The Role of Emotion Regulation in Autism Spectrum Disorder

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Objective: Autism spectrum disorder (ASD) is associated with amplified emotional responses and poor emotional control, but little is known about the underlying mechanisms. This article provides a conceptual and methodologic framework for understanding compromised emotion regulation (ER) in ASD. **Method:** After defining ER and related constructs, methods to study ER were reviewed with special consideration on how to apply these approaches to ASD. Against the backdrop of cognitive characteristics in ASD and existing ER theories, available research was examined to identify likely contributors to emotional dysregulation in ASD. **Results:** Little is currently known about ER in youth with ASD. Some mechanisms that contribute to poor ER in ASD may be shared with other clinical populations (e.g., physiologic arousal, degree of negative and positive affect, alterations in the amygdala and prefrontal cortex), whereas other mechanisms may be more unique to ASD (e.g., differences in information processing/perception, cognitive factors [e.g., rigidity], less goal-directed behavior and more disorganized emotion in ASD). **Conclusions:** Although assignment of concomitant psychiatric diagnoses is warranted in some cases, poor ER may be inherent in ASD and may provide a more parsimonious conceptualization for the many associated socioemotional and behavioral problems in this population. Further study of ER in youth with ASD may identify meaningful subgroups of patients and lead to more effective individualized treatments. J. Am. Acad. Child Adolesc. Psychiatry, 2013;52(7):679–688. **Key Words:** arousal, autism spectrum disorder, comorbidity, emotion regulation, psychiatric disorders

Autism spectrum disorder (ASD), a neurodevelopmental condition diagnosed based on impairments in socialization and social communication and the presence of restricted and repetitive behaviors,[1] is estimated to affect 1 in 88 in school-age children.[2] An emerging consensus has indicated that ASD involves disruptions in connectivity and processing across brain regions.[3] Thus, impairments associated with ASD are pervasive, including a significant impact on emotional functioning.[3] Common associated problems include irritability, aggression, self-injury, anxiety, and impulsivity.[4]

Emotion regulation (ER) is a construct that may provide explanatory power for understanding the observed emotional and behavioral problems in ASD. ER is generally defined as the automatic or intentional modification of a person's emotional state that promotes adaptive or goal-directed behavior.[5] Individuals with ASD may fail to use adaptive ER strategies and instead react impulsively to emotional stimuli with tantrums, aggression, or self-injury.[6] Such behaviors are often interpreted as deliberate or defiant but may be due to inadequate management of emotion.[7]

A common presenting complaint that exemplifies impaired ER in ASD includes intense reactions to stress or outbursts that are colloquially referred to as "meltdowns."[8] The concern about meltdowns can be inferred by the popularity of resources on this topic. For example, using the search words *meltdown* and *autism* on www.amazon.com identified 50 books. By contrast, a PsychInfo search of peer-reviewed articles with *emotion regulation* and *autism* in the abstract produced only 15 articles (as of October 12, 2012). This suggests a marked discrepancy between the perceived importance of these problems in ASD and the empirical investigation of the phenomenon. This article aims to accelerate research on the *processes* underlying the expression and experience of impaired ER in ASD.

TERMS AND CONCEPTS

The study of ER can be traced to early research on temperament. *Temperament* is a broader construct than ER, encompassing biologically based individual differences in affective, attentional, and motor reactivity or response.[9] The scientific study of ER as a distinct construct emerged only a few decades ago.[10,11] Since then, there has been a burgeoning of research and proposed clinical approaches devoted to ER.[12]

Despite its widespread use, the definition of ER is not straightforward. Emotions allow rapid appraisal of situations (external or internal stimuli) and preparation of subsequent actions to maintain well-being. Thus, emotions emanate from an individual's evaluation of a stimulus.[13,14] The initial strength (i.e., intensity, speed) of this emotional response is often referred to as *emotional reactivity* or *emotionality*.[15] The term *regulation* implies efforts to modify or control the intensity of the emotional reaction, usually in the service of an individual goal.[5] The relation of emotionality to ER has been debated, with some researchers supporting a clear distinction[16] and others contending that emotion/emotionality and ER are inseparable.[17] It is also important to distinguish *mood* (e.g., depression) from *emotion* (e.g., sadness). Mood is a stable and enduring state, whereas emotion is usually tied to the appraisal of a specific situation and a subsequent behavioral response.[13]

