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The Need for a Broader Approach to Emotion Regulation Research in Autism

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Abstract

Maladaptive emotional reactions are common among individuals diagnosed with an autism spectrum disorder (ASD) and often impair functioning. Most research on emotional processes in ASD has focused on the recognition of emotion in others. This article argues for a broader approach to affective research in ASD, one that includes investigations into emotional reactivity and regulation. For example, research has typically looked at perseveration in ASD from a cognitive or perceptual perspective, yet perseveration also appears to have emotional aspects. This article discusses examples of emotion regulation research in other populations to illustrate how this approach could inform understanding of perseveration in ASD, particularly related to affective interference with cognitive control. More broadly, it highlights the potential contributions of emotion regulation research in ASD in relation to improving treatment specificity, increasing understanding of individual differences and diagnostic conceptualizations, and, potentially, contributing to a deeper understanding the neurobehavioral underpinnings of ASD.

Keywords

Autism; Emotion regulation; Perseveration; Affective interference; Cognitive control

Parents of infants who later receive an autism spectrum disorder (ASD) diagnosis often describe them as having less positive affect, more negative affect, and more difficulty controlling attention and behavior than their non-ASD siblings and typically developing children (Garon et al., 2009). These concerns develop alongside, and often before, the more widely recognized repetitive behaviors and impairments in social interaction and communication (Dawson, 2008). Higher levels of negative affect persist into childhood, adolescence, and adulthood, with evidence of more anxiety, emotional lability, and anger than both typically developing and developmentally delayed people (Bradley & Isaacs, 2006; Capps, Kasari, Yirmiva, & Sigman, 1993; White, Oswald, Ollendick, & Scahill, 2009).

Although negative affect can be helpful in some situations, too much can hurt interpersonal relationships, attention, problem solving, and communication abilities (Laurent & Rubin, 2004). Thus, it is necessary to maintain an optimum level of arousal in order to meet individual and social goals (Chambers, Gullone, & Allen, 2009). Most individuals do this through emotion regulation processes that modulate the intensity and timing of both positive

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and negative responses to fit the situation (Gross, 2008; Thompson, 1994). Clinical accounts suggest a gross failure to do so in ASD, resulting in not only increased negative affect, but also rapidly escalating and seemingly out-of-proportion reactions typically called “meltdowns” (e.g., Baker, 2008). In general, these are quite impairing, and often involve increases in repetitive behaviors, further withdrawal, aggression, or self-injuring behaviors.

Given the consequences of maladaptive emotional reactions in ASD and the integral relationship between emotional state and social functioning, it is surprising how little research has focused on emotion regulation in ASD. Some affective research has examined the use of emotion words and description of emotions (Bauminger, 2002; Hill, Berthoz, & Frith, 2004; Rieffe, Terwogt, & Kotronopoulou, 2007; Yirmiya, Sigman, Kasari, & Mundy, 1992), the ability to express emotions (Capps, Yirmiya, & Sigman, 1992), and autonomic nervous system responses to and self-reports of emotion stimuli (e.g., Anderson, Colombo, & Shaddy, 2006; Shalom et al., 2006). These studies point to a potential deficit in ASD in the ability to name and label one’s own emotions (e.g., Rieffe et al., 2007), as well as a disconnect between self-reported emotion and expected physiological patterns (e.g., Shalom et al., 2006). Despite this, the behavioral and neural underpinnings of emotion regulation processes in ASD remain poorly characterized.

Given the large body of work on differences in face processing in ASD (see Sasson, 2006 for review), most emotion perception research has focused on the recognition of emotional reactions from others’ faces. The findings have been quite heterogeneous: some studies find poor emotion recognition skills, whereas others indicate intact abilities. Not surprisingly, task demands (such as use of static versus dynamic presentations of facial expression, or matching versus naming) and demographics explain many of the inconsistencies (Harms, Martin, & Wallace, 2010). Perhaps most interesting, however, is that nearly all of the eye-tracking, neuroimaging, and ERP studies demonstrated group differences even when behavioral performance is normal, which suggests a difference in the underlying mechanism for emotion perception (Harms et al., 2010). This research indicates the possible use of compensatory strategies such as feature-based learning, and also points to different neural activation patterns, including more activation of the anterior cingulate cortex and precuneus and less activation of the amygdala and fusiform gyrus than controls (see Harms et al., 2010 and Pelphrey, Adolphs, & Morris, 2004 for review).

