Behaviour Research and Therapy 49 (2011) 867–874

Contents lists available at SciVerse ScienceDirect

Behaviour Research and Therapy

journal homepage: www.elsevier.com/locate/brat

Perseverative thought: A robust predictor of response to emotional challenge in generalized anxiety disorder and major depressive disorder

Ayelet Meron Ruscio*, Allison E. Seitchik [1], Emily L. Gentes, Jason D. Jones [2], Lauren S. Hallion

Department of Psychology, University of Pennsylvania, 3720 Walnut Street, Philadelphia, PA 19104, USA

ARTICLE INFO

Article history:
Received 21 March 2011
Received in revised form
19 August 2011
Accepted 4 October 2011

Keywords:
Perseveration
Worry
Rumination
Generalized anxiety disorder
Major depression
Comorbidity

ABSTRACT

Generalized anxiety disorder (GAD) and major depressive disorder (MDD) frequently co-occur, yet the reasons for their comorbidity remain poorly understood. In the present experiment, we tested whether a tendency to engage in negative, repetitive thinking constitutes a common risk process for the two disorders. A mixed sample of adults with comorbid GAD–MDD ($n = 50$), GAD only ($n = 35$), MDD only ($n = 34$), or no lifetime psychopathology ($n = 35$) was administered noncontingent failure and success feedback on consecutive performance tasks. Perseverative thought (PT), measured by negative thought intrusions during a baseline period of focused breathing, emerged as a powerful prospective predictor of responses to this experimental challenge. Participants reporting more frequent negative thought intrusions at baseline, irrespective of thought content or diagnostic status, exhibited a stronger negative response to failure that persisted even after subsequent success. Higher PT over the course of the experiment was associated with later behavioral avoidance, with negative affect and other traits closely linked to anxiety and depression, and with the presence and severity of GAD and MDD. These findings provide evidence for a broadly-defined PT trait that is shared by GAD and MDD and contributes to adverse outcomes in these disorders.

A sizable literature has shown that anxiety and mood disorders co-occur at levels far greater than chance (Brown, Campbell, Lehman, Grisham, & Mancill, 2001; Kessler et al., 1996). While the extent of comorbidity is well-established, the mechanisms underlying comorbidity are poorly understood. Prior attempts to explain comorbidity have focused on the shared influence of broad temperament dimensions such as negative affect or neuroticism (Brown, Chorpita, & Barlow, 1998; Goldberg, Krueger, Andrews, & Hobbs, 2009; Mineka, Watson, & Clark, 1998) but have stopped short of describing *how* broad dimensions increase risk for emotional disorders. Specifying behaviorally measurable processes through which risk is manifested is essential for understanding, treating, and ultimately preventing comorbidity and its associated disability. In recent years, a growing research base has drawn attention to functional processes that cut across disorders and may contribute to comorbidity (Harvey, Watkins, Mansell, & Shafran, 2004; Sanislow et al., 2010). Studying such processes directly—in samples that encompass multiple, relevant disorders—has been advocated for identifying improved phenotypes that map onto neural systems and intervention targets more readily than traditional disorders (Sanislow et al., 2010). Treating these common processes—rather than individual disorders—has also been advocated for increasing treatment flexibility and transportability (McHugh, Murray, & Barlow, 2009) and for enhancing clinical outcomes, especially among persons with comorbid psychopathology (Barlow, Allen, & Choate, 2004; Fairburn et al., 2009).

