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The form and function of depressive rumination☆,☆☆

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ABSTRACT

Rumination is a symptom of depression that refers to intense, distraction-resistant thinking. Although rumination is widely considered maladaptive, the analytical rumination hypothesis (ARH) proposes that rumination is an adaptive cognitive process where depression first promotes rumination on the causes of problems ("causal analysis"), which in turn promotes rumination on solving problems ("problem-solving analysis"). Effective problem-solving then feeds back to reduce depressive symptoms. To test this cyclical model, a scale with both problem-solving and causal analysis components is required. There are two candidates: (1) the widely used Ruminative Responses Scale (RRS); and (2) the Analytical Rumination Questionnaire (ARQ)—a new scale based on the ARH. These instruments were administered to five samples (Total $N = 1414$) from two different cultures (Canada, Czech Republic) with different clinical statuses (nonclinical, hospitalized). Latent factor analysis of the ARQ supported the existence of both causal analysis and problem-solving analysis factors, making it suitable for testing ARH predictions. Using the ARQ, we found consistent support for the predicted covariance pattern between depression, causal analysis, and problem-solving analysis. However, we found no evidence that either of the RRS factors were related to problem-solving. Moreover, we were systematically unable to detect the predicted covariance pattern between depression and the RRS factors. We conclude that the ability to detect functional relationships between depression and rumination requires the researcher to consider both function (a correct hypothesis for how rumination and depression are adaptively related to each other) and form (valid measures of those constructs). Understanding rumination as a two-stage problem-solving process may help explain why most depressive episodes eventually resolve without treatment.

1. Introduction

It is a maxim of biology that the form of a trait is related to its function (e.g., Williams, 1966). However, the utility of this maxim for determining evolved function depends on an objective assessment of a trait's form. Since thoughts and feelings are not directly observable, psychologists usually study hypothetical constructs of psychological traits, rather than the traits themselves. These constructs are frequently measured with questionnaires and scales that may not carve psychological nature at its joints (Barrett, 2017; Cronbach & Meehl, 1955). The lack of valid psychological constructs has made it difficult to accurately distinguish between adaptive and disordered mental states (Cuthbert & Kozak, 2013; Wakefield, 2013). For instance, rumination refers to a cognitive symptom of depression that is commonly thought to be maladaptive. However, the ability to detect functional relations between depression and rumination requires the use of valid rumination constructs as well as a correct hypothesis for how they are functionally related to depression. To demonstrate this, we evaluate and compare the psychometric properties of two rumination scales—a widely used

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scale, and a new scale based on evolutionary theory—and we use both scales to test a prediction for how rumination may be adaptively related to depression.

The term "depression" refers to multiple phenotypes that share in common the symptoms of sadness and anhedonia (a loss of interest in activities that are normally pleasurable), but vary in other thoughts, behaviors, physiology, and situational precipitants (Andrews & Durisko, 2016; Keller & Nesse, 2006). While typically viewed as pathological, there are several reasons to consider depressive symptomology from an adaptationist perspective. First, some depressive phenotypes are clearly adaptations (Andrews & Durisko, 2016). Infection and starvation both trigger adaptive depressive phenotypes, the function of which is sometimes described as "energy conservation". But the function of depression in these contexts is more accurately described as "energy reallocation" (Andrews & Durisko, 2016). Fighting an infection requires *increasing* overall energy expenditure as the body mobilizes the immune system (Lochmiller & Deerenberg, 2000). Depressive symptoms help reallocate energy to immune function by suppressing growth, physical activity, and reproductive effort (Dantzer, 2001; Hart, 1988). In starvation, the adaptive problem is to preserve brain function while searching for food, and this requires suppressing growth, reproduction, and immune function and upregulating foraging activity. Depressive symptoms promote energy reallocation by suppressing interest in sex, social interaction, and humor, but increasing interest in food (Andrews & Durisko, 2016). Neither of these phenotypes would likely be given a diagnosis of Major Depressive Disorder (MDD) in the Diagnostic and Statistical Manual of Mental Disorders (DSM; APA, 2013). They would more likely be excluded as medical conditions. But they demonstrate the principle that severe or prolonged bouts of depressive symptoms can be adaptive. Moreover, they represent ancient phenotypes from which other phenotypes may have evolved (Andrews & Durisko, 2016).

Second, evolutionary theory supports concerns that DSM diagnostic criteria tend to pathologize normal emotional responses (Frances & Nardo, 2013; Spitzer & Wakefield, 1999). DSM episodes of MDD are most common during the reproductive years (Kessler, Berglund, Demler, Jin, & Walters, 2005), and cumulative incidence rates for young adults may reach 50% in longitudinal studies (Rohde, Lewinsohn, Klein, Seeley, & Gau, 2013). Since natural selection disfavors somatic dysfunction during the reproductive years (Kirkwood & Austad, 2000), only a few explanations for the high rates of depression in young adults are consistent with disorder (infection, physical injury, evolutionary mismatches), none of which are strongly supported (E. H. Hagen, 2011). For example, many stressors that are depressogenic in modern environments (social conflict, pregnancy) were ancestrally common (Andrews & Durisko, 2016; E. H. Hagen, 2003, 2011). Thus, the writers of the fifth edition of the DSM provoked complaints by eliminating the bereavement exemption for MDD, because bereavement is common and evolutionarily relevant (Volk & Atkinson, 2013), and grief is a common emotional response (Thieleman & Cacciatore, 2014; Wakefield, 2013). As another example, human cognitive abilities are thought to have evolved, at least in part, to manage conflicts within cooperative relationships, yet such conflicts commonly cause depression (Andrews & Thomson Jr, 2009). It seems doubtful that the brain would be most vulnerable to malfunction precisely when it is needed most. It is plausible that depressive phenotypes evolved as adaptive responses to such stressors (Keller & Nesse, 2005, 2006; Rosenström et al., 2017).