The emotional response involves multiple domains, such as behavior, subjective experience, and physiology. Definitions of ER also have included a consideration of attention and motivation.[18] A point of difference between emotionality and ER involves the integration of this information to control emotional responses through unconscious or conscious processes. ER may decrease (i.e., downregulate), increase (i.e., upregulate), or simply maintain positive or negative emotions. These processes can be response focused or occur before the emotional response (antecedent focused).[13]

METHOD TO STUDY ER

Behavioral/Clinical Method

Few studies have examined ER assessment methods for individuals with ASD. A general recommendation for the measurement of ER is multimethod assessment.[19,20] Widely used observational methods for children include requiring a child to wait to open a surprise[21] and free play in an unstructured setting.[22] Another approach is the disappointment paradigm,[23] during which the child receives an undesirable prize. In these methods, emotions are quantified based on ratings of facial and vocal indices.[24] Other coded behaviors may include asking for information for reassurance and disruptive behaviors. The application of such approaches in children with ASD may be difficult to interpret if not evaluated in the context of the child's baseline behaviors and emotional expressions. Abnormal use of nonverbal communication, which is a diagnostic criterion for ASD, may explain unusual or limited facial expressions. In addition, the observable behaviors reflecting self-regulation may differ. For example, adults with ASD have commented that self-stimulatory behaviors (e.g., rocking) or intense focus on routines/rituals/circumscribed interests can be used for self-soothing.[25] By contrast, these same behaviors may reflect distress.[26] Thus, an extended baseline period of observation and historical information may be needed to accurately classify behavior as manifestations of distress versus deliberate self-soothing. Moreover, despite the value of observational methods in developmental research, these methods may miss the internal components of ER.[19]

Interviews and questionnaires have been widely used in non-ASD populations to assess relevant internal processes. The advantages of interviews include the opportunity for the respondent to describe internal states and ER strategies in an open-ended manner. In addition, the examiner can ask for clarification or elaboration. Self-report questionnaires designed to study the internal processes of ER or putative cognitive biases related to emotional responses also have been used in non-ASD populations. However, there is limited research to support the psychometric properties or clinical utility of such self-report questionnaires in ASD. For example, a study of adolescents with ASD found no association between the Dysfunctional Attitudes Scale for Children and self-reported depressive symptoms.[27] This finding stands in sharp contrast to consistent evidence of an association in non-ASD populations.[28] Whether this difference is due to differences in the mechanism underlying depression in ASD or reflects poor validity of this questionnaire in ASD is not clear. Another limitation of self-report questionnaires in ASD is the difficulty of applying them to nonverbal individuals, individuals with intellectual disability, and young children. Taken together, these considerations imply that measurements of ER validated in the general population warrant careful reassessment in youth with ASD.

Biologically Based Method

The autonomic nervous system is relevant to understanding the physiologic processes of ER and offers the possibility of an objective assessment of ER.[29] Heart rate variability (variation in interbeat intervals) is an index of autonomic flexibility.[29,30] There are multiple

measurements of parasympathetically mediated heart rate variability, such as respiratory sinus arrhythmia (RSA).[27] Studies examining RSA have suggested that higher baseline levels of RSA are associated with flexible emotional responses.[31,32] However, the interpretation of *changes* in RSA from baseline differs depending on the context, which may be a particular consideration in ASD. Specifically, RSA suppression (i.e., *decrease* in RSA from baseline) may reflect effective coping in response to stressful situations.[33] Conversely, in contexts that are nonthreatening, effective ER is thought to be associated with an *increase* in RSA above baseline.[34]