It is unknown at this point whether similar patterns would be found in studies focused on the expression and regulation of one’s own emotion in ASD, but it seems possible that the neural response may differ. For example, Dalton and colleagues (2005) reported increased activation of the amygdala at the specific times when individuals with ASD fixate on another person’s eyes. They speculate that the avoidance of eye contact in ASD may be a coping mechanism for a heightened emotional response to eyes (Dalton et al., 2005). Further, individuals with ASD who are more socially anxious also have greater amygdala activation (Kleinhans et al., 2010). Thus, it may be that individuals with ASD have an exaggerated amygdala response when experiencing negative emotions and a dampened response when perceiving or judging emotions in others. To further consider this possibility and to demonstrate the usefulness of an emotion regulation focus in ASD, we present an example below that describes perseveration in ASD from an emotional framework.

AN EMOTIONAL COMPONENT TO PERSEVERATION?

Perseveration refers to a maladaptive tendency, common among individuals with ASD, to focus intently and excessively on one thing. There has been a large amount of research on the perceptual and cognitive aspects of perseveration. Several ASD studies required participants to shift visual attention from one target to another (e.g., Courchesne et al., 1994;

Leekam, Lopez, & Moore, 2000), which is more of a measure of stimulus-dependent reflexive orienting. Other designs have explicitly attempted to tease apart the perceptual and cognitive components involved with shifting attention, with support for the notion that behavioral problems with perseveration are due to cognitive rather than perceptual deficits (Ozonoff, Strayer, McMahon, & Filloux, 1994). Specifically, studies show that children with autism can shift attention between stimuli when shifting continuously is the only task demand (e.g., Pascualvaca, Fantie, Papageorgiou, & Mirsky, 1998). Difficulties in shifting attention emerged only when they were required to shift after they were already involved in another activity or way of thinking about things (Ozonoff et al., 1994; Pascualvaca et al., 1998).

This research advances our understanding of perseveration in ASD by suggesting a problem with cognitive control, but no previous work has considered the emotional aspects of perseveration. Individuals with ASD may be more susceptible to affective influences on attention-shifting problems, which may manifest as a tendency to overly focus on distressing stimuli and difficulty shifting to a more positive affect after activation of a negative emotion. Although the actual trigger for emotional distress is typically quite idiosyncratic and varied among children with ASD, there seems to be a pattern of difficulty disengaging thought from the situation and switching to a more positive emotional state after activation of the negative emotion. For example, the mother of a 16-year-old with high-functioning ASD said of her son, “once he starts to have a meltdown, it continues to escalate and there is no talking him down.”

The experience of a 12-year-old with Asperger’s disorder (whom we will call Arthur) provides a perfect example of emotional perseveration in ASD. Arthur was a happy child who was virtually always smiling. Other children teased him, but even that did not cause him emotional distress. However, when something did bother him, he remained so focused on it and his related “bad mood” that it would interfere for days or weeks on end. For example, he became distraught about grub worms being crushed on the sidewalk outside of his school. For weeks, Arthur could think of nothing else: he wrote very emotional stories about the misunderstood grub worms for all of his writing assignments at school, he was late to class because he was outside protecting grub worms, and he instigated fights with other children who paid less attention to the grub worms than he did. The thoughts of grub worms were associated with an increase in his level of emotionality: he cried in session when he discussed them, and he was moved to tears about the grub worms’ plight any time a teacher attempted to suggest that he not worry as much about them. When grub worms were not the topic of discussion, he remained much more emotionally labile than usual. Arthur’s strong feelings about the grub worms seemed to be interfering with his cognitive control.

