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Cognitive Vulnerability to Depression Can Be Contagious

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Abstract

Cognitive vulnerability is a potent risk factor for depression. Individual differences in cognitive vulnerability solidify in early adolescence and remain stable throughout the life span. However, stability does not mean immutability. We hypothesized that cognitive vulnerability would be susceptible to change during major life transitions when social milieus undergo significant changes (e.g., moving to college). Specifically, we tested the hypothesis that cognitive vulnerability could change via a contagion effect. We tested this hypothesis using a prospective longitudinal design with a sample of randomly assigned college freshmen roommate pairs (103 pairs). Results supported the hypotheses. Participants who were randomly assigned to a roommate with high levels of cognitive vulnerability were likely to “catch” their roommate’s cognitive style and develop higher levels of cognitive vulnerability. Moreover, those who experienced an increase in cognitive vulnerability had significantly greater levels of depressive symptoms over the prospective interval than those who did not.

Keywords

cognitive vulnerability, depression, contagion, rumination

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The cognitive theories of depression are among the most clearly articulated and well-supported models of depression. According to these theories (e.g., Abramson, Metalsky, & Alloy, 1989; Nolen-Hoeksema, 1991), some individuals have a *cognitive vulnerability* that interacts with stress to produce depression. Specifically, people are vulnerable to depression because they have a tendency to generate interpretations of stressful life events (and dysphoric moods) that have negative implications for their future and for their self-worth.

Recent research has provided direct and compelling support for the cognitive vulnerability hypothesis (see Haefel et al., 2008, and Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008, for reviews). Prospective studies have consistently found that cognitive vulnerability interacts with stressful events to predict the development of depressive symptoms and depressive disorders (Abramson et al., 1999; Haefel et al., 2008; Nolen-Hoeksema, 2000; Nolen-Hoeksema et al., 2008). These studies have shown that it is possible to take a group of never depressed individuals and predict which of them are most likely to develop a first episode of clinically significant depression based

solely on individual differences in their cognitive style for interpreting life events (i.e., their level of cognitive vulnerability). The cognitive model of depression is also supported by prevention and treatment studies. Interventions designed to modify cognitive vulnerabilities are among the most effective interventions available for depression. For example, cognitive therapy is as effective as medication and may even have a prophylactic effect (Hollon, Stewart, & Strunk, 2006; Hollon, Thase, & Markowitz, 2002).

Taken together, prior studies indicate that cognitive vulnerability is a potent risk factor for depression. Thus, it is critical to understand how one “acquires” a cognitive vulnerability. It is likely that a variety of factors (e.g., genetic, biological, and environmental) contribute to developing a cognitive vulnerability; however, many researchers have converged on the idea that early exposure to negative interpersonal contexts is a particularly

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influential antecedent. For example, both negative parenting practices (e.g., emotional abuse) and direct inferential feedback from significant others (e.g., teachers, peers, and parents) predict the development of cognitive vulnerability (e.g., Alloy et al., 2001; Cole, Jacquez, & Maschman, 2001; Dweck, Davidson, Nelson, & Enna, 1978; Garber & Flynn, 2001; Lau, Belli, Gregory, Napolitano, & Eley, 2012; L. Murray, Woolgar, Cooper, & Hipwell, 2001). Of importance, it appears that these early contexts do not take long to exert their influences on the development of cognitive vulnerability. By early adolescence it is possible to detect meaningful and stable individual differences in how individuals cognitively interpret stressful life events. Indeed, longitudinal and cross-sectional studies indicate that cognitive vulnerability can predict depression in children as young as 12 years of age (Cole et al., 2008; Nolen-Hoeksema, Girgus, & Seligman, 1992).

Once cognitive vulnerability forms and stabilizes in early adolescence, it confers risk for depression throughout the life span (see Romens, Abramson, & Alloy, 2009, for a review). Research shows that cognitive vulnerability exhibits moderate to high stability during high school (Hankin & Abramson, 2002), college (Alloy et al., 2000), and the rest of adulthood (Burns & Seligman, 1989; Haefel et al., 2005). These findings suggest that by early adulthood one is saddled (or blessed) with a level of cognitive risk that is relatively impervious to changes in environmental conditions (Hankin, 2008).

Given its stability over time, cognitive vulnerability is often viewed similarly to a genetic diathesis. One either possesses the risk factor or one does not. And one's risk status remains the same throughout one's life. However, it is important to recognize that stability is not the same as immutability (Haefel et al., 2005; Just, Abramson, & Alloy, 2001). Although highly stable, there may be unique environmental situations during which cognitive vulnerability can be altered. One situation is when it is directly targeted by a prevention or treatment intervention. Results of studies testing cognitive interventions for depression (e.g., cognitive therapy) demonstrate that cognitive vulnerability can indeed be changed (e.g., Clark & Beck, 2010; DeRubeis & Hollon, 1995; Seligman, Schulman, DeRubeis, & Hollon, 1999). Moreover, reductions in cognitive vulnerability appear to decrease risk for the onset or recurrence of depression (e.g., Clark & Beck, 2010; DeRubeis et al., 1990; Segal et al., 2006; Tang & DeRubeis, 1999). Results from these studies suggest that cognitive vulnerability is not completely resistant to change. However, targeted interventions such as cognitive therapy are the exception rather than the rule for most people. Thus, the question remains as to whether there are naturally occurring environmental conditions that can alter (either increase or decrease) this potent risk factor.

The purpose of this study was to test the hypothesis that cognitive vulnerability can change via a contagion effect. Specifically, we proposed that cognitive vulnerability is susceptible to change during life transitions when social milieus undergo significant changes. Previous research indicates that early social contexts have a significant influence on the development of cognitive vulnerability. We suspect that social contexts continue to influence cognitive vulnerability even into adulthood, but their influence is typically masked by the continuity and stability of the environment over time (e.g., Mehl & Pennebaker, 2003). However, during situations in which a person is continually exposed to a novel social context (e.g., going to college, moving to a new state, entering an assisted living center), we should be able to detect its influence on cognitive vulnerability once again. Specifically, we predicted that individuals would be influenced directly by the cognitive vulnerability levels of those around them (e.g., through direct inferential feedback or modeling). In other words, the level of cognitive vulnerability of those around a person might "rub off" and be contagious. Moreover, we hypothesized that contagion-induced changes in cognitive vulnerability would have implications for risk for future depressive symptoms.

