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Research Report

The role of negative mood induction on working memory capacity in individuals putatively at risk for bipolar disorder: A pilot study

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ABSTRACT

Objective: Bipolar disorder (BD) is characterized by cognitive deficits. Usually individuals at risk for BD do not exhibit such deficits but they might be evident under cognitive or emotionally stressful conditions. To our knowledge this is the first study examining working memory capacity under mood induction in individuals at risk for BD.
Methods: Using the Hypomanic Personality Scale (HPS) 68 participants out of an initial pool of 148 students were divided into groups at high and low risk for BD. They completed twice a Dual Task Paradigm (DTP) task assessed under high and low cognitive load prior to and following a negative mood induction.
Results: As expected stimuli incongruency, high cognitive load and mood induction increased response times. Contrary to our hypothesis the mood induction did not differentially affect at-risk individuals. However, they generally reacted faster to neutral stimuli compared to those at low risk.
Conclusions: While we replicated former results related to the DTP, we did not find evidence for the hypothesis that individuals putatively at risk for BD will be more affected by negative mood when doing such a cognitive task. Replication using a larger sample is needed which should also examine whether changes in positive mood might more relevant in the context of risk for mania.

1. Introduction

Individuals with bipolar disorder (BD) consistently display mild to moderate deficits in response inhibition, verbal memory and attention (Bora et al., 2009). Such impairments persist across mood phases but are enhanced during the manic and depressive states (Robinson et al., 2006). Few studies have focused on first-degree relatives of BD patients; however findings from two meta-analyses suggest that healthy first-degree relatives of BD patients display problems in verbal memory, sustained attention and executive functions (e.g. set-shifting, response inhibition) (Arts et al., 2008; Bora et al., 2009). Similarly, Thermenos and colleagues (Thermenos et al., 2010) found that both BD patients and their healthy relatives performed less accurately (percentage of correct responses) and required additional time than healthy controls in a working memory task. Although working memory is not the primary cognitive deficit in BD, Lee et al. (2014)'s meta-analysis in first-episode bipolar disorder found medium to large effect size deficits in attention and working memory, compared to healthy controls. Similar deficits have also been observed in non-affected first-degree relatives of individuals with BD which may indicate that this deficit is a candidate cognitive marker for BD (Ferrier et al., 2004). Further, while compared to schizophrenia cognitive deficits observed in remitted depression and bipolar disorder are relatively small (Altshuler et al., 2004; Krabbendam et al., 2005; Martínez-Arán et al., 2004), problems with cognitive functioning emerge or become much more pronounced during acute phases of BD (Quraishi and Frangou, 2002).

While the before mentioned literature looked at cognitive functioning, there is also some evidence for information processing biases. More specifically there may be a bias towards negative information in both adults with BD (Matsubara et al., 2014; Pavuluri et al., 2008) and unaffected offspring of patients following a negative mood induction (Gotlib et al., 2005).

Given the strong genetic component of bipolar disorder (BD), the majority of studies in high-risk individuals have focused on first-degree relatives and offspring of persons with BD (Goodwin and Jamison, 2007). However, up to 90% of individuals with BD do not have a first-degree relative with BD (Goodwin and Jamison, 2007). This means that BD is not always associated with a high familial risk to develop the disorder. Thus, current studies focusing

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on siblings and offspring of BD patients may have so far only identified a subgroup of vulnerable people.

Although most of current research links stress to depression, mania can be triggered by a range of naturally occurring events (Johnson, 2005) including negative life experiences (Hosang et al., 2012; Kessing et al., 2004) and threat to self-esteem (Mansell and Pedley, 2008). Along the same line, there is increasing evidence that specific types of personality and temperament factors involving proneness to hypomanic symptoms and mood lability (Angst et al., 2003; Blechert and Meyer, 2005) constitute vulnerability factors for the development of mood disorders in individuals who are not at high genetic risk for BD (Kwapil et al., 2000; Meyer and Hautzinger, 2003). One of the most widely used measures to assess such temperament traits is the Hypomanic Personality Scale (HPS) (Eckblad and Chapman, 1986). Previous studies have shown that high HPS scores are associated with a greater number of affective symptoms (Bentall et al., 2011), and also other signs of vulnerability to develop BD such as increased variability in circadian and social rhythms (Meyer and Maier, 2006), alcohol use (Krumm-Merabet and Meyer, 2005; Meyer and Maier, 2006) or impulsivity (Johnson et al., 2013). Furthermore, many studies show a clear association between HPS scores and current depressive symptoms (Klein et al., 1996; Meyer, 2002). Thus, although the HPS scale is sometimes conceptualised as measuring hyperthymic temperament, it does not just assess vulnerability to increased positive affect and energy. In addition there is evidence that mania and hypomania often include features which are rather thought as of being related to depression (Lex et al., 2011; Lyon et al., 1999; Vieta and Valentí, 2013), and the same has been demonstrated in individuals with a high HPS score (French et al., 1996; Klein et al., 1996). Nevertheless, individuals scoring high on the HPS have been found to have an increased risk of developing hypomanic symptoms (Meyer and Blechert, 2005). In particular, 25% of these individuals have been found to fulfil the diagnostic criteria for BD in a 13-year follow-up (Kwapil et al., 2000). This finding indicates some specificity of the HPS scale for mood disorders. Furthermore the HPS scores have been linked to familial aggregation which is in line with the well-established genetic and biological vulnerability associated with BD (Meyer and Hautzinger, 2001).

