Behaviour Research and Therapy 48 (2010) 275–281

Contents lists available at ScienceDirect

Behaviour Research and Therapy

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

Cognitive reactivity mediates the relationship between neuroticism and depression

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ARTICLE INFO

Article history:
Received 1 July 2009
Received in revised form
8 December 2009
Accepted 9 December 2009

Keywords:
Depression
Neuroticism
Rumination
Cognitive reactivity

ABSTRACT

Although neuroticism has long been established as an important risk factor for depression, the mechanisms through which this temperamental predisposition translates into the occurrence of symptoms are still relatively unclear. This study investigated cognitive reactivity, i.e. the ease with which particular patterns of negative thinking are reactivated in response to mild low mood, as a potential mediator. Individuals with ($N = 98$) and without a previous history of depression ($N = 83$) who had provided neuroticism scores six years previously were assessed for cognitive reactivity and current symptoms of depression using self-report questionnaires. Tendencies to respond to mild low mood with ruminative thinking mediated the relation between neuroticism and current symptoms of depression in both groups. Reactivation of hopelessness and suicidal thinking occurred as an additional mediator only in those with a history of previous depression. The results suggest that neuroticism predisposes individuals to depression by generally increasing the likelihood of ruminative responses to low mood. In those with a history of depression in the past, neuroticism additionally increases risk of recurrence by facilitating reactivation of previously associated patterns such as suicidal thinking and hopelessness. These findings suggest potential targets for interventions to help preventing the occurrence, or recurrence of depression in those who due to their temperamental predisposition are at an increased risk.

Introduction

Neuroticism is considered to be a temperamental factor that predisposes individuals for a range of emotional psychopathologies and other aversive outcomes (see for example, Clark, Watson, & Mineka, 1994). Research on depression suggests that neuroticism predicts onset of depressive disorders (De Graaf, Bijl, Ravelli, Smit, & Vollebergh, 2002; Kendler, Gatz, Gardner, & Pedersen, 2006; Kendler, Neale, Kessler, Heath, & Eaves, 1993; Ormel, Oldehinkel, & Vollebergh, 2004); that those who are high in neuroticism are likely to suffer from more chronic episodes of depression (Duggan, Lee, & Murray, 1990; Hirschfeld, Klerman, Andreasen, & Clayton, 1986; Rhebergen et al., in press; Weissman, Prusoff, & Klerman, 1978); and that neuroticism modifies the impact of life events, that is the experience of stressful life events is more likely to lead into depression in those who are high in neuroticism than those who are low in neuroticism (Ormel, Oldehinkel, & Brilman, 2001; van Os & Jones, 1999). These are well established and important findings. However, their implications for the understanding and treatment of vulnerability for depression are disappointingly limited (cf. Ormel, Rosmalen, & Farmer, 2004). Neuroticism is defined as a temperamental factor that is presumed to be relatively stable over time, and as such amenable to therapeutic interventions only to a limited degree. Furthermore, the multi-facetted nature of the construct and its overlap with measures of distress themselves have made it difficult to draw conclusions regarding the particular vulnerability mechanisms it indexes. In order to better understand such mechanisms, it would be helpful to learn more about the factors that mediate the relationship between neuroticism and depression, in particular, how neuroticism relates to more proximal, potentially malleable factors that research has already shown to be implicated in vulnerability for depression.

Neuroticism reflects a global dimension of negative emotionality that encompasses the tendencies to experience negative affect in the face of minor stressors, to be aroused quickly and for arousal to fall slowly following stimulation. It also reflects tendencies towards worrying and post-event processing, tendencies to appraise events as stressful and an inability to control urges (Widiger, Hurt, & Frances, 1984). A core feature of neuroticism is a difficulty in emotion regulation. According to Eysenck and

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0005-7967
doi:10.1016/j.brat.2009.12.005

Eysenck (1991) someone who is high on N is "overly emotional, reacting too strongly to all sorts of stimuli, and finds it difficult to get back on an even keel after each emotionally arousing experience" (Eysenck & Eysenck, 1991, p. 4). It is easily conceivable how such temperamental features may lay the ground for the development of maladaptive reactions and strategies more specifically related to the occurrence of depression. Recent evidence has particularly highlighted the role of rumination as a factor accounting for the relation between neuroticism and depression (Kuyken, Watkins, Holden, & Cook, 2006; Muris, Roelofs, Rassin, Franken, & Mayer, 2005; Roelofs, Huibers, Peeters, & Arntz, 2008; Roelofs, Huibers, Peeters, Arntz, & van Os, 2008). Rumination has been defined as a "mode of responding to distress that involves repetitively and passively focusing on symptoms of distress and on the possible causes and consequences of these symptoms" (Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008, p. 400). A large body of research has demonstrated relations between rumination and the occurrence of depression (for overviews see Nolen-Hoeksema et al., 2008; Smith & Alloy, 2009). At the same time, rumination may easily arise out of tendencies towards post-event processing. In fact, repetitive thinking, a hallmark of rumination, has been described by some commentators as a cognitive manifestation of neuroticism (Segerstrom, Tsao, Alden, & Craske, 2000). As described above, recent research supports the assumption of a meditational role of rumination (Kuyken et al., 2006; Muris et al., 2005; Roelofs, Huibers, Peeters, & Arntz, 2008; Roelofs, Huibers, Peeters, Arntz, & van Os, 2008). However, the results of these studies also showed that rumination only partially mediated the relation between neuroticism and depression, suggesting that further factors need to be taken into account.