We tested the contagion hypothesis in a sample of randomly assigned college freshmen roommate pairs. Freshmen are an ideal sample for testing the hypotheses because they are experiencing a major life transition that involves a significant change to their social environment, are at the peak age for developing depression, and can be randomly assigned to a roommate. The randomization of roommate pairs ensures that our results are not due to a self-selection bias, in which participants select (or create) their own social environment. Participants completed measures of cognitive vulnerability (as featured in two prominent cognitive theories of depression), depressive symptoms, and stressful life events at three time points over a 6-month prospective interval.

Method

Participants

Participants were 108 college freshmen roommate pairs (42 male pairs, 66 female pairs; M age = 18.00) from a selective, private, midsized university in the midwestern United States. Five roommate pairs ($n = 10$) dropped out after the baseline assessment (note that these roommate pairs were not different from pairs who remained in the study on their baseline measures of cognitive vulnerability or depressive symptoms; all $ps > .46$). Thus, the final sample consisted of 103 roommate pairs. Of the remaining participants, 90% completed at least two of three questionnaire sessions (with all participants completing

the baseline assessment). Of the sample, 80% reported their ethnicity as Caucasian, 9% as Hispanic, 6% as Asian, 3% as African American, and 2% as "other."

To obtain the sample, 700 freshmen were randomly selected from the freshmen directory and contacted via e-mail asking if they and their roommate would like to participate in the study. Freshmen indicated interest in the study by responding affirmatively to the e-mail. To ensure that the roommates also agreed to participate, freshmen were required to copy their roommate on the corresponding e-mail. We paid participants \$5 for each questionnaire session that they completed. It is important to note that all freshmen at this university are required to live in an on-campus dormitory, and the university housing agency uses a computer to randomly assign freshmen to both a roommate and a dormitory.

Measures

Cognitive vulnerability. We measured the cognitive vulnerability factors featured in two prominent cognitive theories of depression: response styles theory (Nolen-Hoeksema, 1991) and hopelessness theory (Abramson et al., 1989). Response style theory defines cognitive vulnerability as the tendency to focus attention on one's negative mood and the implications of that mood (i.e., ruminate). Individuals who have this tendency are at increased risk for more severe and enduring depressive responses. Response style theory's cognitive vulnerability factor is measured with the Ruminative Response Scale (RRS; Nolen-Hoeksema & Morrow, 1991). It is composed of 22 items that assess participants' ruminative responses to depressed moods. Items are rated on a Likert-type scale from 1 to 4. Consistent with prior research, we used the RRS Brooding subscale, which has demonstrated the greatest association with depression. The RRS has demonstrated good internal consistency (alphas typically greater than .80; Nolen-Hoeksema & Morrow, 1991) and predictive validity (Nolen-Hoeksema, Parker, & Larson, 1994; Treynor, Gonzalez, & Nolen-Hoeksema, 2003). For example, numerous prospective studies have found that the RRS Brooding subscale interacts with measures of negative events to predict the development of depression (e.g., Nolen-Hoeksema et al., 1994; Nolen-Hoeksema, Larson, & Grayson, 1999; Nolen-Hoeksema, Morrow, & Fredrickson, 1993). Internal consistency for the Brooding subscale in the current sample was good ($\alpha = .90$); test-retest reliability over 6 months was .54.

We also measured the cognitive vulnerability factor featured in the hopelessness theory of depression. Hopelessness theory defines cognitive vulnerability as the tendency of an individual to make particular kinds of inferences about the cause, consequences, and self-worth

implications of negative life events. Specifically, when faced with a negative life event, an individual who has a cognitive vulnerability is likely to (a) attribute the event to stable and global causes, (b) view the event as likely to lead to other negative consequences, and (c) construe the event as implying that he or she is unworthy or deficient. Individuals who generate these negative inferences are hypothesized to be at risk for hopelessness, which is viewed as a proximal and sufficient cause of depression. Hopelessness theory's cognitive vulnerability factor is measured with the Cognitive Style Questionnaire (CSQ; Haefel et al., 2008). It assesses participants' inferences for 12 hypothetical negative events on dimensions of cause, consequences, and self-worth. The CSQ has demonstrated excellent internal consistency (coefficient alpha typically $> .90$; Haefel et al., 2008), strong test-retest reliability over months and even years (e.g., 1-year test-retest reliability is .80; Alloy et al., 2000), and predictive validity (Haefel et al., 2008). Prospective studies have consistently found that the CSQ interacts with measures of negative events to predict the development of depressive symptoms (e.g., Haefel et al., 2007; Metalsky & Joiner, 1992) and depressive disorders (e.g., Alloy et al., 2006; Hankin, Abramson, Miller, & Haefel, 2004). Internal consistency in the current sample was good ($\alpha = .89$); test-retest reliability over 6 months was .62.

Stressful life events. The Acute Life Events Questionnaire (ALEQ; Haefel et al., 2007) was used to assess 30 naturally occurring acute stressful life events important to college students. Items assessed a broad range of life events from achievement to interpersonal. Participants were instructed to indicate which of the stressful life events had occurred to them over the previous 5 weeks. Scores range from 0 to 30, with higher scores indicating the occurrence of more negative events. Prior research indicates that the ALEQ has good reliability (test-retest correlations typically range from .60 to .80; Haefel, 2010; Haefel, Eastman, & Grigorenko, 2012) and predictive validity. For example, prospective studies have found that the ALEQ predicts the development of future depressive symptoms (Doom & Haefel, 2013; Haefel & Vargas, 2011). Internal consistency in the current sample was acceptable ($\alpha = .77$); test-retest reliability over 3 months was .57.

Depressive symptoms. The Beck Depression Inventory (BDI; Beck, Rush, Shaw, & Emery, 1979) is a widely used 21-item self-report inventory that assesses depressive symptoms. Participants rate symptoms of depression (e.g., negative mood, pessimism, sleep disturbance) on 0 to 3 scales. Total scores on the BDI can range from 0 to 63, with higher scores indicating greater levels of depressive

symptoms. The BDI has high internal consistency (coefficient alpha is typically greater than .8), good test-retest reliability ($r = .60-.83$ for nonpsychiatric samples), and validity with both college and psychiatric samples (see Beck, Steer, & Garbin, 1988, for a review). Internal consistency in the current sample was good ($\alpha = .87$); test-retest reliability over 6 months was $.57$.

Procedure

Prior to arriving on campus, the university housing agency randomly assigned participants to both a roommate and a dormitory. Within 1 month of arriving on campus, freshmen completed the first of three online questionnaire sessions (i.e., the baseline assessment). Participants completed measures of cognitive vulnerability (CSQ, RRS) and depressive symptoms (BDI). Participants completed these same measures 3 months and 6 months later; they also completed a measure of stressful life events (ALEQ) at the two prospective time points.

