Journal of Autism and Developmental Disorders (2018) 48:3858–3870
https://doi.org/10.1007/s10803-018-3647-1

ORIGINALPAPER

Intrinsic and Extrinsic Predictors of Emotion Regulation in Children with Autism Spectrum Disorder

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Published online: 20 June 2018

Abstract
Difficulties regulating emotion have been linked to comorbid psychopathology in children with autism spectrum disorder (ASD), but little empirical work has examined predictors of dysregulation in this population. Forty-six families of children with ASD participated in a laboratory visit that included direct measurement of children's IQ, ASD symptoms, and psychophysiological reactivity. Child emotion regulation was observed during independent and co-regulatory tasks, and parental scaffolding was rated in the dyadic context. ASD symptom severity emerged as the strongest predictor of child emotion dysregulation across contexts. Child age and parental scaffolding also uniquely predicted child dysregulation in the dyadic task. Implications for conceptualizing intrinsic and extrinsic influences on emergent emotion regulation in children with ASD are discussed, as are applications to intervention.

Keywords Autism spectrum disorder · Emotion regulation · Co-regulation · Autism symptomatology · Psychophysiology · Intellectual disability

Introduction

Diagnostic criteria for autism spectrum disorder (ASD) focus on core deficits in social communication and the presence of restricted and repetitive behaviors (American Psychiatric Association 2013). However, individuals with ASD also experience high rates of co-occurring emotional and behavioral challenges (Brereton et al. 2006; Georgiades et al. 2013), which has prompted efforts to better understand mechanisms underlying heterogeneity in this population. Emotion regulation, or the ability to modulate emotional states to achieve a goal (Gross 2008; Thompson 1994), has been posited to play a central role in the manifestation of individual differences in core ASD-related symptomatology and comorbid problems (Mazefsky et al. 2013, 2012; Mazefsky and White 2014; Weiss 2014; White et al. 2014).

Emotion Regulation in ASD

Research on emotion regulation in individuals with ASD has generally focused on group differences in regulatory strategies between individuals with ASD and those with neurotypical development (Jahromi et al. 2012; Mazefsky et al. 2014; Nuske et al. 2017; Samson et al. 2015, 2012). Findings generally suggest that children with ASD utilize fewer adaptive emotion regulation strategies in independent regulatory contexts than do children without ASD. Specifically, children with ASD have been observed to display greater resignation, avoidance, and venting behaviors in the context of frustrating tasks (Jahromi et al. 2012), and less use of cognitive reappraisal as a response to problem-solving vignettes than children with neurotypical development (Samson et al. 2015). Children and adolescents with ASD also self-report higher levels of rumination, avoidance, and emotion suppression (Mazefsky et al. 2014; Patel et al. 2017; Samson et al. 2012), and reduced frequency of a host of adaptive strategies, including approach-oriented coping, cognitive

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reappraisal, relaxation, and acceptance (Rieffe et al. 2014; Samson et al. 2015, 2012).

Although evidence suggests greater reliance on maladaptive strategies among children with ASD, studies have rarely tested assumptions regarding associations between purported regulatory efforts and indices of actual regulation quality, represented by certain parameters of emotion process (e.g., lability, intensity, duration, and soothability) and associated behaviors (Cole et al. 2004, 1994; Thompson 1994). In a notable exception, Jahromi et al. (2012) used sequential analyses and revealed that regulatory strategies were significantly less effective in promoting reduction of negativity and resignation for children with ASD than for children with neurotypical development. These findings are compelling in suggesting that difficulties experienced by children with ASD in modulating negative arousal may stem not only from deficits in strategy selection, but also from difficulties with effective implementation. Further work is needed to enhance understanding of process-level indicators of regulatory quality.

A growing body of research has centered on functional outcomes associated with regulatory difficulties or dysregulation in children with ASD. Specifically, a number of studies have examined emotion regulation as a predictor of internalizing and externalizing problems in this population (Mazefsky et al. 2014; Pouw et al. 2013; Rieffe et al. 2014; Samson et al. 2015). Endorsement of maladaptive regulatory strategies has been associated with internalizing symptoms and problem behaviors in verbally-fluent older children and adolescents with ASD, with rumination figuring prominently in relation to depressive and related internalizing symptoms (Mazefsky et al. 2014; Patel et al. 2017; Rieffe et al. 2014). More broadly, several studies have implicated parent-reported emotion dysregulation in the manifestation of anxiety and difficulties with peer interaction in individuals with ASD (Berkovits et al. 2017; Jahromi et al. 2013; Swain et al. 2015; White et al. 2014). In contrast, children's ability to generate adaptive regulatory strategies in response to hypothetical scenarios has been linked to reduced parent-reported externalizing behavior problems in this population (Ting and Weiss 2017).