Accumulated results from functional magnetic resonance imaging studies have provided insight into the neural circuitry of ER in clinical and nonclinical populations.[35] In ASD, the challenge of finding ecologically valid means of eliciting emotion in a neuroimaging scanner is difficult given the idiosyncratic nature of emotional triggers in this population.[36] Once an emotion is elicited, it may be difficult to differentiate the emotional response from efforts to regulate emotion.[17] To date, functional magnetic resonance imaging studies in ASD have used predominantly higher-functioning individuals, perhaps because of the difficulties involved in engaging subjects with intellectual disability and those with receptive language deficits in functional magnetic resonance imaging tasks.

ER RESEARCH IN ASD

Research in ASD has focused more on emotional experience than on ER. In general, this research has suggested that youth with ASD compared with non-ASD controls have poorly differentiated emotional responses, exhibit more negative and less positive affect, and experience the physiologic consequences of emotion with limited cognitive insight.[37] Children with high-functioning ASD tend to rely on overt cues to describe their emotions (e.g., I was sad because I was crying) and provide nonspecific accounts of their emotional experiences.[38] Although limited by small samples and the assessment of predominantly high-functioning samples, the available research has suggested that individuals with ASD generally lack the emotional insight needed for effective ER.

Survey data in a few small studies have supported the presence of poor ER in ASD.[37,39] Moreover, although the link has yet to be empirically established, ER deficits in children with ASD likely contribute in part to an increased use of psychiatric services. A year-long study of the health care costs and use of 33,582 children (3,053 with ASD) found that children with ASD had almost 12 times more psychiatric hospital days than children without ASD, at almost 11 times the cost.[40] Similarly, as many as 60% of children with ASD have been prescribed psychotropic medications despite scant evidence that any medication is effective for the core features of ASD.[41,42]

An emerging body of evidence has supported the presence of inadequate ER strategies in ASD. For example, adults with ASD have reported less use of cognitive reappraisal and greater use of suppression than adults without ASD.[39] This pattern persisted even after controlling for differences in emotional experiences and alexithymia (difficulty identifying and describing emotions).[37] In addition, 2 separate observational studies of young children with ASD found that they were less likely to use adaptive coping strategies compared with children without ASD.[43,44] No ER studies have included adolescents with ASD.

CONCEPTUALIZING POOR ER IN ASD

One possible explanation for inadequate ER in ASD is the co-occurrence of a psychiatric disorder that accounts for the deficit. Alternatively, poor ER may be intrinsic to ASD. Another possibility is that specific psychiatric disorders and ER deficits in ASD share clinical or neurobiological features in common, making it difficult to disentangle the source of the behavioral disturbance.

Psychiatric Comorbidity

There is a reciprocal relation between maladaptive ER and psychiatric disorders, such that poor ER increases the risk for developing a psychiatric disorder and having a psychiatric disorder interferes with adaptive regulation. In addition, poor ER is a common mechanism across many disorders, and its behavioral manifestation is often what prompts referral for treatment.[20] The presence of emotion dysregulation in ASD is often interpreted as evidence of an additional psychiatric disorder.[36,45] Among community samples, 71% to 80% of children with ASD have been reported to have at least 1 concurrent psychiatric diagnosis and 41% at least 2.[46] Even higher rates have been observed in clinically referred samples. For example, children with ASD seen in a psychopharmacology clinic were diagnosed with an average of 6.4 concurrent psychiatric disorders using a standard psychiatric interview.[47]

However, co-occurring psychiatric disorders may be overdiagnosed in this population when using standard diagnostic measurements. A study of 35 high-functioning adolescents with ASD found that most (~60%) prior psychiatric diagnoses were not supported by a psychiatric interview that was modified to take ASD-related impairment into account.[48] The results of this pilot study highlight the need for validated diagnostic measurements in ASD to separate the behavioral correlates of ASD from symptoms of other psychiatric disorders.[49]

This issue of diagnostic boundaries shares some of the challenges of severe mood dysregulation and irritability in non-ASD samples.[50] The new diagnostic category, disruptive mood dysregulation disorder, aims to differentiate individuals with impaired mood regulation from individuals with bipolar disorder. It is not yet clear how this diagnosis will apply to individuals with ASD and emotion dysregulation. In a study of 91 adolescents with ASD, more than 25% of the ASD group had severe mood dysregulation, but the neurocognitive correlates associated with severe mood problems differed from non-ASD samples.[45] Therefore, manifestations of emotion dysregulation in ASD may be somewhat distinct from those in non-ASD youth.

