

Abstract

Activation and Habituation of the Cingulate Cortex during Emotion Processing in Healthy Controls, Borderline, and Schizotypal Personality Disorder

by

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Disturbances in emotional functioning are central features of the clinical profiles of both borderline and schizotypal personality disorder (BPD and SPD, respectively). BPD is characterized by a high sensitivity to emotional stimuli and unusually strong and long-lasting reactions, indicative of impaired habituation to emotional stimuli (Linehan, 1993). Previous research suggests that SPD patients demonstrate limbic hyper-reactivity to unpleasant stimuli, at least initially, but intact habituation to repeated presentation of unpleasant stimuli (Hazlett et al., 2012). The cingulate cortex supports various aspects of emotion processing and regulation, and abnormalities of this region have been related to emotion dysfunction in SPD and BPD (Koenigsberg, Fan, et al., 2009; Modinos, Ormel, & Aleman, 2010b). However, findings of functional cingulate abnormalities in the context of emotion processing have been inconsistent in BPD and limited in SPD. The current study utilized event-related functional magnetic resonance imaging (fMRI) in three groups, BPD patients, SPD patients, and healthy control individuals, during a task involving an intermixed series of unpleasant, neutral, and pleasant pictures, each presented twice within their respective trial. This approach permitted the examination of reactivity to emotional stimuli and habituation of emotional responses within the cingulate. Blood-oxygen-level dependent response values were obtained within the manually delineated anterior cingulate cortex (ACC) and posterior cingulate cortex (PCC) and compared across groups. Compared to healthy controls and SPD patients, BPD patients exhibited significantly

greater activity in the ACC during the presentation of neutral pictures. Heightened activity in the BPD group persisted across the initial and repeated presentations of neutral pictures. On the other hand, SPD patients exhibited greater activity in the ACC compared to healthy controls and BPD patients during the initial presentation of unpleasant pictures, but activity normalized when the pictures were repeated. The two patient groups demonstrated heightened ACC activation, but these abnormalities were differentiated by associated picture-type (neutral versus unpleasant) and attenuation of the response upon repeated presentation of the stimuli. Diagnostic differences in PCC activation did not reach significance. Overall results suggest unique involvement of the ACC in BPD and SPD symptomatology.