

Susan Nolen-Hoeksema

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Thinking About Rumination: The Scholarly Contributions and Intellectual Legacy of Susan Nolen-Hoeksema

Sonja Lyubomirsky,[1] Kristin Layous,[1] Joseph Chancellor,[2] and S. Katherine Nelson[1]

[1]Department of Psychology, University of California, Riverside, California 92521; email: sonja.lyubomirsky@ucr.edu

[2]Department of Experimental Psychology, University of Cambridge, Cambridge, CB1 2PG United Kingdom

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Abstract

Our article reviews and celebrates Susan Nolen-Hoeksema's remarkable contributions to psychological and clinical science, focusing on her vast body of theoretical and empirical work and her influence on colleagues and students. Susan spent her career trying to understand how and why a style of regulating emotions called rumination increases vulnerability to depression and exacerbates and perpetuates negative moods. More broadly, we describe research by Susan and her colleagues on the predictors of depression in childhood and adolescence; gender differences in depression and rumination in adolescence and adulthood; roots, correlates, and adverse consequences of ruminative response styles; and rumination as a transdiagnostic risk factor for not only depression but also a host of psychological disorders, including anxiety, substance abuse, and eating disorders. Susan's intellectual legacy is evident in her impressive publication and citation record, the clinical applications of her work, and the flourishing careers of the students she mentored.

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INTRODUCTION

Psychological and clinical science suffered an enormous loss on January 2, 2013, when Susan Nolen-Hoeksema, the founding editor of the *Annual Review of Clinical Psychology*, died following heart surgery at Yale–New Haven hospital. Susan had been at the apex of her career, chair of her department at Yale, mentor to numerous devoted students, mother, wife, and loyal friend to many. Over her tenure at three distinguished psychology departments—at Stanford University, the University of Michigan, and Yale University—she amassed an extraordinary body of work on how a style of regulating emotions called rumination ("overthinking" for nonscientists) increases vulnerability to depression and exacerbates and perpetuates negative moods. In this article, we describe in detail Susan's and her colleagues' research on the predictors of depression in childhood and adolescence; gender differences in depression and rumination in adolescence and adulthood; the roots, correlates, and adverse consequences of ruminative response styles; and rumination as a transdiagnostic risk factor for not only depression but also a host of psychological disorders, including anxiety, substance abuse, and eating disorders.

The arc of Susan's life's work was deep and broad, yet also decidedly programmatic. In addition to the core themes of gender differences, depression, rumination, and emotion regulation, she explored a variety of other questions that are beyond the scope of this article—for example, coping with loss and childhood sexual abuse, gender differences in achievement settings, effects of parental styles on helplessness in children, and predictors of suicidality and self-harm—and was passionate about applying her work to underrepresented groups vis-à-vis ethnicity, race, and sexual orientation.

Susan was a first-class thinker—lucid, elegant, wise, and methodical. For her students, she had an uncanny ability to give direction and freedom at the same time—she challenged and energized them to work harder and to think in a more rigorous and sophisticated fashion. Her approach to science was to conduct careful studies and experiments and eliminate hypotheses one by one. Not surprisingly, her work inspired many new lines of inquiry and application from her former advisees and colleagues to new investigators who never knew her, as well as clinicians, practitioners, and laypeople.

NOLEN-HOEKSEMA'S EARLY WORK: PREDICTORS OF DEPRESSION IN CHILDREN AND ADOLESCENTS

As a graduate student, Susan's research focused primarily on understanding the predictors of depression among children and adolescents. Together with her doctoral advisor, Martin Seligman, she conducted rigorous longitudinal studies to explore the role of learned helplessness, pessimistic explanatory style, and adverse life events in the development of depression.

Learned helplessness, pessimistic explanatory style, and negative life events are interrelated: Learned helplessness and pessimistic explanatory style are both ways that people respond to negative life events, and a pessimistic explanatory style involves interpreting the causes of negative events as being stable across time, global in effect, and internal to the individual. For example, a child who performs poorly on a spelling test might interpret his bad grade as being due to his own incompetence (internal), indicating that he will never be a good speller (stable), and suggesting that he will perform poorly in other subjects as well (global). Learned helplessness develops after people have had repeated experiences with uncontrollable bad events, leading them to believe that they have little or no control over their future outcomes. These expectations of low control lead to myriad deficits in motivation, cognition, and emotion. For example, after experiencing the death of a loved one and the divorce of her parents, a child may generalize her sense of lacking control to her schoolwork and subsequently withdraw efforts to work on difficult assignments and demonstrate less persistence at those assignments (i.e., motivational deficits); she may fail to see that asking her teacher for help would give her an opportunity to improve and therefore gain control over her outcomes (i.e., cognitive deficits); and she may experience feelings of sadness and low self-esteem (i.e., emotional deficits).

To examine the interplay among life events, explanatory style, and depression, Susan assessed school-age children five times during the course of one year (Nolen-Hoeksema et al. 1986). This research revealed a reciprocal relationship between explanatory style and depression over time, such that a pessimistic explanatory style at one time point predicted increases in depression, and depression predicted increases in pessimistic explanatory style. In addition, explanatory style interacted with negative life events in this study to predict increases in depressive symptoms (Nolen-Hoeksema et al. 1986). This latter finding indicates that experiencing negative life events is associated with increases in depressive symptoms only when children interpret those events as being internal, global, and stable.

In another study (Nolen-Hoeksema et al. 1992), Susan assessed children's explanatory style, helplessness behaviors, negative life events, and depression every six months for five years (beginning when the children were in third grade) to understand developmental changes in the predictors of depression. This study revealed that among younger children (i.e., third- and fourth-graders), negative life events, but not the explanatory style that children used to interpret these events, predicted later depressive symptoms. As the children grew older, however, explanatory style, and the interaction between explanatory style and negative life events, predicted increases in depression. In

addition, at both ages, children who were more depressed at one time point also became more pessimistic and more helpless in achievement and social situations six months later (Nolen-Hoeksema et al. 1992). Consistent with these latter findings, other work indicates that depressive symptoms are also associated with an increased likelihood of experiencing negative life events (Cole et al. 2006). Together, these studies suggest that depressive episodes may generate a downward spiral, whereby individuals experiencing depressive episodes ultimately experience more negative life events and adopt a more pessimistic explanatory style, which further exacerbates depression.

Finally, Susan extended her work on explanatory style to other outcomes beyond depression, such as academic achievement (Nolen-Hoeksema et al. 1986) and athletic performance (Seligman et al. 1990). For example, in one study, varsity swimmers with pessimistic explanatory styles had more unexpectedly poor performances than those with optimistic styles. Moreover, pessimistic swimmers also showed relatively greater difficulty in recovering from a poor performance, suggesting one possible mechanism by which a pessimistic explanatory style impacts athletic performance (Seligman et al. 1990).