Results

We tested two primary hypotheses. First, we predicted that cognitive vulnerability to depression would be contagious between roommates. Second, we predicted that changes in cognitive vulnerability would lead to changes in risk for future depressive symptoms. As expected due to random assignment, there was no association between roommates' levels of cognitive vulnerability at baseline: The intraclass correlation for cognitive vulnerability as measured by the RRS was $-.04$, $p = .66$; the intraclass correlation for cognitive vulnerability as measured by the CSQ was $-.06$, $p = .73$.

Cognitive vulnerability to depression is contagious

We used the actor-partner interdependence model (APIM; Kenny, 1996) to test whether cognitive vulnerability was contagious between roommates. Specifically, we used multilevel modeling for indistinguishable dyads to test the stability of cognitive vulnerability over time and determine how a participant's vulnerability level was influenced by his or her roommate's vulnerability level. APIM is superior to traditional statistical strategies for testing interpersonal phenomena for a number of reasons. First, unlike traditional statistical techniques, APIM does not assume independence of observations. This is important because the assumption of independence is often violated in studies of interpersonal relationships; this violation can then lead to biased statistical significance tests (Cook & Kenny, 2005). Second, APIM accounts

for bidirectional effects; it estimates both the "actor's" effect on the "partner" as well as the "partner's" effect on the "actor."

In our analyses, the dependent variable was cognitive vulnerability (as measured by the RRS and CSQ, respectively) at 3 months and 6 months. We controlled for baseline levels of depressive symptoms (BDI) and recent stressful life events (ALEQ) to ensure the results could not be attributed to any individual differences in negative mood or the shared experience of stressful life events. The independent variable was level of cognitive vulnerability at baseline (as measured by the RRS and CSQ, respectively).

Consistent with prior research, results showed that cognitive vulnerability was relatively stable across the prospective interval. The test-retest correlations for both cognitive vulnerability measures ranged between $.5$ and $.7$ for the prospective interval. Participants' level of cognitive vulnerability at baseline was a robust predictor of their level of cognitive vulnerability at both 3 months and 6 months (see Figs. 1 and 2). Of importance, the results supported our main hypothesis that cognitive vulnerability would be contagious between college roommates. As shown in Figure 1, a person's future level of cognitive vulnerability was significantly affected by his or her roommate's baseline level of cognitive vulnerability (and vice versa) as measured by the RRS. Specifically, individuals who were randomly assigned to a roommate with high initial levels of cognitive vulnerability experienced increases in their own level of cognitive vulnerability over time, whereas those who were assigned to a roommate with low initial levels of cognitive vulnerability experienced decreases in their levels of cognitive vulnerability. This contagion effect was significant for the RRS at both the 3-month, $b = .17$, $t(155) = 2.34$, $p = .02$, and 6-month intervals, $b = .16$, $t(132) = 2.13$, $p = .04$, even after controlling for baseline levels of cognitive vulnerability, depressive symptoms, and stressful life events for both members of the roommate pair (note that the results also hold if roommates' level of depressive symptoms at follow-up [3 months and 6 months, respectively] is also controlled for). The contagion effect was not significant when cognitive vulnerability was measured with the CSQ. As shown in Figure 2, the cognitive vulnerability as measured by the CSQ was more stable across time, and there was not a significant contagion effect at 3 months, $b = .01$, $t(154) = 0.07$, $p = .94$, or at 6 months, $b = .12$, $t(133) = 1.83$, $p = .07$. It is important to note that neither roommates' levels of depressive symptoms nor their levels of stress was a significant predictor of changes in participants' levels of cognitive vulnerability (either the CSQ or RRS) at either 3 months or 6 months.

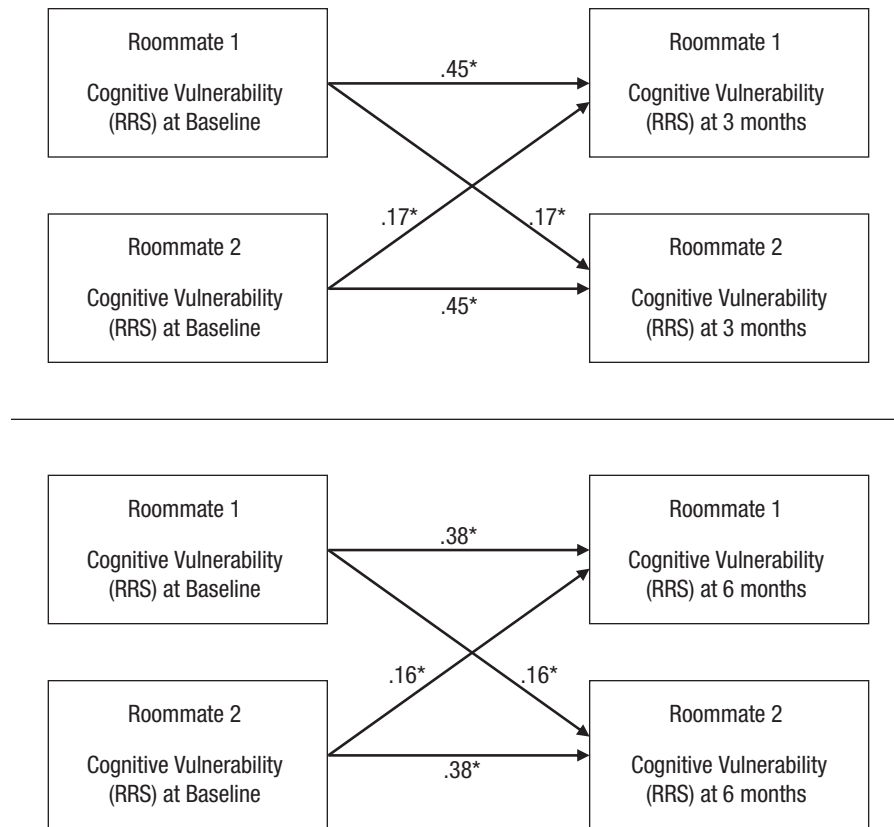


Fig. 1. Results of the actor-partner interdependence model testing whether cognitive vulnerability, as measured by the Ruminative Response Scale (RRS), was contagious between randomly assigned roommates at 3 months (top panel) and 6 months (bottom panel) controlling for depressive symptoms and negative life events (for both members of the roommate pair).

* $p < .05$.

