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Social-cognitive, physiological, and neural mechanisms underlying emotion regulation impairments: understanding anxiety in autism spectrum disorder

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ABSTRACT

Anxiety is one of the most common clinical problems among children, adolescents, and adults with autism spectrum disorder (ASD), yet we know little about its etiology in the context of ASD. We posit that emotion regulation (ER) impairments are a risk factor for anxiety in ASD. Specifically, we propose that one reason why anxiety disorders are so frequently comorbid with ASD is because ER impairments are ubiquitous to ASD, stemming from socio-cognitive, physiological, and neurological processes related to impaired cognitive control, regulatory processes, and arousal. In this review, we offer a developmental model of how ER impairments may arise in ASD, and when (moderating influences) and how (meditational mechanisms) they result in anxiety.

Emotion regulation (ER) refers to the ability to modulate experienced and expressed emotion in the service of goal-directed or value-based behavior. For example, becoming transiently angry when someone takes your parking space may be a normative response, but it is not adaptive to act on that anger in an aggressive manner. In such situations, optimal ER skills allow for the down-regulation of negative emotions to proceed with the task at hand (i.e., find another place to park). An impoverished ability to cope with and control intense, especially negative, emotions is associated with a range of psychiatric conditions and symptoms (Aldao et al., 2010; Berking et al., 2012; Berking and Wupperman, 2012), and the goal of this review is to explore ER impairment in relation to manifest anxiety in autism spectrum disorder (ASD).

Over the last several years, there has been burgeoning scientific interest in the possibility that ER impairments are nearly ubiquitous in ASD (Mazefsky et al., 2013). Impaired ER may underlie many of the behavior problems commonly seen in children and adults with ASD, such as aggression, irritability, and anxiety (Mazefsky and White, 2013). Anxiety is among the most commonly observed and impairing associated (i.e., non-core) symptoms in ASD (e.g., White et al., 2009). As many as four out of five children with ASD are diagnosed with comorbid psychiatric disorders (Simonoff et al., 2008) and, although there is evidence that diagnostic practices that take into account the ASD-related impairments would result in fewer comorbid diagnoses (Mazefsky et al., 2012a), children, adolescents, and adults with ASD are clearly at increased risk of experiencing a range of secondary behavioral and emotional problems.

In this review, we consider socio-cognitive, physiological, and neural mechanisms that may serve as mediators of ER impairments in ASD. We then explore possible pathways between disrupted ER and the behavioral manifestations of anxiety in ASD, with a focus on intra-individual moderating influences. The goals of this paper are to synthesize the extant literature related to specific processes involved in ER impairments in ASD and to identify factors that may cause ER deficits to manifest as anxiety. We conclude with recommendations for future research to systematically evaluate the plasticity of ER mechanisms and thereby reduce anxiety in ASD.

1. Overview: emotion regulation and anxiety in ASD

Anxiety is an emotional response with both affective and physiologic components. Anxiety can be defined as nervous tension and

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autonomic arousal, often accompanied by general distress (Clark and Watson, 1991). The *regulation* of emotion is distinct from the *experience* of that emotion. For example, a child might cope with anxiety about speaking in front of peers by reminding himself that most other children his age also get anxious or by feigning illness on the morning of a class presentation. Reappraisal and avoidance behaviors such as these are strategies to modulate or regulate the emotion responses. Emotional experience is automatic and often intense, whereas the regulatory strategies used for coping with the situation are often explicit and intentional.

ER is a broad, multilevel process, involving inter-related systems (e.g., attention, physiology, neurological processes; e.g., Calkins, 2010). Regulatory strategies are typically used to increase, or heighten, an emotional experience (up-regulation), decrease the emotion itself, or dampen the outward expression of the emotion (down-regulation). Derived from Gross (2001) process model of ER, strategies can also be categorized in relation to emotional experience. Regulation can occur prior to the experience of the emotion or in response to the emotion, termed antecedent-focused and response-focused strategies, respectively. For example, avoidance of situations likely to trigger an unpleasant emotion is an antecedent strategy whereas suppression of an emotion is response-focused and occurs after the emotion generating experience. Additionally, some regulatory strategies are explicit and intentional (e.g., reappraisal, suppression), whereas others are implicit, or occurring without conscious intention (e.g., affect labeling; Gyurak et al., 2011).

A person's 'regulatory style' is associated with general well-being (Gross and John, 2003) and, in some cases, the development of psychopathology (Cicchetti et al., 1995). Developmentally, regulatory ability improves dramatically during the first few years of life (Calkins, 2010) and is predictive of positive outcomes. A child's ability to effectively manage emotions, for example, predicts better adaptive behavior and social relationships (Eisenberg and Fabes, 2006). People who consistently engage in adaptive cognitive reappraisal tend to have stable, close interpersonal relationships and more positive emotions than do people who do not readily engage in reappraisal. Those who habitually suppress emotion, on the other hand, experience more negative affect and often experience inauthenticity, or a sense of externally presenting a false persona (Gross and John, 2003).