AFFECTIVE INTERFERENCE WITH COGNITIVE CONTROL

Why was Arthur so stuck on grub worms, and why did he remain distraught about them for so long? One explanation could be that increased and sustained amygdala reactivity interfered with his cognitive control. A similar finding has been reported with rumination (prolonged elaborative processing of negative emotional information), an information-processing bias that has been implicated in the development and maintenance of depression (Papageorgiou & Siegle, 2003). Specifically, a study to clarify the mechanisms underlying this sustained processing found that depressed individuals displayed amygdala responses to negative words that lasted throughout nonemotional processing tasks, in contrast to a fast decay of amygdala responses in individuals who were not depressed (Siegle, Steinhauer, Thase, Stenger, & Carter, 2002). Further, their findings suggested that sustained amygdala reactivity may be due in part to decreased inhibition of the amygdala by the prefrontal

cortex, given an inverse relationship between the amygdala and dorsolateral prefrontal cortex, highlighting the connection between cognition and emotion.

Many theories implicate structures related to executive function, such as the prefrontal cortex, in the control of affective responses (e.g., Zelazo & Müller, 2002). Generally, these theories propose an inverse relationship between effortful control and negative affect, such that highly intense negative emotions may interfere with the allocation of executive attention and the ability to effectively regulate responses (Evans & Rothbart, 2009). Indeed, evidence from delayed gratification tasks demonstrates that negative emotion can interfere with a child's ability to control behavior, particularly when the child's attention is directly manipulated to focus more on the salient emotional cue or when the motivational significance of the cue is enhanced (Mischel & Baker, 1975; Prencipe & Zelazo, 2005). In an oversimplified application of this framework, the grub worms are quite motivationally significant to Arthur, thereby dampening his cognitive flexibility and reducing opportunities for more adaptive reprocessing of information such as disengaging or reappraisal.

The Iterative Reprocessing Model presents a neural circuitry to explain how strong emotional salience may undermine executive function and result in poorer emotion regulation. This model proposes that there is a bidirectional flow of information from an interconnected neural network, beginning in the thalamus and amygdala and interacting with many areas of prefrontal cortex (as described in more detail in Zelazo & Cunningham, 2007). The importance of this neural model is that information is continually reprocessed and, when it works efficiently, it prompts regulation by amplification or suppression of attention to certain stimuli. Although a strong and negative emotional context may weaken this reprocessing, continued development of the prefrontal cortical networks throughout childhood in typical populations corresponds to improvements in the ability to reprocess information even in more emotionally salient situations (Zelazo, Qu, & Kesek, 2010). The idea of this process being disrupted in ASD would be consistent with the now generally accepted notion of underdeveloped connectivity with the frontal cortex in ASD (Schipul, Keller, & Just, 2011). Thus for Arthur, the initial salience of the grub worms that caused his amygdala to react—or potentially overreact—may be maintained through a failure of the connection with the prefrontal cortex and the reprocessing of information that it would have influenced.

IMPLICATIONS

If the above models do apply to Arthur's emotional perseveration, then how would this affect the overall conceptualization of his concerns? The implications of deriving a more specific understanding of the mechanisms underlying problematic emotional and behavioral control in ASD include advancing our understanding in the areas of treatment, diagnosis, individual differences, and neurobehavioral underpinnings. Below, we provide examples of how a greater focus on emotion regulation could advance our conceptualization of ASD.

First, interventions, or intervention supplements, that directly target specific aspects of emotion regulation and accompanying neurobiological mechanisms would enable clinicians to better match specific treatments to individuals and would lead to improved response rates (Siegle, Ghinassi, & Thase, 2007). Treatment of problematic emotional responses in ASD is generally nonspecific and often includes multiple psychotropic medications or even hospitalization in more extreme cases (Morgan, Roy, & Chance, 2003). Other common approaches, such as packaged programs to teach verbal individuals with ASDs a language for describing emotions so that they can begin to identify and regulate their emotions (e.g., Buron & Curtis, 2003), make common sense but are untested in research and are not directly connected to any theoretical model or understanding of the mechanism behind the concern.