Changes in cognitive vulnerability predict future depressive symptoms

We used a hierarchical multiple regression analysis (Cohen, Cohen, West, & Aiken, 2003) to test whether increases in cognitive vulnerability from baseline to 3 months later increased risk for depressive symptoms at the 6-month assessment under conditions of stress. The dependent variable was level of depressive symptoms at the 6-month assessment. Covariates (depressive symptoms at baseline and stressful life events at 3 months) were entered into the first step of the regression equation. In the second step, the main effects of stressful life events (ALEQ at 6 months) and changes in cognitive vulnerability (e.g., RRS change score from baseline to 3 months) were entered. Last, the Vulnerability \times Stress interaction term was entered (RRS change score \times ALEQ at 6 months). Consistent with hypotheses, there was a significant RRS \times ALEQ interaction, $b = -.07$, $t = -2.00$, $pr = -.17$, $p = .048$, (model $R^2 = .46$). As shown in Figure 3, participants who experienced an increase in cognitive

vulnerability during the first 3 months of college exhibited significantly greater levels of depressive symptoms at 6 months than did participants who did not experience an increase in cognitive vulnerability, particularly under conditions of high stress.

The results of the regression analysis supported our hypothesis; however, a limitation of this analysis was that we could not determine if the portion of change in cognitive vulnerability due to contagion was what predicted future depression. To address this limitation, we conducted a complementary analysis using the APIM. If the contagion effect is responsible for changes in risk for depression, then changes in cognitive vulnerability should mediate the association between roommates' level of cognitive vulnerability and participants' future depressive symptoms. In other words, roommates' level of cognitive vulnerability at baseline should predict changes in participants' future level of depression because of its effect on participants' own level of cognitive vulnerability. Following the criteria for mediation set forth by Baron and Kenny (1986), the following four conditions should hold if

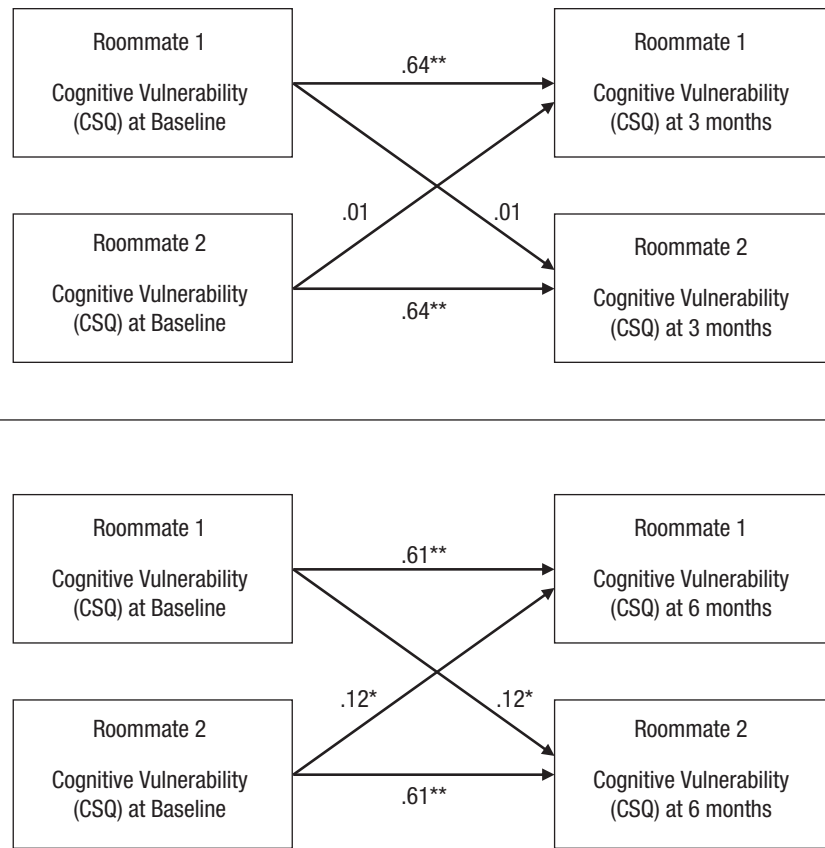


Fig. 2. Results of the actor-partner interdependence model testing whether cognitive vulnerability, as measured by the Cognitive Style Questionnaire (CSQ), was contagious between randomly assigned roommates at 3 months (top panel) and 6 months (bottom panel) controlling for depressive symptoms and negative life events (for both members of the roommate). * $p < .10$. ** $p < .05$.

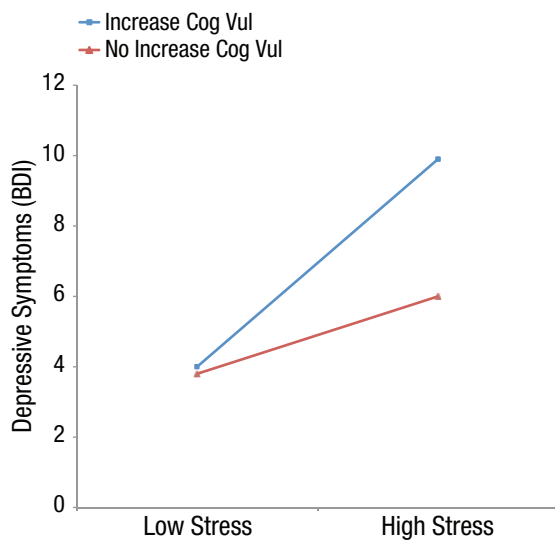


Fig. 3. Depressive symptoms at 6 months as a function of change in cognitive vulnerability (increase vs. no increase in RRS score from baseline to 3 months) and stress (low vs. high).

mediation is present: (a) Roommates' baseline level of cognitive vulnerability should predict participants' future level of depressive symptoms, (b) roommates' baseline level of cognitive vulnerability should predict changes in participants' level of cognitive vulnerability, (c) changes in participants' level of cognitive vulnerability should predict future depressive symptoms when controlling for roommates' baseline level of cognitive vulnerability, and (d) roommates' baseline level of cognitive vulnerability in the third condition should no longer be a significant predictor when controlling for changes in cognitive vulnerability. Note that Condition b was already supported in the earlier analysis; thus, these analyses focused on Conditions a, c, and d.

