

The role of emotion regulation and core autism symptoms in the experience of anxiety in autism

Autism
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Abstract

Youth with autism spectrum disorder are at elevated risk for impaired emotion regulation and clinically impairing anxiety. A prior developmental framework posited that impaired emotion regulation leads to co-occurring psychiatric conditions such as anxiety, with outcome determined in part by autism spectrum disorder-specific moderating factors. Using measures developed and validated in autism spectrum disorder, this study evaluated (1) the association between emotion regulation and anxiety in a large, community-based sample of youth with autism spectrum disorder and a wide range of intellectual and verbal abilities and (2) whether greater core autism spectrum disorder symptoms strengthened the association between impaired emotion regulation and anxiety. Parents of 1107 children with a community diagnosis of autism spectrum disorder (881 boys; age 6–17 years) participated in an online survey assessing their child's emotion regulation, anxiety, and autism spectrum disorder symptoms. Emotion regulation impairment significantly predicted whether participants had elevated levels of anxiety, after controlling for demographic variables and autism spectrum disorder symptoms; however, there was no interaction of emotion regulation and autism spectrum disorder symptoms. This study is the first to support the anxiety–emotion regulation association with measures developed and validated specifically for autism spectrum disorder, in a large sample with co-occurring intellectual disability and minimally verbal youth with autism spectrum disorder.

Lay abstract

Many children with autism spectrum disorder have problems with managing their emotions (emotion regulation) and anxiety. In this study, over 1000 parents completed an online survey which showed that emotion regulation and anxiety are closely linked. Although emotion regulation and anxiety are inter-connected, the results also show that autism symptoms play an important role in anxiety in autism spectrum disorder. Emotion regulation problems may be an important target for the treatment of anxiety in autism.

Keywords

anxiety, core autism spectrum disorder symptoms, emotion regulation, reactivity

Co-occurring psychiatric conditions are more common in youth with autism spectrum disorder (ASD) than in the general population (Joshi et al., 2010; Simonoff et al., 2008; van Steensel et al., 2013). The high rates of co-occurring anxiety disorders in youth and adults with ASD are well established (van Steensel et al., 2011; White et al., 2009). In a meta-analysis of 83 studies, van Steensel and Heeman (2017) reported that youth with ASD have significantly higher levels of anxiety when measured dimensionally than youth in the general population and youth without ASD ascertained from clinical settings. These findings suggest that ASD may increase the risk of anxiety.

There has been increasing interest in the role of impaired emotion regulation (ER) in the manifestation of anxiety and other psychiatric conditions in ASD (Cai et al., 2018;

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White et al., 2014). ER is defined as the ability to modulate arousal and emotional responses in the service of adaptive and socially appropriate behavior (Gross & Thompson, 2006). In ASD, youth report using fewer ER strategies and employing them less flexibly (Cai et al., 2018; Khor et al., 2014). They also tend to rely on potentially maladaptive strategies such as rumination, avoidance, and denial (Khor et al., 2014; Mazefsky et al., 2014). Available data, focused mostly on verbal samples, have shown an association between greater anxiety and impaired ER (Cai et al., 2018). Impairments in ER have also been implicated in disruptive behaviors such as irritability, tantrums, and aggression as well as mood disorders in youth with ASD (Mazefsky & White, 2014; Weiss, 2014).

White and colleagues (2014) proposed a developmental framework in which impaired ER can lead to a host of co-occurring psychopathologies (i.e. multifinality), with the type of psychopathology partially determined by characteristics associated with ASD. For example, biases in attention, sensory sensitivities, alexithymia, repetitive interests, restricted behaviors and intolerance of uncertainty, and insistence on sameness may promote avoidance of certain situations. These biases may accentuate the relationship between ER impairment and anxiety (Rodgers et al., 2012; Wigham et al., 2015). ASD-related social impairments may play a pivotal role in the ER-anxiety association. For example, the association between social communication deficits and social anxiety in the general population of youth is well established (Pickard et al., 2017). In ASD, one study found that engaging in characteristically adaptive ER strategies was associated with less social impairment among youth with ASD (Goldsmith & Kelley, 2018), while another observed that higher social impairment was associated with higher ER impairment (Samson et al., 2014). Overall, it may be helpful to consider how impaired ER effects anxiety in ASD, including how ASD core symptoms affect the ER-anxiety relationship.

