ALEQ). Four weeks later at Time 2 (T2) participants again completed measures of depressive symptoms and life stress. If participants did not answer at least 80% of the items on any of the measures, then a score was not calculated for that individual.

Results

Means, standard deviations, and intercorrelations of the study measures are summarized in Table 1. Consistent with prior research using undergraduates, CSQ and PSQI sleep quality scores were normally distributed whereas BDI and ALEQ scores were positively skewed. Average depressive symptom scores at baseline were in the minimal range (average BDI scores <13). We hypothesized that individuals with cognitive vulnerability would be at greater risk for increases in

Table 1
Study 1: Means, Standard Deviations, and Correlations

Variable	1	2	3	4	5	6	7
1. CSQ	—						
2. PSQI_QUALITY	.07	—					
3. PSQI_HOURS	−.10	**−.35**	—				
4. ALEQ T1	.05	**.21**	.05	—			
5. BDI T1	**.30**	**.35**	**−.28**	**.27**	—		
6. ALEQ T2	.14	**.17**	.05	**.57**	**.21**	—	
7. BDI T2	**.32**	.16	−.06	**.49**	**.63**	**.53**	—
Mean	3.92	2.04	6.52	2.24	5.86	1.02	3.67
SD	.93	.65	1.04	2.81	4.95	2.74	6.10

Note. $N = 132$. CSQ = Cognitive Style Questionnaire; PSQI_QUALITY = Pittsburgh Sleep Quality Index self-reported sleep quality; PSQI_HOURS = Pittsburgh Sleep Quality Index self-reported average hours actually slept; ALEQ T1 = Acute Life Events Questionnaire at Time 1; BDI T1 = Beck Depression Inventory at Time 1; ALEQ T2 = Acute Life Events Questionnaire at Time 2; BDI T2 = Beck Depression Inventory at Time 2. Higher scores on the CSQ, PSQI_HOURS, ALEQ, and BDI indicate greater levels of the construct being measured. In contrast, higher scores on the PSQI_QUALITY indicate lower levels of the construct being measured (i.e., worse sleep quality). Correlations in bold are significant at the .05 level.

depressive symptoms after life stress if they experienced good, rather than poor, sleep quality. Two hierarchical multiple regression equations were used to test these hypotheses (one equation with sleep operationalized as self-reported quality and one equation with sleep operationalized as self-reported number of hours). Continuous independent variables were centered and individual variables within a given set were not interpreted unless the set as a whole was significant, thereby reducing Type I errors (Cohen et al., 2003). In all analyses, the Time 1 depression measure (T1 BDI) was entered in the first step of the regression equation to create a residual change score for the same Time 2 measure (T2 BDI; dependent variable). Baseline level of stress (T1 ALEQ) was also entered in the first step to control for individual differences in baseline levels of stress. In the second step, the main effects of cognitive vulnerability (CSQ), sleep (PSQI Quality subscale or PSQI Hours subscale) and recent stressful life events (T2 ALEQ) were entered. Next, the two-way interaction terms were entered. And, finally, the three-way Vulnerability × Sleep × Stress interaction term was entered (e.g., CSQ × PSQI Quality × ALEQ).

Results of the first step of the regression analysis (see Table 2) found that there were significant main effects of cognitive vulnerability, stress, and sleep (both self-reported quality and self-reported hours). Participants with high levels of cognitive vulnerability, high levels of stress, and poor sleep, respectively, experienced the greatest levels of depressive symptoms over the prospective interval. There was also a significant two-way interaction between cognitive vulnerability and life stress. Replicating prior research, individuals with a cognitive vulnerability were most likely to experience increases in depressive symptoms when also experiencing high levels of life stress. These results were qualified by the hypothesized three-way interaction among cognitive vulnerability, life stress, and self-reported quality of sleep; the effect size was in the small range (partial correlation = −.22; change in $R^2 = .02$). The three-way interaction was not significant when sleep was operationalized as hours slept per night, $b = .16$, $t = 1.80$, $p = .07$, partial correlation = .16; change in $R^2 = .02$.

To graphically depict the cognitive vulnerability-by-stress-by-sleep quality interaction, Time 2 depressive symptoms scores were computed by inserting specific values for the predictor variables (i.e., 1 *SD* above and below the mean) into the regression equation (Cohen et al., 2003). The pattern of the three-way interaction corroborated the hypotheses. As can be seen in Figure 2, individuals with high levels of cognitive vulnerability were most likely to experience increases in depression when faced with stress if they also reported good sleep quality. Tests of simple slopes showed that, depending on level of stress, the gradient of the slope for those with high levels of cognitive vulnerability and poor sleep quality was significantly different than those with (a) high levels of cognitive vulnerability and good sleep quality ($t = -2.40$, $p = .02$), (b) low levels of cognitive vulnerability and poor sleep quality ($t = 4.18$, $p < .001$), and (c) low levels of cognitive vulnerability and good sleep quality ($t = 3.62$, $p < .001$). Although depressive symptom scores were in the minimal to mild range, it is important to note that cognitively vulnerable individuals reporting good sleep quality experienced twice the number of depressive symptoms as any other vulnerability-sleep combination. To further determine the pattern of the interaction, a Johnson-Neyman regions of significance test was conducted. Results showed that the point of transition on the perceived sleep quality scale (PSQI) between a statistically significant and nonsignificant effect of the interaction was 2.24 (84% below). This means that the effect of the cognitive vulnerability by stress interaction on increasing depressive symptoms was significant when participant rated their sleep quality as fairly good or very good.

Discussion

As hypothesized, results showed that cognitively vulnerable individuals were at increased risk for future depressive symptoms during times of stress if they also reported good sleep quality. Put another way, individuals with high levels of cognitive vulnerability were no longer at greater risk for depression than individuals with low levels of cognitive vulnerability during times of stress if they reported poor

Table 2
Study 1: Cognitive Vulnerability × Stress × Sleep Quality Interaction Predicting Prospective Levels of Depressive Symptoms

Predictor	*b*	*SE*	*pr*	*t*	R^2 Change
Step 1					.51*
BDI T1	.66	.08	.59	8.41*	
ALEQ T1	.73	.14	.42	5.32*	
Step 2					.10*
CSQ	.77	.39	.17	1.99*	
PSQI_Quality	−1.26	.58	−.19	4.59*	
ALEQ T2	.70	.15	.38	−2.18*	
Step 3					.05*
CSQ × PSQI_Quality	−.28	.48	−.05	−.59	
CSQ × ALEQ T2	.44	.15	.26	2.99*	
PSQI_Quality × ALEQ T2	−.16	.33	−.04	−.48	
Step 4					.02*
CSQ × PSQI × ALEQ T2	−.94	.39	−.22	−2.44*	
Model $R^2 = .68$, $F(9, 123) = 28.38$, $p < .001$					

Note. BDI = Beck Depression Inventory; ALEQ = Acute Life Events Questionnaire. CSQ = Cognitive Style Questionnaire; PSQI_Quality = Pittsburgh Sleep Quality Index self-reported sleep quality.
* $p < .05$.

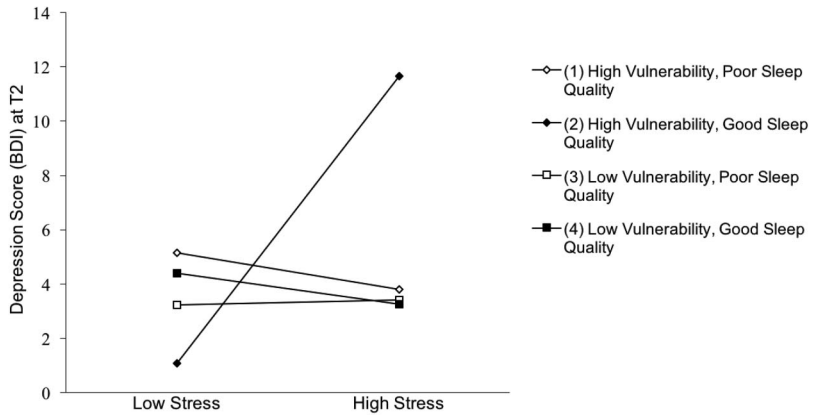

Figure 2. Study 1: Level of depressive symptoms at Time 2 as a function of cognitive vulnerability, self-reported sleep quality, and stress. Time 2 depressive symptoms scores were computed by inserting specific values for the predictor variables (i.e., 1 *SD* above and below the mean) into the regression equation. BDI = Beck Depression Inventory.

sleep quality. The findings were specific to self-reported quality of sleep rather than self-reported hours of sleep (p value = .07). These findings provide preliminary support for the theory that poor sleep can mitigate the depressogenic effects of cognitive vulnerability.