First we tested if roommates' level of cognitive vulnerability (RRS) at baseline predicted participants' level of depressive symptoms at 6 months (Condition a). As hypothesized, the APIM analyses found that individuals' future levels of depressive symptoms were directly influenced by their roommates' baseline level of cognitive

vulnerability. Participants who were randomly assigned to a roommate with high initial levels of cognitive vulnerability experienced greater levels of depressive symptoms over time than those assigned to a roommate with low initial levels of cognitive vulnerability experienced (even after controlling for baseline levels of cognitive vulnerability, depressive symptoms, and stressful life events for both members of the roommate pair), $b = .32$, $t(124) = 2.36$, $p = .02$. Next, we tested for the mediation effect by adding RRS change score (i.e., the mediator) to the APIM to determine if it reduced the power of roommates' level of cognitive vulnerability to predict participants' future depressive symptoms (Conditions c and d). Results confirmed the mediating effect of changes in cognitive vulnerability (Sobel's t test $p = .054$). When controlling for changes in participants' level of cognitive vulnerability from baseline to 3 months, roommates' baseline level of cognitive vulnerability was no longer a significant predictor of participants' depressive symptoms at 6 months. Changes in cognitive vulnerability (i.e., the mediator) remained a significant predictor of depressive symptoms, $b = .35$, $t(123) = 2.23$, $p = .03$.

It is important to note that roommates' level of depressive symptoms was not a significant predictor of changes in participants' level of depressive symptoms at either 3 months ($p > .40$) or 6 months ($p > .40$). In other words, there was not a contagion effect for depressive symptoms.

Discussion

The results of the study support the hypothesis that cognitive vulnerability to depression can be contagious. We found that participants' level of cognitive vulnerability was significantly influenced by their roommates' level of cognitive vulnerability and vice versa. Participants who were randomly assigned to a roommate with high levels of cognitive vulnerability (specifically a ruminative response style) were likely to "catch" their roommate's cognitive style and develop higher levels of cognitive vulnerability over the prospective interval. This contagion effect was detectable after only 3 months of cohabitation. It is important to emphasize that these results cannot be explained by participants' and roommates' levels of depressive symptoms or by the occurrence of stressful life events (neither roommates' level of depressive symptoms nor their level of stress was a significant predictor of changes in cognitive vulnerability). Not only was cognitive vulnerability contagious, but changes in vulnerability affected risk for future depressive symptoms. Participants who experienced an increase in cognitive vulnerability levels during the first 3 months of college had nearly twice the level of depressive symptoms at 6 months than did participants who did not experience an increase in cognitive vulnerability, particularly under conditions of high stress.

To date, there has been little reason to view cognitive vulnerability as anything other than highly stable, akin to a genetic diathesis. However, the results of this study indicate that it may be time to rethink how cognitive vulnerability is conceptualized. Our study demonstrates that cognitive vulnerability has the potential to wax and wane over time depending on the social context. This means that cognitive vulnerability should be thought of as plastic rather than immutable. Our findings are consistent with a growing number of studies that have found that many psychological and biological factors previously thought to be "set in stone" by adulthood continue to be malleable. For example, it was assumed for nearly a century that the central nervous system became structurally stable in early childhood and that the brain was not capable of generating new neurons in adulthood. But there is now strong support for neurogenesis in adult mammals (Gould & Gross, 2002). In addition, it was once believed that a second language could not be readily acquired after a critical period in childhood. However, we now know that given the appropriate environmental context (e.g., immersion), adults can very successfully achieve proficiency in a second language (e.g., Flege, Yeni Komshian, & Liu, 1999). Along these lines, we found that cognitive vulnerability can still be altered by the social environment (via contagion) years after its consolidation in early adolescence. Our finding opens the door for an entirely new line of research investigating how cognitive vulnerability changes over the life span, the mechanisms by which change occurs, and their effect on developmental changes in rates of depression.

We suggest two high-priority areas of study to advance our understanding of the cognitive vulnerability contagion phenomenon and, in turn, risk for depression. One priority is to determine the scope of the contagion effect. This study used a unique sample and study design to maximize sensitivity for detecting the hypothesized contagion effect. Participants in our study were experiencing a dramatic change in their social context. For many of them, moving away to college was the first time that they had to live apart from their family and friends for an extended period. In addition, participants were randomly assigned to roommates. This naturalistic experimental enabled us to make definitive conclusions about the presence of a contagion effect and rule out alternative explanations such as a self-selection bias, in which participants choose their own social context. The sample and experimental design are significant strengths of the study as they optimized our ability to detect the contagion effect and make strong conclusions about our results. At the same time, however, this methodology does not capture the typical person's social environment. Thus, research is needed to determine the generalizability of the contagion effect. One strategy is to test whether the findings apply to other life transitions such as moving

to a new city or entering a nursing home. It is likely that the contagion effect will generalize to such situations because they are akin to moving to college. They represent a new life stage (e.g., starting a first “real job”) and a major transition to a new living environment.

However, given the relatively short amount of time in which cognitive vulnerability changed in the present study, we suspect that the contagion effect may not be restricted to major life transitions. Indeed, we posit that a contagion effect may be detected even in a much more ubiquitous social context—a person’s social media network. Social media platforms such as Facebook and Twitter are changing the interpersonal landscape. One in seven people are active Facebook users (Facebook Inc., 2012), and more than 100 million people use Twitter (Miller, 2011). People are now able to develop relationships (both platonic and romantic) with millions of individuals from all over the world. This means that a person’s social milieu has the potential to be much larger and much more fluid than ever before. No longer is a person’s social environment restricted to his or her immediate surroundings. It is now possible to alter one social network with the click of a button (e.g., adding a new “follower” on Twitter or accepting a friend request on Facebook). We suspect that as a person’s social media network changes over time, so will his or her levels of cognitive vulnerability. Preliminary research has already demonstrated the potential power of social networks in creating contagion effects. For example, scientists have found social media networks to exhibit contagion-like effects for mood, smoking habits, and even obesity (Christakis & Fowler, 2013). A recent study of reciprocal-reply networks on Twitter found that happiness appears to be contagious up to three degrees away from the user (Bliss, Klourmann, Harris, Danforth, & Dodds, 2012). These results add to a growing number of studies that suggest that psychological constructs can be contagious within social media networks and that the effects are not merely due to homophily (Christakis & Fowler, 2013).

Social media networks may be particularly influential in altering cognitive vulnerability levels due to their increased activity during major life events. For example, deaths of high-profile figures, natural disasters, and other highly stressful events are among the most tweeted events of each year. These types of events are fodder for cognitive vulnerability. During these times, social media users are exposed to the cognitive reactions and coping strategies of potentially hundreds of people around the world. A question that is ripe for future research is whether the tone and consistency of comments in individuals’ social media network have the potential to alter their cognitive vulnerability levels over their life span and, in turn, influence their risk for depression.

