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How does reactivity to frustrative non-reward increase risk for externalizing symptoms?☆,☆☆

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ABSTRACT

Frustration is a normative affective response with an adaptive value in motivating behavior. However, excessive anger in response to frustration characterizes multiple forms of externalizing psychopathology. How a given trait subserves both normative and pathological behavioral profiles is not entirely clear. One hypothesis is that the magnitude of response to frustration differentiates normative versus maladaptive reactivity. Disproportionate increases in arousal in response to frustration may exceed normal regulatory capacity, thus precipitating aggressive or antisocial responses. Alternatively, pathology may arise when reactivity to frustration interferes with other cognitive systems, impairing the individual's ability to respond to frustration adaptively. In this paper we examine these two hypotheses in a sample of kindergarten children. First we examine whether children with conduct problems (CP; $n = 105$) are differentiated from comparison children ($n = 135$) with regard to magnitude of autonomic reactivity (cardiac and electrodermal) across a task that includes a frustrative non-reward block flanked by two reward blocks. Second we examine whether cognitive processing, as reflected by magnitude of the P3b brain response, is disrupted in the context of frustrative non-reward. Results indicate no differences in skin conductance, but a greater increase in heart rate during the frustration block among children in the CP group. Additionally, the CP group was characterized by a pronounced decrement in P3b amplitude during the frustration condition compared with both reward conditions. No interaction between cardiac and P3b measures was observed, suggesting that each system independently reflects a greater sensitivity to frustration in association with externalizing symptom severity.

1. Introduction

Theoretical models of affective neuroscience focus on the evolutionary function of emotions, the most basic of which are not only consistently evident across cultures but across a broad range of mammalian species (e.g. Panksepp, 2012). Emotions can be viewed as the activation of primary motivational drives to pursue rewards, defend safety, or withdraw from uncertainty (Harkness et al., 2014). However, extreme, chronic, or contextually dissonant emotions are viewed as the foundation of many major mental health disorders. Thus research is needed to understand how affective processes that support basic behavioral systems can lead to psychopathology. The Research Domain Criteria (RDoC) generated by the National Institute of Mental Health identifies 5 distinct subconstructs of negative affect believed to contribute to a range of mental health disorders from depression to aggression. Among these subconstructs is "frustrative non-reward", defined as the removal of, or impediment to obtaining, a previously available award.

2. Frustrative non-reward

The omission of an expected reward results in a decrease in striatal dopamine that signals the discrepancy between the actual versus predicted outcome, and facilitates an adaptive learning response

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(Porter-Stransky et al., 2013). Theoretical models of attention-deficit/hyperactivity disorder (ADHD) posit that deficient processing of non-reward cues contributes to the resistance of behavioral symptoms to normal operant conditioning (Sagvolden et al., 2005). Imaging data provides support for this model, demonstrating that adolescents across multiple externalizing disorders, including ADHD and conduct disorder (CD), fail to show changes in neural activation from rewarded to non-rewarded blocks, in contrast to their normally developing peers (Gatzke-Kopp et al., 2009). These findings suggest that individuals with externalizing disorders have difficulty in extinguishing previously rewarded behaviors, but do not address the affective domain of externalizing behaviors. Negative affectivity is proposed to be a key component of externalizing psychopathology, particularly conduct disorder, and thus further attention is warranted to the psychophysiological responses to non-reward (Eckhardt and Kassinove, 1998; Hubbard et al., 2002; Roberton et al., 2012; Sanderlin, 2001).

In addition to the effects on adaptive learning, reward omission, or frustration, is typically accompanied by an affective reaction of anger, which is associated with patterns of neural activity distinct from those that mediate learning. Omission of reward is associated with greater activation in the anterior cingulate, and greater right-lateralized activation in the anterior insula and ventral prefrontal cortex (Abler et al., 2005). These regions have documented roles in the processing of affect, with right-lateralized regions thought to contribute to negative affect in particular (Davidson and Irwin, 1999). In addition, regions such as the dorsal anterior cingulate and anterior insula are integrally involved in translating cognitive representations of affect into physiological arousal through autonomic activation (Critchley, 2005). Thus activation of these neural regions likely contributes to the visceral responses to frustration. Sympathetic activation (skin conductance) has been shown to be reactive to conditions of perceived unfairness, particularly when the unfairness is relevant to personal goals (Civai et al., 2010).