Study 2

Method

Overview. The purpose of Study 2 was to replicate and extend the results from Study 1 by using an objective measure of sleep. The self-report measure of sleep used in Study 1 (PSQI) required participants to retrospectively make a subjective judgment about their recent sleep, which can be influenced by memory biases (Nisbett & Wilson, 1977). It is also possible that participants' levels of cognitive vulnerability might have influenced their self-reported ratings of sleep quality. Thus, it is necessary to determine whether the positive findings of Study 1 hold when the possible confounding of sleep and vulnerability is eliminated. In Study 2, actigraphy was used to measure sleep. Actigraphs are devices, usually placed on the wrist, that measure body motion. The body motion is then translated into sleep data. Research shows that actigraphs are a highly reliable method of assessing sleep and wake patterns (Ancoli-Israel et al., 2003). Further, actigraphy allows the assessment of sleep in participants' natural environment, which likely allows them to most closely follow their normal sleep schedules.

Study 2 used a 2-week prospective design. Participants wore an actigraph for the first week of the study to create a baseline assessment of objective sleep. During the second week of the study, participants completed daily measures of depressive symptoms and event-specific cognitions. Sleep was assessed the week prior to event-specific cognitions and depressive symptoms (rather than concurrently) to ensure temporal precedence and to avoid disrupting participants' natural sleep habits. If participants were asked to concurrently make ratings about stress, event-specific cognitions and mood, it might have altered their natural sleep habits (e.g., increase awareness of stress, exacerbate ruminative tendencies, etc.). Analyses tested the unique and interactive effects of objectively measured sleep and cognitive vulnerability on future depressive symptoms and negative cognitions about daily stress controlling for baseline levels of depressive symptoms. We hypothesized that individuals with high levels of cognitive vulnerability would be at greatest risk for future depressive symptoms if they experienced longer (rather than shorter) durations of sleep.

Participants. Participants were 47 (19 males, 26 females) unselected undergraduates (M age = 20.87) from a medium size private university in the midwest. The ethnicity of the sample was 64% Caucasian, 13% Hispanic, 13% Asian, 9% African American, and 1% Other. Participants were recruited through an online sign-up procedure and were given extra credit points for their participation.

Power analysis. Based on prior research examining moderators of cognitive vulnerability (Doom et al., 2013; Haeffel & Hames, 2014), we expected a medium effect size for the two-way interaction of cognitive vulnerability and sleep. A larger effect size was expected in Study 2 than was expected in Study 1 because two-way interactions typically have larger effect sizes than three-way interactions. Further, a more objective measure of sleep was used in Study 2. A review by Uher and McGuffin (2010) found that objective measures of environmental factors tend to yield more consistent and robust relations with future depressive symptoms than do more subjective measures of the same factors. The sample size of 47 was used for the statistical power analyses (Zhang & Yuan, 2015) and a 4 predictor variable equation was used as a baseline. The recommended effect sizes used for this assessment of multiple regression were as follows: small (R^2 = .02), medium (R^2 = .13), and large (R^2 = .26). The alpha level used for this analysis was $p < .05$. The analysis revealed the statistical power for detecting a small effect was .52 whereas the power was .92 for detecting a medium effect. Thus, there was more than adequate power (i.e., power * .80) for the expected medium effect size level, but less than adequate for a small effect size.

Measures.

Cognitive vulnerability. The Cognitive Style Questionnaire (CSQ; Haeffel et al., 2008) was used to assess cognitive vulnera-

bility (as featured in the hopelessness theory of depression). Coefficient alpha for the CSQ at baseline in the current study was .93.

Event-specific negative cognitions. Daily event-specific cognitions were measured using the Particular Inference Questionnaire (PIQ; Haeffel, 2011; Metalsky, Halberstadt, & Abramson, 1987). The PIQ is a four-item questionnaire that assesses participants' inferences for daily life stress. The PIQ uses the same exact format as the CSQ to asses participants' inferences about the cause, consequences and self-worth implications of the most stressful event reported for that day. Participants are instructed to "Think about their most stressful event experienced that day" and then write down the one major cause of the stressor. Then, they make ratings on dimensions of stability and globality, the probable consequences of each event, and the self-worth implications of each event. An individual's PIQ score is their average rating across the four dimensions (stability, globality, consequences, and self-worth characteristics). Scores can range from 1 to 7, with higher scores reflecting a greater degree of event-specific negative inferences. The PIQ was administered via a daily diary during the 2nd week of the study. Daily PIQ scores were averaged to create a composite score for the prospective interval; coefficient $\alpha = .83$.

Depressive symptoms. The Beck Depression Inventory (BDI; Beck et al., 1979) was used to assess depressive symptoms. Coefficient alpha for the BDI at baseline in the current study was .84.

Prospective levels of depressive symptoms were assessed via daily diary. Participants rated their level of depressive symptoms each day during the 1-week prospective interval. Specifically, participants rated, on a 1–5 Likert scale, how much they experienced each of the nine depressive symptoms that compose depressive disorder as defined by DSM. Example items included "problems concentrating," "felt slowed down," and so forth. Participants' responses were summed each day, and then averaged for the 7-day prospective interval. Participants' scores could range from 9 to 45. Coefficient alpha for the 7-day aggregate depression score was .83.

Sleep. Actigraphy was used as an objective measure of sleep. The actigraph was a small wristband that participants put on before entering their bed. Actigraphs measure body motion, which is then translated by computer software into sleep data. Research shows that actigraphs are a highly reliable method of assessing sleep and wake patterns (Ancoli-Israel et al., 2003). Further, actigraphy allows the assessment of sleep in participants' natural environment, which likely allows them to most closely follow their normal sleep schedules. Thus, actigraphy is likely more valid than Polysomnography for assessing typical sleep. Sleep duration (minutes slept) was evaluated each night for one week. An aggregate score was then used create a reliable measure of a participant's average number of minutes slept per night; coefficient $\alpha = .73$.

Procedure. The study consisted of two 1-week phases. The baseline assessment occurred during the first week. After reading the informed consent form and agreeing to participate, participants were administered self-report measures of cognitive vulnerability (CSQ) and depressive symptoms (BDI). They were then fitted with an actigraph and given instructions to wear it nightly for the next seven nights. After returning the actigraph, participants completed a daily diary for the next seven days to assess prospective daily depressive symptoms and event-specific negative cognitions (PIQ). If participants did not answer at least 80% of the items on any of the measures, then a score was not calculated for that individual.

Results

Means, standard deviations, and intercorrelations of the study measures are summarized in Table 3. Consistent with prior research using undergraduates, the distribution of CSQ, PIQ, and objectively measured sleep scores were normally distributed whereas depressive symptoms scores were positively skewed. Depressive symptoms at both time points were in the minimal range (average BDI scores <13). We hypothesized that individuals with cognitive vulnerability would be at greater risk for increases in depressive symptoms and more negative event-specific inferences if they experienced longer, rather than shorter, sleep durations. Two hierarchical multiple regression equations were used to test these hypotheses (with depressive symptoms and event-specific negative cognitions as the dependent variables, respectively). Continuous independent variables were centered and individual variables within a given set were not interpreted unless the set as a whole was significant, thereby reducing Type I errors (Cohen et al., 2003). In all analyses, the dependent variable was Time 2 depressive symptom scores (T2 BDI). The Time 1 depressive symptom measure (T1 BDI) was entered in the first step of the regression equation to control for individual differences in initial levels of depression. In the second step, the main effects of cognitive vulnerability (CSQ) and sleep (average minutes slept per night as determined by actigraphy) were entered. Next, the two-way interaction between cognitive vulnerability and sleep was entered.

