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Emotion dysregulation and schizotypy

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Abstract

In schizophrenia, blunted affect has been argued to reflect difficulties with the amplification of emotion expressive behavior. The aim of the present study was to assess whether ostensibly healthy individuals vulnerable to schizophrenia present with similar difficulties. In the first component of the study, 843 non-clinical participants completed the Schizotypal Personality Questionnaire, of which 27 scoring in the upper 15% (high schizotypy group) and 27 scoring in the lower 15% (low schizotypy group) were asked to watch amusing film clips, whilst engaging in different emotion regulatory strategies, and specifically, amplify the expression of an experienced emotion ('amplification') or suppress the expression of an experienced emotion ('suppression'). The results indicate that highly schizotypal participants present with specific difficulties with the amplification (but not suppression) of emotion expressive behavior. These difficulties are significantly correlated with total negative schizotypy, particularly blunted affect. In the second component of the study, an individual differences approach was used to assess the interrelationship between self-reported use of suppression and schizotypy in an independent sample of 204 community volunteers. The results suggest that, although blunted affect is associated with increased use of suppression, it cannot be regarded as the primary mechanism underpinning this disturbance. Implications for understanding blunted affect in schizophrenia and related disorders are discussed.

Keywords: Schizotypy; Psychosis-proneness; Schizophrenia; Emotion regulation; Emotional blunting

1. Introduction

Blunted affect in schizophrenia is characterized by markedly reduced emotion expressivity, alongside apparently normal emotion experience (Berenbaum and Oltmanns, 1992; Kirkpatrick and Fischer, 2006). Thus, although anhedonia is a core clinical feature of the disorder, recent evidence supports a distinction between anticipatory and consummatory (or in-the-moment) pleasure, and suggests that only the former may be deficient (Gard et al., 2007; Horan et al., 2006). Consequently, most participants with schizophrenia exhibit limited outward expression across multiple channels, i.e. face, voice and gestures, and across both negative and positive emotions, despite reported consummatory emotional experience being normal in terms of valence and arousal (Berenbaum and Oltmanns, 1992; Kring and Neale, 1996; Kring and Werner, 2004). Since any discrepancy

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between the emotion experienced and the emotion expressed has the potential to cause communicative misunderstandings during social interactions (Aghevli et al., 2003; Troisi et al., 2007), identification of the mechanisms underpinning blunted affect in schizophrenia is of considerable importance.

Recent evidence suggests that this clinical feature may at least partially reflect emotion dysregulation (Henry et al., 2007, 2008; Kring and Werner, 2004). A prominent theoretical account of emotion regulation identifies two broad types of regulatory strategy that can be distinguished according to their temporal initiation in relation to the emotional response (Gross, 2001). Antecedent-focused strategies are applied early in the emotion generation process and thus influence not only what is expressed behaviorally but also what is experienced subjectively. In contrast response-focused emotion regulatory strategies occur after the emotion response has been triggered, and require management of the ongoing emotional experience, expression and physiological response. A typical example of the former strategy is *reappraisal* of the emotion-eliciting situation (i.e. cognitive transformation of the stimulus), while examples of the latter include *suppression* (the conscious inhibition of ongoing emotion expressive behavior) and *amplification*, (the behavioral augmentation of an already initiated emotion, see; Denmaree et al., 2004; Gross, 2001; Kring and Werner, 2004).

In their model of emotion dysregulation, Kring and Werner (2004) note that dysregulation may not only involve a deficiency in regulatory processes, but also maladaptive use of otherwise intact processes. In the case of schizophrenia, it has therefore been suggested that blunted affect may reflect a deficiency in the regulatory process of amplification. As Kring and Werner (2004) note, suppression of expressive behavior is usually associated with increased autonomic nervous system activity. However, a selective increase in physiological responding to emotional stimuli for individuals with schizophrenia has not been identified. Although patients with schizophrenia have demonstrated greater skin conductance reactivity to emotional films, increased physiological activity is also observed in response to *neutral* films (Kring and Neale, 1996). Henry et al. (2008) also found that individuals with schizophrenia did not differ from non-clinical volunteers with regard to their reported habitual use of suppression in day to day life. Further, using an experimental methodology in which the regulatory demands of the emotional task were directly manipulated, Henry et al. (2007) found that individuals with schizophrenia demonstrated significant deficits in the behavioral augmentation of an already initiated emotion, or *amplification* of emotion expressive behavior, and deficits in this capacity were significantly correlated with clinical levels of blunted affect. This suggests that individuals with schizophrenia may not have the capacity to express a level of emotion that is commensurate with their subjective experience, as they experience problems producing outwardly observable displays of experienced emotion (see; Kring and Werner, 2004).

The aim of the present study was to test whether individuals who score highly on schizotypy also demonstrate specific disturbances in the behavioral amplification of emotion expression, and whether they differ from their low schizotypy counterparts with respect to their habitual reported use of suppression. 'Schizotypy' refers to a range of enduring, biologically determined, personality and cognitive traits that indicate a predisposition to schizophrenia (Claridge, 1990; see also, Lenzenweger, 2006). Research of schizotypy in ostensibly healthy participants and relatives of schizophrenia patients has revealed similar neurological soft signs and impairments on measures of emotional, social and cognitive functioning that are generally intermediate between individuals with schizophrenia and healthy controls (Berenbaum et al., 2006; Raine, 2006). Stirling et al. (2007), for example, found that individuals who scored highly on schizotypy exhibited 'heightened' metacognitive processing that was comparable to the patterns reported in individuals who meet diagnostic criteria for schizophrenia.

In the current article, a replication of the schizophrenia study of Henry et al. (2007) is reported, but with non-clinical volunteers scoring either high or low on schizotypy as the participants. Thus, an experimental methodology was used in which the regulatory demands of the emotional task were directly manipulated. Whilst the modulation of both positive and negative emotion expression behavior is clearly of enormous adaptive significance (Gross, 2001), given that anhedonia is a core feature of schizophrenia, the present study focused on capacity to up- and down-regulate positive emotion expressive behavior, and specifically, amusement. It was predicted that, as was shown for individuals with schizophrenia, individuals who score highly on the dimension of schizotypy will present with significant deficits in the *amplification* (but not the suppression) of emotion expressive behavior. Thus, it will be tested whether the *ability* to use these different regulation strategies is affected by the presence of schizotypal traits. However, in addition, and as per the study of Henry et al. (2008), in the second component of the study, an individual differences approach will be used to assess whether habitual use of suppression differs