Shared Risk Factors With Other Disorders

Impaired ER has been associated with several disorders, including anxiety and mood disorders and borderline personality disorder.[51] Some mechanisms thought to underlie inadequate ER in these populations also may have relevance to ASD. The tripartite model of anxiety and depression and some key findings regarding the neural circuitry implicated in ER are considered here.

Tripartite Model. The tripartite model of anxiety and depression holds that general distress is a shared factor but that physiologic hyperarousal is specific to anxiety and anhedonia is specific to depression.[52] General distress, or high baseline levels of negative affect, also may play an important role in ASD. Indeed, greater irritability and lower positive affect have been observed in many infants who later develop ASD.[53] Although mixed, some evidence has suggested hyperarousal in ASD.[54] In support of emotion dysregulation associated with physiologic hyperarousal in ASD, multiple studies have shown increased basal heart rate and decreased basal heart rate variability or RSA in individuals with ASD compared with healthy controls, suggesting decreased vagal regulation of the heart or sympathetic and limbic activation.[55-58] If ASD is associated with physiologic hyperarousal, the presence of anxiety in ASD is easier to understand and may inform treatment given a well-established connection between hyperarousal and anxiety in non-ASD samples.[59] Although not always clearly articulated, the management of arousal is an element in most cognitive-behavioral therapy approaches for anxiety.[60] Encouraging results from studies of cognitive-behavioral therapy in youth with ASD have supported the potential relevance of overarousal in ASD.[61] A focus on decreasing arousal also has been successfully integrated into other ASD treatment programs, such as Social Communication, Emotional Regulation, and Transactional Support (SCERTS).[62]

Affective Neuroscience. ER typically activates several areas of the prefrontal cortex (PFC). The subgenual anterior cingulate cortex, for example, plays an important role in the modulation of affective experiences.[63,64] Modulation of the amygdala (involved in the experience of emotion) by the ventromedial PFC (including the anterior cingulate cortex) is a prominent theme in affective neuroscience, with abnormalities in ventromedial PFC/amygdala connectivity implicated in mood and anxiety disorders.[35,64] ER roles also have been attributed to ventrolateral PFC and more superior aspects of the medial PFC.[63] The distinct roles of each of these prefrontal areas to ER is an area of active investigation in nonclinical and mood/anxiety disorder samples.

Some aspects of the ER neuroscience literature fit well within current models of neural dysfunction in ASD. For example, abnormal PFC function is a recurring theme in neuroimaging studies of ASD, although often associated with abnormal perspective taking rather than ER.[65,66] However, PFC deficits could account for impaired perspective taking and ER deficits in ASD. A related area of interest is PFC/amygdala connectivity. The few studies that have explicitly examined this connectivity in ASD in the context of emotion have yielded inconsistent findings. Swartz *et al.* reported decreased connectivity,[67] whereas Monk found increased connectivity.[68] Although the affective neuroscience literature generally implicates *decreased* connectivity between the PFC and amygdala in populations with clinical anxiety, Monk raised the intriguing possibility that PFC upregulates amygdala function in ASD,[68] decreasing the capacity to

adaptively regulate emotion. These data call for a more sophisticated model of PFC/amygdala connectivity that moves beyond a strictly inhibitory relation—one that also considers findings of abnormal white matter development in ASD.[69,70]

The numerous reports of abnormal medial PFC activity in ASD are consistent with data on PFC contributions to ER and, by extension, findings of ER deficits in ASD. Although many of these reports have implicated dorsal medial PFC deficits in ASD (Brodmann areas 9 and 10[71]), a growing number has focused on the same ventromedial PFC areas that have been implicated in ER (namely the orbitofrontal and subgenual anterior cingulate cortices).[67,72,73] Given the equivocality in findings on the PFC regions involved in ER within ASD, the authors contend that it will be important to examine ASD deficits and ER processes together. For example, the localization of ASD deficits to specific PFC areas implicated in ER may advance the understanding of individual differences in ER functions and behavior in ASD.