Previous research in the general pediatric population suggests that impaired ER leads to different forms of psychopathology, including anxiety (Aldao et al., 2010). A longitudinal study of a community sample of youth reported that impaired ER predicted later anxiety symptoms (Schneider et al., 2018). Although findings on the association between core ASD symptoms and ER impairment are mixed (White et al., 2014), there is evidence that children with more severe ASD symptoms have increased ER impairment (Samson et al., 2014). Characteristics of ASD, such as inflexibility, high demand for daily routine, as well as decreased capacity to read social cues, recognize emotions of others, and difficulties with perspective taking can all contribute to impaired ER (Jahromi et al., 2012; Konstantareas & Stewart, 2006; Mazefsky & White, 2014; Rieffe et al., 2011).

Several studies have examined the impact of social impairment on the association between anxiety and ER in youth with ASD. Maddox and White (2015) posited that

increased desire for social interaction could account for heightened social anxiety in ASD. Swain and colleagues (2015) examined the interaction between social motivation, impaired ER, and social anxiety in young adults with ASD using self- and parent-report measures. Decreased social motivation was associated with greater social anxiety, suggesting that social anxiety may dampen social motivation. Low social motivation can contribute to social avoidance, increased social anxiety, and greater social impairment. Social impairment, misreading social cues, and uncertainty in social situations may undermine ER skills. Thus, it is possible that greater social impairment amplifies the relationship between ER and anxiety.

ASD symptoms such as restricted interests, repetitive behaviors and sensory sensitivities, and insistence on sameness are associated with anxiety in youth with ASD (Rodgers et al., 2012; Uljarević & Evans, 2016). In an adult sample comparing ASD and an age- and IQ-matched comparison group, ASD symptoms were significantly associated with greater intolerance of uncertainty, lower emotional acceptance, and increased alexithymia. However, intolerance of uncertainty was not a significant predictor of increased anxiety (Maisel et al., 2016). When core ASD symptoms and ER impairment were examined in a sample of 56 youth with ASD, all core symptom domains were associated with increased ER impairment; restricted and repetitive behaviors were the strongest predictor of ER impairment (Samson et al., 2014). Taken together, available data suggest a complex relationship between ASD severity, anxiety, and ER impairment in ASD.

Current study

Previous research on ER and anxiety in ASD has almost entirely focused on samples without significant language delays and without intellectual disability (ID) (Cai et al., 2018; van Steensel & Heeman, 2017). Thus, little is known about youth with ASD with ID and those who are minimally verbal (Bal et al., 2016). In addition, the instruments used to measure anxiety and ER in ASD were not designed to measure these constructs in youth with ASD and are highly reliant on verbal expression, which may affect their ability to capture the full range of anxiety and ER impairment manifestations in ASD (Bearss et al., 2016; Kerns & Kendall, 2012; Mazefsky, Day, et al., 2018; Scahill et al., 2019). Given challenges in the differential diagnosis of anxiety in ASD (Kerns et al., 2015), it may be useful to consider dimensional approaches to anxiety and explore its relationship to ER using measures validated in ASD (Mazefsky, Yu et al., 2018; Scahill et al., 2019).

In the current study, we used measures developed and validated for ASD to examine the association between ER impairment and anxiety as well as the effect of core ASD symptoms on the ER-anxiety relationship in a large sample of youth with ASD. The sample included youth with

and without co-occurring ID. First, we tested whether ER impairment was associated with increased anxiety, and whether ER impairment significantly predicted elevated anxiety after controlling for demographic factors, ID, social impairment, and restricted interests or repetitive behaviors. The second aim was to determine whether more severe core autism symptoms (e.g. social impairment, restricted interests) strengthened the association between ER impairment and anxiety.

Methods

Procedures

The data were collected in partnership with the Interactive Autism Network (IAN). IAN is a national, validated, and verified autism registry consisting of a representative sample of children in the United States with community diagnoses of ASD (Daniels et al., 2012). Parents of children on the IAN registry, with a score on the Social Communication Questionnaire (Rutter et al., 2003) of 12 or higher, between the ages of 6 and 17 years, were invited to participate in the study. All IAN registry members who met inclusion criteria (11,648) were invited to participate by email. Of the 1323 parents who consented to participate, 1107 completed the study measures (see Table 1 for demographics data). ID diagnoses were based on parental selection: “does not have ID,” “mild ID,” or “severe ID”. Responses were dichotomized to either no ID or ID for this study’s analyses.