Emotion dysregulation is a transdiagnostic risk factor (i.e., a fundamental process underlying multiple disorders or symptoms) for psychopathology. In the developmental psychopathology literature, for example, there is a strong association between ER impairments and development of internalizing problems, such as anxiety and depression (e.g., Southam-Gerow and Kendall, 2000). McLaughlin and colleagues (2011) demonstrated, in one of the few longitudinal studies in the field, that ER deficits prospectively predicted not only changes in adolescents' anxiety but also aggressive behavior and eating pathology. Conversely, psychopathology did not predict subsequent ER deficits.

Although there is little empirical research on ER in ASD, there has been considerable study of the experience and expression of emotion in ASD. This research has demonstrated that people with ASD often have poorly differentiated emotional responses and self-knowledge, tend to experience more negative emotion, and are harder to soothe once aroused, relative to peers without ASD (e.g., Konstantareas and Stewart, 2006). From infancy through adulthood, individuals with ASD are often described as having low levels of positive affect along with heightened negative affect and dysregulated behavior, compared to peers without ASD (Garon et al., 2009; Mazefsky et al., 2012b). Children with ASD, when frustrated, display more intense and prolonged periods of resignation (i.e., giving up) and less effective ER strategies (e.g., more avoidance) compared to typical peers (Jahromi et al., 2012). They tend to show poor emotional insight and struggle to adequately express emotion via verbalization, facial expression, or other nonverbal means (e.g., Losh and Capps, 2006).

Our goal is to offer an evidence-informed heuristic for understanding ER impairments as a risk factor that may manifest as anxiety in people with ASD. Among psychiatric comorbidities seen in ASD, anxiety disorders are the most common (de Bruin et al., 2007; Joshi et al., 2010; White et al., 2009), affecting about 40% of children and adolescents with ASD (van Steensel et al., 2011). As noted above, we propose that ER impairments, through a host of biological and nonbiological mechanisms, are fundamentally related to ASD. In this model, ER impairment is viewed as a transdiagnostic risk factor affected by multiple mechanisms, which are etiologically linked to ASD (i.e., equifinality), whereas anxiety is only one possible, though likely, outcome of impaired ER in ASD (i.e., multifinality).

Most clinical affective neuroscience research has focused on emotions as discrete categories, where each emotion is thought to emerge from independent neurobiological systems (for a review, see Posner et al., 2005), and this approach has yielded important advances in the understanding of neurobiological mechanisms of affect. However, there is strong evidence that discrete emotion categories are constructed of more general brain networks, suggestive of a dimensional model that conceptualizes that all affective states arise from common, overlapping neurophysiological systems (Lindquist et al., 2012). Dimensional models of affect have a long history in psychological research (Russell, 2003) including the circumplex model of affect that proposes that all affective states arise from two fundamental neurobiological systems defined by the near-orthogonal dimensions of valence (i.e., pleasure-displeasure) and arousal (i.e., alertness or vigor; Russell, 1980). This framework suggests that every emotion may be represented on the basis of varying degrees of valence and arousal. For example, feeling tense is the product of a negatively valenced and highly aroused emotional state; whereas feeling serene is the product of a positively valenced and low arousal state.

The circumplex model of affect suggests that dysregulated arousal systems would have a direct impact on the experience of all emotions, and Tseng and colleagues (2013) recently found that a circumplex model of experienced affect is characteristic of individuals with ASD. One potential mechanistic account for impaired ER, and the resultant increased anxiety, in ASD is that individuals on the spectrum may experience heightened levels of basal or reactive arousal. Arousal is regulated by connections between the reticular formation in the brainstem and the limbic system, including the amygdala, and the thalamus and parietal lobes, and there is evidence of impairments in functional and structural connectivity between the brainstem and limbic system in ASD (Elison et al., 2013; Fatemi et al., 2012). Additionally, a comprehensive functional neuroimaging literature implicates impaired processing of emotional stimuli in ASD (for a review, see Dichter, 2012). To the extent that arousal systems may be dysregulated in ASD, this would have a direct influence on the experience of affective states, potentially contributing to impaired ER in ASD.

In addition to neural and physiological mechanisms that affect regulatory capacity, there are cognitive and social processes that influence the experience of, and ability to intentionally alter, emotion. In typical development, ER emerges rapidly, largely through interactions between the child and parent. Just as altered interactions between a child (or infant) and the social environment can affect, and be affected by, the child's temperament (Dawson, 2008), this interaction can also affect ER development. In the following sections, we explore these broad domains of mediational mechanisms – socio-cognitive, neural, and physiological, as they relate to understanding impaired ER in people with ASD.