The preponderance of literature on the amygdala and emotion dysregulation in non-ASD clinical samples has implicated hyperactivation,[63,64,68] whereas traditional models of amygdala dysfunction in ASD have implicated hypoactivation.[74] The putatively "social" and "emotional" functions of the amygdala are likely to be strongly related, but ASD has been associated with anxiety *and* decreased amygdala activation (generally interpreted as relating to social anhedonia).[75] However, there are more imaging studies that have found *hyperactivation* of the amygdala in ASD during emotional face perception.[68,76] In one of the only ASD studies directly relating anxiety symptoms to brain function, Kleinhans *et al.* found a positive correlation between amygdala activation and social anxiety symptoms.[78] Increased amygdala activation recently has been associated with patterns of eye gaze avoidance in ASD, leading to an intriguing confluence among amygdala function, anxiety, and social avoidance in ASD.[79,80]

Although further investigation into the parameters surrounding amygdala activation in ASD is needed, 3 points warrant mention. First, individual differences are integral to understanding brain function in ASD. Specifically, the level of anxiety in the ASD samples may influence the amygdala findings, thereby influencing interpretations of the "social" functions of amygdala. Second, recent studies have underscored the importance of attentional mechanisms in the examination of emotion systems in ASD.[68,76,79-81] When given the opportunity, individuals with ASD and elevated anxiety may be more prone to avoid the most potent affective information in a visual display, thereby affecting the signal in biological measurements. Third, measurement of mood, anxiety, and emotionality in ASD research in general and neuroimaging studies in particular is essential to build the evidence base.

The case for amygdala and PFC involvement in ER dysregulation in ASD is strong. Nevertheless, more complex models are likely needed to fully understand the (atypical) development of ER abilities in ASD. In particular, the regulation of amygdala function likely involves more than the PFC. Although the case of PFC regulation of the amygdala is compelling (including extensive evidence from human and animal studies), it has been argued persuasively that emotion processes (including ER) need to be understood in the context of broader networks and more complex modulatory connections between structures.[77] Network approaches to understanding ER are strongly relevant to ASD, given the evidence for broad abnormalities in multiple gray and white matter structures.[66,69] Furthermore, there are many additional brain areas that may play an important role in the understanding of ER in ASD. For example, recent studies have implicated the insula in the experience and anticipation of negative outcomes—functions that fit well within the ER framework.[82-84] Functional and structural imaging studies of ASD have identified abnormalities in this area[73,85-87] A similar overlap can be identified in the striate cortex, which plays an important role in the reward components of ER[72] and has been shown to function abnormally in ASD.[75] Given that ER and ASD deficits involve multiple, interconnected brain circuits, the search for key structures is likely to require a great deal of future research.

Characteristics of ASD That Influence ER

The functionalist perspective of ER posits that a person consciously seeks to regulate positive and negative emotions to attain set goals.[18] Clinical observations in ASD support a tendency to react to emotional stimuli intensely without clear goal directedness. The few ER studies done to date in ASD have suggested the use of suppression,[37] resignation, and avoidance,[44] which lends some support to the notion that ER in ASD lacks the motivational component

thought to be integral to ER in non-ASD populations. This apparent lack of motivation for ER may be compounded by poor emotional insight and self-monitoring.