A promising place to begin the search for common processes is the relationship between generalized anxiety disorder (GAD) and major depressive disorder (MDD). GAD and MDD share perhaps the strongest association of all emotional disorders (Kessler, 1997; Watson, 2005), with upwards of 60% of those with GAD developing MDD in their lifetime (Kessler et al., 2008; Moffitt et al., 2007). GAD and MDD also share virtually all of their genetic risk (Kendler, 1996; Kendler, Gardner, Gatz, & Pederson, 2007; Kendler, Neale, Kessler, & Heath, 1992; Roy, Neale, Pedersen, & Mathé, 1995), suggesting that what is inherited is not the syndromes themselves, but common processes or traits that increase risk for both syndromes. What might these common processes be? One candidate is negative, repetitive thinking. Worry, the defining feature of GAD (American Psychiatric Association, 1994), involves a negative, relatively uncontrollable chain of thoughts concerning future

* Corresponding author. Tel.: +1 215 473 4113; fax: +1 215 898 7301.
E-mail address: ruscio@psych.upenn.edu (A.M. Ruscio).
[1] Present address: Department of Psychology, Northeastern University, Boston, MA, USA.
[2] Present address: Department of Psychology, University of Maryland, College Park, MD, USA.

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doi:10.1016/j.brat.2011.10.001

events whose outcome is uncertain (Borkovec, Robinson, Pruzinsky, & DePree, 1983). Rumination, a vulnerability factor and associated feature of depression (Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008; Wisco & Nolen-Hoeksema, 2008), similarly involves a negative, repetitive, passive style of thinking, partly about present symptoms (Nolen-Hoeksema, 1991) or concerns (Martin & Tesser, 1996) but mainly about the past (Thomsen, 2006), especially past loss or failure (Nolen-Hoeksema et al., 2008; Papageorgiou, 2006).

Although worry and rumination are hypothesized to differ in several ways (Nolen-Hoeksema et al., 2008), they appear to share many more similarities than differences (McLaughlin, Borkovec, & Sibrava, 2007; Segerstrom, Tsao, Alden, & Craske, 2000; Watkins, Moulds, & Mackintosh, 2005). Both are abstract, primarily verbal-linguistic, and negatively self-focused—all features which have been shown to perpetuate negative thought and affect (Holmes & Mathews, 2010; Mor & Winquist, 2002; Stokes & Hirsch, 2010). Both involve difficulty extricating attention from negative material, as evident in patterns of neural activation (Hoehn-Saric, Lee, McLeod, & Wong, 2005; Johnson, Nolen-Hoeksema, Mitchell, & Levin, 2009) and in subjective experiences of negative thoughts as "stuck," persistent, and hard to control (Morrow & Nolen-Hoeksema, 1990; Ruscio & Borkovec, 2004). Finally, both reflect passive, unproductive fixation on largely unsolvable problems in ways that hamper effective coping (Borkovec, Ray, & Stöber, 1998; Nolen-Hoeksema et al., 2008), with similar negative consequences for mood, cognition, interpersonal function, and physical health (Watkins, 2008; Wisco & Nolen-Hoeksema, 2008).

Similarities like these have led to suggestions that worry and rumination represent the same core process of perseverative thought (PT) applied to different content in GAD and MDD (Harvey et al., 2004; McEvoy, Mahoney, & Moulds, 2010). The defining feature of the process is thought to be a tendency to engage in negative thinking in a repetitive, dyscontrolled manner, irrespective of the specific content or temporal focus of the thoughts (Ehring & Watkins, 2008). Evidence for a fundamentally similar thought process in GAD and MDD would have direct implications for treatment. It would draw clinical attention to commonalities across a wide array of thoughts that might efficiently be addressed by a core set of interventions. It would raise the possibility that established interventions for one disorder and thought type could profitably be transferred to the other. It would suggest that treating PT in the context of one disorder may help address or even prevent the onset of the second, comorbid disorder. Given the frequent co-occurrence of GAD and MDD, a modifiable process that cuts across these disorders may constitute an especially profitable target for treatment. However, while parallel findings for worry and rumination provide indirect support for such a process, large gaps in the literature have left important questions unanswered about PT and its role in GAD and MDD.