Measures

Emotion Dysregulation Inventory. The Emotion Dysregulation Inventory (EDI) is a 30-item, caregiver report designed to measure ER impairment in individuals with ASD. It was developed based on guidelines from the NIH Patient-Reported Outcomes Measurement Information System (PROMIS) (Mazefsky, Day et al., 2018; Mazefsky, Yu, et al., 2018). The items in the final version of the scale were the product of factor analysis and item response theory (IRT) analyses using data from 1755 youth with ASD. None of the final items had evidence of differential item functioning (e.g. psychometric biases) by gender, age, intellectual ability, or verbal ability, making it suitable for use across heterogeneous populations. Prior research demonstrated test–retest reliability in a treatment stable sample of 901 youth with ASD between the ages of 6 and 18 years (mean difference of 0.05 and effect size of –0.06 for paired *t* tests) (Mazefsky, Yu, et al., 2018). Items are rated on a 5-point scale based on behavior during the previous 7 days (0=not at all, 1=mild, 2=moderate, 3=severe, and 4=very severe). The EDI’s primary scale measures reactivity (intense, rapid escalating, sustained, and poorly regulated emotional reactions), which is available as a 24-item

Table 1. Demographics and clinical characteristics of youth in online parent survey (N= 1107).

	Mean = 12.08 (standard deviation = 3.18) Range = 6–17.80
Age	
Gender	N(%)
Male	881 (79.6)
Female	226 (20.4)
Race	
White	1020 (92.1)
Black	65 (5.9)
Asian	24 (2.2)
Native American	23 (2.1)
Other	45 (4.1)
Unknown	4 (0.4)
Ethnicity	
Hispanic	106 (9.6)
Non-Hispanic	1000 (90.3)
Currently prescribed medication	435 (39.3)
Antipsychotic	198 (17.9)
Mood Stabilizer	91 (8.2)
Stimulant	176 (15.9)
Antidepressant	223 (20.1)
ID ^a	
None	460 (41.6)
Mild ID	397 (35.9)
Severe ID	188 (17.0)
Missing	62 (5.6)
	Mean (standard deviation)
EDI Reactivity Short Form theta score	–0.27 (0.85)
SCQ total score	22.93 (6.53)
SRS-2 total <i>t</i> -score	86.82 (13.54)
SRS-2 Social Communication Index <i>t</i> -score	86.36 (21.59)
SRS-2 Restricted Repetitive Behaviors <i>t</i> -score	20.60 (6.41)
PRAS-ASD total score	24.47 (13.69)

ID: intellectual disability; EDI: Emotion Dysregulation Inventory; SCQ: Social Communication Questionnaire; SRS-2: Social Responsiveness Scale, Second Edition; PRAS-ASD: Parent-Rated Anxiety Scale–Autism Spectrum Disorder.

^aVia parent report.

long form or seven-item short form. The long and short forms were correlated 0.99 in the EDI’s autism psychometric sample. The EDI also includes a secondary 6-item dysphoria (low positive affect, nervousness and sadness) scale, which was not used for this study due to the overlap with anxiety symptoms. EDI Reactivity Short Form theta scores, which have a mean of 0 and standard deviation (*SD*) of 1 and provide superior discriminative ability to raw scores (Hamleton, 1991), were used in analyses. EDI

Reactivity reliabilities in the original sample were high ($\alpha=0.97$) (Mazefsky, Yu, et al., 2018). For this study, internal consistency of the EDI Reactivity Short Form was also high ($\alpha=0.94$; non-ID subgroup $\alpha=0.94$; ID subgroup $\alpha=0.94$).

Parent-Rated Anxiety Scale–Autism Spectrum Disorder. The Parent-Rated Anxiety Scale–Autism Spectrum Disorder (PRAS-ASD) is a 25-item parent-report on anxiety symptoms rated on a 4-point scale (0=not present; 1=present sometimes, not a real problem; 2=often present and a problem; 3=very frequent and a major problem) (Scahill et al., 2019). Potential PRAS-ASD items were developed via a series of family focus groups and expert panel review. Systematic factor and item analysis in a sample of 990 youth with ASD, also ascertained through the IAN registry, identified 25 items which loaded onto a single factor. Internal consistency was excellent ($\alpha=0.93$). The distribution of scores in the sample appeared normal with a mean of 29 ($SD=14.9$). A sample of 116 youth (5–17 years of age) received a detailed clinical assessment of anxiety, ASD, and intellectual functioning to evaluate the validity and test–retest reliability of the PRAS-ASD. There was no difference in PRAS-ASD scores in those with and without ID. The PRAS-ASD demonstrated convergent validity with measures of anxiety and divergent validity with indices of ASD symptom severity and irritability. Test–retest reliability in this sample was high. Across these two samples, the mean score on the PRAS-ASD was approximately 30 ($SD=15$). Thus, a score of 45 (i.e. 1 SD above the mean) was used as a clinical cutoff in this study.