EMERGENCE OF GENDER DIFFERENCES IN DEPRESSION IN EARLY ADOLESCENCE

In her investigations of the predictors of depression in children and adolescents, Susan began exploring gender differences in depression. Studies she conducted in graduate school revealed that among children, boys demonstrate higher rates of depressive symptoms than girls, in part due to boys' greater reliance on pessimistic explanatory styles (Nolen-Hoeksema et al. 1991). Interestingly, the pattern of gender differences in depression reverses by the time children reach adolescence—teenage girls demonstrate higher rates of depression than teenage boys (Hilt et al. 2010, Twenge & Nolen-Hoeksema 2002).

Susan took a rigorous theoretical approach to explain why female adolescents showed relatively greater rates of depression, including the systematic elimination of alternative hypotheses (Nolen-Hoeksema & Girgus 1994). First, her research was beginning to establish that ruminative responses to negative mood (i.e., perseverative thoughts about the symptoms of depression) exacerbate depressive episodes (Nolen-Hoeksema 1987; see Response Styles Theory section below). These findings suggested that one potential explanation for the emergence of gender differences in depression is that the determinants of depression (e.g., rumination, pessimistic explanatory style) are the same among young boys and girls, but that these determinants become more prevalent among girls when they enter adolescence. This theory, however, was undermined by evidence that girls demonstrate higher rates of risk factors for depression than boys prior to entering adolescence, before adolescent girls' higher rates of depression have actually emerged.

Second, Susan considered that the causes of depression might differ between boys and girls (e.g., boys may become depressed due to pessimistic explanatory style, whereas girls may become depressed due to incessant rumination) and that in early adolescence, the causes of girls' depression become more prevalent than the causes of boys' depression. She eliminated this theory, however, on account of evidence that the predictors of depression are similar among boys and girls. For example, ruminative responses to depressed mood and pessimistic explanatory styles similarly predict depression among both boys and girls (Nolen-Hoeksema et al. 1991; cf. Nolen-Hoeksema et al. 1993).

Finally, she considered that girls are more likely than boys to carry risk factors (e.g., rumination) for depression even before early adolescence, but these risk factors lead to depression only in the face of challenges (e.g., academic and social pressure at school) that increase in prevalence in early

adolescence. Indeed, substantial evidence supports this third model of the emergence of gender differences in depression (Nolen-Hoeksema 1994).

Girls confront greater challenges than boys do as they enter early adolescence: Girls are more likely to be sexually abused; their physical pubertal changes (e.g., increases in body fat in girls versus increases in muscle tone in boys) are viewed more negatively by society (and by the girls themselves); and they are met with relatively greater expectations by parents and peers to conform to gender roles. In turn, evidence suggests that when these challenges are coupled with girls' tendencies to dwell on their emotions, girls are more likely to experience depressive episodes (Hilt et al. 2010, Nolen-Hoeksema 1994, Nolen-Hoeksema & Girgus 1994). Supporting these findings, a meta-analysis indicated that girls were more likely to ruminate than boys, and, in turn, rumination predicted more depressive symptoms over time among both children and adolescents (Rood et al. 2009).

In sum, the research that Susan initiated early in her career created a strong foundation for her scientific approach to investigating mental illness. For example, her interest in gender differences in depressive episodes and symptoms was not only a theme in her early work but also one that permeated her entire career, as she made several major contributions to the psychological understanding of gender (e.g., Nolen-Hoeksema 2010). More important, her efforts to explain the gender differences in depression steered her to the observation that girls and women ruminate more on their problems and symptoms and motivated more than two decades of research on rumination.

GENDER DIFFERENCES IN RUMINATION AND DEPRESSION IN ADULTS

Building on her work examining gender differences in depression among children and adolescents, Susan developed an extensive program of research to understand why, beginning in adolescence and persisting throughout the life span, women are twice as likely to experience depression as men (Nolen-Hoeksema 1987, 2001). Indeed, the gender differences in depression are striking: Women experience the onset of depression more frequently than do men, they demonstrate relatively longer durations of depressed moods, and they are relatively more likely to experience a recurrence of depressed mood (Nolen-Hoeksema 1987). Accordingly, women are 70% more likely to experience depression in their lifetimes than are men (Kessler et al. 2003).

As with her approach to understanding the emergence of gender differences in childhood and adolescence, Susan systematically considered numerous lay and now esoteric explanations for the gender differences in depression among adults (Nolen-Hoeksema 1987), and finding supportive evidence for these explanations lacking, ultimately developed her own theory to fill the explanatory void. First, she considered several methodological explanations—namely, that women hold a lower socioeconomic status than men, that women are more willing to report and seek help for depressive symptoms, and that women's depressive symptoms take a different form than men's (e.g., sadness and crying versus alcoholism and acting out). These artifactual explanations, however, were challenged by findings that (*a*) women report greater depressive symptoms than men even after controlling for socioeconomic status (Radloff 1975); (*b*) men are no less willing to disclose their symptoms of depression in public than in private (King & Buchwald 1982) and are equally likely to seek help for depressive symptoms when they experience them at similar levels as women (Amenson & Lewinsohn 1981); and (*c*) for both genders, the onset of depression typically occurs at least 10 years after the onset of alcoholism (Cadoret & Winokur 1974), suggesting that alcoholism may be a cause but not a symptom of depression in men.

Second, Susan considered biological explanations for women's greater prevalence of depression—namely, hormonal fluctuations and genetic factors (Nolen-Hoeksema 1987). She ruled out these explanations due to little evidence of increased rates of depression during periods of hormonal imbalance and a lack of consistent evidence that women have a greater genetic predisposition for depression than men have.

A third account of women's higher rates of depression evokes psychoanalytic explanations of female psychosexual development. According to classic psychoanalytic theory, young girls' realization that they are deprived of many privileges held by males leads them to experience hostility toward other women and envy toward men, as well as to experience low feelings of self-worth (Mitchell 1974). Despite these theoretical suggestions, virtually no empirical evidence has supported psychoanalytic explanations of gender differences in depression.

Fourth, sex role theories suggest several possible explanations for the greater prevalence of depression among women. For example, one hypothesis involves women's tendencies to overvalue interpersonal relationships. Yet research indicates that social relationships are actually protective against depression and positively associated with well-being. A second hypothesis evokes a role conflict account—namely, because of their tendency to focus on relationships, women will experience more conflict when they enter the workforce, where they are expected to express more masculine behaviors. Counter to this hypothesis, studies find that working women experience fewer depressive symptoms than nonworking women (Radloff 1975).