Adaptive ER strategies are contextually dependent and applied selectively, based on the situation. By contrast, maladaptive strategies tend to be universally applied.[51] Given the problems with cognitive flexibility and difficulties in modulating behavior in people with ASD, the greater use of maladaptive strategies and the less effective implementation of adaptive strategies are not surprising. In addition, deficits in perspective taking[88] may limit the ability to evaluate the responses of others, lead to misunderstanding and frustration, and hinder accurate reappraisal of a situation.[37]

Differences in information processing and perception in ASD, ranging from slower processing to a heightened sensitivity to environmental influences (e.g., sensory sensitivity, resistance to change), also may affect ER in ASD. For example, the drive to modulate sensory input to a comfortable level may override and interfere with ER. A lower threshold for sensory information (hypersensitivity) may lead to intense and easily triggered reactions, whereas hyposensitivity (higher sensory thresholds) may be associated with disruptive and dysregulated behavior.[89] A recent study found that cortical responses to sensory stimuli in ASD are markedly unreliable, with large within-subject differences in amplitude across trials.[90] This may help to explain observations of aberrant and inconsistent responses to sensory information in people with ASD. It also raises the question of whether evoked emotional responses are similarly unreliable (e.g., whether neural responses to emotion are variable across time). If so, this would interfere with the ability to identify and respond to emotional experiences appropriately.

DISCUSSION

Despite the many challenges involved in studying ER in ASD, this area of inquiry has the potential to improve the understanding of the neural circuitry of ASD and pave the way for new and more effective treatments. Therefore, ER warrants assessment in clinical and research settings. However, many questions remain regarding the mechanisms that lead to impaired ER in ASD and the implications for treatment (Figure 1). The authors' framework proposes that neural mechanisms shared with other psychiatric disorders, in combination with ASD-related behavioral and cognitive characteristics, interact to produce the heterogeneous presentations of emotion dysregulation in ASD (Figure 2). The inclusion of ASD characteristics that are likely to influence ER disruption makes this model specific to ASD, yet the authors posit that ER is a dimensional construct that cuts across disorders (compared with research domain criteria). It may be possible

FIGURE 1 Key research and clinical questions related to processes that interact to produce impaired emotion regulation (ER) in autism spectrum disorder (ASD).