Finally, in community samples, 89–98% of people who meet DSM criteria for major depression recover within a year of onset (Kendler, Walters, & Kessler, 1997; McLeod, Kessler, & Landis, 1992), and most of these remissions probably occur without treatment (Lewinsohn, Clarke, Seeley, & Rohde, 1994). Spontaneous remission has long puzzled clinicians, and it is difficult for disorder hypotheses to explain, but adaptationist hypotheses can easily account for it.

The phenotypic heterogeneity of depression probably requires multiple adaptationist explanations (Andrews & Durisko, 2016; Durisko, Mulsant, & Andrews, 2015). There are many such hypotheses, and several excellent literature reviews exist (Nesse, 2000; E.H. Hagen, 2011; Durisko et al., 2015). Most of these are conceptually linked to the hypothesis that negative or painful emotions evolved to draw attention to threats in the environment and promote corrective action (Thornhill & Thornhill, 1989). This is a general hypothesis for all negative emotions (e.g., fear, anger), not just depressive phenotypes per se. Each negative emotion affects attention, information-processing, physiology, and behavior in different ways because different threats require different solutions (Tooby & Cosmides, 1990).

One important hypothesis for depressive symptoms proposes a social leveraging or signaling function (E. H. Hagen, 2003, 2011; Watson & Andrews, 2002). Similar hypotheses have been proposed for self-harm and suicidal behavior (Andrews, 2006; E. H. Hagen, Watson, & Hammerstein, 2008; Syme, Garfield, & Hagen, 2016; Watson & Andrews, 2002), which provides a possible explanation for the association between depression and suicide. Other important hypotheses propose that depression evolved to promote disengagement from unachievable goals (Nesse, 2000; Wrosch & Miller, 2009), minimize the risk of aversive social events, such as ostracism (Allen & Badcock, 2003; Badcock, Davey, Whittle, Allen, & Friston, 2017), or manage the problems associated with a subordinate social status (Price, Sloman, Gardner, Gilbert, & Rohde, 1994).

The various hypotheses make different predictions about specific symptom profiles, situational precipitants, and cognitive or behavioral effects (Durisko et al., 2015). Here, we focus on the *analytical rumination hypothesis* (ARH), which makes predictions about the features of depressive rumination (Andrews & Thomson Jr, 2009).

1.1. The conventional view on depressive rumination

Depressive rumination refers to persistent, distraction-resistant thoughts associated with the circumstances surrounding a depressive episode (Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008; Watkins, 2008). Diagnostically, rumination is not present in all depressive phenotypes, but its presence is considered supportive evidence of a depressive episode (APA, 2013).

Rumination has been most widely studied using the *Ruminative Responses Scale* (RRS), where it was first conceptualized as a stable, unproductive cognitive style for coping with depressed mood (Nolen-Hoeksema, 1991; Nolen-Hoeksema, Morrow, & Fredrickson, 1993). The original RRS consisted of 22 items, but 12 were eliminated because they were confounded with depressive symptoms (Treynor, Gonzalez, & Nolen-Hoeksema, 2003). Principal components analysis (PCA) of the remaining 10 items (listed in Table 1) revealed two five-item factors: *brooding* and *reflective pondering* (Treynor et al., 2003). Higher levels of brooding positively predict depressive symptoms both concurrently and longitudinally (Nolen-Hoeksema et al., 2008). This, coupled with other evidence, led many to conclude that brooding is a maladaptive rumination style (Joormann, Dkane, & Gotlib, 2006; Watkins, 2009). Pondering is sometimes, but not always, positively associated with depressive symptoms when measured concurrently; and it is sometimes, but not always, associated with lower symptoms longitudinally (Hasegawa, Koda, Kondo, Hattori, & Kawaguchi, 2013; Junkins & Haeffel, 2017; Nolen-Hoeksema & Davis, 2004; Treynor et al., 2003). For this reason, pondering is sometimes thought to be an adaptive problem-solving rumination style (Nolen-Hoeksema et al., 2008).

However, brooding and pondering may not divide rumination along natural fault lines. First, the use of PCA to analyze the factor structure of the RRS is problematic because PCA assumes that items have no measurement error and that factors are uncorrelated, and both assumptions are frequently violated in psychological research (Fabrigar, Wegener, MacCallum, & Strahan, 1999). For instance, research has consistently found that brooding and pondering are correlated (Armey et al., 2009; Griffith & Raes, 2015; Schoofs, Hermans, & Raes, 2010; Treynor et al., 2003; Whitmer & Gotlib, 2011). It has been suggested that exploratory

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