Social Communication Questionnaire, lifetime version. The SCQ is a 40-item caregiver-report questionnaire of binary questions (Yes/No) that measures social, language, and communication, and repetitive and stereotyped behaviors characteristic of ASD over a person’s lifetime (Rutter et al., 2003). The SCQ has shown strong discriminative validity between those with and without ASD (sensitivity=0.88, specificity=0.72) (Chandler et al., 2007). In this study, the SCQ cutoff of ≥ 12 was used to determine eligibility into the study. Reliability of the measure was acceptable in this sample ($\alpha=0.82$; non-ID subgroup $\alpha=0.96$; ID subgroup $\alpha=0.86$).

Social Responsiveness Scale, Second Edition. The Social Responsiveness Scale, Second Edition (SRS-2) is a 65-item parent-report measure of autism symptoms with five expert-derived subscales (Constantino & Gruber, 2012). Items are rated on a 4-point scale (1=not true, 2=sometimes true, 3=often true, 4=almost always true). Scales are converted to t -scores, where 60–65 is the mild range, 66–75 is the moderate range, and ≥ 76 is the severe range. Internal consistency of the Total Score of the SRS-2 has been reported to range between 0.92 and 0.97

in previous school-aged ASD samples (Constantino & Gruber, 2012). The SRS-2 authors do not recommend the use of the individual social subscales outside of treatment planning (Constantino & Gruber, 2012, p. 22). Thus, in this study, we used the Social Communication Index (SCI; $\alpha=0.92$; non-ID subgroup $\alpha=0.93$; ID subgroup $\alpha=0.91$), Restricted and Repetitive Behavior Scale (RRB; $\alpha=0.80$; non-ID subgroup $\alpha=0.84$; ID subgroup $\alpha=0.77$), and Total Score ($\alpha=0.94$; non-ID subgroup $\alpha=0.95$; ID subgroup $\alpha=0.93$).

Analysis

To assess the ER–anxiety association (aim 1), we calculated the correlation between the EDI Reactivity and PRAS-ASD total scores. On the PRAS-ASD, the sample was dichotomized at ≥ 45 as the clinical cutoff for elevated anxiety (Scahill et al., 2019). Independent sample t tests compared EDI Reactivity scores for youth with elevated anxiety symptoms to those without elevated anxiety. Logistic regression models were used to determine whether EDI Reactivity scores and SRS-2 variables significantly contributed to the probability of participants having elevated anxiety symptoms on the PRAS-ASD, after controlling for gender, age, race, and ID status. We also conducted a supplementary linear regression model with the PRAS-ASD as a continuous measure as the dependent variable, and EDI Reactivity, and SRS-2 Total Score as independent variables, controlling for gender, age, race, and ID status. Finally, we examined the role of ID in supplemental analyses within ID and non-ID subgroups separately, using the EDI Reactivity and SRS-2 Total Score variables in a logistic regression.

Multiple linear regression analyses were run to determine whether SRS-2 Total Score, SCI, or RRB moderated the relationship between EDI Reactivity scores and PRAS-ASD total scores (aim 2). In separate regression models for each SRS-2 scale, age, gender, and ID status were entered as covariates, with EDI Reactivity and the SRS-2 Total Score, SCI, or RRB (and their interaction) entered next, and anxiety as the dependent variable. SRS-2 scores were mean-centered prior to analysis. The PROCESS macro for SPSS (Hayes, 2013) was used to probe significant interactions, including post hoc tests of simple slopes at 1 SD above and below the moderator’s mean value. We also used the Johnson–Neyman technique (Johnson & Fay, 1950) to test for regions of significance across the range of observed data.

Results

The average age of participants was 12.08 ($SD=3.18$) years; 79.6% were male and 92.1% were White (see Table 1). Table 1 also contains descriptive statistics for the EDI Reactivity, PRAS-ASD, and SRS-2. The PRAS-ASD and EDI Reactivity scores were significantly correlated ($r=0.49$, $p<0.001$). Participants with PRAS-ASD score ≥ 45 ($n=96$)

Table 2. Logistic regression of EDI reactivity and SRS-2 onto PRAS-ASD clinical cut-off groups (above/below).