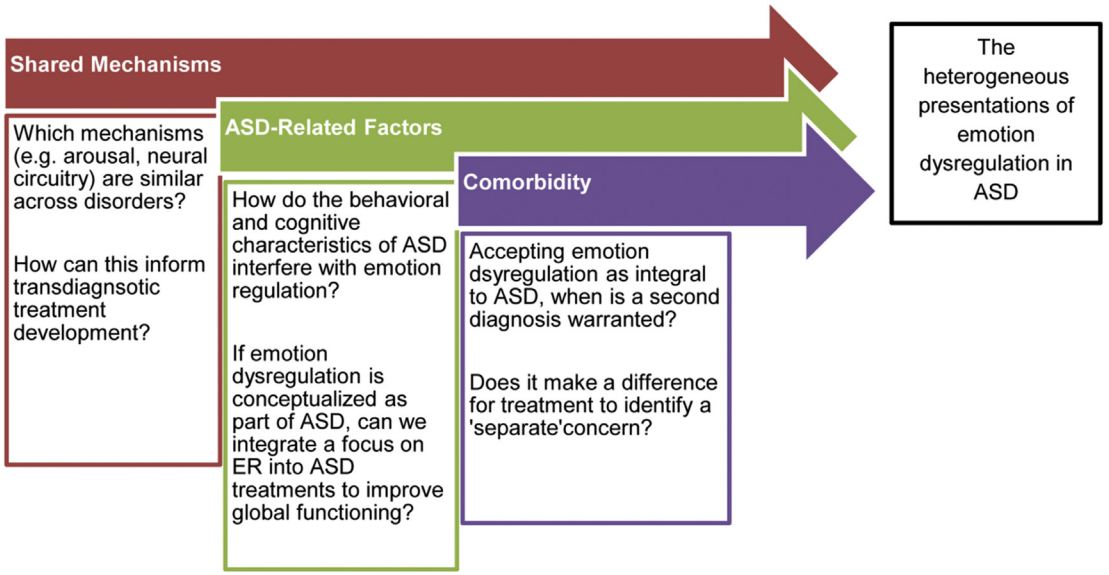
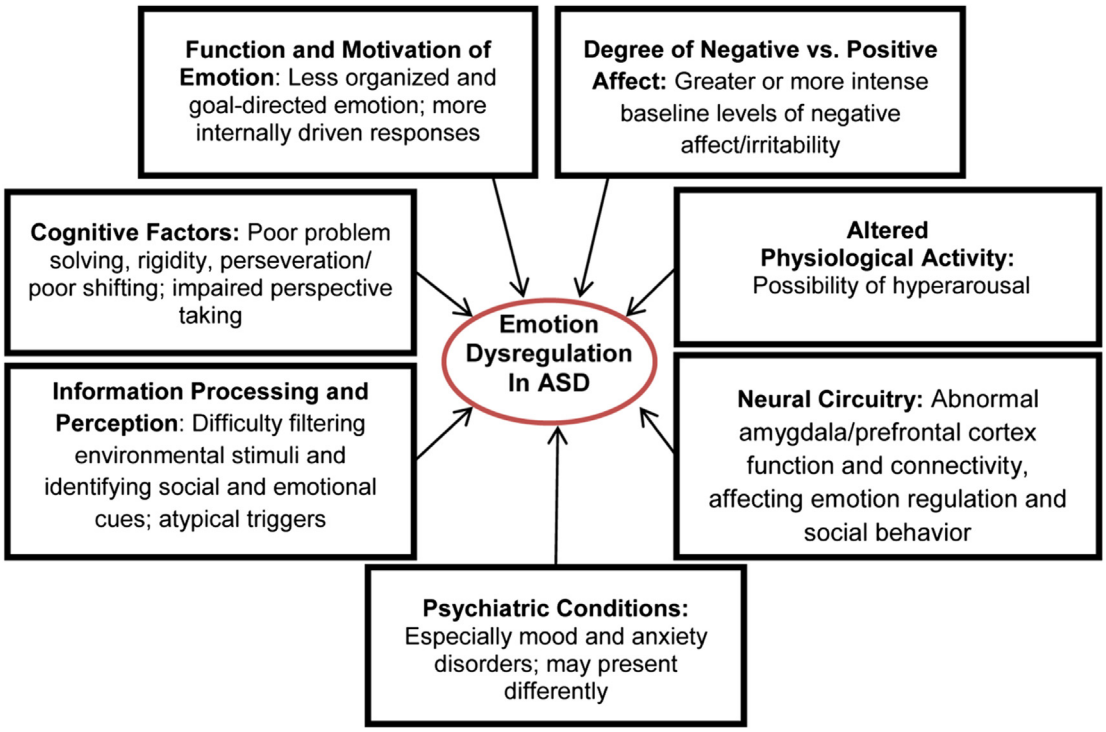


FIGURE 2 Factors that may contribute to emotion dysregulation in autism spectrum disorder (ASD).



to develop transdiagnostic treatments for ER that are effective for many diagnostic populations, with modifications included to address the unique aspects of emotion dysregulation in ASD.

The authors propose that impaired ER may be intrinsic to ASD. However, this may increase the risk for developing a psychiatric disorder and co-occurring psychiatric disorders contribute to the clinical picture in some cases. Nonetheless, the reported prevalence of concurrent psychiatric disorders in individuals with ASD may be overstated to explain the behavioral manifestations of emotion dysregulation.[48] Impaired ER may be a more parsimonious explanation for the degree to which problems with anger and aggression (externalizing behaviors) and anxiety and depression (internalizing behaviors) are seen in ASD. A balance between understanding emotion dysregulation inherent to ASD (perhaps through the identification of subtypes) and the presence of a separate psychiatric disorder is needed. Situations marked by a new constellation of behaviors and symptoms that reflect a clear change in the individual's functioning may call for a second diagnosis (e.g., an episodic mood disorder).[49] Greater clarity will occur when researchers understand how the developmental, neural, cognitive, and behavioral features of these psychiatric disorders differ in ASD compared with the non-ASD population.