Finally, learned helplessness theory has been used to explain women's higher rates of depression. Accordingly, when people have the expectation that they cannot control outcomes, they exhibit learned helplessness, a set of motivational, cognitive, and affective deficits that have been associated with increases in depressive symptoms (Seligman 1975). However, despite this link to depression, studies of learned helplessness and depression had been primarily conducted with samples (e.g., college students) with no observable gender differences in depression. As a result, those investigations could not test whether learned helplessness explained the greater prevalence of depression among women.

After reviewing the available evidence for each of these potential explanations for the gender differences in depression, Susan concluded that none of them were strongly supported. Instead, she proposed an alternative—namely, that the gender differences can best be explained by women's greater reliance on a ruminative style of coping with depressed mood (Nolen-Hoeksema 1987). In other words, when women experience negative moods, they tend to dwell on those bad moods, their causes, and their implications. Men, by contrast, tend to engage in activities to distract themselves from their negative moods. In turn, women's style of coping exacerbates their depressed moods, whereas men's style of coping diminishes their depressed moods (Nolen-Hoeksema et al. 1993).

The development of this hypothesis led Susan to launch a series of investigations of gender differences in rumination and depression. After demonstrating that a ruminative response style explains the gender differences in depression (Nolen-Hoeksema 1987), she shifted her focus to address the question, "Why do women ruminate more than men?" One series of investigations demonstrated that part of the answer is that they choose to. In these studies, men and women were induced into a sad mood and then offered the option to engage in an emotion-related task or a non-emotion-related task. In one study, 92% of the women chose the emotion-related task, whereas only 46% of the men did (Butler & Nolen-Hoeksema 1994), and, in another study, adolescent girls engaged in more self-focused thoughts than adolescent boys did, even when they were not in a depressed mood (Sethi & Nolen-Hoeksema 1997).

Additional research investigated the roles of social status, perceived ability to control emotions, and chronic strain as predictors of gender differences in rumination and depression. For example, women's subordinate social status and the presence of uncontrollable stressors may teach them

that any actions they would take to improve their situation would be unsuccessful (e.g., Watkins & Nolen-Hoeksema 2014). As a result, women are more inclined to focus on their emotions in the context of stressful situations (Nolen-Hoeksema & Jackson 2001). In addition, the experience of uncontrollable stressors and the belief that negative emotions cannot be managed may perpetuate rumination and depression. Indeed, in one investigation, women were more likely than men to experience chronic strain, low mastery, and rumination, and, in turn, these characteristics explained the gender differences in depression (Nolen-Hoeksema et al. 1999). Moreover, chronic strain and rumination demonstrated reciprocal relationships with one another over time, and depressive symptoms contributed to more rumination and less mastery over time, suggesting that the interplay among these variables can create a downward spiral.

In her work on gender differences in rumination and depression, Susan emphasized that rumination does not have a stronger impact on depressive episodes experienced by women than men, but that because women engage in rumination more frequently, they are more likely to experience depressive episodes in the first place. Indeed, as previously mentioned, rumination similarly predicts depression among males and females (Rood et al. 2009). Accordingly, much of Susan's work later in her career focused on the harmful effects of rumination among both men and women across a wide range of outcomes. Moreover, her influence on the psychological understanding of gender is apparent across many domains. In addition to exploring gender differences in rumination and depression, she found that women use a wider range of emotion-regulation strategies than do men (Nolen-Hoeksema 2012), that women are relatively more influenced by evaluative feedback (Roberts & Nolen-Hoeksema 1989, 1994), and that men are relatively more likely to abuse alcohol to cope with their negative emotions, in part because men are more likely to carry risk factors for alcohol abuse (e.g., impulsivity) and to have fewer protective factors (Nolen-Hoeksema & Harrell 2002, Nolen-Hoeksema & Hilt 2006).

RESPONSE STYLES THEORY

Susan devoted the better part of her life's work to understanding the relationship between rumination and depression—specifically, how rumination can exacerbate negative moods, triggering and prolonging depressive episodes (Nolen-Hoeksema 1991, Nolen-Hoeksema et al. 2008). According to her response styles theory (RST), rumination is a maladaptive pattern of responding to distress by repetitively and passively focusing on the meanings, causes, and consequences of one's depressive symptoms rather than actively working toward a solution to resolve the circumstances surrounding these symptoms (Nolen-Hoeksema 1991, Nolen-Hoeksema et al. 2008). Susan's focus on rumination as a robust predictor of the onset and duration of depression attracted other scientists to advance research in this area, resulting in today's extensive body of literature on rumination, its outcomes and correlates, and its role in depression.

Notably, a wealth of data has shown that rumination is associated with a host of negative outcomes. For example, rumination is linked to maladaptive cognitive styles, such as negative attributional styles, hopelessness, pessimism, self-criticism, neediness, sociotropy (i.e., excessive investment in relationships), and neuroticism (Ciesla & Roberts 2002, Flett et al. 2002, Lam et al. 2003, Lyubomirsky & Nolen-Hoeksema 1995, Lyubomirsky et al. 1999, Nolen-Hoeksema & Davis 1999, Nolen-Hoeksema & Jackson 2001, Nolen-Hoeksema et al. 1999, Robinson & Alloy 2003, Spasojevic & Alloy 2001), and predicts depression above and beyond these negative cognitive styles (Flett et al. 2002, Nolen-Hoeksema et al. 1994, Spasojevic & Alloy 2001). Importantly, although negative thoughts arise during rumination, rumination is not defined simply by the content of thoughts, but rather by their repetitive and passive nature (Nolen-Hoeksema 1991).

Rumination is also correlated with harmful behaviors and behavioral intentions, such as nonsuicidal self-injury (Hilt et al. 2008), recklessness (i.e., driving dangerously or taking recreational drugs; Nolen-Hoeksema & Morrow 1991), and delay in seeking diagnosis when presented with adverse health symptoms (Lyubomirsky et al. 2006a). Furthermore, rumination is associated with potentially detrimental emotion-regulation strategies, such as attempts at thought suppression (Wenzlaff & Luxton 2003) that are typically unsuccessful (Joorman 2004) and avoidance of adaptive interpersonal and intrapersonal efforts (e.g., attending social gatherings or making decisions about the future; Moulds et al. 2007) (see also Emotion Regulation and Psychopathology section below).