Intrinsic Predictors of Emotion Regulation in ASD

ASD Symptoms

Multiple factors have been theorized to underlie regulatory difficulties in children with ASD, including core ASD-related symptomatology and associated social-cognitive and executive functioning deficits. As outlined by Mazefsky, White, and colleagues (see Mazefsky et al. 2013; Mazefsky and White 2014; White et al. 2014 for reviews), characteristics commonly associated with, or intrinsic to, ASD likely predispose children to experiencing challenges with emotion regulation. For example, emotion understanding, perspective taking, and executive functioning are closely intertwined with emotion regulation in children with neurotypical development (Fox and Calkins 2003; Izard et al. 2011; Zelazo and Cunningham 2007), and these domains are recognized areas of difficulty for children with ASD (Geurts et al. 2014; Heerey et al. 2003; O'Hearn et al. 2008). Children with ASD also often exhibit problems with abstract reasoning, weak central coherence, and behavioral rigidity (D'Cruz et al. 2013; Pellicano et al. 2006; Williams et al. 2006), which may impede accurate assessment of situational contexts and effective enactment of regulatory efforts. Moreover, core ASD-related deficits in social communication and the presence of repetitive behaviors may challenge the delivery of vital external co-regulatory support. Transactional effects are likely, with emotion regulation difficulties further exacerbating underlying vulnerabilities in this population.

Given the pervasiveness of problems with emotion regulation in individuals with ASD, clarifying the nature of the relationship between ASD symptoms and emotion dysregulation has the potential to inform understanding of foundational developmental processes as well as approaches to clinical intervention. However, few investigations have empirically examined predictors of individual differences in regulatory competence in this population, and the handful of existing studies have yielded inconsistent results. Several studies examining observed and self-reported regulatory strategies have not found associations with ASD symptom severity, as measured by observer ratings and parent report (Konstantareas and Stewart 2006; Patel et al. 2017). In contrast, parent report of global emotion regulation and of certain internalizing and externalizing behavior problems thought to be indicative of dysregulation have been linked to parent-reported ASD symptoms (Berkovits et al. 2017; Samson et al. 2014). Advancing this line of research using more direct assessments that eliminate shared informant variance and increase the specificity of the emotion regulation construct is of particular importance.

Intellectual Functioning

Level of intellectual functioning may be related to differences in neural circuitry that are implicated in self-regulation in children with ASD (Bachevalier and Loveland 2006). Although IQ has occasionally been considered as a covariate (Jahromi et al. 2013; Samson et al. 2015; Ting and Weiss 2017), this construct has rarely been featured in research on emotion regulation in this population. Existing work has predominantly included samples characterized by intellectual functioning in the low-average range or above,

with inclusion criteria often requiring a minimum measured IQ score of 80. In these studies, associations between IQ and emotion regulation generally either have not been reported or have been found to be non-significant, perhaps due to truncated range (Jahromi et al. 2012; Ting and Weiss 2017). Of those studies employing more cognitively diverse samples of children with ASD, most investigations have not found IQ to be a significant predictor of self- or parent-reported regulation and relevant behavior problems (Berkovits et al. 2017; Samson et al. 2015, 2014). However, methodology may be a significant factor, given that some evidence suggests positive associations between scores on a developmental screener and observed adaptive regulatory strategies in children with ASD (Konstantareas and Stewart 2006). The relationship between intellectual functioning and regulatory skills in this population remains a central and understudied research question.

Chronological Age

Theoretical accounts propose a developmental progression in children's emotion regulation abilities characterized by greater complexity, effectiveness, and independence over time, which parallels corresponding maturational advances in cognition and neurobiology, as well as the changing nature of social influences (Calkins 1994; Cole et al. 1994; Kopp 1982, 1989; Thompson 2011). Findings from a handful of longitudinal studies provide evidence to support age-related improvement in the selection and use of regulatory strategies over time for children with neurotypical development as well as children with general developmental delays, although the latter group continue to show comparative deficits (Gerstein et al. 2011; Gullone et al. 2010; Mangelsdorf et al. 1995; Vaughn et al. 1984). Some findings from studies of children and adolescents with ASD suggest the potential for age-related increases in the quality of self-reported regulatory strategies (Samson et al. 2015) whereas other results indicate non-significant associations between chronological age and self-reported regulation (Cai et al. 2018; Pouw et al. 2013). In addition, a study utilizing observational measures of emotion regulation found age to be significantly less predictive of regulatory processes for children with ASD than children with neurotypical development (Konstantareas and Stewart 2006). For children with ASD, the potential influence of chronological age upon regulatory processes is likely complex due to wide variation in intellectual functioning, ASD symptom severity, and related contributing characteristics.