Logistic predictors (<i>n</i> = 1107)	<i>B</i>	Wald χ^2	Odds ratio	95% CI
With SRS-2 Total Score				
Age	0.01	0.09	0.99	0.92–1.06
Gender	–24	0.74	1.28	0.73–2.22
Race	0.47	0.76	0.63	0.22–1.80
Ethnicity	0.82	5.10*	0.44	0.22–0.90
Intellectual disability	–50	3.94*	1.65	1.01–2.70
SRS-2 Total Score	0.03	8.64**	1.03	1.01–1.05
EDI Reactivity Short Form theta score	1.22	53.51***	3.39	2.45–4.71
With SRS-2 Index Scores				
Age	0.01	0.05	0.99	0.92–1.07
Gender	–57	4.22*	1.77	1.03–3.05
Race	0.45	0.70	0.64	0.22–1.83
Ethnicity	0.88	5.79*	0.42	0.20–0.85
Intellectual disability	–48	3.57	1.61	0.98–2.65
SRS-2 Social	0.003	0.09	1.00	0.99–1.02
Communication Index				
SRS-2 Restricted and Repetitive Behaviors	0.07	5.52*	1.07	1.01–1.14
EDI Reactivity Short Form theta score	1.21	52.30***	3.36	2.42–4.67

EDI: Emotion Dysregulation Inventory; SRS-2: Social Responsiveness Scale, Second Edition; PRAS-ASD: Parent-Rated Anxiety Scale–Autism Spectrum Disorder; CI: confidence interval.

* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$.

had an EDI Reactivity theta score of 0.52 ($SD=0.73$) which was significantly higher than those with PRAS-ASD <45 ($n=1009$) (EDI Reactivity theta score -0.35 ; $SD=0.82$) ($t(1103)=-10.11$, $p < 0.001$). Youth with elevated anxiety symptoms on the PRAS-ASD also had significantly higher mean scores on the SCI (99.08, $SD=21.91$ vs 85.09, $SD=21.10$; $t(1103)=-6.19$, $p < 0.001$), RRB (24.68, $SD=5.76$ vs 20.20; $SD=6.33$; $t(1103)=-6.67$, $p < 0.001$), and SRS-2 Total Scores (123.76, $SD=26.41$ vs 105.29, $SD=26.24$; $t(1103)=-3.88$, $p < 0.001$).

The logistic regression model to predict PRAS-ASD clinical cut-off groups with EDI Reactivity and SRS-2 Total Score as independent variables (Wald $\chi^2=458.03$, $p < 0.001$) explained 41% of the variance in anxiety (Cox & Snell $R^2=0.103$; $-2\log$ likelihood=495.25). The model correctly identified 91.5% of the participants >45 on the PRAS-ASD (Table 2). Higher EDI Reactivity ($\beta=1.22$, $p < 0.001$) and SRS-2 ($\beta=0.03$, $p=0.003$) scores significantly predicted exceeding the clinical cut-off for anxiety on the PRAS-ASD. Bivariate odds ratios (ORs) indicated that EDI Reactivity (OR=3.39; confidence interval (CI): 2.45–4.71) more strongly predicted anxiety than did SRS-2 Total Score (OR=1.03; CI: 1.01–1.05).

The same model with the SRS-2 subscales (SCI and RRB) as independent variables instead of the SRS-2 Total

Score (Wald $\chi^2=495.89$, $p < 0.001$) explained 41% of the variance in anxiety (Cox & Snell $R^2=0.102$; $-2\log$ likelihood=495.89). The model correctly identified 91.5% of the participants >45 on the PRAS-ASD (Table 2). Higher levels of EDI Reactivity ($\beta=1.21$, $p < 0.001$) and RRB ($\beta=0.07$, $p=0.03$), but not SCI ($\beta=0.003$, $p=0.63$), were significant predictors of elevated anxiety on the PRAS-ASD. Bivariate ORs indicated that EDI Reactivity (OR=3.36; CI: 2.42–4.67) more strongly predicted anxiety than did RRB (OR=1.07; CI: 1.01–1.14) or SCI (OR=1.00; CI: 0.99–1.02).