A second priority for future research is to determine if the contagion effect can be leveraged to create resilience to depression. Until now, it appeared that the only way to alter cognitive vulnerability to depression was to target it directly via cognitive therapy. However, our findings suggest that it may be possible to use an individual’s social environment as part of the intervention process (either as a supplement to existing cognitive interventions or possibly as a stand-alone intervention). An intervention that capitalizes on the contagion effect would require an evaluation of the patient’s interpersonal context including family, friends, and perhaps even his or her social media network. The primary purpose of this evaluation would be to ensure that the patient is surrounding himself or herself with an adaptive cognitive social context. The therapist could assess if people in the patient’s life are modeling and providing adaptive cognitive feedback about stress and negative life events. The therapist could then provide those with maladaptive cognitive styles with information about the contagion effect along with training that would help them identify maladaptive thought patterns and provide examples of more adaptive ways of thinking (see Dobkin et al., 2007, for an example of a program designed to train partners in adaptive cognitive feedback). Surrounding a person with others who exhibit an adaptive cognitive style should help to facilitate cognitive change in therapy.

A second benefit of developing interventions that capitalize on the contagion effect is that such interventions could be used in experimental designs to determine the mechanisms by which contagion occurs. There are a number of social processes that might contribute to the contagion of cognitive vulnerability. For example, people might contract high levels of cognitive vulnerability by observing (or modeling) others around them. Another possibility is that a person learns maladaptive cognitive styles on the basis of inferences that significant others make for his or her (i.e., the person’s) behavior. In this case, the person develops a cognitive style consistent with the feedback that he or she receives. By using an experimental intervention design, it will be possible to determine which of these processes underlie the contagion effect. Participants could be randomly assigned to different social context influences such as direct feedback, modeling, or positive self-disclosure. Identifying more precisely the mechanisms driving the contagion effect would enable researchers to create a more focused, potent, and time efficient intervention.

Although the goal of the present study was to test the hypothesis that cognitive vulnerability could be contagious, the findings also call into question the literature on the contagion of depressive symptoms. Prior research suggests that depression itself might be contagious

between individuals (Joiner & Katz, 1999). However, we did not detect a contagion effect for depressive symptoms. Roommates' baseline levels of depressive symptoms were not significantly associated with the participants' levels of depressive symptoms at either 3 months or 6 months. Rather, the strongest predictors of prospective changes in participants' level of depressive symptoms were (a) their own baseline level of depressive symptoms, (b) their own baseline level of cognitive vulnerability, and (c) their roommates' baseline level of cognitive vulnerability. Thus, the results of this study suggest that the contagion is not depression per se, but rather the cognitive factors that increase risk for depression. Although it is true that contracting higher levels of cognitive vulnerability might then lead individuals to develop more depression, this increase in depression would not be due to their roommate's level of depression, but rather to their roommate's cognitive vulnerability. In addition to this indirect effect via cognitive vulnerability, there are also at least two additional differences between the present study and previous studies that may explain why we did not find a depressive contagion effect. First, we used a more appropriate data analytic strategy for testing dyadic effects (APIM; Kenny, 1996). Prior studies testing depressive contagion effects have relied on traditional statistical techniques, which assume the nonindependence of observations. Because the nonindependence assumption is often violated in studies of close interpersonal relationships, the test statistics in previous studies may have been biased (Cook & Kenny, 2005). Second, we had random assignment of participants to roommates. Prior studies testing depressive contagion effects did not randomly assign participants to dyads, so these studies cannot rule out the influence of participants self-selecting their interpersonal environments in their effects.

It is important to note that the rumination vulnerability factor was less stable over time (and more likely to be contagious) than the hopelessness theory vulnerability factor. The rumination vulnerability factor was contagious at both the 3-month and 6-month time points, whereas the hopelessness theory vulnerability was not. These results are consistent with a study by Hankin (2008), which showed that the hopelessness theory cognitive vulnerability factor was highly stable over time, whereas the rumination vulnerability factor exhibited more moderate stability. One explanation for why the two vulnerabilities exhibit different temporal stabilities is that they focus on different cognitive attributes. The hopelessness theory vulnerability factor focuses on cognitive *content* (i.e., the particular types of interpretations that people generate about stress), whereas response styles theory focuses on the *process* by which people respond to negative moods (i.e., brooding vs. distraction). We suspect that cognitive processes might be more

amenable to change than cognitive content. For example, learning a new process such as distraction (e.g., playing a sport rather than brooding) in response to a negative mood seems more readily attainable for someone to learn than altering his or her cognitive content, which is likely part of a well-organized latent schematic structure. However, the issue of stability remains an empirical question. Future research is needed to determine why the hopelessness vulnerability factor is more stable than the rumination vulnerability.

In conclusion, the goal of this study was to establish the presence of the contagion phenomenon for cognitive vulnerability to depression. Although it is difficult to fathom the idea of people suddenly "catching" a different style of interpreting their world, the current study demonstrates that people's cognitive vulnerability can affect the cognitive vulnerability of those around them, at least when people are going through relatively large-scale changes in their interpersonal environment. Using a natural experimental design with three time points, we found that when people change from a home context to a college context they are susceptible to the influence of the cognitive styles of those around them, even when the others are initially strangers. This effect was shown at both 3 and 6 months for the RRS, and the results cannot be explained by a depression contagion effect or by increased stress. Moreover, the contagion effect has depressive consequences. Those who experienced an increase in cognitive vulnerability had increased risk for future depressive symptoms. These results call for a change in how scientists conceptualize cognitive vulnerability and provide clues for understanding developmental fluctuations in risk and resilience to depression.

Declaration of Conflicting Interests

The authors declared that they had no conflicts of interest with respect to their authorship or the publication of this article.