Psychophysiological Reactivity

Efforts to understand psychobiological components of emotion processing in individuals with ASD have increasingly turned to measures of psychophysiology. Findings in this area are decidedly mixed and vary considerably depending upon the chosen physiologic index, methodology, and outcome measurement (Lydon et al. 2016; White et al. 2014). Electrodermal activity (EDA), a measure of sympathetic nervous system activation related to the behavioral inhibition system (BIS), has emerged as a promising marker of individual differences. Various metrics have been utilized, including baseline EDA, EDA response to discrete stimuli, and more recently, the frequency of nonspecific fluctuations in skin conductance responses (NSCRs; Boucsein et al. 2012; Beauchaine et al. 2015). The latter has emerged as an especially useful metric when examining symptom presentation in individuals with ASD. Heightened overall EDA variability as measured by NSCRs has been found to predict greater ASD symptom severity (Fenning et al. 2017) whereas hypoactivation, particularly during compliance contexts, may be a marker of risk for comorbid externalizing problems (Baker et al. 2018). Although much remains to be learned about associations between EDA and symptomatology in children with ASD, even less is understood about relations with the potentially more proximal construct of emotion regulation.

Extrinsic Predictors of Emotion Regulation in ASD

Extrinsic influences on children's emotion regulation, particularly in the form of parental co-regulatory support, have long been recognized as central to the development of children's independent regulatory abilities (Cole et al. 1994; Kopp 1982, 1989; Morris et al. 2007; Thompson and Meyer 2007). Direct assistance in managing emotional arousal may be especially critical for children with developmental problems given heightened vulnerability to dysregulation in this population and the potential for parenting behavior to exert stronger influence upon social-emotional functioning for children with developmental vulnerabilities than for children with neurotypical development (Baker et al. 2007; Guralnick 1999). Indeed, mothers' ability to effectively scaffold their children's modulation of emotion through provision of sensitive structuring and emotional and motivational support has been linked with observed regulation quality and important social skills outcomes in children with general developmental delays (Baker et al. 2007).

Investigations have begun to focus on the role of parents in regulatory processes in children with ASD. Emerging evidence suggests that parental presence may be conducive to regulation in young children with ASD, even in the absence of direct co-regulatory support (i.e., as when the parent is rendered unavailable by requirements of a laboratory protocol; Jahromi et al. 2012). Of the few studies to examine co-regulation, one study of toddlers revealed associations between parental co-regulatory support and observed

response to distress in a dyadic free-play context (Gulsrud et al. 2010). However, a study of school-aged children did not find expected relations between the quality and form of co-regulation during a parent–child emotion discussion task and children's regulation strategies generated independently in response to hypothetical vignettes (Ting and Weiss 2017). Discrepancies underscore the importance of further examining child characteristics, particularly child age, and measurement context when considering the role of co-regulation in the emergence of regulatory skills in children with ASD.

Current Study

Theoretical conceptualizations of emotion regulation emphasize the importance of differentiating activated emotion and putative regulatory strategies from indices of regulatory quality (Cole et al. 2004, 1994; Thompson 1994). Although existing research on individuals with ASD has enhanced understanding of regulatory strategies and associated implications for comorbid symptomatology, relatively little attention has been devoted to characterizing process-level indicators or to examining predictors of emotion regulation in this population. In addition, most investigations have relied upon a single measure of emotion regulation in individuals scoring within the normative range of intellectual functioning (Weiss et al. 2014).

The current investigation directly addressed these areas of need by examining predictors of global features of emotion regulation in a developmentally-diverse sample of children with ASD using a multi-method approach. Specifically, we tested the prediction of child characteristics (age, intellectual functioning, ASD symptom severity, and psychophysiological reactivity) and parental scaffolding to observed emotion dysregulation in independent and co-regulatory contexts. We expected that children would display greater dysregulation in independent relative to dyadic contexts. Building upon conceptualizations of emotion regulation as central to ASD (Mazefsky et al. 2013), we anticipated that ASD symptom severity would emerge as the strongest predictor of observed dysregulation and would predict beyond the effects of other important intrinsic and extrinsic factors. Parental scaffolding was hypothesized to emerge as an important inverse predictor of dysregulation, particularly in contexts providing opportunities for co-regulatory support.

In order to address the nuances of the association between IQ and emotion dysregulation, we tested the relationship using a continuous measure of IQ and a metric dichotomized according to the IQ criterion for intellectual disability (ID; APA 2013), the latter of which we expected to be a more powerful predictor. Drawing upon work suggesting inverse associations between intellectual functioning and ASD symptom severity (Lecavalier et al. 2011; Matson and Shoemaker 2009) and evidence of important differences in the regulation profiles of children with significant cognitive delays and children with neurotypical development (Baker et al. 2007; Norona and Baker 2014), we expected that intellectual functioning would be negatively related to dysregulation in children with ASD. Specifically, we expected that IQ would be inversely predictive of dysregulation, and that children meeting the IQ criterion for ID would display greater dysregulation than those with measured IQ in the normative range.

Regarding psychophysiology, it was anticipated that greater EDA reactivity would be positively related to observed emotion dysregulation. This hypothesis was informed by evidence of associations between increased EDA reactivity and ASD symptom severity in children with ASD (Fenning et al. 2017). Previous findings have also linked heightened EDA reactivity with observed emotionality and subsequent behavior problems in preschool-aged children (Cole et al. 1996), a pattern that Cole and colleagues referred to as suggesting potential "emotional impulsivity" and "underregulation."