In general, the most recommended approach for handling uncontrolled emotions and behaviors in ASD includes conducting a functional behavior assessment and applying positive behavioral supports (Baker, 2008; Mazefsky & Handen, 2011). Although this can be successful and is grounded in a long history of applied behavior analysis, it often targets single problematic behaviors and may not result in generalization for long-term emotional stability.

Many aspects of emotion regulation would provide treatment-related information, but Arthur's example suggests a need to clarify the latency, onset, offset, and duration of the emotional responding process in ASD, such as whether there is a sustained negative emotional response. This type of information can help identify the types of interventions needed. For example, a study of young adults with and without dysphoric symptoms found that although both groups exhibited similar initial reactivity and peak positive emotional response to positive stimuli, dysphoric individuals were less able to maintain positive emotions (McMakin, Santiago, & Shirk, 2009). This suggests that common cognitive-behavioral approaches to treating depression—such as learning how to reappraise a situation in a more positive light—may be insufficient, and that there is also a need for a targeted treatment module that teaches strategies for *sustaining* positive affective states (McMakin, Siegle & Shirk, 2010). Gaining more specific information on the time course of emotional responding in ASD may similarly lead to more targeted treatments that would better address the root of the problem and thereby be more likely to generalize across situations.

Another clinical implication of improved understanding of emotion regulation processes in ASD is related to diagnosis. Often, higher-functioning children with ASD first receive treatment in mood or behavioral clinics, while their ASD goes unrecognized and untreated (e.g., Gilmour, Hill, Place, & Skuse, 2004; Towbin, Pradella, Gorrindo, Pine, & Leibenluft, 2005). It is difficult to determine when a child with ASD also has a mood or anxiety disorder, or whether these concerns are better conceptualized as emotion dysregulation that is part and parcel of having an ASD. Many children with ASD receive multiple psychiatric diagnoses (Gjevik, Eldevik, Fjaeran-Granum, & Sponheim, 2011; Leyfer et al., 2006; Siminoff et al., 2008). It is possible that, at least in some cases, this represents mislabeling of impairment related to the ASD itself, which then leads to inappropriate or unnecessary treatments (Mazefsky, Oswald, Eack, Minshew, & Lainhart, submitted).

More research explicitly focused on emotion regulation and reactivity in ASD would help inform diagnostic boundaries. Infant studies demonstrating early emotion-related temperament differences in ASD (Garon et al., 2009) and increasing evidence that ASD is a distributed neural systems disorder (Minshew & Keller, 2010) both suggest that emotion regulation processes would be affected in ASD. Although our suggestion that perseveration is related to possible affective interference with cognitive control is not a concept specific to ASD, it would be important to determine whether it is an integral part of ASD's underlying neural circuitry and behavioral presentation rather than something secondary or comorbid.

However, one would not expect a single emotion regulation or reactivity profile to characterize ASD. Rather, just as symptoms in other domains are highly variable, emotional presentation in ASD is also heterogeneous. For example, some children with ASD are like Arthur and are generally stable unless something triggers a serious negative reaction; others have more blunted emotional reactions overall; and still others are more consistently irritable, anxious, or volatile. Thus emotion regulation research also provides a unique opportunity to better understand individual differences and influences on outcomes. Concepts such as attribution processes, motivation, and self-monitoring have been shown to predict variability in the behavioral expression of the ASD and its underlying neurobiological processes (Mundy, Henderson, Inge, & Coman, 2007), in a manner similar

to how emotion regulation research can be used to explain heterogeneity in presentation and outcome in ASD.

Identifying various emotion regulation styles and impairments in ASD may also help determine ASD's biological basis. Heterogeneity has been a problem for genetics research in ASD, and emotion regulation and activity in the neural circuitry supporting emotion regulation processes may represent an important endophenotype. Further, better understanding of specific emotion deficits may point to particular neural pathways. For instance, as we noted in the example above, perhaps the connections between the limbic system and prefrontal cortex deserve more attention.