Results of the first regression equation (see Table 4) predicting future depressive symptoms found that (in the first step of the regression equation) there was a significant main effect of baseline depression, but not cognitive vulnerability or objective sleep. Consistent with hypotheses, the interaction of cognitive vulnerability and objectively measured sleep was a significant predictor of future depressive symptoms, when controlling for baseline depression (medium to large effect size; partial correlation = .41). To graphically depict the cognitive vulnerability-by-minutes slept in-

Table 3
Study 2: Means, Standard Deviations, and Correlations

Variable	1	2	3	4	5
1. CSQ	—				
2. BDI	**.39**	—			
3. SLEEP MINUTES	.09	−.05	—		
4. COGNITIONS T2	**.48**	**.49**	.10	—	
5. DEP SYMPTOMS T2	.28	**.61**	.14	**.52**	—
Mean	4.12	9.02	386.73	3.77	12.67
SD	.79	6.45	49.12	1.08	3.74

Note. $N = 45$. CSQ = Cognitive Style Questionnaire; BDI = Beck Depression Inventory; SLEEP MINUTES = Average minutes slept per night at baseline (assessed via actigraphy); COGNITIONS T2 = Average event-specific negative inference score during prospective interval; DEP SYMPTOMS T2 = Average depressive symptoms score during prospective interval. Higher scores on the CSQ, BDI, SLEEP MINUTES, COGNITIONS, and DEPRESSIVE SYMPTOMS indicate greater levels of the construct being measured. Correlations in bold are significant at the .05 level.

Table 4
Study 2: Cognitive Vulnerability × Objectively Measured Sleep Duration Interaction Predicting Prospective Level of Depressive Symptoms

Predictor	*b*	*SE*	*pr*	*t*	R^2 Change
Step 1					.36*
BDI at Baseline	.35	.08	.60	4.66*	
Step 2					.02
CSQ	.05	.69	.01	.08	
Sleep Minutes	.01	.01	.19	1.18	
Step 3					.10*
CSQ × Sleep Minutes	.04	.02	.41	2.67*	
Model $R^2 = .48$, $F(4, 36) = 8.44$, $p < .001$					

Note. BDI = Beck Depression Inventory; CSQ = Cognitive Style Questionnaire; Sleep Minutes = Average minutes slept per night at baseline assessed via actigraphy.
* $p < .05$.

teraction, depressive symptoms scores were computed by inserting specific values for the predictor variable (i.e., 1 *SD* above and below the mean) into the regression equation (Cohen et al., 2003). A simple slope analysis showed that the gradient of the simple slope for those with "low" and "high" durations of sleep was significantly different depending level of cognitive vulnerability, $t = 2.40$, $p = .02$. As predicted, participants with high levels of cognitive vulnerability and greater duration of sleep, experienced the greatest levels of depressive symptoms over the prospective interval (see Figure 3). Depression scores for these participants were in the mild range (BDI scores 14–19). To further define the pattern of the interaction a Johnson-Neyman regions of significance test was conducted. Results showed that there were two points of transition for number of minutes sleeping (438 min and 328 min). Participants with higher levels of cognitive vulnerability reported greater levels of depressive symptoms than those with lower levels of cognitive vulnerability when sleeping more than about 7 [1/2] hours. Those with higher levels of cognitive vulnerability reported lower levels of depressive symptoms than those with lower levels of cognitive vulnerability when sleeping less than about 5 [1/2] hours.

Results of the second regression equation predicting future event-specific negative cognitions found that there was a significant main effect of baseline depression ($p = .02$) and cognitive vulnerability ($p = .04$), but not objective sleep ($p = .53$). The interaction of cognitive vulnerability and objectively measured sleep did not predict future event-specific cognitions, when controlling for baseline depression, $b = .01$, $t = 1.85$, $p = .07$, partial correlation = .30; change in $R^2 = .06$. Although not statistically significant, the pattern of results conformed to hypotheses. Participants with high levels of cognitive vulnerability and longer average sleep durations reported the most negative event-specific inferences during the prospective interval.

Discussion

The purpose of Study 2 was to replicate and extend the results from Study 1 by using an objective measure of sleep. Results corroborated those of Study 1 showing that individuals with high, but not low, levels of cognitive vulnerability were most likely to exhibit increases in depressive symptoms when they slept more minutes rather than less minutes. These results provide further support for the hypothesis that good sleep is necessary for cognitive vulnerability to exert its depressogenic effects. When sleeping less, individuals with high cognitive vulnerability were not more likely to experience depressive symptoms than those with low levels of cognitive vulnerability. We hypothesize that less sleep prevents the strengthening of negative cognitions about stress. However, this hypothesis was not supported. The interaction of cognitive vulnerability and objectively measured sleep was not a significant predictor of negative cognitions ($p = .07$). That said, the pattern of the interaction conformed to hypotheses; those with high levels of cognitive vulnerability and shorter sleep durations reported the lowest level of negative event-specific inferences during the prospective interval.

Study 3

Method

Overview. The results of both Study 1 and Study 2 corroborated the theory that sleep plays a critical role in whether or not cognitive vulnerability creates risk for depression. Those with high levels of cognitive vulnerability were at no greater risk for increases in depressive symptoms than those with low vulnerability when sleeping less. However, a limitation of these studies is that they used correlational designs. The two studies establish temporal precedence for the interaction of cognitive vulnerability and sleep predicting depression, but they do not establish causal evidence. The purpose of Study 3 was to use an experimental design to test the causal influence of sleep on future depression in those with high levels of cognitive vulnerability.

Participants. Participants were 40 undergraduates (32 women, 8 men; mean age = 19.1, standard deviation = .93) from a medium size private university who were chosen because they reported high levels of cognitive vulnerability (i.e., scored greater than the 70th percentile on the CSQ). The ethnicity of the sample was 68% Caucasian, 12% Hispanic, 12% Asian, and 8% African American. Participants were recruited through an online sign-up procedure and were given extra credit points for their participation.

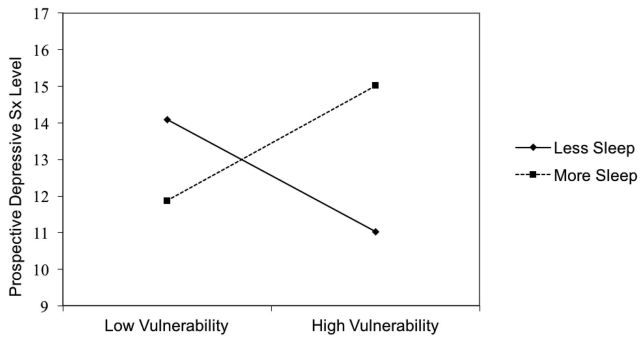

Figure 3. Study 2: Level of depressive symptoms during prospective interval as a function of cognitive vulnerability and objectively measured sleep. Depressive symptoms scores were computed by inserting specific values for the predictor variable (i.e., 1 *SD* above and below the mean) into the regression equation.

Power analysis. Based on prior research examining the effect of sleep restriction on psychological and cognitive outcomes (e.g., Payne et al., 2008; van Heugten–van der Kloet, Giesbrecht, & Merckelbach, 2015), we expected a large effect size for the main effect of sleep condition. Also, if sleep restriction is to have promise as potential intervention for preventing depression, a large effect size would be needed for the intervention to be clinically relevant. The sample size of 40 was used for the statistical power analyses (Zhang & Yuan, 2015) for a one-way ANOVA with two groups. The recommended effect sizes used for this assessment of multiple regression were as follows: small ($\eta_p^2 = .01$), medium ($\eta_p^2 = .06$), and large ($\eta_p^2 = .14$). The alpha level used for this analysis was $p < .05$. The analysis revealed the statistical power for detecting a small to medium effect was .46 whereas the power exceeded .99 for detecting a large effect. Thus, there was more than adequate power (i.e., power * .80) for the expected large effect size level, but less than adequate for a small to medium effect size.

Measures.

Cognitive vulnerability. The Cognitive Style Questionnaire (CSQ; Haeffel et al., 2008) was used to screen participants for high levels of cognitive vulnerability. Coefficient alpha for the CSQ in the current study was .82.

Depressive symptoms. The Beck Depression Inventory (BDI; Beck et al., 1979) was used to assess depressive symptoms. The BDI has demonstrated strong reliability and validity (Beck et al., 1988). Coefficient alpha for the BDI in the current study was .84 at baseline and .82 postintervention.