As her work on rumination matured, Susan brought further conceptual clarity to rumination to differentiate it from related constructs such as worry. For example, although rumination is related to worry (another type of repetitive thought; Fresco et al. 2002, Watkins 2004), the two are distinct (Fresco et al. 2002, Watkins et al. 2005). As Susan cogently explained, although worry and rumination are typically correlated, they differ in their time orientation (i.e., rumination is focused on the past; worry is focused on the future), topic focus (i.e., rumination is focused on themes of loss, meaning, and lack of self-worth; worry is focused on anticipated threats), degree of certainty and controllability (i.e., rumination involves viewing events as certain and uncontrollable; worry involves viewing events as uncertain and potentially controllable), and conscious motives (i.e., ruminators seek to gain insight; worriers seek to anticipate and prepare for threat) (Nolen-Hoeksema et al. 2008).

Similarly, Susan and her colleagues responded to critiques of their primary measure of rumination (the Ruminative Responses Scale; Nolen-Hoeksema & Morrow 1991) by eliminating items that overlapped with depression inventories and revealing two subtypes of rumination: brooding and pondering (Treynor et al. 2003). These subtypes mapped on to previous distinctions in the literature between rumination and its less nefarious counterpart in self-focus, intellectual self-reflection (Trapnell & Campbell 1999). Brooding is characterized by a focus on obstacles and abstract, passive thoughts such as "Why can't I handle things better?" whereas pondering is characterized by more general self-reflective tendencies, such as writing down and analyzing one's feelings to problem solve. Brooding is related to depression in both the short term and long term, whereas pondering is related to depression only in the short term, possibly because pondering facilitates effective problem solving (Treynor et al. 2003). Susan's adeptness at continuously elucidating the construct of rumination and RST in light of emerging research innovations helped the scientific conversation about rumination to evolve and stay relevant.

Susan also inspired other researchers to further fine-tune the family of constructs that includes rumination, general self-focus, and nonmaladaptive forms of repetitive thought. As one example, a meta-analysis of 226 effect sizes found that general self-focus was related to negative affect, but this was especially so when the style of thought was ruminative (i.e., repetitive and passive; Mor & Winquist 2002). In another study, investigators distinguished between repetitive thought focused on negative and positive content and found that repetitive thought was only maladaptive to the extent that it focused on negative material (Segerstrom et al. 2003; see also Lyubomirsky et al. 2006b). In addition, researchers have also differentiated between abstract and concrete rumination, the former focused on abstract, evaluative thoughts about one's emotions and current circumstances (like brooding) and the latter involving a nonevaluative awareness of present experiences (also called mindful experiencing/being; Watkins 2008).

Rumination Maintains and Exacerbates Depression

Susan has frequently been praised for her creativity and rigor in designing experiments to test her theories. Her skill as a methodologist is evident in the body of empirical work supporting the

main tenet of RST—that rumination maintains and exacerbates depression by enhancing negative mood-congruent thinking, impairing problem solving and instrumental behavior, and deterring social support (Nolen-Hoeksema 1991, Nolen-Hoeksema et al. 2008).

Susan's approach was to employ a powerful quasi-experimental 2 × 2 design in which dysphoric and nondysphoric participants are randomly assigned to engage in laboratory rumination or distraction tasks, a design originally developed in collaboration with her graduate student Jannay Morrow (Nolen-Hoeksema & Morrow 1993). For the rumination task, participants were prompted to fixate on the meanings, causes, and consequences of their current feelings for eight minutes (e.g., "Think about the level of motivation you feel right now," "Think about the long-term goals you have set," "Think about what your feelings might mean"). Although these items have previously been coded as neutral and are seemingly innocuous, such instructions send dysphoric individuals into an emotional tailspin, as Susan suspected. By contrast, participants assigned to the distraction task were asked to focus on non-self-relevant neutral topics (e.g., "Visualize the layout of the post office," "Think about a boat slowly crossing the Atlantic," "Visualize clouds forming in the sky"). Thus, Susan was able to compare the effects of rumination (versus distraction) among people experiencing negative moods (dysphoric people) and those who are not experiencing negative moods (nondysphoric people) to test whether rumination prolongs and worsens depressive symptoms as well as other adverse outcomes.

Rumination enhances the effect of depressed mood on negative thinking. Susan's prediction that rumination can promote negative thoughts among people in negative moods has been borne out by copious empirical evidence. For example, dysphoric participants induced to ruminate recalled more negative autobiographical memories and reported more frequent negative life events than did either dysphoric participants induced to distract themselves or nondysphoric participants (Lyubomirsky et al. 1998; see also McFarland & Buehler 1998, Pyszczynski et al. 1989). In another set of experiments, dysphoric ruminators spontaneously brought up personal problems such as financial or interpersonal woes, used a negative tone, and engaged in self-blame and self-criticism relatively more than their distracted-dysphoric and nondysphoric counterparts (Lyubomirsky et al. 1999). Furthermore, dysphoric ruminators (versus distracted-dysphoric or nondysphoric participants) interpreted even hypothetical negative life events through a more negatively biased lens (e.g., overgeneralizing from failures) (Lyubomirsky & Nolen-Hoeksema 1995; see also Greenberg et al. 1992) and reported lower expectations for ostensibly fun, positive events (Lyubomirsky & Nolen-Hoeksema 1993, 1995). In sum, rumination prompts people who are in a negative mood to have negative thoughts, which reinforces their negative mood.

Rumination interferes with effective problem solving and instrumental behavior. Although ruminators report that they think about their problems in an effort to solve them (Papageorgiou & Wells 2001, 2003), evidence strongly suggests that rumination actually impedes problem solving. Dysphoric participants assigned to ruminate deemed their problems relatively more overwhelming and unsolvable (Lyubomirsky et al. 1999) and came up with less-effective solutions (Lyubomirsky & Nolen-Hoeksema 1995, Lyubomirsky et al. 1999) than either dysphoric participants who distracted themselves or nondysphoric participants. Moreover, even when dysphoric ruminators generated a potentially efficacious solution, they were less confident that it would actually work (Ward et al. 2003) and reported lower intentions to implement it (Lyubomirsky et al. 1999). The real-world implications of rumination were highlighted in a laboratory experiment focused on a common intellectual and work-related activity—namely, reading comprehension. After receiving negative feedback on an academic task, dysphoric students assigned to ruminate took longer in a subsequent reading assignment and remembered less of what they had read

than distracted-dysphoric or nondysphoric students (Lyubomirsky et al. 2011). Thus, dysphoric ruminators' emotional debris hindered their ability to bounce back and concentrate after a failure.

Evidence also suggests that even when ruminators are aware of a solution, they lack the motivation and initiative to carry it out. In one example, even though dysphoric ruminators acknowledged that a distracting activity might boost their mood, they did not wish to try it (Lyubomirsky & Nolen-Hoeksema 1993; see also Wenzlaff et al. 1988). As another example, women with chronic ruminative styles were more likely to delay seeking a diagnosis after discovering a breast lump (Lyubomirsky et al. 2006a). Similarly, correlational evidence demonstrates that a tendency toward rumination is related to lower adherence to treatment among cancer patients (Aymanns et al. 1995). Taken together, these findings indicate that ruminators seem to have an impaired cognitive capacity to generate solutions to their problems and reduced motivation to implement solutions that could improve their mental or physical health.