A supplemental analysis using linear regression with PRAS-ASD scores a continuous dependent variable and the EDI Reactivity and SRS-2 Total Score as independent variables yielded the same pattern of results (see Supplemental Material). Finally, as a follow-up analysis, the primary model predicting PRAS-ASD scores from EDI Reactivity, SRS-2 Total Score, and their interaction was re-run in ID and non-ID subgroups separately (see Supplemental Material). Higher elevated SRS-2 Total Scores were associated with elevated anxiety only in the non-ID group ($\beta=0.04$, $p=0.02$), and higher EDI Reactivity was significantly associated with elevated anxiety on the PRAS-ASD.

Moderation analyses

The linear regression with PRAS-ASD continuous score as the dependent variable and EDI Reactivity, SRS-2 Total Score, and their interaction as dependent variables, controlling for age, gender, and ID status accounted for 55% of the variance in PRAS-ASD (see Table 3). Anxiety was significantly associated with younger age ($\beta=-0.09$, $p=0.003$), non-ID status ($\beta=-0.13$, $p < 0.001$), higher EDI Reactivity ($\beta=0.32$, $p < 0.001$), and higher SRS-2 Total Score ($\beta=0.19$, $p < 0.001$). There was no interaction between EDI Reactivity and SRS-2 Total Score ($\beta=0.15$, $p=0.50$).

A linear regression model with EDI Reactivity, SRS-2 SCI, and their interaction, as independent variables, controlling for demographic factors, and PRAS-ASD continuous scores as the dependent variable accounted for 54.5% of variance in anxiety scores. Anxiety scores were significantly and positively associated with younger age ($\beta=-0.09$, $p=0.001$), not having ID ($\beta=-0.13$, $p < 0.001$), higher EDI Reactivity ($\beta=0.36$, $p < 0.001$), and higher SRS-2 SCI ($\beta=0.16$, $p < 0.001$). There was no interaction between EDI Reactivity and SCI ($\beta=0.12$, $p=0.66$).

The linear regression model with EDI Reactivity, SRS-2 RRB, and their interaction as independent variables, controlling for demographic factors, explained 55.4% of the variance in PRAS-ASD scores. PRAS-ASD scores were significantly associated with young age ($\beta=-0.08$, $p=0.004$), not having ID ($\beta=-0.12$, $p < 0.001$), higher EDI Reactivity ($\beta=0.36$, $p < 0.001$), and higher SRS-2

Table 3. Moderation analyses.

Dependent variable	Predictors	B	β	T	F change	R ²
PRAS-ASD total	Age	-0.36	-0.09	-3.20**		
	Gender	1.37	0.04	1.51		
	Intellectual disability	-3.51	-0.13	-4.75***		
	SRS-2 Total t-score	0.19	0.19	5.16***		
	EDI Reactivity Short Form theta score	5.03	0.32	3.50***		
	SRS-2 Total Score \times EDI Reactivity	0.001	0.15	1.54		
	Overall model				75.64***	0.31***
PRAS-ASD total	Age	-0.37	-0.09	-3.25**		
	Gender	-0.44	-0.01	-0.50		
	Intellectual disability	-3.43	-0.13	-4.61***		
	SRS-2 Social Communication Index t-score	0.10	0.16	4.09***		
	EDI Reactivity Short Form theta score	5.71	0.36	4.74***		
	SRS-2 SCI \times EDI Reactivity	0.001	0.12	1.42		
	Overall model				73.54***	0.30***
PRAS-ASD total	Age	-0.34	-0.08	-3.08**		
	Gender	-0.68	-0.02	-0.76		
	Intellectual disability	-3.17	-0.12	-4.36***		
	SRS-2 Restricted Repetitive Behaviors t-score	0.38	0.18	4.70***		
	EDI Reactivity Short Form theta score	5.69	0.36	5.27***		
	SRS-2 RRB \times EDI Reactivity	0.003	0.12	1.54		
	Overall model				76.84***	0.31***

PRAS-ASD: Parent-Rated Anxiety Scale–Autism Spectrum Disorder; SRS-2: Social Responsiveness Scale, Second Edition; EDI: Emotion Dysregulation Inventory; SCI: Social Communication Index; RRB: Restricted and Repetitive Behavior Scale.

* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$.

RRB ($\beta = 0.18$, $p < 0.001$). There was no significant interaction between EDI Reactivity and SRS-2 RRB ($\beta = 0.12$, $p = 0.42$) (see Table 3 and Figure 1).