First, with few exceptions, worry and rumination have been studied in separate literatures using measures specific to each construct (e.g., Hayes, Hirsch, & Mathews, 2008; Nolen-Hoeksema, Morrow, & Fredrickson, 1993). This has challenged efforts to compare the core process of PT across disorders. Second, worry and rumination have typically been assessed using global self-report questionnaires (e.g., Meyer, Miller, Metzger, & Borkovec, 1990; Nolen-Hoeksema & Morrow, 1991) rather than measures that track the experience of PT in particular situational contexts. Assessing PT in real time, as it unfolds in interaction with the environment, may illuminate pathways through which PT influences symptoms. Third, despite particular interest in the role played by rumination and worry in emotional disorders, investigations of PT have been carried out mainly with healthy or analog samples (e.g., Moulds, Kandris, Starr, & Wong, 2007; Oathes, Siegle, & Ray, 2011). There is a need for research characterizing broadly-defined PT in GAD and MDD, both in comorbid and pure cases. Finally, most studies have assessed worry or rumination concurrently with outcomes (e.g., Fresco, Frankel, Mennin, Turk, & Heimberg, 2002; Muris, Roelefs, Rassin, Franken, & Mayer, 2005). This has made it difficult to evaluate PT as a potential risk factor rather than merely an epiphenomenon, correlate, or consequence of emotional distress. The case for PT as a risk mechanism would be strengthened if PT can be shown to predict subsequent adverse outcomes, especially following stress. The case would further be enhanced by demonstrating substantial incremental validity of PT over GAD and MDD diagnoses in predicting relevant outcomes.

To begin addressing these gaps, we investigated broadly-defined PT in a mixed sample of GAD and MDD cases and healthy controls. We chose this sample based on recommendations for studying transdiagnostic mechanisms (Sanislow et al., 2010) which call for sampling across clinical conditions in which the mechanism is implicated and for treating the mechanism—rather than diagnostic groupings—as the independent variable of interest. Including cases as well as controls, and comorbid as well as pure cases, enabled us to examine the predictive value of PT beyond the presence of one or both disorders with which it is most closely associated. PT was assessed "on-line" using thought sampling within the context of a focused breathing task. Baseline levels of naturally-occurring PT were used to predict responses to a subsequent emotional challenge. Associations of PT with clinical, course, and temperament measures were examined to further describe this putative risk mechanism in relation to anxiety and depression and to other risk dimensions (e.g., behavioral inhibition, intolerance of uncertainty) previously linked with these conditions. Consistent with a view of PT as a risk mechanism (e.g., Nolen-Hoeksema, 1991), we hypothesized that, regardless of diagnostic status, persons with elevated baseline PT would respond more negatively to a personal experience of failure and would continue to display more negative affect, cognition, and behavior even after an ensuing experience of success. Consistent with a view of PT as a shared mechanism for GAD and MDD (e.g., Ehring & Watkins, 2008), we hypothesized that PT would be elevated in both GAD and MDD relative to controls, associated with the severity and persistence of both disorders, and related to other vulnerability traits previously implicated in these disorders.

Methods

Participants

Participants were 154 adults recruited from the Philadelphia community ($n = 119$) and from the student body of a private northeastern university ($n = 35$). Participants were recruited through electronic and print media and, in the case of student participants, through a website maintained by the psychology department. They were assigned to one of four mutually exclusive groups based on current, primary (most severe) diagnosis: (1) comorbid GAD–MDD ($n = 50$) met criteria for both GAD and MDD; (2) GAD only ($n = 35$) met criteria for GAD, but not MDD; (3) MDD only ($n = 34$) met criteria for MDD, but not GAD; and (4) healthy controls ($n = 35$) had no past or current psychopathology. Persons with a primary diagnosis other than GAD or MDD, current substance abuse or dependence, active psychosis, or active suicidal intent were excluded from the study.

The final sample was 60% female and ranged in age from 18 to 80 years ($M = 31.51$, $SD = 12.62$). Most participants were never married (73%) and relatively well-educated (87% completed at least some college). Racial/ethnic composition was 66% Caucasian, 17% African-American, 11% Asian or Pacific Islander, 3% Hispanic, and 3% other ethnicity. Race, sex, and marital status did not differ