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. doi:10.1037//0033-295X.96.2.358.
- Alloy, L. B., Abramson, L. Y., Hogan, M. E., Whitehouse, W. G., Rose, D. T., Robinson, M. S., . . . Lapkin, J. B. (2000). The Temple-Wisconsin Cognitive Vulnerability to Depression (CVD) Project: Lifetime history of Axis I psychopathology in individuals at high and low cognitive vulnerability to depression. *Journal of Abnormal Psychology, 109*, 403–418. doi:10.1037/0021-843X.109.3.403

- Alloy, L. B., Abramson, L. Y., Tashman, N. A., Berrebbi, D. S., Hogan, M. E., Whitehouse, W. G., . . . Morocco, A. (2001). Developmental origins of cognitive vulnerability to depression: Parenting, cognitive, and inferential feedback styles of the parents of individuals at high and low cognitive risk for depression. *Cognitive Therapy and Research*, *25*, 397–423. doi:10.1023/A:1005534503148
- 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. doi:10.1037/0021-843X.115.1.145
- Baron, R. M., & Kenny, D. A. (1986). The moderator–mediator variable distinction in social psychological research: Conceptual, strategic and statistical considerations. *Journal of Personality and Social Psychology*, *51*, 1173–1182. doi:10.1037/0022-3514.51.6.1173
- Beck, A. T., Rush, A. J., Shaw, B. F., & Emery, G. (1979). *Cognitive therapy of depression*. New York, NY: Guilford.
- Beck, A. T., Steer, R. A., & Garbin, M. G. (1988). Psychometric properties of the BDI: Twenty-five years of evaluation. *Clinical Psychology Review*, *8*, 77–100. doi:10.1016/0272-7358(88)90050-5
- Bliss, C. A., Klourmann, I. M., Harris, K. D., Danforth, C. M., & Dodds, P. S. (2012). Twitter reciprocal reply networks exhibit assortativity with respect to happiness. *Journal of Computational Science*, *3*, 388–397. doi:10.1016/j.jocs.2012.05.001
- Burns, M. O., & Seligman, M. E. P. (1989). Explanatory style across the life span: Evidence for stability over 52 years. *Journal of Personality and Social Psychology*, *56*, 471–477. doi:10.1037/0022-3514.56.3.471
- Christakis, N. A., & Fowler, J. H. (2013). Social contagion theory: Examining dynamic social networks and human behavior. *Statistics in Medicine*, *32*, 556–577. doi:10.1002/sim.5408
- Clark, D. A., & Beck, A. T. (2010). Cognitive theory and therapy of anxiety and depression: Convergence with neurobiological findings. *Trends in Cognitive Sciences*, *14*, 418–424. doi:10.1016/j.tics.2010.06.007
- Cohen, J., Cohen, P., West, S. G., & Aiken, L. S. (2003). *Applied multiple regression/correlation analysis for behavioral sciences* (3rd ed.). Mahwah, NJ: Erlbaum.
- Cole, D. A., Ciesla, J. A., Dallaire, D. H., Jacquez, F. M., Pineda, A. Q., LaGrange, B., . . . Felton, J. W. (2008). Emergence of attributional style and its relation to depressive symptoms. *Journal of Abnormal Psychology*, *117*, 16–31. doi:10.1037/0021-843X.117.1.16
- Cole, D. A., Jacquez, F. M., & Maschman, T. L. (2001). Social origins of depressive cognitions: A longitudinal study of self-perceived competence in children. *Cognitive Therapy and Research*, *25*, 377–395. doi:10.1023/A:1005582419077
- Cook, W. L., & Kenny, D. A. (2005). The actor-partner interdependence model: A model of bidirectional effects in developmental studies. *International Journal of Behavioral Development*, *29*, 101–109. doi:10.1080/01650250444000405
- DeRubeis, R. J., Evans, M. D., Hollon, S. D., Garvey, M. J., Grove, W. M., & Tuason, V. B. (1990). How does cognitive therapy work? Cognitive change and symptom change in cognitive therapy and pharmacotherapy for depression. *Journal of Consulting and Clinical Psychology*, *58*, 862–869. doi:10.1037/0022-006X.58.6.862
- DeRubeis, R. J., & Hollon, S. D. (1995). Explanatory style in the treatment of depression. In G. M. Buchanan & M. E. P. Seligman (Eds.), *Explanatory style* (pp. 99–111). Hillsdale, NJ: Erlbaum.
- Dobkin, R. D., Allen, L. A., Alloy, L. B., Menza, M., Gara, M. A., & Panzarella, C. (2007). Adaptive inferential feedback partner training for depression: A pilot study. *Cognitive and Behavioral Practice*, *14*, 350–363. doi:10.1016/j.cbpra.2006.09.007
- Doom, J. R., & Haefel, G. J. (2013). Teasing apart the effects of cognition, stress, and depression on health. *American Journal of Health Behavior*, *37*, 610–619.
- Dweck, C. S., Davidson, W., Nelson, S., & Enna, B. (1978). Sex differences in learned helplessness: II. The contingencies of evaluative feedback in the classroom and III. An experimental analysis. *Developmental Psychology*, *14*, 268–276. doi:10.1037/0012-1649.14.3.268
- Facebook Inc. (2012). *Key facts*. Retrieved from <http://newsroom.fb.com/Key-Facts>
- Flege, J. E., Yeni Komshian, G. H., & Liu, F. (1999). Age constraints on second-language acquisition. *Journal of Memory and Language*, *41*, 78–104.
- Garber, J., & Flynn, C. (2001). Predictors of depressive cognitions in young adolescents. *Cognitive Therapy and Research*, *25*, 353–376. doi:10.1023/A:1005530402239
- Gould, E., & Gross, C. G. (2002). Neurogenesis in adult mammals: Some progress and problems. *Journal of Neuroscience*, *22*, 619–623.
- Haefel, G. J. (2010). Cognitive vulnerability to depressive symptoms in college students: A comparison of traditional, weakest-link, and flexibility operationalizations. *Cognitive Therapy and Research*, *34*, 92–98. doi:10.1007/s10608-008-9224-8
- Haefel, G. J., Abramson, L. Y., Brazy, P., Shah, J., Teachman, B., & Nosek, B. (2007). Explicit and implicit cognition: A preliminary test of a dual-process theory of cognitive vulnerability. *Behaviour Research and Therapy*, *45*, 1155–1167. doi:10.1016/j.brat.2006.09.003
- Haefel, G. J., Abramson, L. Y., Voelz, Z. R., Metalsky, G. I., Halberstadt, L., Dykman, B. M., . . . Alloy, L. B. (2005). Negative cognitive styles, dysfunctional attitudes, and the remitted depression paradigm: A search for the elusive cognitive vulnerability to depression factor among remitted depressives. *Emotion*, *5*, 343–348. doi:10.1037/1528-3542.5.3.343
- Haefel, G. J., Eastman, M., & Grigorenko, E. L. (2012). Using a cognitive endophenotype to identify risk genes for depression. *Neuroscience Letters*, *510*, 10–13. doi:10.1016/j.neulet.2011.12.060
- Haefel, G. J., Gibb, B. E., Abramson, L. Y., Alloy, L. B., Metalsky, G. I., Joiner, T., . . . Swendsen, J. (2008). Measuring cognitive vulnerability to depression: Development and validation of the Cognitive Style Questionnaire. *Clinical Psychology Review*, *28*, 824–836. doi:10.1016/j.cpr.2007.12.001
- Haefel, G. J., & Vargas, I. (2011). Resilience to depressive symptoms: The buffering effects of enhancing cognitive style and positive life events. *Journal of Behavior Therapy*