Method

Participants

The present sample included 46 children between the ages of 4 and 11 years and their primary caregivers (see Table 1 for demographics; only one child was 11 years of age). Children with an existing diagnosis of ASD provided by a physician or psychologist were recruited from the community. One participating child was reported to have a chromosomal deletion (region 7q35), which has been implicated in risk for

Table 1 Sample demographic information (n = 46)

Variable	
Child	
Mean age	6.39 (1.95)
Mean estimated IQ	84.73 (22.71)
Range estimated IQ	47–139
Male (%)	80%
Race/ethnicity	
Caucasian, non-hispanic	44%
Caucasian, hispanic	24%
Asian American	11%
African American	9%
"Other"	11%
Primary caregiver	
Married (%)	85%
Father was primary caregiver (%)	5%
Median annual family income	US $50,000–$70,000

ASD in emerging research (Peñagarikano and Geschwind 2012); no other participating children were reported to have underlying genetic variants or neurological disorders of known etiological significance to ASD. Exclusionary criteria for the child also included measured IQ below 40 (to address task validity considerations) and motor impairment that would prevent independent ambulation.

Procedures

All procedures were approved by our institutional review board. After obtaining consent from parents and assent from the children, wireless EDA sensors were placed on the right wrist of each child, as recommended for the current purpose (Picard et al. 2016). A short introductory period followed in which the child became accustomed to the sensors while the experimenter presented the parent with an overview of research procedures. Dyads then engaged in a series of established laboratory tasks, which included our focal co-regulatory and independent frustration tasks. The laboratory visit concluded with direct assessments of children's intellectual functioning and ASD-related symptoms performed by a licensed clinical psychologist with expertise in ASD (see Fenning et al. 2017 for a complete description of study methods and procedures).

Co-Regulation Task (Baker et al. 2018; Fenning et al. 2017). The parent–child dyad was positioned at the corner of a table, with the parent to the child's left. The dyad was provided with 32 colorful block tiles and a photo of a completed fish puzzle. The child was instructed to make the structure depicted in the photo. The parent was asked to let the child try it on his or her own, and then to provide any help that the parent deemed necessary. The experimenter returned after 4 min.

Independent Regulation Task (Fenning et al. 2017; Goldsmith et al. 2001; Jahromi et al. 2012). Children participated in a 5-min "locked box" independent frustration task. The child was asked to select a favorite item from a variety of prizes and the item was placed in a translucent hard-plastic box, which was then locked with a padlock. The experimenter demonstrated using a key to open the lock and retrieve the prize. The child was given a keychain with 15 visually similar, non-functional keys. The experimenter instructed the child to try to find a key that would open the box and allow him/her to get the toy. After 5 min, the experimenter returned and informed the child that he or she was given incorrect keys. The child was then provided with a set of appropriate keys and he or she had the opportunity to retrieve the prize.

Measures

Child IQ

An estimate of child IQ was obtained using the SB5 ABIQ (Roid 2003). The ABIQ is comprised of two subscales with high loading on *g*. A Matrix Reasoning task assesses non-verbal fluid reasoning and a Vocabulary task evaluates expressive word knowledge. The SB5 has sound psychometric properties, including good test–retest reliability, for preschool- and school-aged children and has demonstrated validity for children with ASD (Fenning et al. 2017; Matthews et al. 2015; Roid 2003). Two variables were derived to represent intellectual functioning in the current study: the continuous ABIQ score (*IQ*) and the ABIQ score dichotomized according to the DSM-5 IQ criterion for ID (IQ < 76; APA 2013), subsequently referred to as *ID range*.

ASD Symptoms

Level of ASD symptoms was assessed through direct testing with the Autism Diagnostic Observation Schedule-2 (ADOS-2; Lord et al. 2012), a semi-structured assessment that facilitates observation and recording of child behaviors related to language, social communication, play, repetitive behaviors, and restricted interests. The ADOS-2 comparison score was used to index overall ASD symptom severity. The comparison score allows for examination of symptomatology across different modules, with 1 indicative of minimal to no evidence of ASD-related symptoms, and 10 reflecting a high level of symptoms.