Procedure. The study had two phases - screening and intervention. First, the CSQ was used to screen 574 extracredit volunteer participants for high levels of cognitive vulnerability; 172 participants scored above the 70th percentile on the CSQ. These cognitively vulnerable participants were contacted via email (in order of most vulnerable to least vulnerable) and invited to participate in the intervention portion of the study. Participants with high levels of cognitive vulnerability were used because according to the theory (and prior research) these individuals are at especially heightened risk for increases in future depressive symptoms, and most likely to benefit from a sleep restriction intervention.

Participants who agreed to participate in the intervention portion of the study completed the BDI and then were randomly assigned to one of two intervention conditions: sleep restriction ($n = 20$) or active control ($n = 20$). Both intervention conditions lasted two weeks, and all participants were given a 14-day instructional booklet. Each day, participants were instructed to record their sleep times from the prior night and rate their current level of stress on a 0 (*no stress*) to 10 (*high stress*) scale. If daily stress was rated a 7 or higher, participants were then instructed to perform one of two intervention tasks. Participants randomly assigned to the sleep restriction condition were instructed to restrict their sleep by one hour (i.e., go to bed one hour later and get up at the same time) on days of high stress. The 1-hr sleep restriction time frame was used because we wanted a time frame that would have a low probability of chronically sleep depriving our participants. Moreover, the results of Study 2 indicated that 1-hr time frame fell within the region of significance for the desired effect. Participants randomly assigned to the active control condition were instructed to restrict eating their favorite snack that night. Snack restriction was chosen as the active control condition because it required participants to perform a restriction activity during times of stress (like the sleep-restriction intervention), but would also not affect future depressive symptoms. Participants followed the instructions for two weeks. Participants then returned to the laboratory to complete the BDI again. Randomization appears to have been successful as participants in the sleep restriction and active control conditions did not differ significantly on any of the baseline variables (CSQ, $p = .88$; BDI, $p = .63$). There was also no difference between conditions ($p = .62$) in the number of days in which participants rated their daily stress as a seven or greater Sleep restriction $M = 3.6$ days, $SD = 2.72$; Snack restriction $M = 3.2$ days, $SD = 2.28$). Examination of participants' self-reported sleep duration indicated that the manipulation was successful. Participants in the sleep restriction condition reported an average of 6 hours of sleep during restriction nights (i.e., when stress was rated as a seven or greater) whereas they reported an average of 7 [1/2] hours sleep during nights in which restriction was not required. Participants in the active control condition reported an average of 7 hours 20 min of sleep when stress was rated a seven or greater and an average of 7 [1/2] hours sleep during times of low stress. If participants did not answer at least 80% of the items on any of the measures, then a score was not calculated for that individual.

Results

Means, standard deviations, and intercorrelations of study measures are summarized in Table 5. The average BDI score at baseline (6.3) is considered in the minimal range. Analyses were designed to test the hypothesis that restricting sleep during times of stress would reduce risk for future depression in those with high levels of cognitive vulnerability. Specifically, it was predicted that participants in the sleep restriction condition would exhibit fewer depressive symptoms postintervention than those in the active control condition (i.e., snack restriction). The hypothesis was tested using analysis of covariance (ANCOVA) with condition (sleep restriction vs. active control) as the independent variable and BDI score postintervention as the dependent variable. BDI score at baseline was included as a covariate to control for any individual differences in initial levels of depressive symptoms. As predicted (see Figure 4), results showed a significant main effect of condition, $F(1, 35) = 4.73$, $p = .037$, $\eta_p^2 = .12$. Participants in the sleep restriction ($M = 6.30$; $SE = .64$) reported significantly fewer depressive symptoms postintervention than participants in the active control condition ($M = 8.28$; $SE = .64$) controlling for baseline levels of depressive symptoms. Level of depression symptoms for partici-

Table 5
Study 3: Means, Standard Deviations, and Range of Baseline Measures as a Function of Condition

	Restriction			Control		
Measure	*M*	*SD*	Range	*M*	*SD*	Range
1. CSQ	4.57	.38	4.72–5.21	4.59	.32	4.77–5.25
2. BDI	6.05	4.96	0–17	5.95	4.14	1–16
3. STRESSORS	3.20	2.28	0–9	3.60	2.22	0–10

Note. $N = 40$. CSQ = Cognitive Style Questionnaire; BDI = Beck Depression Inventory; STRESSORS = Number of days on which stress was rated at least a 7 on scale of 10 (i.e., perceived high stress). There were no significant differences between conditions on levels of any of the measures. Higher scores on the CSQ, BDI, and STRESSORS indicate greater levels of the construct being measured.

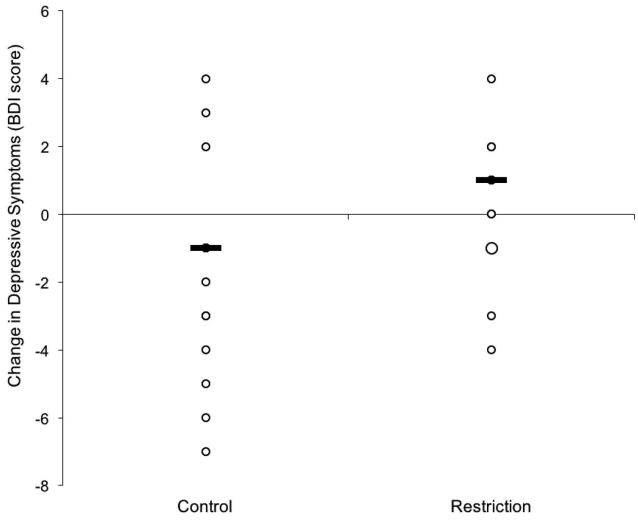

Figure 4. Study 3: Change in depressive symptoms pre to post as a function of condition. Positive symptom change scores indicate a decrease in depressive symptoms whereas negative scores indicate an increase. BDI = Beck Depression Inventory.

pants in both conditions were in the minimal range. The difference in BDI scores between groups is considered in the large range.

Discussion

Study 3 extended the results of Studies 1 and 2 by providing causal evidence for the role of sleep in conferring risk for future depression in those with high levels of cognitive vulnerability. Results showed that restricting sleep during times of high perceived stress had a large effect on reducing risk for future depressive symptoms compared with an active control condition in which sleep was not manipulated.

General Discussion

The purpose of the current research was to test a novel theory of depression that integrates work on cognitive vulnerability and sleep. The theory is both specific and falsifiable. Specifically, we theorize that good sleep is *necessary* for cognitive vulnerability to create risk for depression. Without good sleep, negative cognitions and emotions about a stressor weaken in memory, leading to fewer depressive symptoms. The results of the current research provide preliminary support for the theory. Studies 1 and 2 showed that sleep moderates the depressogenic effect of cognitive vulnerability. For individuals with high, but not low, levels of cognitive vulnerability, poor sleep reduced their risk for future depressive symptoms during times of stress. This finding held for self-reported sleep quality and objectively obtained sleep duration.

Study 3 provided the first causal evidence that sleep mitigates the depressogenic effects of cognitive vulnerability. Cognitively vulnerable participants randomly assigned to a sleep restriction condition reported significantly fewer depressive symptoms postintervention than participants randomly assigned to an active control condition. These results provide proof of principle that it may be possible to prevent future depressive symptoms in cognitively at-risk individuals by restricting their sleep during times of stress. Using this strategy, the sleep-restriction intervention would resemble a "take as needed" medication.

The current findings also have relevance for understanding prior work examining the effects of total sleep deprivation (typically 36 hours without sleep) on depression (Schulte, 1959). This work shows that a majority of patients deprived of sleep (typically 36 hours without sleep) experience a rapid reduction in depressive symptoms. However, total sleep deprivation can also trigger seizures in some individuals and the effects are short-lived. Upwards of 95% of responders relapse once normal sleep resumes (Dallaspezia et al., 2015). Moreover, despite the sleep deprivation phenomenon being documented since the late 1950s, the mechanism of action is still not understood. Researchers hypothesize that it could be attributable to effects on neurotransmitter systems or changes in sleep/wake biological clock. In contrast, we theorize that the positive effects of total sleep deprivation may be due to the disruption of memory processes associated with negative content. The current theory can also explain why responders relapse when normal sleep returns; this is because the negative emotional memories and cognitions would start being strengthened and consolidated once again (Wagner et al., 2006).