Anger in the face of frustrative non-reward is a normative affective response, and the associated increase in arousal facilitates behavioral activation needed to overcome obstacles to goal achievement (Dixon et al., 2013; Otis and Ley, 1993). The individual's perception of this behavioral activation can manifest in prosocial (e.g. determination) or antisocial (e.g. anger) states (Harmon-Jones et al., 2011). Anger is considered a key factor in behaviors such as reactive aggression, which characterize several clinical conditions. In adults, borderline personality disorder (BPD) is viewed as resulting from intense and dysregulated negative affect in response to frustration that can include physical violence and suicidal gestures (Harkness et al., 2014; Linehan and Dexter-Mazza, 2008). In children these behaviors are best captured in oppositional defiant disorder and conduct disorder (CD) in which intense negative affect can lead to pathological non-compliance, fighting behaviors, and destruction of property (Cappadocia et al., 2009).

3. Disproportionate affective arousal

It is possible that these clinical conditions are distinguished from normative responses to frustration by the magnitude of affective distress. In the context of acute stress, neural processing in limbic regions becomes prioritized to facilitate sensitivity to affective information (Oei et al., 2012). This re-prioritization can come at the expense of activation in the dorsolateral cortex, a region involved in working memory and other executive systems (Krause-Utz et al., 2012). Researchers have found that individuals with BPD show a greater increase in amygdala activation than controls when presented with emotional distractors during a working memory task, as well as a significant increase in reaction times, indicating greater interference of affective arousal with cognitive processing (Krause-Utz et al., 2012). Thus more extreme reactions to frustration may effectively re-prioritize neural processing away from systems engaged in regulatory control. Interestingly, a recent study examining the mechanisms of response to an intervention focused on anger management among men with trauma histories found that successful reduction in anger symptoms was mediated by increasing skills in reducing physiological arousal and not related to cognitive-based coping skills (Mackintosh et al., 2014). These findings support a model whereby psychopathology might arise in the context of hypersensitivity toward affective arousal that competes with regulatory systems.

Some evidence suggests that children with externalizing tendencies react with greater degrees of sympathetic arousal when frustrated. Among a sample of 2nd grade children, high levels of teacher-rated reactive aggression were associated with greater increases in both heart rate and skin conductance in response to an anger-inducing game in which the child loses to another child who has cheated (Hubbard et al., 2004). Further research found that the tendency for individuals with a history of aggression to be more physiologically reactive was specific to conditions of anger compared to other forms of negative affect (Wang et al., 2007).

4. Disrupted cognitive control

From the behavioral surface, it is difficult to determine exactly what mechanisms underlie the tendency of individuals with externalizing disorders to resort to aggressive behavioral responses to this type of emotional arousal. Because anger in response to frustration is a normal emotion, it is not clear whether it is the affective experience (e.g. anger), or the behavioral response to it (e.g. aggression), that differentiates individuals with externalizing problems. In other words, individuals with externalizing symptoms could have stronger anger reactions wherein the magnitude of physiological arousal exceeds that of typical individuals, or they could have comparable levels of affective arousal to frustration, but lack appropriate cognitive resources to direct their increased behavioral activation in socially appropriate and adaptive ways. Although not mutually exclusive, each pathway lends itself to specific hypotheses.

As mentioned above, frustration is a common experience, and most individuals encounter frustrating or goal blocking situations on a regular basis. One of the primary goals of development is learning to regulate responses to frustration in socially appropriate ways. For instance, imagine a child playing with her favorite toy when her older sibling comes along and snatches it from her hands. Anger would be a normal and fully appropriate emotional response. The objective would be for the child to resist any inclination to respond aggressively but employ a more prosocial strategy, such as appealing to an adult for help. Children with externalizing behaviors characteristically fail to engage the non-aggressive response. Recent theories have posited that this results from disruptions in cognitive function, specifically in motivationally salient contexts. Individuals with externalizing behavior problems appear to prioritize motivationally relevant information for attentional processing, which detracts from executive function resources needed to regulate arousal (Baskin-Sommers et al., 2012). In a small study of 27 children treated for externalizing behavior disorders, those who showed clinical improvement in response to treatment also showed improvement in cortical brain potentials specific to the temporal window of inhibitory control from pretest to posttest (Lewis et al., 2008). The authors interpret this change as reflecting improvement in cognitive control rather than a decrease in emotional arousal. This perspective highlights the importance of examining cognitive processing within affective contexts.

The study of cognition × affect interactions is facilitated by the use of event-related potential (ERP) studies in which individual trials are embedded within affective blocks in order to examine the state-dependent effects of context on cognitive processing. Studies of this type have reported an association between context-specific effects on ERP amplitudes and symptoms of psychopathology, suggesting that the extent to which affect disrupts cognitive processing may be a unique feature that discriminates between normative and pathological affective responses. One study of children diagnosed with bipolar disorder

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