Rumination reduces social support. On top of its other adverse outcomes, rumination may also be isolating, because ruminators shun social interaction in favor of thinking or writing perseveratively about their feelings or repel others by repeatedly and perhaps annoyingly sharing how bad they feel. Indeed, rumination has been linked with a variety of undesirable interpersonal characteristics, such as dependency (Spasojevic & Alloy 2001), neediness (Spasojevic & Alloy 2001), sociotropy (Gorski & Young 2002, Nolen-Hoeksema & Jackson 2001), revenge seeking (McCullough et al. 2001), and aggression following provocation (Collins & Bell 1997), that could render ruminators unwelcome company. A study of bereaved adults showed that although ruminators reached out for social support more often than nonruminators, they were less likely to report actually receiving emotional support and more likely to report relational friction and feeling isolated from support providers (Nolen-Hoeksema & Davis 1999). Thus, ruminators believed that they were asking for the support they desperately needed but not receiving it.

A recent longitudinal study of adolescents also demonstrated that baseline ruminative tendencies predict self-reported relational victimization (i.e., social exclusion) and reputational victimization (i.e., being the target of rumors) at a seven-month follow-up (McLaughlin & Nolen-Hoeksema 2012). Furthermore, incidences of peer victimization mediated the relationship between baseline rumination and follow-up internalizing symptoms (i.e., depression and anxiety), indicating that interpersonal stress can explain the link between rumination and clinical symptomatology.

The Effect of Rumination on Onset Versus Duration of Depression

Although considerable evidence links rumination to the onset of clinical depression (e.g., Abela & Hankin 2011, Just & Alloy 1997, Nolen-Hoeksema 2000), the evidence is mixed on the main hypothesis of RST—that is, whether rumination also predicts the duration of depressive episodes. Some studies suggest that it does (e.g., Kuehner & Weber 1999), and others demonstrate that it does not (Just & Alloy 1997, Nolen-Hoeksema 2000). Further evidence indicates that an interaction between rumination and negative cognitive styles predicts the duration of a depressive episode, but rumination alone does not (Ciesla & Roberts 2002, Robinson & Alloy 2003). Therefore, passive and repetitive thoughts predict the duration of depressive episodes only to the extent that they are negatively biased and irrational.

Although research on the effect of rumination on the duration of clinical depressive episodes is conflicting, evidence regarding the effect of rumination on the duration of subclinical depressed mood is much more consistent. For example, participants induced into a depressed mood and then randomly assigned to ruminate maintained their depressed mood throughout the study,

whereas participants who distracted themselves became even less depressed than they were before the mood induction (Morrow & Nolen-Hoeksema 1990; see also Nolen-Hoeksema & Morrow 1993). Similarly, one study asked participants to keep track of their moods and responses to their moods for 30 consecutive days and found that the more ruminative responses participants engaged in, the longer their depressed moods lasted, even after controlling for initial severity of the mood (Nolen-Hoeksema et al. 1993). In addition, in a nonclinical sample of college students, those who exhibited elevated levels of depression and a ruminative response style 2 weeks before the occurrence of a natural disaster (the Loma Prieta earthquake) were relatively more likely to report depressive symptoms both 10 days and 7 weeks after the disaster (Nolen-Hoeksema & Morrow 1991). Therefore, evidence suggests that a ruminative style can prolong depressed moods and depressive symptoms in nonclinical samples.

Susan and her colleagues (2008) offered several ideas for why rumination might prolong a depressed mood but not clinically diagnosed depressive episodes. One explanation involves a potential restriction of range in rumination among individuals meeting criteria for major depression. Indeed, rumination and depression are highly correlated, so samples of depressed people might be relatively homogenous for rumination. A second explanation is that rumination might push people from dysphoria into a major depressive episode (i.e., rumination predicts onset of depression), but once people are in the midst of such an episode, other processes emerge to control its duration. By elegantly integrating the mixed literature, this theoretically driven account highlights how Susan continued to reanalyze and fine-tune her ideas in light of new empirical findings.

Rumination as a Mental Habit

Most recently, Susan and her colleague Ed Watkins conceptualized rumination as a mental habit (Watkins & Nolen-Hoeksema 2014), a framing that alleviates some of the previous tension between characterizing repetitive thought as arising from a particular discrepancy between one's desired and actual situation (i.e., as a temporary state; Martin & Tesser 1989, 1996) versus as a chronic tendency (i.e., as a trait; Nolen-Hoeksema 1991). Habits are defined as "learned dispositions to repeat past performance" (Wood & Neal 2007, p. 843), so conceiving rumination as a mental habit does not diverge far from conceiving it as a trait. However, Watkins and Nolen-Hoeksema also drew on the habit–goal literature for this characterization, thereby suggesting the process by which repetitive thought elicited by goal discrepancies can become maladaptive. That is, state rumination can evolve from a benign, isolated incident to a learned pattern of associations (i.e., a habit) if negative mood and repetitive thinking are paired often over time (Watkins & Nolen-Hoeksema 2014).

According to control theory (Martin & Tesser 1989, 1996), state rumination is triggered by perceived discrepancies between people's goals and their present reality (i.e., wanting to buy a home but barely making ends meet), and ruminative states persist until individuals resolve such discrepancies or abandon the goal. Indeed, unattained personally valued goals predict repetitive thought, but evidence indicates that these thoughts can be either constructive or unconstructive, depending on the context (Watkins 2008). Specifically, Watkins (2008) suggested that state rumination is unproductive if one's thoughts are passive (i.e., not action or solution oriented), negative, and abstract (i.e., focused on the meaning and implications of the goal discrepancies versus the concrete details). Watkins & Nolen-Hoeksema (2014) extended this argument to hypothesize that pathological depressive rumination might result when people consistently use this pattern of passive, negative, and abstract repetitive thought to cope with goal discrepancies. Furthermore, because goal discrepancies and negative mood often co-occur, over time, negative mood might trigger a ruminative response even in the absence of a goal discrepancy.

ORIGINS OF RUMINATION

Framing rumination as a mental habit suggests a possible process by which past associations of negative emotions and repetitive thought might instill a chronic tendency to ruminate. This framework allows for many different developmental triggers of rumination. Susan explored two examples—learned passivity and stressful life events—in her own work, and others she later conceptualized as "distal risk factors" (see Broadening Transdiagnostic Models section below).