Electrodermal Activity (EDA)

EDA during the independent and co-regulatory tasks was recorded in microsiemens (*us*) at 8 Hz using wireless *Affectiva* Q-Sensors (Picard et al. 2016). The sensors utilized Ag/AgCl dry disc electrodes and data were recorded and stored within the sensor itself, then processed using Q Software (Affectiva 2014) and SPSS (IBM 2016). The degree of variability of EDA for each child within each task was indexed by non-specific skin conductance responses (NSCRs). NSCRs (Beauchaine et al. 2015; Dawson et al. 2000) were calculated with the use of an algorithm that determined the number of EDA increases of at least .03*us* over a period of three seconds. Data cleaning procedures included visual inspection of NSCRs as well as procedures to detect and omit extreme scores indicative of increases in EDA that occurred too quickly (e.g., > 10–30 *us*/3 s) or exhibited intensities greater than would likely reflect the relevant biological processes (e.g., > 60 *us*). To address the possibility of the sensors momentarily losing connection and mimicking an NSCR upon re-establishing contact, we also omitted any

NSCRs that followed a drop of 1 *SD* (based on a particular child's mean EDA) over the course of 3 s (see also Fenning et al. 2017 for additional details regarding EDA processing). NSCRs derived from the two regulation tasks were highly correlated, $r = .63$, $p < .001$, and were composited to better reflect the conceptualization of EDA as representing trait-like underlying arousal tendencies that could contribute to regulatory behavior. The sensors also recorded movement data across three axes, which were averaged across tasks and considered as a potential covariate.

Scaffolding

Parental co-regulatory support was coded from videotape of the dyadic task, using the Parental Scaffolding Observation System (Hoffman et al. 2006). This system considers parents' ability to provide motivational, emotional, and technical support to their children during a challenging activity. Motivational scaffolding includes the ability of the parent to recruit the child's attention to the task, to foster enthusiasm for the task, and to refocus the child should he or she become distracted. Emotional scaffolding scores reflect the parent's ability to provide co-regulatory emotional support to the child (e.g., through affective attunement, modeling, calming, and/or direct emotion coaching) and to contribute to the child's feelings of accomplishment. Technical scaffolding evaluates the parent's ability to provide structure and support for the child with regard to the task through instruction, guidance, prompting, and/or modification of the task or goal. Each of these subscales are rated from 1 (*very low or absent support*) to 5 (*characteristically high support*). Although these subscales can be used individually, the scores are highly positively correlated and the measure is most commonly used as a single overall score (Baker et al. 2007, 2018). This system has been used in families of children with and without developmental challenges (Hoffman et al. 2006), but scaffolding scores have demonstrated particular utility for children with delays and/or ASD (Baker et al. 2007, 2018; Fenning and Baker 2012; Gulsrud et al. 2010; Ting and Weiss 2017). Inter-rater reliability based on 43% of cases yielded an intraclass correlation coefficient (ICC) of .73.

Emotion Dysregulation

The quality of child emotion dysregulation was coded from videotape using the Dysregulation Coding System (Hoffman et al. 2006). Adapted from parameters presented by Cole and colleagues (Cole et al. 1994), the system was designed to measure key indices of dysregulation/regulation, including overall lability and soothability as well as the appropriateness of the type, duration, and intensity of emotional expressions. The system also measures the behavioral manifestation of poor regulatory control that impedes task engagement.

The overall Dysregulation score ranges from 0 (*no evidence of dysregulation*) to 4 (*significant dysregulation*). A score of 1 reflects a low degree of dysregulation and describes individuals who: (a) displayed only one or two brief emotional expressions that were inappropriate to the situation and who were able to regroup or (b) displayed one or two brief instances of emotional lability and/or variability in intensity of emotional expression and recovered quickly from inappropriate emotional experiences. Behaviorally, a child receiving a score of 1 displayed only one or two brief interfering behaviors during the segment, with no instances of intense behavior disruption. In contrast, a score of 4 was provided to children showing significant dysregulation evidenced by several intense emotional expressions or less intense but frequent emotional expressions for the majority of the segment. Children receiving a score of 4 were virtually unable to regroup and were very labile, showing extreme variability in the intensity of emotion and/or very slow recovery from emotional experiences. The children who received the highest rating also displayed several intense disruptive behaviors or displayed less intense, but frequent disruptive behaviors for the majority of the segment.

Validity of the system is supported by prior studies of preschool- and school-aged children with and without neurodevelopmental disorders (Baker et al. 2007; Hoffman et al. 2006; Norona and Baker 2014). Specifically, ratings of dysregulation have been linked with child externalizing problems (Hoffman et al. 2006), and have been found to partially mediate associations between early developmental delay and later social skills (Baker et al. 2007). Interrater reliability for the present study was based upon 30% of tapes and resulted in an ICC of .90. Although coding for the two different tasks occurred months apart, a portion of the same coders rated dysregulation for both tasks. All coders were blind to the children's scores on all other variables.