Along these same lines, the current results can also help explain the seemingly contradictory finding that sleep not only causes fear extinction, but also fear generalization (Pace-Schott, Germain, & Milad, 2015). According to the current theory and findings, "cognitive content" determines whether sleep will be harmful or beneficial. If an individual is prone to negative thinking patterns or has an attentional bias toward fearful stimuli fearful, then sleep will likely strengthen and generalize newly learned fearful associations. In contrast, if an individual has positive thinking patterns and an attentional bias away from fearful stimuli, then sleep will likely facilitate extinction of a newly learned fearful association. In other words, individual differences in sleep-memory outcomes are due to content differences rather than process differences.

We suggest two important directions for future research to further advance our understanding of sleep's influence on cognitive vulnerability and, in turn, risk for depression. One priority is to determine the mechanism by which sleep reduces risk for depression in cognitively vulnerable individuals. According to the theory, sleep restriction works because it prevents the strengthening and consolidation of negative event-specific cognitions. However, the results of Study 2 did not support this theorized mechanism of action. The results of this study showed that poor sleep did not significantly reduce levels of event-specific negative cognitions in those with high levels of vulnerability. Thus, it is important to consider alternative explanations for why sleep restriction had a prophylactic effect on depressive symptoms. One possibility is that sleep restriction caused decreases in REM sleep (Banks & Dinges, 2007). Research shows that individuals with depression and even those with subclinical levels of depression have greater amounts of REM sleep than nondepressed individuals (see review by Tsuno, Besset, & Ritchie, 2005). And, reductions in REM sleep is associated with improvements in depression. Almost all antidepressant medications suppress REM sleep, which some researchers consider to be a critical therapeutic feature of these drugs (Sandor & Shapiro, 1994). Thus, sleep restriction might have had an

antidepressant-like effect because it reduced duration of REM sleep (Gillin et al., 2001). A second explanation for the efficacy of sleep restriction is that it might have reduced problematic sleep patterns. Sleep restriction is a component of cognitive–behavioral therapy for insomnia (CBT-I). CBT-I reduces time in bed based on an individual's sleep efficiency. Sleep restriction, as used in CBT-I, leads to improvements in a variety of sleep parameters including sleep efficiency and sleep latency (Spielman, Saskin, & Thorpy, 1987). Thus, it is possible that sleep restriction worked in our study because it improved sleep for participants. A third explanation for the efficacy of sleep restriction is that it caused participants to be more tired, which shortened sleep onset latency, and, in turn, left less time for participants to ruminate about stress. Fourth, it is possible that participants used the extra hour afforded by sleep restriction to socialize or do an enjoyable activity, which might increase mood. It is also possible that participants used the extra hour to study more for their classes, which could have reduced academic stress. A fifth alternative explanation is that sleep restriction reduced the consolidation of emotional memories, and thus, reduced the negative affect that accompanies these memories (Wagner et al., 2006). Finally, a recent study by Fischer, Diekelmann, & Born (2011) found that sleep, particularly REM sleep, made it difficult for people to suppress unwanted memories. Thus, it is possible that sleep restriction enhanced participants' ability to suppress negative memories associated with stress, which decreased depressive symptoms. All of these alternative explanations are speculation, but may be important avenues to pursue in future research to identify the mechanism by which sleep restriction mitigates the depressogenic effects of cognitive vulnerability.

A second priority is to determine the optimal timing, frequency, and duration of sleep restriction. In Study 3, we chose to restrict initial sleep (by delaying bedtime) because prior work on memory and sleep in humans and animals has typically found their effects when restricting sleep immediately after learning. However, it is unknown whether sleep restriction during other times of the night might be useful. Diekelmann, Wilhelm, and Born (2009) concluded that the consolidating effects of sleep depend on the type of memory information and the specific phase of sleep. REM sleep appears to play a large role in consolidating emotional memories whereas slow wave sleep is most likely to influence declarative memories. In light of this work, it might be possible to create a highly targeted sleep restriction intervention that only needs to disrupt sleep for a very short period of time during a specific phase of sleep during the night.

Similarly, it will be necessary to determine how often participants should engage in sleep-restriction. According to the theory, those at cognitive risk for depression should only have to restrict their sleep during times of high stress (because this is when the generation of negative cognitions is most likely to occur). In the current study, participants restricted their sleep an average of 3 days over a 2-week time interval. However, the possibility of chronic stress is a concern. Research suggests that continued sleep restriction would not be beneficial because chronic sleep deprivation is linked to severe cognitive and emotional deficits (Brown, 2012; Killgore, 2010). It will be important to understand how frequently sleep restriction can be used before becoming iatrogenic.

It will also be critical to determine the proper duration of sleep restriction. Participants were instructed to restrict their sleep for only one hour because did not want to create a situation in which participants are chronically sleep deprived. However, it is possible that longer durations of sleep restriction may produce even stronger results. Studies that manipulate duration of sleep restriction will be important for identifying the tipping point at which prophylactic effects start to dissipate. It will also be important to understand how stress naturally affects sleep duration. The current research used measures of sleep (PSQI ratings and objectively measured sleep) that captured how people typically sleep, which includes times of life stress. In essence, participants provide a "sleep sample," which was thought to reveal their general sleep pattern. The next step in testing the current theory is to start conducting more finely grained analyses of the interplay between stress and sleep (e.g., use momentary assessment or a conduct a sleep study in a laboratory). This will help identify individual differences in reactions to life stress, mechanisms of action, as well as the time frame for how long these variables impact one another. For example, individual differences in how people sleep immediately following stress might predict fluctuations in depressive symptoms. Similarly, repeated stress might change natural sleeping habits over time, which might increase risk for depression depending on one's cognitive vulnerability level.

The current research had both strengths and limitations. A significant strength of the present work is the use of three independent studies to test the theory. The studies used different designs as well as different sleep measures. The first two studies used prospective longitudinal designs to establish temporal precedence for the interaction of sleep and cognitive vulnerability predicting future depressive symptoms. Moreover, the theory was corroborated using both a self-report and objective measure of sleep. Study 3 provided the most rigorous test of our theory by using an experimental design. By using an experiment it was possible to make causal conclusions about the role of sleep in reducing depressive symptoms in cognitively high-risk individuals. According to Lykken (1968), this type of constructive replication (testing the original hypothesis with different methodology rather than the same procedures) places a theory at high risk of refutation. That said, direct replication is also necessary, and look forward to learning the results of both direct and constructive replication tests of the theory. An additional strength of the experiment was the use of an active control condition. This allows us to rule out the possibility that sleep restriction worked because people simply were instructed to behave differently than normally.

The current study also had limitations. First, it would be unwarranted to make conclusions about clinically significant depression because the current studies only assessed depressive symptoms, which tended to be low in severity. Also, the extent to which the theory and findings would apply to individuals with clinically significant levels of depressive symptoms is unknown. Cognitive theories of depression such as the hopelessness theory (Abramson, Metalsky, & Alloy, 1989; Nolen-Hoeksema & Morrow, 1991) and response styles theory are models of etiology. They help explain individual differences in depressive reactions to life stress. The current theory is based on these models, and thus the theory might apply best to the prevention rather than treatment of depression. It is unclear whether the theory will hold given the changes in sleep and stress reactivity that those with full blown depressive disorders experience. It is also important to recognize that depression is a heterogeneous disorder with multiple causes; the theory and sleep restriction intervention may only apply to possible cognitive subtypes of depression. Second, the

studies examined undergraduates from a private university. Undergraduates are appropriate for testing prevention interventions because they are at the peak age for developing depression (Hankin et al., 1998) and are likely to experience sufficient levels of stress. However, future work is needed to determine whether the results hold in community or clinical samples. Indeed, the sleep patterns of college students tend to be different than general community samples (college students sleep less on average; Lund et al., 2010). However, even among community samples, 1 in 3 adults report getting less than 6 hours of sleep per night. A 2005 Gallup poll in the United States found that the average self-reported sleep duration for a general adult sample was 6.8 hours on weekdays. We suspect that the findings will hold because the results of studies using college samples often do generalize to community samples, particularly when basic processes (e.g., cognition, memory) are being studied (e.g., Anderson et al., 1999). However, future studies are needed in this area. Third, these three studies are the first to test the newly proposed theory of depression. Although the studies provide "proof of principle," replication is needed before definitive conclusions can be made about the efficacy of sleep restriction in preventing depression. Given the specific hypotheses, results of recent research on sleep and memory in the area of anxiety, and the consistency in the direction and size of the results, the most parsimonious explanation is that the sleep intervention worked as hypothesized. Finally, future tests of the theory should include a no-instruction control condition. It is possible that differences between the sleep-restriction and active control group conditions were attributable to an iatrogenic effect of the snack-restriction condition rather than a benefit of the restriction condition. For example, participants might have a favorite snack that is soothing during times of stress. However, it is important to note that it was still possible for participants to eat their second favorite snack, which could also be used to alleviate stress. Also, prior studies using a high vulnerable sample have shown similar increases in depressive symptoms over similarly short-longitudinal time frames (Doom & Haeffel, 2013; Haeffel et al., 2007).