Another process that has been found to be particularly relevant for the understanding of vulnerability to depression is cognitive reactivity. Cognitive reactivity describes the finding that, once they have become established, negative patterns of thinking can easily be reactivated through only minor triggers such as subtle changes in mood. A number of studies have demonstrated this phenomenon in individuals at risk for depression (for overviews see Lau, Segal, & Williams, 2004; Scher, Ingram, & Segal, 2005). Theoretical accounts have taken such findings to suggest that negative patterns of thinking become associated with negative mood during previous experiences of depression, and that these associations then remain latent during times of normal mood ("differential activation" hypothesis, Teasdale, 1988). Prospective research has demonstrated that those who remain reactive during times of recovery are more likely to relapse (Segal, Gemar, & Williams, 1999; Segal et al., 2006). Given that neuroticism is characterized by an increased sensitivity to emotional stimuli, cognitive reactivity may play a particular role in those who are high in neuroticism. It is conceivable that those who are high in neuroticism are at an increased risk to both acquire negative patterns of thinking and experience their reactivation in response to mild triggers. This may be the case especially when individuals have been depressed in the past given that associations between negative thinking and mood are particularly likely to be formed during times when both of them are predominant. The current study was aimed at investigating this mechanism and to look at the degree to which reactivation of particular aspects of negative thinking and rumination mediate the relation between neuroticism and current symptoms of depression in a sample of individuals with and without a history of depression. In order to assess cognitive reactivity, the study used a self-report questionnaire called the Leiden Index of Depression Sensitivity (LEIDS, Van der Does, 2002).

The LEIDS is based on the assumption that important aspects of cognitive reactivity are accessible to self-report. It assesses cognitive reactivity by asking participants to describe how they would feel and think if they were to experience a low mood. The different sub-scales of the questionnaire assess both cognitive processes and contents that may occur as a response to negative mood such as rumination or thoughts relating to hopelessness or thoughts relating to attempts at harm avoidance. Importantly, previous research has shown that the LEIDS not only differentiates between previously depressed and never-depressed samples when they are in normal mood (Moulds, Kandris, Williams, Lang, Yap, & Hoffmeister, 2008; Van der Does, 2005) but also predicts changes in thinking following negative mood induction. In a study assessing cognitive reactivity in individuals with and without previous history of depression, Van der Does (2002) found that LEIDS scores predicted change in dysfunctional attitudes following mood induction. Studies in previously depressed groups with a history of suicidality have found that self-reported cognitive reactivity predicts changes in a cognitive indicator of hopelessness following mood induction (Williams, Van der Does, Barnhofer, Crane, & Segal, 2008) and that reactivity profiles can differentiate between patients with and without suicidal ideation during previous episodes (Antypa, Van der Does, Penninx, in press).

Most of the research so far that has investigated potential cognitive mediators of the relation between neuroticism and depression has relied on cross-sectional assessments (Kuyken et al., 2006; Muris et al., 2005; Roelofs, Huibers, Peeters, & Arntz, 2008; Roelofs, Huibers, Peeters, Arntz, & van Os, 2008). This is problematic as many of the items of neuroticism scales ask about current distress and reactions to distress with only vague time specifiers and assessing neuroticism at the same time as mediator and criterion variables may, therefore, artificially inflate associations (Ormel, Oldehinkel, et al., 2004; Ormel, Rosmalen, et al., 2004). For the current study we were able to investigate a sample in which neuroticism had been assessed 6 years before we re-contacted them, thus allowing investigations of how neuroticism related prospectively to current symptoms of depression and cognitive reactivity. We hypothesized that (1) neuroticism would be positively associated with current symptoms of depression and (2) that cognitive reactivity as assessed by the LEIDS would mediate the relation between neuroticism and current levels of depression in participants both with and without a history of depression. In order to investigate relative contributions of particular patterns of cognitive reactivity, the different sub-scales of the LEIDS were entered simultaneously into a multiple mediator model. Separate models were computed for participants with and without a history of depression and any differences between models followed up by formally testing moderating effects of previous history.

Method

Participants

Participants were recruited by contacting individuals who had previously taken part in a study on neuroticism that had established a large randomly-ascertained family cohort in southwest England ($N = 88.000$; Martin et al., 2000) and who had given their written permission to be contacted again for participation in further research. As part of the previous research, individuals had provided information on neuroticism 6 years before the start of the current study. In addition, diagnostic information on previous history of depression was available from answers to a questionnaire screening for Major Depression that contained questions about each of the DSM-IV criteria. This measure had been specifically designed for the initial genetic study. Prior diagnostic status had been derived using the DSM-IV algorithm except that questions referred to a period of 4 weeks rather than 2 weeks making diagnoses generally more conservative. In recruiting participants for the