In sum, research on emotion regulation processes in ASD could lead to advances in conceptualization of treatments, diagnosis, individual differences, and the biological basis of ASD. We provided an example to demonstrate how to apply emotion regulation theories to concepts typically studied from a strict cognitive angle in ASD. The example focused on perseveration on emotionally arousing stimuli because (a) perseveration is often associated with maladaptive emotional responses and is very interfering; (b) the affective aspects of perseveration have not been considered in ASD; (c) many theories from other populations describe how affect may interfere with and reduce cognitive control and these theories may be relevant to understanding the mechanisms behind perseveration in ASD; and (d) clarifying the time course of emotional responding and associated neural mechanisms in ASD can inform the development of new and specific treatments. This example, as well as the illustrative theories from other populations that we chose, are not exhaustive of the countless potentially relevant theories and bodies of research in affective neuroscience. In fact, it is the complexity of emotion regulation that makes it ideal for providing a window to hone our understanding of maladaptive processes in ASD.

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REFERENCES

- Anderson CJ, Colombo J, Shaddy JD. Visual scanning and pupillary responses in young children with autism spectrum disorder. *Journal of Clinical Experimental Neuropsychology*. 2006; 28:1238–1256.
- Baker, J. Positive strategies for managing and preventing out-of-control behavior: No more meltdowns. Future Horizons; Arlington, TX: 2008.
- Bauminger N. The facilitation of social-emotional understanding and social interaction in high-functioning children with autism: intervention outcomes. *Journal of Autism and Developmental Disorders*. 2002; 32:283–298. [PubMed: 12199133]
- Bradley EA, Isaacs BJ. Inattention, hyperactivity, and impulsivity in teenagers with intellectual disabilities, with and without autism. *Canadian Journal of Psychiatry*. 2006; 51:598–599.
- Buron, KD.; Curtis, M. The incredible 5 point scale. Autism Asperger Publishing Company; Shawnee, KS: 2003.
- Capps L, Kasari C, Yirmiya N, Sigman M. Parental perception of emotional expressiveness in children with autism. *Journal of Consulting and Clinical Psychology*. 1993; 61:475–484. [PubMed: 8326050]
- Capps L, Yirmiya N, Sigman M. Understanding of simple and complex emotions in non-retarded children with autism. *Journal of Child Psychology and Psychiatry*. 1992; 33:1169–1182. [PubMed: 1400699]