Results

Mean child IQ fell within the borderline range of intellectual functioning, with 36% of children exhibiting IQ scores within the ID range (< 76; APA 2013). The sample was diverse with regard to race/ethnicity and income level, and demonstrated, on average, overall ASD symptom levels in the moderate to high range based upon the ADOS-2 comparison score. One family needed to leave prior to administration of assessments of estimated intellectual functioning and ASD-symptom severity. Emotion regulation data for two additional children were excluded from final analyses because the children did not appear to adequately understand the goal of the independent frustration task. Given that the

Table 2 Descriptive statistics and correlations among variables of interest

	Valid *n*	1	2	3	4	5	6	7	Mean (*SD*)
1. Age (in months)	46	–							81.51 (24.18)
2. Intellectual functioning (IQ)	45	−.04	–						84.73 (22.71)
3. ID range	45	−.11	−.80***	–					.36 (.48)[a]
4. ASD symptom level	45	.03	−.26+	.28+	–				7.20 (2.17)
5. Electrodermal activity (EDA)	45	−.05	.04	.00	.13	–			.02 (.94)
6. Scaffolding	44	−.06	.23	−.18	−.11	−.19	–		3.33 (1.12)
7. Dysregulation: independent	44	−.08	−.08	.24	.44**	.11	−.13	–	1.83 (1.11)
8. Dysregulation: dyadic	44	−.24	−.33*	.20	.38*	.13	−.38*	.36*	1.02 (1.21)[b]

ID intellectual disability, *ASD* autism spectrum disorder

+$p < .10$; *$p < .05$; **$p < .01$; ***$p < .001$

[a]The distribution of ID range was dichotomous according to the DSM-5 IQ criterion: 64% No ID (Code = 0), 36% ID (Code = 1)

[b]Descriptive information reflects unstandardized and uncorrected data

amount of missing data was minimal and that missing data involved the primary predictor or outcome variables, a decision was made not to estimate these data, rendering a sample size of 43 for the final regression analyses.

Table 2 presents descriptive statistics and bivariate correlations among the primary variables of interest. Demographics including child gender and race/ethnicity, family income, and marital status were not significantly related to child emotion dysregulation and were not considered in further analyses. However, sensor movement, averaged across tasks, was related to child EDA ($r = .44$, $p < .01$) and to dysregulation during the independent regulatory task ($r = .33$, $p < .05$), and was therefore controlled in the relevant regression. Data for dysregulation during the parent–child problem solving task exhibited significant kurtosis; the variable was standardized and corrected using a square root transformation.

Children displayed significantly higher levels of dysregulation during the independent regulation task in comparison to the dyadic co-regulation task, $t(43) = 6.61$, $p < .001$. As expected, ASD symptom severity was positively related to observed emotion dysregulation across contexts. ASD symptom severity was also linked with IQ and functioning in the ID range at the level of a trend, with children in the ID range displaying higher levels of ASD symptoms in comparison to children scoring above the IQ criterion for ID, $t(43) = -1.89$, $p = .07$, $d = .62$. No significant relations emerged between IQ or functioning in the ID range and dysregulation in the independent task. However, IQ was inversely correlated with observed child dysregulation in the dyadic parent–child context. Subsequent analyses therefore included the continuous measure of IQ rather than ID range. Parental scaffolding was significantly associated with dysregulation in the dyadic task. No significant bivariate

Table 3 Linear regressions predicting child dysregulation (n = 43)

	Dysregulation					
	B	*SE*	*β*	*t*	*p*	R^2
Independent task						.34
Sensor movement	.63	.25	.38	2.53	.02	
Age	−.01	.01	−.22	−1.53	.14	
IQ	.00	.01	.05	.37	.71	
ASD symptom level	.24	.07	.48	3.38	.00	
Electrodermal activity	−.19	.18	−.15	−1.00	.32	
Scaffolding	−.09	.14	−.09	−.63	.53	
Co-regulatory task						.39
Age	−.01	.01	−.32	−2.47	.02	
IQ	−.01	.01	−.23	−1.69	.10	
ASD symptom level	.15	.06	.34	2.51	.02	
Electrodermal activity	−.03	.15	−.02	−.17	.87	
Scaffolding	−.27	.12	−.30	−2.28	.03	

ASD autism spectrum disorder

associations emerged with child age or with psychophysiological reactivity as indexed by EDA.

Two linear regressions were performed to examine hypotheses regarding predictors of child dysregulation in independent and co-regulatory contexts. As seen in Table 3, ASD symptom severity was the strongest predictor of observed emotion dysregulation across contexts. Other than the movement covariate, ASD symptom level emerged as the only significant correlate of dysregulation in the independent frustration task. Given that movement may better represent an effect of dysregulation, a conservative estimate of the explanatory power of the hypothesized predictors is almost one quarter of the total variance ($R^2 = .22$). In contrast, greater ASD symptom severity, younger age, and lower quality scaffolding were all associated with higher levels of dysregulation in the dyadic context, accounting for over one-third of the variance. Of note, substitution of ID range for continuous IQ in these regressions did not affect the nature or pattern of significant findings.