Overall, higher social impairment, RRB symptoms, and ER impairment were significantly associated with elevated PRAS-ASD scores. There were no significant interaction effects between SRS-2 scores and EDI Reactivity on anxiety.

Discussion

The high prevalence of ER impairment and anxiety disorders in youth with ASD is well established (Samson et al., 2015; van Steensel et al., 2013). The current study examined the association between ER and anxiety in a large sample of youth with ASD that included participants with and without ID. As expected, ER impairment predicted clinical levels of anxiety as measured on the PRAS-ASD after controlling for indices of ASD severity, and ER impairment remained strongly associated with anxiety. The medium correlation between ER and PRAS-ASD, however, suggests that these instruments are not measuring the same construct. Although this was a cross-sectional study, we propose that ER impairment may reflect a mechanism, or pathway, for the development of anxiety. If so, ER may be a target for intervention to prevent or remediate anxiety in youth with ASD.

This study also examined whether measures of ASD symptoms such as social impairment, restricted interests, and repetitive behaviors would strengthen the association between ER impairment and anxiety. All SRS-2 scores and ER impairment were significantly associated with higher anxiety scores, suggesting that social impairment, restricted interests and repetitive behaviors, and ER impairment are each associated with anxiety in ASD. These findings are consistent with prior research showing that social impairment (Swain et al., 2015) is associated with increased social anxiety in ASD. These results also concur with prior reports suggesting that the second cluster of ASD symptoms, including sensory sensitivities, insistence on sameness, restricted interests, and repetitive behaviors, is associated with anxiety in youth with ASD (Rodgers et al., 2012; Wigham et al., 2015). However, there were no interaction effects between ASD symptom indices on the SRS-2 and ER impairment on anxiety symptoms. Thus, future research should incorporate ASD symptom severity and ER impairment to deepen our understanding on the emergence of anxiety in youth with ASD.

Findings of the current study are consistent with prior work linking core ASD symptoms and ER impairment (Samson et al., 2014). ASD features such as inflexibility, decreased capacity to read social cues, and difficulties with perspective taking appear to increase the risk of ER impairment in ASD (Mazefsky & White, 2014). If biological