Learned Passivity

Watkins & Nolen-Hoeksema (2014) propose that parents might teach their children patterns of passivity that can lead to habitual rumination. Indeed, 5- to 7-year-old children of overcontrolling mothers (i.e., mothers who tell their children what to do and do not encourage them to try different approaches to problem solving) were relatively more helpless and passive when faced with a frustrating situation (Nolen-Hoeksema et al. 1995). Similarly, college students who retrospectively report that their parents were controlling exhibit elevated levels of rumination (Spasojevic & Alloy 2002). Learned passivity might be more common in women owing to differences in the socialization of gender role identities (i.e., girls are encouraged to focus on their emotions and be passive), which could explain the consistent finding that girls and women engage in rumination more frequently than boys and men (Nolen-Hoeksema et al. 1999). One study found that regardless of gender, 9- to 12-year-old children who strongly identified with feminine stereotypes reported more trait rumination (Broderick & Korteland 2004), thus implying that the socialization of girls and women and not their actual gender promotes ruminative tendencies.

Stressful Life Events

Decisive evidence demonstrates that significant stressful life events can trigger rumination and exacerbate its effects. In one example, exposure to stress, such as divorce and serious illness, predicted rumination, which in turn predicted depression and anxiety one year later (Michl et al. 2013). A second study mirrored these results in adolescents, but rumination mediated only the relationship between stressful life events (e.g., getting suspended from school or parents getting divorced) and anxiety, not depression (Michl et al. 2013).

Further evidence of the detrimental effects of rumination after negative life events comes from studies exploring people's adjustment after trauma (e.g., a natural disaster or sexual abuse) (Nolen-Hoeksema & Morrow 1991, Sarin & Nolen-Hoeksema 2010) and bereavement (Nolen-Hoeksema & Larson 1999; Nolen-Hoeksema et al. 1994, 1997). For example, controlling for baseline rumination, students who ruminated more frequently in the ten days following a nearby natural disaster (i.e., the 1989 Loma Prieta earthquake) also reported relatively elevated levels of depression at the seven-week follow-up (Nolen-Hoeksema & Morrow 1991). Thus, even controlling for habitual tendencies to ruminate, the act of rumination following a trauma predicts later depressive symptoms. Additionally, people who report relatively greater rumination following bereavement also show relatively higher depression at follow-up six months or one year later (Nolen-Hoeksema et al. 1994, 1997). In sum, research demonstrates that stress can trigger rumination and that rumination after stressful life events predicts detrimental mental health outcomes.

THE TRANSDIAGNOSTIC RISK FACTOR FRAMEWORK

For three decades, treatment and research on mental illness have revolved around clusters of symptoms codified as disorders in the *Diagnostic and Statistical Manual of Mental Disorders* (DSM;

e.g., Am. Psychiatr. Assoc. 2000). Although the widespread adoption of the DSM standardized the diagnosis of disorders for research, its success led to an overreliance on frequently revised and overlapping classifications—an approach that may have impeded further discovery of the "true" causes of mental illness (Seligman 2014). Susan was an early adopter of a new search for unifying processes that cut across multiple disorders (i.e., transdiagnostic processes) to explain a variety of distressing psychological symptoms—a step that she was uniquely poised to take given the suitability of rumination (which she had linked to numerous psychological disorders) as a unifying core process in psychopathology.

Broadening Transdiagnostic Models

Susan and Ed (Nolen-Hoeksema & Watkins 2011) developed a heuristic to expand the explanatory power of transdiagnostic models in accounting for why the same risk factor may be implicated in multiple disorders (i.e., multifinality) and simultaneously why individuals with the same risk factor eventually succumb to different disorders (i.e., divergent trajectories). In other words, they offered organizing principles to clarify how individuals' trajectories can be both functionally similar (i.e., the same risk factor is linked to multiple disorders) and yet terminally different (i.e., individuals take one path but not another).

To aid in the formulation of these expansive models, Susan and Ed divided known risk factors into two temporal categories: distal and proximal. Distal risk factors appear early in the causal chain of psychological impairment and act indirectly on present-day psychological distress; thus, they are difficult to modify or temper. Distal risk factors can include childhood trauma, neglectful parenting, or genetic susceptibilities to mental illness. Proximal risk factors, however, appear close in time to (if not concurrently with) present-day distress, directly cause psychological impairment, and may be controllable or modifiable. Proximal risk factors might include a dysregulated stress response, a deficit in working memory, or a personality trait such as neuroticism. Temporally separating risk factors aids theorists in delineating specific mechanisms by which distal risk factors lead to proximal risk factors (e.g., a genetic abnormality causes deficits in attention and working memory) and proximal risk factors lead to disorders (e.g., deficits in attention and working memory lead to psychotic symptoms).

Their heuristic also identified moderators that could explain individual differences in psychopathology by interacting with proximal risk factors to determine the particular disorders an individual will actually develop. A moderator of a proximal risk factor could be a chronically threatening environment (e.g., a high-crime neighborhood) or the presence of maladaptive role models (e.g., an older sibling who is a gang member). Moderators are thus crucial in investigating and understanding individuals' divergent trajectories. For example, an individual with dysregulated emotions and alcoholic parents would likely develop an alcohol abuse disorder, whereas another person with dysregulated emotions and whose parents died unexpectedly might develop depression.

By constructing a model that includes distal risk factors, proximal risk factors, and moderators, theorists can uncover gaps in existing knowledge and formulate new avenues of investigation. In essence, the work of Susan and Ed on transdiagnostic models bridges a modern focus on psychopathology's underlying mechanisms with decades of prior disorder-specific knowledge.

Rumination as a Risk Factor for Mood, Substance Abuse, and Eating Disorders

Susan and Ed offered rumination as an exemplar of the explanatory power of a proximal risk factor within a broad transdiagnostic model (Nolen-Hoeksema & Watkins 2011). As previously discussed, Susan and her colleagues' research had already shed light on the roots and developmental

triggers of rumination (i.e., the distal risk factors; see Origins of Rumination section above). The tendency to ruminate has been linked to disorders such as depression, anxiety, substance abuse, and eating disorders as well as moderators (e.g., social stress) that may increase the likelihood of developing one disorder over another.

Depression. Rumination exacerbates depression, and the mechanisms by which it does so have been exhaustively investigated by Susan, her collaborators, and other researchers (for reviews, see Lyubomirsky & Tkach 2004, Nolen-Hoeksema et al. 2008, Watkins 2008). Rumination leads to depression via at least three pathways (for more detail, see Rumination Maintains and Exacerbates Depression section above). First, rumination amplifies a bidirectional circuit between negative mood and negative cognitions—increasing the availability of negative cognitions when individuals are in a negative mood and increasing their affective reactivity to adverse events. Second, rumination interferes with problem solving by leading individuals to think more pessimistically and fatalistically. Third, rumination reduces social support by creating situations (e.g., excessive neediness, friction) that repel potential helpmates. Finally, rumination interferes with instrumental behavior—that is, even when individuals know which actions can directly bring about a resolution to a problem, they cannot muster the motivation to actually enact them. A key moderator that makes rumination more likely to lead to depression is a recurring theme of loss or rejection in one's life, such as losing an adult sibling or experiencing a bitter divorce (Nolen-Hoeksema & Larson 1999).