- Chambers R, Gullone E, Allen NB. Mindful emotion regulation: An integrative review. *Clinical Psychology Review*. 2009; 29:560–572. [PubMed: 19632752]
- Courchesne E, Townsend J, Akshoomoff NA, Saitoh O, Yeung-Courchesne R, Lincoln AJ, et al. Impairment in shifting attention in autistic and cerebellar patients. *Behavioral Neuroscience*. 1994; 108:848–865. [PubMed: 7826509]
- Dalton KM, Nacewicz BM, Johnstone T, Schaefer HS, Gernsbacher MA, Goldsmith HH, et al. Gaze fixation and the neural circuitry of face processing in autism. *Nature Neuroscience*. 2005; 8:519–526.
- Dawson G. Early behavioral intervention, brain plasticity, and the prevention of autism spectrum disorder. *Development and Psychopathology*. 2008; 20:775–803. [PubMed: 18606031]
- Evans DE, Rothbart MK. A two-factor model of temperament. *Personality and Individual Differences*. 2009; 47:565–570. [PubMed: 20161172]
- Garon N, Bryson SE, Zwaigenbaum L, Smith IM, Brian J, Roberts W, Szatmari P. Temperament and its relationship to autistic symptoms in a high-risk infant sib cohort. *Journal of Abnormal Child Psychology*. 2009; 37:59–78. [PubMed: 18704676]
- Gilmour J, Hill B, Place M, Skuse D. Social communication deficits in conduct disorders: A clinical and community survey. *Journal of Child Psychology and Psychiatry*. 2004; 45:967–978. [PubMed: 15225339]
- Gjeverik E, Eldevik S, Fjæraan-Granum T, Sponheim E. Kiddie-SADS reveals high rates of DSM-IV disorders in children and adolescents with autism spectrum disorders. *Journal of Autism and Developmental Disorders*. 2011; 41:761–769. [PubMed: 20824493]
- Gross, JJ. Emotion regulation. In: Lewis, M.; Haviland-Jones, JM.; Barrett, LF., editors. *Handbook of emotions*. 3rd ed. Guilford; New York: 2008.
- Gross, JJ.; Thompson, RA. Emotion regulation: Conceptual foundations. In: Gross, JJ., editor. *Handbook of emotion regulation*. Guilford; New York: 2007.
- Harms MB, Martin A, Wallace GL. Facial emotion recognition in autism spectrum disorders: A review of behavioral and neuroimaging studies. *Neuropsychology Review*. 2010; 20:290–322. [PubMed: 20809200]
- Hill E, Berthoz S, Frith U. Brief report: Cognitive processing of own emotions in individuals with autistic spectrum disorder and in their relatives. *Journal for Autism and Developmental Disorders*. 2004; 34:229–235.
- Kleinhans NM, Richards T, Weaver K, Johnson LC, Greenson J, Dawson G, Aylward E. Association between amygdala response to emotional faces and social anxiety in autism spectrum disorders. *Neuropsychologia*. 2010; 48:3665–3670. [PubMed: 20655320]
- Laurent AC, Rubin E. Challenges in emotional regulation in Asperger syndrome and high-functioning autism. *Topics in Language Disorders*. 2004; 24:286–297.
- Leekam SR, Lopez B, Moore C. Attention and joint attention in preschool children with autism. *Developmental Psychology*. 2000; 36:261–273. [PubMed: 10749083]
- Leyfer OT, Folstein SE, Bacalman S, Davis NO, Dinh E, Morgan J, et al. Comorbid psychiatric disorders in children with autism: Interview development and rates of disorders. *Journal for Autism and Developmental Disorders*. 2006; 36:849–861.
- Mazefsky, CA.; Handen, BL. Addressing behavioral and emotional challenges in school-aged children with ASD. In: Lubetsky, M.; McGonigle, J.; Handen, B., editors. *Autism spectrum disorder*. Oxford University Press; 2011.
- Mazefsky, CA.; Oswald, DP.; Minshew, NJ.; Eack, SM.; Lainhart, JE. Is it ASD, a psychiatric disorder, or both? Psychiatric diagnoses in high-functioning adolescents with ASD. (submitted)
- McMakin DL, Santiago CD, Shirk SR. The time course of positive and negative emotion in dysphoria. *Journal of Positive Psychology*. 2009; 4:182–192. [PubMed: 20190879]
- McMakin D, Siegle G, Shirk S. Positive Affect Stimulation and Sustainment (PASS) module for depressed mood: A preliminary investigation of treatment-related effects. *Cognitive Therapy and Research*. 2010:1–10. doi: 10.1007/s10608-010-9311-5.
- Mischel W, Baker N. Cognitive appraisals and transformations in delay behavior. *Journal of Personality and Social Psychology*. 1975; 31:254–261.