In conclusion, there is now converging evidence from both the area of depression (the current set of studies) and the area of anxiety/fear that less sleep has the potential to be beneficial. The results from three independent studies that show that reductions in sleep significantly increased resiliency to future depressive symptoms in individuals with cognitive vulnerability. These findings support the new theory, and indicate that minimally invasive, easy, and cost-effective sleep based interventions are a potentially efficacious new strategy for mitigating depression. However, replication is needed. No single set of studies can validate a theory. Indeed, Popper (1959) has argued that a theory can never actually be "proven" regardless of the number of positive findings. We subscribe to a philosophy of science where progress comes from testing and falsifying theories (Meehl, 1978; Popper, 1959). The present work makes a significant scientific contribution because a specific and falsifiable theory has been put forth that explains how sleep influences risk for depression among those with cognitive vulnerability. The theory is based on basic research from the area of memory and sleep, and it has passed its first series of tests. We look forward to continued work that puts our new theory at risk of refutation.

References

Abramson, L. Y., Alloy, L. B., Hogan, M. E., Whitehouse, W. G., Donovan, P., Rose, D., . . . Raniere, D. (1999). Cognitive vulnerability to depression: Theory and evidence. *Journal of Cognitive Psychotherapy, 13,* 5–20.

Abramson, L. Y., Metalsky, G. I., & Alloy, L. B. (1989). Hopelessness depression: A theory-based subtype of depression. *Psychological Review, 96,* 358–372. http://dx.doi.org/10.1037/0033-295X.96.2.358

Alloy, L. B., Abramson, L. Y., Walshaw, P., & Neeren, A. (2006). Cognitive vulnerability to unipolar and bipolar mood disorders. *Journal of Social and Clinical Psychology, 25,* 726–754. http://dx.doi.org/10.1521/jscp.2006.25.7.726

Alloy, L. B., Abramson, L. Y., Whitehouse, W. G., Hogan, M. E., Panzarella, C., & Rose, D. T. (2006). Prospective incidence of first onsets and recurrences of depression in individuals at high and low cognitive risk for depression. *Journal of Abnormal Psychology, 115,* 145–156. http://dx.doi.org/10.1037/0021-843X.115.1.145

Ancoli-Israel, S., Cole, R., Alessi, C., Chambers, M., Moorcroft, W., & Pollak, C. P. (2003). The role of actigraphy in the study of sleep and circadian rhythms. *Sleep, 26,* 342–392.

Anderson, C. A., Lindsay, J. J., & Bushman, B. J. (1999). Research in the psychological laboratory: Truth or triviality? *Current Directions in Psychological Science, 8,* 3–9. http://dx.doi.org/10.1111/1467-8721.00002

Backhaus, J., Junghanns, K., Broocks, A., Riemann, D., & Hohagen, F. (2002). Test-retest reliability and validity of the Pittsburgh Sleep Quality Index in primary insomnia. *Journal of Psychosomatic Research, 53,* 737–740. http://dx.doi.org/10.1016/S0022-3999(02)00330-6

Baert, S., De Raedt, R., Schacht, R., & Koster, E. H. W. (2010). Attentional bias training in depression: Therapeutic effects depend on depression severity. *Journal of Behavior Therapy and Experimental Psychiatry, 41,* 265–274. http://dx.doi.org/10.1016/j.jbtep.2010.02.004

Banks, S., & Dinges, D. F. (2007). Behavioral and physiological consequences of sleep restriction. *Journal of Clinical Sleep Medicine, 3,* 519–528.

Beck, A. T. (1976). *Cognitive therapy and the emotional disorders*. New York, NY: International Universities Press.

Beck, A. T., Rush, A. J., Shaw, B. F., & Emery, G. (1979). *Cognitive therapy of depression*. New York, NY: Guilford Press.

Beck, A. T., Steer, R. A., & Garbin, M. G. (1988). Psychometric properties of the Beck Depression Inventory: Twenty-five years of evaluation. *Clinical Psychology Review, 8,* 77–100. http://dx.doi.org/10.1016/0272-7358(88)90050-5

Benca, R. M., Obermeyer, W. H., Thisted, R. A., & Gillin, J. C. (1992). Sleep and psychiatric disorders. A meta-analysis. *Archives of General Psychiatry, 49,* 651–668. http://dx.doi.org/10.1001/archpsyc.1992.01820080059010

Brown, L. K. (2012). Can sleep deprivation studies explain why human adults sleep? *Current Opinion in Pulmonary Medicine, 18,* 541–545. http://dx.doi.org/10.1097/MCP.0b013e3283596740

Buysse, D. J., Reynolds, C. F., Monk, T. H., Berman, S. R., & Kupfer, D. J. (1989). The Pittsburgh Sleep Quality Index: A new instrument for psychiatric practice and research. *Psychiatry Research, 28,* 193–213. http://dx.doi.org/10.1016/0165-1781(89)90047-4

Carskadon, M. A., & Dement, W. C. (1981). Cumulative effects of sleep restriction on daytime sleepiness. *Psychophysiology, 18,* 107–113. http://dx.doi.org/10.1111/j.1469-8986.1981.tb02921.x

Cohen, J., Cohen, P., West, S. G., & Aiken, L. S. (2003). *Applied multiple regression/correlation analysis for behavioral sciences* (3rd ed.). Mahwah, NJ: Erlbaum.

Daches, S., Mor, N., & Hertel, P. (2015). Rumination: Cognitive consequences of training to inhibit the negative. *Journal of Behavior Therapy and Experimental Psychiatry, 49,* 76–83. http://dx.doi.org/10.1016/j.jbtep.2015.01.010

Dallaspezia, S., Suzuki, M., & Benedetti, F. (2015). Chronobiological therapy for mood disorders. *Current Psychiatry Reports, 17,* 95. http://dx.doi.org/10.1007/s11920-015-0633-6

Deliens, G., Gilson, M., & Peigneux, P. (2014). Sleep and the processing of emotions. *Experimental Brain Research, 232,* 1403–1414. http://dx.doi.org/10.1007/s00221-014-3832-1

Diekelmann, S., Wilhelm, I., & Born, J. (2009). The whats and whens of sleep-dependent memory consolidation. *Sleep Medicine Reviews, 13,* 309–321. http://dx.doi.org/10.1016/j.smrv.2008.08.002

Dinges, D. F., Pack, F., Williams, K., Gillen, K. A., Powell, J. W., Ott, G. E., . . . Pack, A. I. (1997). Cumulative sleepiness, mood disturbance, and psychomotor vigilance performance decrements during a week of sleep restricted to 4–5 hours per night. *Sleep, 20,* 267–277.