Anxiety. Rumination can exacerbate any negative mood that an individual is experiencing, not just a depressed or dysphoric one. Thus, individuals who are anxious or whose attention is drawn toward anxiety-producing thoughts will find that rumination multiplies their anxiety (Nolen-Hoeksema & Watkins 2011). Just as in the case of depression, rumination in anxious individuals likewise interferes with their problem solving and instrumental behavior, although depressed individuals tend to act passively (e.g., no motivation to get up in the morning), whereas anxious individuals are more likely to act avoidantly (e.g., eschewing social interactions). Moderators that increase the likelihood of rumination leading to anxiety are threatening environmental stressors (with subtypes of anxiety disorders related to specific kinds of threats) or biological predispositions. For example, rumination in combination with prominent social threats (e.g., childhood teasing or humiliation) may produce social phobia. Conversely, rumination in the presence of uncontrollable, unpredictable, and chronic trauma may lead to posttraumatic stress disorder. Finally, biological predispositions, such as a dysfunction in the fight-or-flight response, could increase the likelihood that rumination will lead to a panic disorder.

Substance abuse. Susan also argued that ruminators are likely to turn to substances to mitigate the negative moods brought about by their overthinking (e.g., Nolen-Hoeksema 2005). Individuals who are particularly at risk of developing a substance disorder are those with a biological predisposition to find substances rewarding or who have seen such behavior modeled by others (i.e., distal risk factors) (Nolen-Hoeksema & Watkins 2011). In the presence of environmental stressors (i.e., moderators), rumination is associated with more alcohol and drug abuse, a relationship that does not appear to vary by gender (Nolen-Hoeksema & Harrell 2002).

Eating disorders. A ruminative response style is a risk factor for bulimic symptoms, which include cycles of overeating (i.e., binge eating) followed by attempts to rid oneself of the food consumed (i.e., purging) by vomiting or taking laxatives. Key moderators increase the likelihood of developing bulimia, such as a relatively high reward sensitivity for food or exposure to a culturally thin ideal. Evidence exists for emotional cascades, whereby rumination heightens negative affect, which then

drives individuals to binge eat (Selby et al. 2008). Finally, early bulimic experiences, and depressive and externalizing symptoms, increase the likelihood that an individual will eventually adopt a ruminative response style (Nolen-Hoeksema et al. 2007), potentially precipitating a downward spiral.

Emotion Regulation and Psychopathology

More recently, rumination has been explored as part of a category of oft-studied emotion-regulation strategies. Along with her graduate student Amelia Aldao, Susan expanded her work on rumination to investigate the associations between various emotion-regulation strategies (e.g., avoidance and reappraisal) and mental health and distress. In a challenge to researchers' assumption that individuals employ a single emotion-regulation strategy at a time in response to a given situation or stimulus, Aldao & Nolen-Hoeksema (2013) found that people often use multiple emotion-regulation strategies at a given moment.

Rumination is considered to be a maladaptive emotion-regulation strategy, a category that also includes avoidance, self-criticism, hiding of expressions, suppression of experiences, and worry. Maladaptive strategies are generally associated with the presence of psychopathology (e.g., depression, anxiety, and alcohol abuse) (Aldao & Nolen-Hoeksema 2012, Nolen-Hoeksema & Aldao 2011). Conversely, adaptive strategies, such as reappraisal and problem solving, are considered beneficial and have been linked to lower levels of psychopathology (Aldao et al. 2010). Compared with maladaptive strategies, adaptive strategies have smaller effect sizes in predicting reduced mental distress. Notably, of all the emotion-regulation strategies (both adaptive and maladaptive) reviewed by Aldao and colleagues, rumination has the strongest association with psychopathology.

Aldao & Nolen-Hoeksema (2012) investigated the ways that individuals employ their emotion-regulation strategies and uncovered individual differences with implications for mental health. Specifically, people tend to overuse maladaptive styles—for example, engaging in avoidance in response to all unpleasant situations, not just particular ones. However, flexibility in how one uses adaptive strategies is associated with better mental health (e.g., reappraising unchangeable situations but problem solving changeable ones)—a finding that may partially explain why the effects of adaptive strategies are often weaker than those of maladaptive strategies in predicting mental distress. Another study found an interaction between the use of adaptive and maladaptive emotion-regulation strategies in women (but not in men), such that adaptive strategies are associated with few symptoms of psychopathology for women with high levels of use of maladaptive strategies (Nolen-Hoeksema & Aldao 2011). In other words, adaptive strategies, such as reappraisal, may work by mitigating the negative consequences of maladaptive strategies, such as rumination, rather than by directly alleviating symptoms.

Evidence suggests that general emotion dysregulation is a cause of psychopathology, not a symptom. For example, in a longitudinal study, emotion dysregulation (operationalized as a lack of emotional understanding, dysregulated expressions of sadness and anger, and ruminative responses to distress) predicted increases in anxiety symptoms, aggressive behavior, and eating pathology (but not depressive symptoms) (McLaughlin et al. 2011). However, viewed from the alternative causal direction, psychopathology did not predict emotional dysregulation, supporting the idea that emotional dysregulation is indeed a transdiagnostic proximal risk factor.

OFFSHOOTS OF SUSAN NOLEN-HOEKSEMA'S WORK

As a mentor, Susan's impact on her doctoral students can be observed in subsequent lines of research that extended and complemented her own. At Stanford University, Susan mentored

two students who would help lead the then-emerging field of positive psychology: Barbara Fredrickson and Sonja Lyubomirsky. Fredrickson's early work on the ability of positive emotions to "undo" (i.e., mitigate) the effects of negative emotions (e.g., Fredrickson et al. 2000a,b; Tugade & Fredrickson 2004) evolved from her prior collaboration with Susan on the duration of negative emotions (Nolen-Hoeksema et al. 1993). Lyubomirsky and Nolen-Hoeksema investigated the adverse effects of rumination on mood, thinking, and problem solving (Lyubomirsky et al. 1998, 1999; Lyubomirsky & Nolen-Hoeksema 1993, 1995), a line of research that steered Lyubomirsky to study the cognitive and hedonic strategies used by naturally happy and unhappy people (Lyubomirsky 2001) and ultimately fostered an interest in positive activity interventions (Lyubomirsky & Layous 2013; see also Layous et al. 2014) and sustainable well-being (Lyubomirsky et al. 2005). In addition, Tomi-Ann Roberts, also a Stanford University student, and Susan investigated gender differences in responses to evaluative feedback (Roberts & Nolen-Hoeksema 1989, 1994). Fredrickson & Roberts (1997) later collaborated in developing objectification theory—the examination of how culture and media negatively influence women's self-perceptions and engender shame, anxiety, eating disorders, depression, and sexual dysfunction.