Research on ER in ASD is likely to require a multimethod approach,[91] preferably combining physiologic or neural measurements with behavioral measurements. An advantage of physiologic and neuroimaging approaches is their ability to detect a response even if other factors (e.g., impaired language or insight, or alexithymia) limit the accuracy of self-report. Paradigms are needed to capture the often idiosyncratic emotional triggers in ASD. Self-reports have obvious pitfalls in ASD but hold potential for obtaining information about specific manifestations of anxiety and internal processes. For this reason, there is a need to develop and validate self-report ER measurements in ASD.

To understand ER in ASD, representative samples with a variety of emotional presentations (from blunted to labile) are needed. Longitudinal studies will help tease apart whether ER development in ASD is delayed versus deviant and will identify age effects in ER. In addition, given the role of language in ER development,[23] ASD samples spanning a range of language and intellectual abilities will be necessary. Integration of research into specialized inpatient psychiatry units that serve a large volume of children with

ASD and other developmental disorders is one way to access large numbers of children with ASD and limited verbal ability.[92] Treatment development studies related to ER also should include participants representing the full range of verbal and cognitive abilities.

As clinicians await research on the feasibility and efficacy of psychosocial, pharmacologic, and other (e.g., biofeedback) treatment approaches for impaired ER in ASD, they must draw pon the extant research from other clinical populations and make adaptations when necessary for clients with ASD. To assess ER in a clinical context, careful observation of behavior may be most informative and applicable regardless of the individual's verbal ability or cognitive function. For example, how does the individual react to disappointment (e.g., parent "forgot" a favorite snack) or uncertainty (e.g., examiner asks the individual to wait while she organizes papers) and to positive emotions, such as receiving a reward? Such observational data combined with parent report and self-report may be feasible in most practice settings and provide information useful to case conceptualization. If it is determined that poor ER is an explanatory factor for clinical presentation, psychoeducation for the patient and family on ER and adaptive and maladaptive strategies can be useful. In some cases, caregivers may not realize their reliance on a maladaptive strategy and that these strategies are modeled for the patient. Psychoeducation about ASD and ER can promote a validating environment and acceptance of the individual's difficulties (similar to dialectical behavior therapy[93]) and recognition of the individual's strengths. Other treatment strategies could include developing the client's awareness of his/her own internal state to prepare for potentially difficult situations. Helping the individual to identify situations when an ER strategy should be used (flexibility in implementation) is essential in addition to specific ER skills (e.g., acceptance, reappraisal).

Approaching externalizing behaviors in ASD from the perspective of impaired ER also suggests alternate pharmacologic approaches, such as the use of α_2 agonists. The α_2 agonists decrease sympathetic tone and thus can be conceptualized as targeted treatment for the emotional reactivity and hyperarousal that are characteristic of poor ER in ASD. Although there is insufficient evidence for α_2 agonists in ASD at this time,[41] a large multisite trial of a long-acting α_2 agonist for children with ASD is underway and may provide further evidence for this pathway.

Application of a validated dimensional measurement reflecting an emotional and behavioral composite, termed *irritability*,[94] in randomized clinical trials led to Food and Drug Administration approval of 2 medications in children with *DSM-IV*–defined autistic disorder.[95,96] These successful drug treatment programs point the way forward and suggest that validating other emotional profiles may affirm important treatment targets. In addition, there is preliminary evidence that psychosocial interventions targeting ER skills can effectively decrease outbursts and negativity in young children with ASD,[97] but much more work in this area is needed. Measurements of ER that are reliable and valid across the full range of youth with ASD and sensitive to change have yet to be developed. The development of such a dimensional measurement requires a better understanding of ER in the entire ASD population and moving beyond strict, categorical approaches. &

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