Doom, J. R., & Haeffel, G. J. (2013). Teasing apart the effects of cognition, stress, and depression on health. *American Journal of Health Behavior, 37,* 610–619. http://dx.doi.org/10.5993/AJHB.37.5.4

Fischer, S., Diekelmann, S., & Born, J. (2011). Sleep's role in the processing of unwanted memories. *Journal of Sleep Research, 20,* 267–274. http://dx.doi.org/10.1111/j.1365-2869.2010.00881.x

Garber, J. (2008). Prevention of depression: Are we there yet? *Clinical Psychology: Science and Practice, 15,* 336–341. http://dx.doi.org/10.1111/j.1468-2850.2008.00145.x

Germain, A. (2013). Sleep disturbances as the hallmark of PTSD: Where are we now? *The American Journal of Psychiatry, 170,* 372–382. http://dx.doi.org/10.1176/appi.ajp.2012.12040432

Gillin, J. C., Buchsbaum, M., Wu, J., Clark, C., & Bunney, W., Jr. (2001). Sleep deprivation as a model experimental antidepressant treatment: Findings from functional brain imaging. *Depression and Anxiety, 14,* 37–49. http://dx.doi.org/10.1002/da.1045

Graves, L. A., Heller, E. A., Pack, A. I., & Abel, T. (2003). Sleep deprivation selectively impairs memory consolidation for contextual fear conditioning. *Learning & Memory, 10,* 168–176. http://dx.doi.org/10.1101/lm.48803

Haeffel, G. J. (2010). When self-help is no help: Traditional cognitive skills training does not prevent depressive symptoms in people who ruminate. *Behaviour Research and Therapy, 48,* 152–157. http://dx.doi.org/10.1016/j.brat.2009.09.016

Haeffel, G. J. (2011). After further deliberation: Cognitive vulnerability predicts changes in event-specific negative inferences for a poor midterm grade. *Cognitive Therapy and Research, 35,* 285–292. http://dx.doi.org/10.1007/s10608-010-9298-y

Haeffel, G. J., Abramson, L. Y., Brazy, P. C., Shah, J. Y., Teachman, B. A., & Nosek, B. A. (2007). Explicit and implicit cognition: A preliminary test of a dual-process theory of cognitive vulnerability to depression. *Behaviour Research and Therapy, 45,* 1155–1167. http://dx.doi.org/10.1016/j.brat.2006.09.003

Haeffel, G. J., Gibb, B. E., Metalsky, G. I., Alloy, L. B., Abramson, L. Y., Hankin, B. L., . . . Swendsen, J. D. (2008). Measuring cognitive vulnerability to depression: Development and validation of the Cognitive Style Questionnaire. *Clinical Psychology Review, 28,* 824–836. http://dx.doi.org/10.1016/j.cpr.2007.12.001

Haeffel, G. J., & Hames, J. L. (2014). Cognitive vulnerability to depression can be contagious. *Clinical Psychological Science, 2,* 75–85. http://dx.doi.org/10.1177/2167702613485075

Haeffel, G. J., Rozek, D. C., Hames, J. L., & Technow, J. (2012). Too much of a good thing: Testing the efficacy of a cognitive bias modification task for cognitively vulnerable individuals. *Cognitive Therapy and Research, 36,* 493–501. http://dx.doi.org/10.1007/s10608-011-9379-6

Hankin, B. L., Abramson, L. Y., Miller, N., & Haeffel, G. J. (2004). Cognitive vulnerability-stress theories of depression: Examining affective specificity in the prediction of depression versus anxiety in three prospective studies. *Cognitive Therapy and Research, 28,* 309–345. http://dx.doi.org/10.1023/B:COTR.0000031805.60529.0d

Hankin, B. L., Abramson, L. Y., Moffitt, T. E., Silva, P. A., McGee, R., & Angell, K. E. (1998). Development of depression from preadolescence to young adulthood: Emerging gender differences in a 10-year longitudinal study. *Journal of Abnormal Psychology, 107,* 128–140. http://dx.doi.org/10.1037/0021-843X.107.1.128

Harvey, A. G., Murray, G., Chandler, R. A., & Soehner, A. (2011). Sleep disturbance as transdiagnostic: Consideration of neurobiological mechanisms. *Clinical Psychology Review, 31,* 225–235. http://dx.doi.org/10.1016/j.cpr.2010.04.003

Hollon, S. D., Areán, P. A., Craske, M. G., Crawford, K. A., Kivlahan, D. R., Magnavita, J. J., . . . Kurtzman, H. (2014). Development of clinical practice guidelines. *Annual Review of Clinical Psychology, 10,* 213–241. http://dx.doi.org/10.1146/annurev-clinpsy-050212-185529

Hollon, S. D., Thase, M. E., & Markowitz, J. C. (2002). Treatment and prevention of depression. *Psychological Science in the Public Interest, 3,* 39–77. http://dx.doi.org/10.1111/1529-1006.00008

Irwin, M. R. (2015). Why sleep is important for health: A psychoneuroimmunology perspective. *Annual Review of Psychology, 66,* 143–172. http://dx.doi.org/10.1146/annurev-psych-010213-115205

Kensinger, E. A., Garoff-Eaton, R. J., & Schacter, D. L. (2007). Effects of emotion on memory specificity: Memory trade-offs elicited by negative visually arousing stimuli. *Journal of Memory and Language, 56,* 575–591. http://dx.doi.org/10.1016/j.jml.2006.05.004

Killgore, W. D. S. (2010). Effects of sleep deprivation on cognition. *Progress in Brain Research, 185,* 105–129. http://dx.doi.org/10.1016/B978-0-444-53702-7.00007-5

Kumar, T., & Jha, S. K. (2012). Sleep deprivation impairs consolidation of cued fear memory in rats. *PLoS ONE, 7,* e47042. http://dx.doi.org/10.1371/journal.pone.0047042

Kuriyama, K., Soshi, T., & Kim, Y. (2010). Sleep deprivation facilitates extinction of implicit fear generalization and physiological response to fear. *Biological Psychiatry, 68,* 991–998. http://dx.doi.org/10.1016/j.biopsych.2010.08.015

Lund, H. G., Reider, B. D., Whiting, A. B., & Prichard, J. R. (2010). Sleep patterns and predictors of disturbed sleep in a large population of college students. *Journal of Adolescent Health, 46,* 124–132. http://dx.doi.org/10.1016/j.jadohealth.2009.06.016

Lykken, D. T. (1968). Statistical significance in psychological research. *Psychological Bulletin, 70,* 151–159. http://dx.doi.org/10.1037/h0026141

Lynch, D., Laws, K. R., & McKenna, P. J. (2010). Cognitive behavioural therapy for major psychiatric disorder: Does it really work? A meta-analytical review of well-controlled trials. *Psychological Medicine, 40,* 9–24. http://dx.doi.org/10.1017/S003329170900590X

Lyubomirsky, S., Tucker, K. L., Caldwell, N. D., & Berg, K. (1999). Why ruminators are poor problem solvers: Clues from the phenomenology of dysphoric rumination. *Journal of Personality and Social Psychology, 77,* 1041–1060. http://dx.doi.org/10.1037/0022-3514.77.5.1041

Maquet, P. (2001). The role of sleep in learning and memory. *Science, 294,* 1048–1052. http://dx.doi.org/10.1126/science.1062856

McClelland, G. H., & Judd, C. M. (1993). Statistical difficulties of detecting interactions and moderator effects. *Psychological Bulletin, 114,* 376–390. http://dx.doi.org/10.1037/0033-2909.114.2.376

McNeil, N. M., & Alibali, M. W. (2005). Why won't you change your mind? Knowledge of operational patterns hinders learning and performance on equations. *Child Development, 76,* 883–899. http://dx.doi.org/10.1111/j.1467-8624.2005.00884.x

Meehl, P. E. (1978). Theoretical risks and tabular asterisks: Sir Karl, Sir Ronald, and the slow progress of soft psychology. *Journal of Consulting and Clinical Psychology, 46,* 806–834. http://dx.doi.org/10.1037/0022-006X.46.4.806

Menz, M. M., Rihm, J. S., Salari, N., Born, J., Kalisch, R., Pape, H. C., . . . Buchel, C. (2013). The role of sleep and sleep deprivation in consolidating fear memories. *NeuroImage, 75,* 87–96.