Due to the relatively large age range of our sample, we conducted post-hoc analyses to determine whether the observed significant association between ASD symptoms and emotion dysregulation differed for older and younger children. An interaction term between ASD symptoms and child age was entered into the regression equations and was not significant, (independent dysregulation $\beta = -.06$, $t = -.41$, $p = .68$; co-regulated dysregulation $\beta = -.14$, $t = -1.04$, $p = .31$). Furthermore, performing analyses within subgroups of children either older or younger than the sample median of 6.5 years ($n's = 21, 22$) revealed similar coefficients for the two age groups (independent dysregulation: younger $\beta = .58$, older $\beta = .37$; co-regulated dysregulation: younger $\beta = .44$ and older $\beta = .31$). Together, these analyses suggest that the link between ASD symptoms and child dysregulation did not appear to differ significantly between older and younger children.

Discussion

The current study adopted a multi-method approach to examine predictors of observed emotion regulation quality in children with ASD, with a focus on the role of child characteristics and parental co-regulatory support. Consistent with a growing consensus on the centrality of emotion dysregulation to the presentation of children with ASD (Mazefsky et al. 2013, 2012; Mazefsky and White 2014; Weiss 2014; White et al. 2014), our results revealed a significant positive association between children's level of ASD symptomatology derived through direct testing and observed emotion dysregulation in both independent and co-regulatory contexts. ASD symptom severity emerged as the strongest predictor of dysregulation, accounting for meaningful variance above and beyond the contributions of other important intrinsic and extrinsic influences. By directly testing an association often assumed but rarely explicitly examined, the present study significantly advances empirical understanding of the close connection between core symptomatology and the manifestation of regulatory behavior in children with ASD. Importantly, our methodology also extends prior work by utilizing a gold-standard direct assessment of ASD symptom severity and an observational measure of emotion dysregulation across independent and co-regulatory contexts, which addresses concerns regarding shared informant variance and the use of less-specific proxies for regulation quality in previous investigations.

Results also highlight the significance of contextual considerations, with children exhibiting more dysregulation when alone than in a co-regulatory context. During the independent regulation task, ASD symptoms emerged as the only significant correlate of dysregulation aside from the movement covariate, whereas additional child characteristics and parent behavior were also predictive in a co-regulatory setting. Specifically, higher quality parental scaffolding was associated with less child dysregulation during the dyadic task, providing further evidence that effective parental scaffolding may significantly assist children with ASD in modulating emotion. However, the benefits of parental co-regulation emerged largely in the moment and were not predictive of independent regulation, suggesting that children with ASD may have difficulty internalizing and generalizing co-regulatory support. It is likely that delay or deviance in the trajectory of emerging regulation may prolong reliance upon the social environment for co-regulatory support in this population. Indeed, many children with ASD benefit from the presence of one-to-one support for regulatory management in learning settings well into middle childhood and beyond. The present findings provide empirical support for this apparent developmental need and underscore the importance of research into the development of strategies for promoting internalization of regulatory processes in children with ASD.

Although clearer evidence of an association between scaffolding and emotion dysregulation in the dyadic task is consistent with theory and study hypotheses, it is important to acknowledge the possibility that shared measurement may have contributed to this finding. Furthermore, the order of the tasks was not counter-balanced. Given that the co-regulatory activity preceded the independent task, children's level of dysregulation in the latter context could have been influenced by a depletion of resources. Relations between parental scaffolding and child emotion regulation are likely bidirectional, with prior studies suggesting that parental co-regulatory strategies appear responsive to the developmental needs of children with ASD (Gulsrud et al. 2010). It will be important for future studies to further address the relative

effect of intrinsic and extrinsic factors on the manifestation of regulatory behavior across contexts, particularly with respect to the relationship between independent and co-regulatory processes in children with ASD. In addition, it may be fruitful to consider the extent to which the regulatory dimensions coded in the present study, particularly those related to lability and soothability, might be separable and differentially influenced by child characteristics and certain forms of external support.

In the present study, IQ was not uniquely predictive of emotion dysregulation, and age only emerged as a significant predictor of dysregulation in the co-regulatory context. If the development of emotion regulation in children with ASD follows the traditional trajectory of a progression from co-regulated support to independent regulatory control, but reflects significant delay, greater variance associated with age may have been observed in the co-regulation task precisely because this is the initial context within which regulatory skills emerge. From this perspective, age effects might emerge in independent regulatory contexts later in development for children with ASD. Although children in our study ranged in chronological age from 4 to 11, the majority of children were around 6 years of age, and a slightly older sample might reveal more age-related influences. It is also possible that co-regulatory support may temper the predominance of ASD symptomatology, allowing for other influences to emerge. Indeed, although the correlations were not significantly different, the association between ASD symptom severity and dysregulation was higher in the independent task. Such contextual effects may help to explain inconsistent findings in prior research.