Similarly to persons with BD individuals with a high HPS score display information processing biases towards emotional information, more specifically for stimuli with negative connotation. A study by French et al. (1996) used the emotional Stroop test and found that individuals with a high HPS score displayed an interference effect for words related to depression but not for those associated with euphoria. This is consistent with the attentional bias for negative information observed in BD (García-Blanco et al., 2014; Gopin et al., 2011). However, when non-emotional attentional functioning was assessed, there was no direct evidence of such deficits (Meyer and Blechert, 2005). A potential explanation for this is that cognitive performance deficits in individuals at risk for BD only become obvious when emotional content interferes with cognitive processing, or when the cognitive load of the task is higher than the individual's working memory capacity.

Cognitive performance has been shown to be dependant on both information load and stimuli congruency. The De Fockert paradigm has been previously used to investigate the role of increasing working memory load on selective attention (De Fockert et al., 2001) which demonstrated a strong interference effect of a high working memory load. In other words, under conditions of high working memory load, irrelevant distractors disrupt information processing to a greater extent than under a low working memory load condition. Thus this task allows researchers to compare performance on more and less difficult tasks within one and the same paradigm.

Along with the task load, the individual's current mood state may modulate both attentional processes and their underlying neural activity. A recent study using a face-word interference task with either low or high attentional demands in healthy controls and individuals with BD showed that the latter were overall slower than healthy controls but depressed patients committed more mistakes than euthymic patients and healthy controls (Rey et al., 2014). Similarly, another study that presented participants with BD with a 2-back working memory task found reduced accuracy levels (percentage of correct responses) following negative mood inductions when compared to the "no mood induction" condition (Deckersbach et al., 2008). The authors interpret their results as indicating that individuals with BD may be overwhelmed by the increasing complexity of the task, when in a negative mood.

Mood induction activates emotion-related information biases and ruminative thoughts that undermine working memory capacity (Curci et al., 2013; Scheibe and Blanchard-Fields, 2009). A recent study showed that individuals putatively at risk for BD performed better on a task of cognitive flexibility after they underwent a positive mood induction compared to a neutral mood induction (Fulford et al., 2013). The authors concluded that positive mood induction improves cognitive performance. However, the effects of a negative mood induction are still unknown since this condition was not included in the study design.

In summary to our knowledge, to date no study has used assessed whether a negative mood interferes with cognitive processing in individuals at risk for BD. Thus, the present pilot study aimed to investigate the effects of negative mood induction on working memory in individuals at high and low risk for BD as measured by the HPS score. We predicted that the performance of individuals at high risk for BD in a working memory/attention task will be affected by a negative mood. Further we expect this effect to be even more pronounced when working memory capacity is drained by high working memory load.

2. Materials and methods

2.1. Participants

Participants were 68 undergraduate psychology students at Newcastle University (19.4 ± 1.24 years, 54 females; 14 males) selected from a sample of 148 undergraduate students who completed several questionnaires assessing the HPS, mood state prior and following mood induction, and levels of anxiety and depression (see below). Being conscious that in this pilot sample the range of scores in HPS might be restricted, we did not use the sample mean to define risk status but rather defined high risk individuals based on the median HPS score obtained in a prior large sample study (Krumm-Merabet and Meyer, 2005). We invited all participants scoring above the median of the prior sample to take part in the laboratory part of the study. Seven of these high scoring participants declined to participate. We matched individuals scoring below the median to the remaining high risk individuals with respect to age and gender. Because of this matching procedure not everyone scoring below the pre-defined median in the current sample was invited back to participate. This led to the final sample of $n=68$.

Participants were healthy individuals with no self-reported past or current history of physical and mental disorders during the informed consent process, and who showed a memory span capacity of at least five digits (see below). The study proposal was peer-reviewed and ethical approval for the study was then obtained from the Newcastle University Faculty of Medical Sciences Ethics Committee.