Metalsky, G. I., Abramson, L. Y., Seligman, M. E. P., Semmel, A., & Peterson, C. (1982). Attributional styles and life events in the classroom: Vulnerability and invulnerability to depressive mood reactions. *Journal of Personality and Social Psychology, 43,* 612–617. http://dx.doi.org/10.1037/0022-3514.43.3.612

Metalsky, G. I., Halberstadt, L. J., & Abramson, L. Y. (1987). Vulnerability to depressive mood reactions: Toward a more powerful test of the diathesis-stress and causal mediation components of the reformulated theory of depression. *Journal of Personality and Social Psychology, 52,* 386–393. http://dx.doi.org/10.1037/0022-3514.52.2.386

Metalsky, G. I., Joiner, T. E., Jr., Hardin, T. S., & Abramson, L. Y. (1993). Depressive reactions to failure in a naturalistic setting: A test of the hopelessness and self-esteem theories of depression. *Journal of Abnormal Psychology, 102,* 101–109. http://dx.doi.org/10.1037/0021-843X.102.1.101

Nisbett, R., & Wilson, T. (1977). Telling more than we can know: Verbal reports on mental processes. *Psychological Review, 84,* 231–259. http://dx.doi.org/10.1037/0033-295X.84.3.231

Nolen-Hoeksema, S., & Morrow, J. (1991). A prospective study of depression and posttraumatic stress symptoms after a natural disaster: The 1989 Loma Prieta Earthquake. *Journal of Personality and Social Psychology, 61,* 115–121. http://dx.doi.org/10.1037/0022-3514.61.1.115

Nolen-Hoeksema, S., Wisco, B. E., & Lyubomirsky, S. (2008). Rethinking rumination. *Perspectives on Psychological Science, 3,* 400–424. http://dx.doi.org/10.1111/j.1745-6924.2008.00088.x

Pace-Schott, E. F., Germain, A., & Milad, M. R. (2015). Effects of sleep on memory for conditioned fear and fear extinction. *Psychological Bulletin, 141,* 835–857. http://dx.doi.org/10.1037/bul0000014

Payne, J. D., Chambers, A. M., & Kensinger, E. A. (2012). Sleep promotes lasting changes in selective memory for emotional scenes. *Frontiers in Integrative Neuroscience, 6,* 108. http://dx.doi.org/10.3389/fnint.2012.00108

Payne, J. D., Stickgold, R., Swanberg, K., & Kensinger, E. A. (2008). Sleep preferentially enhances memory for emotional components of scenes. *Psychological Science, 19,* 781–788. http://dx.doi.org/10.1111/j.1467-9280.2008.02157.x

Peigneux, P., Laureys, S., Fuchs, S., Collette, F., Perrin, F., Reggers, J., . . . Maquet, P. (2004). Are spatial memories strengthened in the human hippocampus during slow wave sleep? *Neuron, 44,* 535–545. http://dx.doi.org/10.1016/j.neuron.2004.10.007

Peterson, M. J., & Benca, R. M. (2006). Sleep in mood disorders. *Psychiatric Clinics of North America, 29,* 1009–1032. http://dx.doi.org/10.1016/j.psc.2006.09.003

Plante, D. T., & Winkelman, J. W. (2008). Sleep disturbance in bipolar disorder: Therapeutic implications. *The American Journal of Psychiatry, 165,* 830–843. http://dx.doi.org/10.1176/appi.ajp.2008.08010077

Poe, G. R., Walsh, C. M., & Bjorness, T. E. (2010). Both duration and timing of sleep are important to memory consolidation. *Sleep, 33,* 1277–1278.

Popper, K. R. (1959). *The logic of scientific discovery*. London, UK: Hutchinson of London. http://dx.doi.org/10.1063/1.3060577

Potthoff, J. G., Holahan, C. J., & Joiner, T. E., Jr. (1995). Reassurance seeking, stress generation, and depressive symptoms: An integrative model. *Journal of Personality and Social Psychology, 68,* 664–670. http://dx.doi.org/10.1037/0022-3514.68.4.664

Rasch, B., & Born, J. (2013). About sleep's role in memory. *Physiological Reviews, 93,* 681–766. http://dx.doi.org/10.1152/physrev.00032.2012

Ray, R. D., Ochsner, K. N., Cooper, J. C., Robertson, E. R., Gabrieli, J. D. E., & Gross, J. J. (2005). Individual differences in trait rumination and the neural systems supporting cognitive reappraisal. *Cognitive, Affective & Behavioral Neuroscience, 5,* 156–168. http://dx.doi.org/10.3758/CABN.5.2.156

Sandor, P., & Shapiro, C. M. (1994). Sleep patterns in depression and anxiety: Theory and pharmacological effects. *Journal of Psychosomatic Research, 38,* 125–139. http://dx.doi.org/10.1016/0022-3999(94)90143-0

Schelling, G., Kilger, E., Roozendaal, B., deq Uervain, D. J., Briegel, J., Dagge, A., . . . Kapfhammer, H. P. (2004). Stress doses of hydrocortisone, traumatic memories, and symptoms of posttraumatic stress disorder in patients after cardiac surgery: A randomized study. *Biological Psychiatry, 55,* 627–633. http://dx.doi.org/10.1016/j.biopsych.2003.09.014

Schulte, W. (1959). Sequelae of sleep deprivation. *Medizinische Klinik, 54,* 969–973.

Shiffrin, R. M., & Schneider, W. (1977). Controlled and automatic human information processing: II. Perceptual learning, automatic attending, and a general theory. *Psychological Review, 84,* 127–190. http://dx.doi.org/10.1037/0033-295X.84.2.127

Spielman, A. J., Saskin, P., & Thorpy, M. J. (1987). Treatment of chronic insomnia by restriction of time in bed. *Sleep, 10,* 45–56.

Tsuno, N., Besset, A., & Ritchie, K. (2005). Sleep and depression. *Journal of Clinical Psychiatry, 66,* 1254–1269. http://dx.doi.org/10.4088/JCP.v66n1008

Uher, R., & McGuffin, P. (2010). The moderation by the serotonin transporter gene of environmental adversity in the etiology of depression: 2009 update. *Molecular Psychiatry, 15,* 18–22. http://dx.doi.org/10.1038/mp.2009.123

van Heugten-van der Kloet, D., Giesbrecht, T., & Merckelbach, H. (2015). Sleep loss increases dissociation and affects memory for emotional stimuli. *Journal of Behavior Therapy and Experimental Psychiatry, 47,* 9–17. http://dx.doi.org/10.1016/j.jbtep.2014.11.002

Vgontzas, A. N., Zoumakis, E., Bixler, E. O., Lin, H. M., Follett, H., Kales, A., & Chrousos, G. P. (2004). Adverse effects of modest sleep restriction on sleepiness, performance, and inflammatory cytokines. *The Journal of Clinical Endocrinology and Metabolism, 89,* 2119–2126. http://dx.doi.org/10.1210/jc.2003-031562

Wagner, U., Gais, S., & Born, J. (2001). Emotional memory formation is enhanced across sleep intervals with high amounts of rapid eye movement sleep. *Learning & Memory, 8,* 112–119. http://dx.doi.org/10.1101/lm.36801

Wagner, U., Hallschmid, M., Rasch, B., & Born, J. (2006). Brief sleep after learning keeps emotional memories alive for years. *Biological Psychiatry, 60,* 788–790. http://dx.doi.org/10.1016/j.biopsych.2006.03.061

Walker, M. P. (2009). The role of sleep in cognition and emotion. *Annals of the New York Academy of Sciences, 1156,* 168–197. http://dx.doi.org/10.1111/j.1749-6632.2009.04416.x

Walker, M. P., & van der Helm, E. (2009). Overnight therapy? The role of sleep in emotional brain processing. *Psychological Bulletin, 135,* 731–748. http://dx.doi.org/10.1037/a0016570

Watkins, E., & Brown, R. G. (2002). Rumination and executive function in depression: An experimental study. *Journal of Neurology, Neurosurgery, & Psychiatry, 72,* 400–402. http://dx.doi.org/10.1136/jnnp.72.3.400

Wehr, T. A., Sack, D. A., & Rosenthal, N. E. (1987). Sleep reduction as a final common pathway in the genesis of mania. *The American Journal of Psychiatry, 144,* 201–204.

Yoo, S. S., Gujar, N., Hu, P., Jolesz, F. A., & Walker, M. P. (2007). The human emotional brain without sleep—A prefrontal amygdala disconnect. *Current Biology, 17,* R877–R878. http://dx.doi.org/10.1016/j.cub.2007.08.007

Yoo, S. S., Hu, P. T., Gujar, N., Jolesz, F. A., & Walker, M. P. (2007). A deficit in the ability to form new human memories without sleep. *Nature Neuroscience, 10,* 385–392. http://dx.doi.org/10.1038/nn1851

Zhang, Z., & Yuan, K.-H. (2015). *WebPower: Statistical power analysis online*. Retrieved from http://webpower.psychstat.org

Received February 20, 2016
Revision received August 3, 2016
Accepted August 16, 2016 ■