In addition, evidence that the association between ASD symptom severity and child dysregulation was similar in magnitude for older and younger children in our study suggests that ASD symptoms may be a salient and sensitive predictor of regulatory quality across a relatively wide age range. It is important to note that though the observational system utilized in the present investigation has been employed with school-aged children (Norona and Baker 2014), it has most often been implemented in studies of preschoolers (e.g., Hoffman et al. 2006; Baker et al. 2007), and it will be important to further validate the system, especially for older school-aged children with ASD. Examining potential transactions between chronological age and intellectual functioning when considering contextual influences and trajectories over time will also be critical. Improved understanding of the predictors of emotion regulation has great potential to enhance intervention efforts, which are increasingly focused on addressing dysregulation in this population (Thomson et al. 2015). Greater insight into core developmental processes may also inform decisions regarding optimal treatment targets and therapeutic modality (e.g., individual versus dyadic or family-focused approaches).

The inclusion of children with a broad range of intellectual functioning permitted both a dimensional and a categorical approach. Present results were similar in direction and magnitude when considering the clinically-meaningful threshold of functioning in the ID range and a continuous measure of IQ. Although IQ was inversely correlated with dysregulation in the dyadic context, ASD symptom severity was by far the most salient and significant predictor of regulatory outcomes, and IQ did not uniquely predict dysregulation in regression analyses. It will be important to continue to emphasize research involving children with diverse developmental profiles in order to advance understanding of the complex relationship between cognition, symptomatology, and regulation in children with ASD.

Psychophysiological reactivity was not significantly related to observed emotion dysregulation in the present study. The current investigation represents one of the first studies of EDA in relation to emotion regulation in children with ASD, and it is important to address considerations related to methodology in attempting to understand these null results. This study used the relatively novel metric of NSCRs to index physiological reactivity, which permitted assessment of EDA variability over the course of structured laboratory tasks. Although this metric has demonstrated good psychometric properties and relates meaningfully to other important outcomes in children with ASD (Baker et al. 2018; Fenning et al. 2017), many previous studies have employed baseline examination of EDA or change in EDA following discrete stimuli (Lydon et al. 2016; White et al. 2014). It is possible that the latter approach focused on magnitude rather than variability in physiological responding may be particularly informative when considering regulatory functioning. Future research would benefit from considering multiple indicators of physiological response in order to better understand measurement implications. Although previous studies that included a large portion of the current sample did identify moderate to high correlations for EDA across laboratory tasks, some evidence for task-specificity in relation to other variables was observed (Baker et al. 2018; Fenning et al. 2017). The current study measured EDA during independent and co-regulatory tasks involving problem-solving with manipulatives, which may activate different regulatory processes than the social stress, mood induction, and experimental procedures that have often characterized the broader literatures examining EDA in the context of regulation (Cole et al. 1996; Egloff et al. 2006; Gross 1998). Protocols that facilitate cross-context assessment of regulatory processes will continue to be helpful in advancing this area of research.

Lastly, EDA is generally considered to reflect arousal reactivity, and other physiological indices may represent processes more proximal to emotion regulation. Respiratory

sinus arrhythmia (RSA) is an index of parasympathetic activity that has emerged as a potential biomarker of poor regulation across diagnostic groups (Beauchaine 2015) and has been linked with emotion recognition, social functioning, and internalizing and externalizing symptoms in children with ASD (Bal et al. 2010; Guy et al. 2014; Neuhaus et al. 2014; Van Hecke et al. 2009; White et al. 2014). Integrating measurement of both sympathetic and parasympathetic activity in future studies will further enhance understanding of regulatory processes in children with ASD.

The intrinsic and extrinsic factors examined in the present study accounted for a considerable proportion of variance in observed emotion dysregulation. Nonetheless, meaningful variance remained unexplained, especially in the independent regulatory context. In addition to the aforementioned considerations, further exploration of the role of child executive functioning, social cognition, and neuroanatomical factors as well as other parental influences, including broader indices of parenting behavior, socialization practices, and disciplinary approaches, may hold additional promise for extending this line of inquiry.

The present study is not without limitations. In addition to issues mentioned previously, a primary consideration relates to sample size, which was adequate for the analyses performed, but limited our ability to consider more complex modeling. A longitudinal perspective would also offer important information regarding transactional effects and individual differences in the unfolding of emotion regulation processes over time.

Acknowledgments This project was supported by intramural funds awarded by the California State University, Fullerton and by a grant from the *Eunice Kennedy Shriver* National Institute of Child Health and Human Development (R15HD087877). Portions of these data were presented at the 2015 Gatlinburg Conference and the 2017 Convention of the American Psychological Association. We wish to thank the families who made this work possible. We also thank Audrey Rodriguez, Christopher Murakami, Mariann Howland, and Alyssa Bailey for their contributions to data collection and observational coding.

Author Contributions RMF and JKB conceived of the study, participated in its design and coordination, performed statistical analyses and data interpretation, and drafted the manuscript; JM participated in the design and coordination of the study, performed aspects of the measurement, and contributed text to the manuscript.

Compliance with Ethical Standards

Conflict of interest The authors declare that they have no conflict of interest.

Ethical Standards All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed Consent Informed consent was obtained from all individual participants included in the study.

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