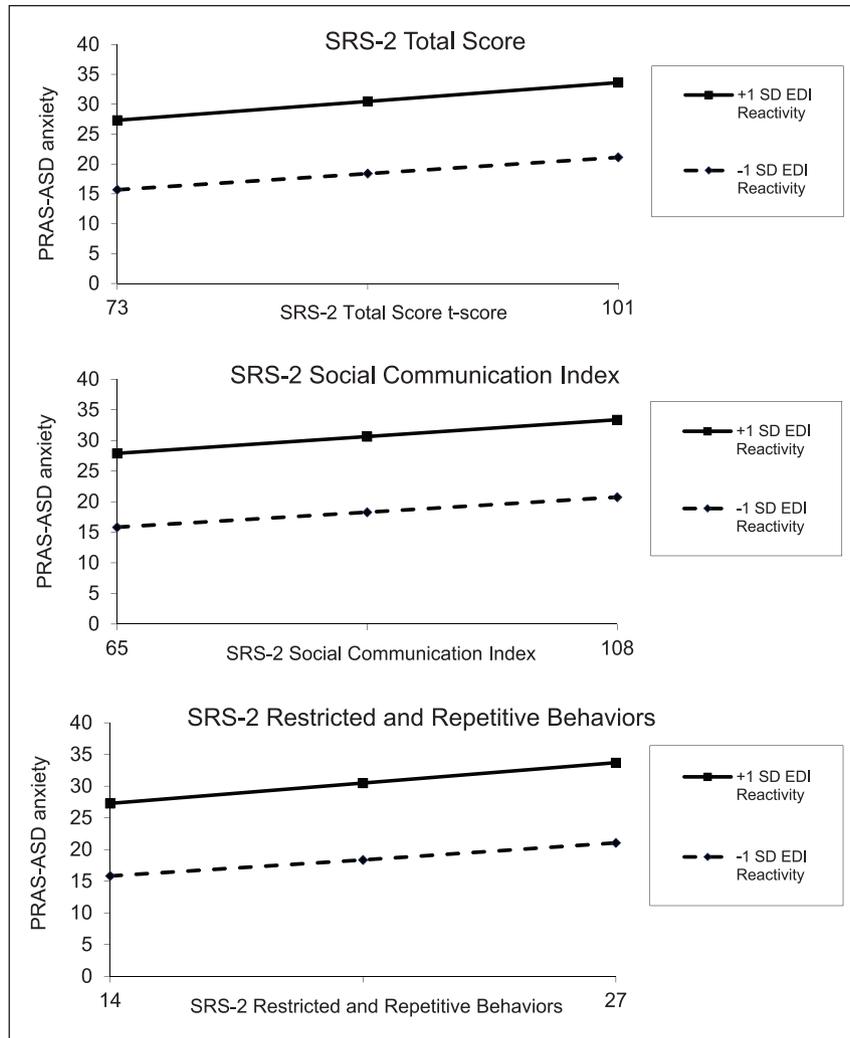


Figure 1. Interaction between SRS-2 and emotion regulation on anxiety symptoms.

SD: standard deviation; SRS-2: Social Responsiveness Scale, Second Edition; PRAS-ASD: Parent-Rated Anxiety Scale–Autism Spectrum Disorder score; EDI: Emotion Dysregulation Inventory.

factors associated with ASD contribute to impaired ER, as suggested by White et al. (2014) and Mazefsky et al. (2014), these biological factors could increase the identification of individuals at elevated risk. A next step could be to develop and test models of how core ASD symptoms, along with impaired ER, inattention, and executive function deficits, affect development of co-occurring psychopathology in ASD.

This study was cross-sectional, which precludes any conclusions about causation. Although our model posits that the level of anxiety may be moderated (i.e. amplified) by social impairment, restricted interests, and repetitive behaviors, it is also possible that anxiety can contribute to social impairment, restricted interests, and repetitive behaviors. Duvekot and colleagues (2018) followed 79 youth with ASD over a 2-year period. Participants with high levels of anxiety at Time 1 had greater social communication impairment at Time 2. A larger study in the

general population showed that social communication deficits predicted increased social anxiety over time between ages 7 and 13 (Pickard et al., 2017). These findings support a dynamic interaction between impaired social communication and social anxiety—but temporal ordering remains uncertain. For example, anxiety in youth with ASD may promote social avoidance, reduce opportunities for social interaction, and hinder the development of social skills. Alternatively, social communication deficits may contribute to failures in social situations followed by social avoidance (Kerns et al., 2015; Scahill et al., 2019; White et al., 2015). Longitudinal studies could move beyond association to confirmation of predisposing and perpetuating factors.

This study also suggested that some other demographic and patient characteristics may play a role in anxiety in ASD, namely younger age and lack of ID were associated with higher parent-reported anxiety. Interestingly, this

finding contrasts both anxiety prevalence in the general population and findings suggesting that risk for anxiety in ASD may be exacerbated by social impairment (Swain et al., 2015; White et al., 2014). We note that the current study relied on parent report of ID status, which may have affected the observed results. In a sample of 116 youth with ASD assessed with the PRAS-ASD and standardized cognitive testing, there was no difference in anxiety level in those with IQ above or below 70 (Scahill et al., 2019). However, prior research has suggested that anxiety is more common among children with autism who have higher intellectual ability (Lecavalier et al., 2019).

There are several limitations of the current study. A community diagnosis of ASD as self-declared in the IAN sample and an SCQ score above clinical cutoff were used to define cases. Although this method has been verified by medical records in a series of IAN participants (Daniels et al., 2012), we could not confirm the ASD diagnosis in our participants. Furthermore, all surveys were parent reported, so biases arising from a single informant cannot be ruled out. Although the sample size was relatively large, respondents were self-selected, predominantly White and non-Hispanic/Latino. Future studies could use more fine-grained cognitive and language assessment to compare how differences in cognitive functioning and age affect the interaction between social disability, ER impairment, and anxiety. Treatment studies could use these characteristics to guide treatment of ER impairment and anxiety.

In summary, this study established the association between ER impairment and anxiety in a large sample that included youth with ASD across a wide range of cognitive function. Higher ASD symptoms were also associated with increased anxiety, in accordance with prior research (Swain et al., 2015; Uljarević & Evans, 2016). Impaired ER, in particular, may be a vital underlying construct for anxiety. The emergence of heightened anxiety may also undermine capacity for ER. Recent research has indeed suggested that improving ER leads to decreased anxiety in ASD (Conner et al., 2019). ER impairment should be considered in our pursuit for mechanistically driven and transdiagnostic, personalized treatments.

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Supplemental material

Supplemental material for this article is available online.

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