- and *Experimental Psychiatry*, 42, 13–18. doi:10.1016/j.jbtep.2010.09.003
- Hankin, B. L. (2008). Stability of cognitive vulnerabilities to depression: A short-term prospective multiwave study. *Journal of Abnormal Psychology*, 117, 324–333. doi:10.1037/0021-843X.117.2.324
- Hankin, B. L., & Abramson, L. Y. (2002). Measuring cognitive vulnerability to depression in adolescence: Reliability, validity and gender differences. *Journal of Clinical Child and Adolescent Psychology*, 31, 491–504.
- 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. doi:10.1023/B:COTR.0000031805.60529.0d
- Hollon, S. D., Stewart, M. O., & Strunk, D. (2006). Enduring effects for cognitive behavior therapy in the treatment of depression and anxiety. *Annual Review of Psychology*, 57, 285–315. doi:10.1146/annurev.psych.57.102904.190044
- Hollon, S. D., Thase, M. E., & Markowitz, J. C. (2002). Treatment and prevention of depression. *Psychological Science in the Public Interest*, 3, 39–77. doi:10.1111/1529-1006.00008
- Joiner, T. E., Jr., & Katz, J. (1999). Contagion of depressive symptoms and mood: Meta-analytic review and explanations from cognitive, behavioral, and interpersonal viewpoints. *Clinical Psychology: Science and Practice*, 6, 149–164. doi:10.1093/clipsy.6.2.149
- Just, N., Abramson, L. Y., & Alloy, L. B. (2001). Remitted depression studies as tests of the cognitive vulnerability hypotheses of depression onset: A critique of conceptual analysis. *Clinical Psychology Review*, 21, 63–83. doi:10.1016/S0272-7358(99)00035-5
- Kenny, D. A. (1996). Models of nonindependence in dyadic research. *Journal of Social and Personal Relationships*, 13, 279–294. doi:10.1177/0265407596132007
- Lau, J. Y. F., Belli, S. D., Gregory, A. M., Napolitano, M., & Eley, T. C. (2012). The role of children's negative attributions on depressive symptoms: An inherited characteristic or a product of the early environment? *Developmental Science*, 15, 568–578. doi:10.1111/j.1467-7687.2012.01152.x
- Mehl, M. R., & Pennebaker, J. W. (2003). The sounds of social life: A psychometric analysis of students' daily social environments and natural conversations. *Journal of Personality and Social Psychology*, 84, 857–870. doi:10.1037/0022-3514.84.4.857
- Metalsky, G. I., & Joiner, T. E. (1992). Vulnerability to depressive symptomatology: A prospective test of the diathesis-stress and causal mediation components of the hopelessness theory of depression. *Journal of Personality and Social Psychology*, 63, 667–675. doi:10.1037/0022-3514.63.4.667
- Miller, G. (2011). Social scientist wade into the tweet stream. *Science*, 333, 1814–1815. doi:10.1126/science.333.6051.1814
- Murray, L., Woolgar, M., Cooper, P., & Hipwell, A. (2001). Cognitive vulnerability to depression in 5-year-old children of depressed mothers. *Journal of Child Psychology and Psychiatry*, 42, 891–899. doi:10.1111/1469-7610.00785
- Nolen-Hoeksema, S. (1991). Responses to depression and their effects on the duration of depressive episodes. *Journal of Abnormal Psychology*, 100, 569–582.
- Nolen-Hoeksema, S. (2000). The role of rumination in depressive disorders and mixed anxiety/depressive symptoms. *Journal of Abnormal Psychology*, 109, 504–511. doi:10.1037/0021-843X.109.3.504
- Nolen-Hoeksema, S., Girgus, J. S., & Seligman, M. E. P. (1992). Predictors of consequences of childhood depressive symptoms: A 5-year longitudinal study. *Journal of Abnormal Psychology*, 101, 405–422. doi:10.1037/0021-843X.101.3.405
- Nolen-Hoeksema, S., Larson, J., & Grayson, C. (1999). Explaining the gender difference in depressive symptoms. *Journal of Personality and Social Psychology*, 77, 1061–1072. doi:10.1037/0022-3514.77.5.1061
- 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. doi:10.1037/0022-3514.61.1.115
- Nolen-Hoeksema, S., Morrow, J., & Fredrickson, B. L. (1993). Response styles and the duration of episodes of depressed mood. *Journal of Abnormal Psychology*, 102, 20–28. doi:10.1037/0021-843X.102.1.20
- Nolen-Hoeksema, S., Parker, L., & Larson, J. (1994). Ruminative coping with depressed mood following loss. *Journal of Personality and Social Psychology*, 67, 92–104. doi:10.1037/0022-3514.67.1.92
- Nolen-Hoeksema, S. N., Wisco, B. E., & Lyubomirsky, S. (2008). Rethinking rumination. *Perspectives on Psychological Science*, 3, 400–423. doi:10.1111/j.1745-6924.2008.00088.x
- Romens, S. E., Abramson, L. Y., & Alloy, L. B. (2009). High and low cognitive risk for depression: Stability from late adolescence to early adulthood. *Cognitive Therapy and Research*, 33, 480–498. doi:10.1007/s10608-008-9219-5
- Segal, Z. V., Kennedy, S., Gemar, M., Hood, K., Pedersen, R., & Buis, T. (2006). Cognitive reactivity to sad mood provocation and the prediction of depressive relapse. *Archives of General Psychiatry*, 63, 749–755. doi:10.1001/archpsyc.63.7.749
- Seligman, M. E., Schulman, P., DeRubeis, R. J., & Hollon, S. D. (1999). The prevention of depression and anxiety. *Prevention and Treatment*, 2, Article 8, doi:10.1037/1522-3736.2.1.28a
- Tang, T. Z., & DeRubeis, R. J. (1999). Sudden gains and critical sessions in cognitive-behavioral therapy for depression. *Journal of Consulting and Clinical Psychology*, 67, 894–904. doi:10.1037/0022-006X.67.6.894
- Treynor, W., Gonzalez, R., & Nolen-Hoeksema, S. (2003). Rumination reconsidered: A psychometric analysis. *Cognitive Therapy and Research*, 27, 247–259. doi:10.1023/A:1023910315561