Susan's former graduate students from Yale University are continuing to extend her research into new areas. Susan and Blair Wisco studied how depression affects memory, as a way of understanding the mechanisms underlying rumination and depression. For example, a common therapeutic technique for treating depression is encouraging a client to reinterpret events as if they were happening to another person, as a way of highlighting and overcoming negativity bias. Wisco & Nolen-Hoeksema (2010a) found that dysphorics indeed show a negativity bias in both the generation and selection of possible interpretations, but that interpretations are more effectively altered in positive directions when considering friends rather than abstract "others." In addition to showing that dysphoria is linked to a negative memory bias, Wisco & Nolen-Hoeksema (2010b) found that adaptive emotion-regulation strategies may render one's memory generally rosier. Specifically, people who frequently use cognitive reappraisal recalled autobiographical memories that were more objectively positive compared with those who infrequently use cognitive reappraisal. Such studies not only replicate prior work on depressed individuals' negatively biased autobiographical memories but also shed light into ways that this tendency may be mitigated or reversed.

McLaughlin, Aldao, Wisco, and Hilt (2014), all former students of Susan, investigated how rumination might explain the comorbidity of internalizing disorders (i.e., depression and anxiety) and externalizing disorders (i.e., aggressive behavior) in adolescents. They found that in adolescent males, rumination explains the transition from internalizing to externalizing symptoms, which provides insight into the origin of male aggressive behavior and further situates rumination as an important transdiagnostic risk factor.

A number of clinical interventions can be directly attributed to or were inspired by Susan and her collaborators' work on rumination. Her longtime collaborator, Ed Watkins, developed a type of cognitive behavior therapy specifically focused on rumination (Watkins et al. 2011). Watkins and colleagues found that assigning participants to rumination-focused cognitive behavioral therapy in addition to normal treatment assuaged symptoms and remission rates, and these effects were mediated by shifts in rumination. Watkins and colleagues also developed self-administered concreteness training to directly target several depressogenic cognitive processes such as rumination and overgeneralization (Watkins et al. 2012). In a randomized trial, individuals meeting criteria for major depression who received concreteness training in addition to standard treatment indeed showed reductions in rumination and overgeneralization relative to treatment as usual. Another intervention that employed Susan's method for experimentally inducing ruminative states found that distraction and mindfulness could successfully interrupt ruminative states in youth

(Hilt & Pollak 2012). Researchers have also trained participants to exert executive control to interrupt ruminative thinking, finding that this training successfully reduces state rumination and the immediate emotional consequences of trait rumination (Cohen et al. 2014). Such intervention research has directly built on and extended Susan's work to benefit those currently experiencing a mood disorder, as well as potentially inoculated at-risk individuals from future mood-related complications.

Finally, Susan and other researchers had begun investigating the biological underpinnings of rumination, an approach that could suggest alternative forms of treatment and shed further light on rumination's origins. Susan's collaboration with Marcia Johnson and colleagues focused on investigating differential activation of brain regions (e.g., medial prefrontal cortex, posterior medial cortex) involved in thinking about one's hopes and aspirations (i.e., promotion focus) versus one's duties and obligations (i.e., prevention focus) (Johnson et al. 2006). A follow-up study with depressed and nondepressed participants suggested that depression involves an inability to both generate positive thoughts from ambiguous cues and disengage from self-reflection when irrelevant to the task at hand (Johnson et al. 2009). Other researchers have continued and extended these lines of inquiry. For example, a recent functional magnetic resonance imaging study of the neural correlates of rumination found that sustained amygdala reactivity is associated with all types of rumination, and specific subtypes of rumination are associated with distinct activity patterns in the hippocampus (Mandell et al. 2014). Thus, in addition to cognitive and behavior therapy, a pharmacological or neurobehavioral intervention might be effective in alleviating rumination by targeting amygdala reactivity. A genetic analysis of the putatively maladaptive component of rumination, known as brooding rumination, found that the *COMT* Val158Met genotype (which is linked to heightened amygdala reactivity and deficits in prefrontal functioning) was indeed associated with brooding rumination as well as heart rate variability (which is part of the same neural circuit) (Woody et al. 2014), adding to the scientific understanding of rumination's many distal risk factors.

Ongoing lines of inquiry conducted by Susan's former students, collaborators, and other researchers highlight the continuing relevance of her work and the methodological rigor that she employed to carry it out. Her research and its direct offshoots proved to be generative across an impressive number of fields, such as clinical psychology, social psychology, developmental psychology, and neuroscience. To be sure, Susan's personal and professional influence is evident in her scientific and academic protégés who are continuing and extending her work.

CONCLUDING WORDS: SUSAN NOLEN-HOEKSEMA'S LEGACY

Susan Nolen-Hoeksema's pioneering contributions were not just in science but in the wider world as well. She took pains to apply and disseminate her research to the general public to help improve people's lives, and especially those who need it most, such as adolescent girls prone to ruminate and at risk for depression. She did not relish media appearances, but she granted them frequently because it was important for her to reach a wider audience than that offered by scholarly journals. She wrote several trade books to this end, such as *Women Who Think Too Much*, in 2003, about rumination and *Eating, Drinking, Overthinking*, in 2005, about the interplay among rumination and mood, substance abuse, and eating disorders, particularly in women. These books revealed something that her empirical articles had not—that Susan was a consummate storyteller with a gift of weaving hard data with revealing anecdotes, compelling stories, and crisp solutions.

As a mentor, Susan schooled her doctoral students to make their way to successful careers; in time, they would look to her as a role model for not only doing science, but also for advising,

teaching, and balancing work and family. She left a mark on students and colleagues at three prestigious universities and won multiple awards for research, mentoring, and teaching.

Susan's research has tremendous implications for how to alleviate rumination and other risk factors and, accordingly, how to treat numerous mental health conditions. Work applying her discoveries is underway, and clinicians and health professionals are already employing in their practices what she found. Her insights, wisdom, and findings leave both scientists and practitioners with a better understanding of the origins of—and strategies for managing—depression in particular and psychopathology in general. Most people strive to leave the world a better place than they entered it, and Susan achieved this with humility and distinction.

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