- Minshew NJ, Keller TA. The nature of brain dysfunction in autism: functional brain imaging studies. *Current Opinion in Neurology*. 2010; 23:124–130. [PubMed: 20154614]
- Morgan CN, Roy M, Chance P. Psychiatric comorbidity and medication use in autism: A community survey. *Psychiatric Bulletin*. 2003; 27:278–281.
- Mundy PC, Henderson HA, Inge AP, Coman DC. The modifier model of autism and social development in higher functioning children with autism. *Research & Practice for Persons with Severe Disabilities*. 2007; 32:1–16.
- Ozonoff S, Strayer DL, McMahon WM, Filloux F. Executive function abilities in autism and Tourette syndrome: An information processing approach. *Journal of Child Psychology and Psychiatry*. 1994; 35:1015–1032. [PubMed: 7995842]
- Papageorgiou C, Siegle GJ. Rumination and depression: Advances in theory and research. *Cognitive Therapy and Research*. 2003; 27:243–245.
- Pascualvaca DM, Fantie BD, Papageorgiou M, Mirsky AF. Attentional capacities in children with autism: Is there a general deficit in shifting focus. *Journal of Autism and Developmental Disabilities*. 1998; 28:467–478.
- Pelphrey K, Adolphs R, Morris JP. Neuroanatomical substrates of social cognition dysfunction in autism. *Mental Retardation and Developmental Disabilities Research Reviews*. 2004; 10(4):259–271. [PubMed: 15666336]
- Prencipe A, Zelazo PD. Development of affective decision-making for self and other: Evidence for the integration of first- and third-person perspectives. *Psychological Science*. 2005; 16:501–505. [PubMed: 16008779]
- Rieffe C, Terwogt MM, Kotronopoulou K. Awareness of single and multiple emotions in high-functioning children with autism. *Journal of Autism and Developmental Disorders*. 2007; 37:455–465. [PubMed: 16868846]
- Sasson NJ. The development of face processing in autism. *Journal of Autism and Developmental Disorders*. 2006; 36:381–394. [PubMed: 16572261]
- Schipul SE, Keller TA, Just MA. Inter-regional brain communication and its disturbance in autism. *Frontiers in Systems Neuroscience*. 2011; 5:1–11. [PubMed: 21347218]
- Shalom DB, Mostofsky SH, Hazlett RL, Goldberg MC, Landa RJ, Faran Y, et al. Normal physiological emotions but differences in expression of conscious feelings in children with high-functioning autism. *Journal for Autism and Developmental Disorders*. 2006; 36:395–400.
- Siegle GJ, Ghinassi F, Thase ME. Neurobehavioral therapies in the 21st century: summary of an emerging field and an extended example of cognitive control training for depression. *Cognitive Therapy and Research*. 2007; 31:235–262.
- Siegle GJ, Steinhauer SR, Thase ME, Stenger A, Carter CS. Can't shake that feeling: Event-related fMRI assessment of sustained amygdala activity in response to emotional information in depressed individuals. *Biological Psychiatry*. 2002; 51:693–707. [PubMed: 11983183]
- Siminoff E, Pickles A, Charman T, Chandler S, Loucas T, Baird G. Psychiatric disorders in children with autism spectrum disorders: Prevalence, comorbidity, and associated factors in a population-derived sample. *Journal of the American Academy of Child and Adolescent Psychiatry*. 2008; 47:921–929. [PubMed: 18645422]
- Thompson, RA. Emotion regulation: A theme in search of definition. In: Fox, NA., editor. *Monographs of the Society for Research in Child Development*. Vol. 59. University of Chicago Press; Chicago: 1994. p. 25–52.
- Towbin KE, Pradella A, Gorrindo T, Pine DS, Leibenluft E. Autism spectrum traits in children with mood and anxiety disorders. *Journal of Child and Adolescent Psychopharmacology*. 2005; 15:452–464. [PubMed: 16092910]
- White SW, Oswald D, Ollendick T, Scahill L. Anxiety in children with autism spectrum disorders. *Clinical Psychology Review*. 2009; 29:216–229. [PubMed: 19223098]
- Yirmiya N, Sigman MD, Kasari C, Mundy P. Empathy and cognition in high-functioning children with autism. *Child Development*. 1992; 63:150–160. [PubMed: 1551323]
- Zelazo, PD.; Cunningham, W. Executive function: Mechanisms underlying emotion regulation. In: Gross, J., editor. *Handbook of emotion regulation*. Guilford; New York: 2007. p. 135–158.

- Zelazo, PD.; Müller, U. Executive function in typical and atypical development. In: Goswami, U., editor. *Handbook of childhood cognitive development*. Blackwell; Oxford, England: 2002. p. 445-469.
- Zelazo, PD.; Qu, L.; Kesek, AC. Hot executive function: Emotion and the development of cognitive control. In: Calkins, SD.; Bell, MA., editors. *Child development at the intersection of emotion and cognition*. American Psychological Association; Washington, DC: 2010. p. 97-111.