SOCIAL ANXIETY AND SOCIAL COGNITION IN YOUTH WITH NEURODEVELOPMENTAL DISORDERS: DIFFERENTIAL PATHS TO FUNCTIONAL IMPAIRMENT

by

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ABSTRACT

Among children with neurodevelopmental disorders, those with Autism Spectrum Disorder (ASD), Attention-Deficit Hyperactivity Disorder (ADHD), and Specific Learning Disorder (SLD) present with notable social impairment, heightened rates of social anxiety, and deficits in social cognition when compared to neurotypical peers. Because parent- and child-reports of social anxiety are frequently discrepant, conventional scientific wisdom dictates the inclusion of these separate reports when measuring symptoms and impairment. Social anxiety and social cognitive deficits are associated with poor functional outcomes, but research has minimally explored these constructs in youth with neurodevelopmental disorders. The present study examined the predictive weight of social anxiety and social cognitive impairment on adaptive functioning in a sample of 99 youth (ages: 7-17 years) with a diagnosis of ASD, ADHD, or SLD. Social cognition significantly predicted adaptive functioning impairments as well as clinician ratings of global functioning and clinical impairment. Parent and child ratings of social anxiety were significantly different; this discrepancy did not predict adaptive functioning, though it was predictive of clinician-rated impairment and approached significance in analyses of clinician-rated global functioning. These findings further clarify differential symptomatic paths to functional impairment in this population and inform mechanism-based treatment research addressing social impairment in youth with neurodevelopmental disorders.

LIST OF ABBREVIATIONS AND SYMBOLS

α	Chronbach's index of internal consistency
ASD	Autism Spectrum Disorder
ADHD	Attention-Deficit Hyperactivity Disorder
β	Beta value: standardized regression coefficient
B	Unstandardized regression coefficient
CGAS	Children's Global Assessment Scale
CGI	Clinical Global Impression – Severity Scale
DSM-5	Diagnostic and Statistical Manual, 5th Edition
F	Fisher's F ratio: A ration of two variances
f^2	Cohen's f^2 measure of effect size: its bias is dependent on the bias of its underlying measurement of variance explained
FSIQ	Full Scale Intelligence Quotient
M	Mean: the sum of a set of measurements divided by the number of measurements in the set
n	Number
p	Probability associated with the occurrence under the null hypothesis of a value as extreme as or more extreme than the observed value
r	Pearson product-moment correlation
R^2	Coefficient of determination
$R^2\Delta$	Change in R^2 value across subsequent steps of a multiple regression model
SA-Child	Child-rated social anxiety symptoms, as measured by the Screen for Child Anxiety and Related Emotional Disorders

SA-Parent	Parent-rated social anxiety symptoms, as measured by the Screen for Child Anxiety and Related Emotional Disorders
SAD	Social Anxiety Disorder
SCARED-C	Child-reported Screen for Child Anxiety and Related Emotional Disorders
SCARED-P	Parent-reported Screen for Child Anxiety and Related Emotional Disorders
SCARED-SA	Social anxiety symptoms, as measured by the Screen for Child Anxiety and Related Emotional Disorders
SD	Standard Deviation
SE	Standard Error: measure of the statistical accuracy of an estimate, equal to the standard deviation of the theoretical population distribution
SLD	Specific Learning Disorder
SRS-2	Social Responsiveness Scale, 2nd Edition
SRS-2-SC	Social Cognition subscale of the Social Responsiveness Scale, 2nd Edition
t	Computed value of *t* test
Tol.	Tolerance: Measurement of multicollinearity in a regression model
VABS	Vineland Adaptive Behavior Scales- III Edition
VIF	Variance Inflation Factor: Measurement of multicollinearity in a regression model
Wald	Wald Chi Square Test; logistic regression model
χ^2	Chi squared value
=	Equal to
<	Less than
%	Percent

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INTRODUCTION

Neurodevelopmental disorders are identified in the American Psychiatric Association's (2013) revised diagnostic handbook (Diagnostic and Statistical Manual, 5th Edition; DSM-5) as those which 1) present in childhood; 2) are typified by developmental deficits; and 3) are associated with impairment in social, personal, academic, and/or occupational domains. This diagnostic grouping includes six specific diagnoses: intellectual disability, specific learning disorder (SLD), communication disorder, motor disorder, Autism Spectrum Disorder (ASD), and Attention Deficit/Hyperactivity Disorder (ADHD). The DSM-5 also identifies nonspecific neurodevelopmental disorders resulting from a medical or genetic condition or environmental factors. Often symptom profiles in these disorders overlap and, importantly, children are frequently diagnosed with comorbid neurodevelopmental disorders. ASD and ADHD, for example, co-occur at a rate of 75% in clinical samples of children (Brookman-Frazee, Stadnick, Chlebowski, Baker-Ericzén, & Ganger, 2017), while SLD has a prevalence estimate of 45% in children diagnosed with ADHD (Pham & Riviere, 2015). Despite this increased rate of co-occurrence and similar pervasive life course, clinical research rarely considers neurodevelopmental disorders together and, instead, focuses specifically on only one or two diagnostic groups.

Nevertheless, these disorders arguably hang together in different groupings, and research points to particularly stark social impairment in youth with ASD, ADHD, and SLD. Individuals with ASD, per diagnostic criteria, are often socially unmotivated (American Psychiatric Association, 2013), and subsequently struggle to understand, develop, and maintain peer

relationships (Mendelson, Gates, & Lerner, 2016). This social impairment is associated with increased reported loneliness (Deckers, Muris, & Roelofs, 2017) as well as depression and low self-esteem (Mazurek, 2015). Meanwhile, children with ADHD also struggle frequently with impairing social deficits (Gentschel & McLaughin, 2000) and report fewer friends than neurotypical peers (Bagwell, Molina, Pelham & Hoza, 2001). These social deficits are thought to stem from characteristic executive functioning deficits associated with ADHD such as behavioral inhibition and inattention (Hilton, Jarrett, McDonald, & Ollendick, 2017). Finally, research has also argued that social skill impairment may be inherent to SLD (see Wiener, 2004). Though research is less consistent, a meta-analysis conducted by Nowicki (2003) found that children with SLD were consistently rated lower than neurotypical peers on measures of social preference. However, a more recent study found no difference in measured social status between children with SLD and neurotypical peers (Bakker, Denessen, Bosman, Krijger, & Bouts, 2007). Despite the co-occurrence of social impairment in these populations, research tends to focus on diagnostic groupings rather than broad analyses of dimensional impairment across diagnoses.

Social anxiety in youth with neurodevelopmental disorders

Among children with neurodevelopmental disorders, those with a diagnoses of ASD, ADHD and/or SLD also present frequently with clinically impairing symptoms of anxiety (Grahame & Rodgers, 2015). Hallmark symptoms of social anxiety disorder (SAD) include fear of negative evaluation from others in social/performance situations and subsequent avoidance of these situations, and as a result, children with SAD frequently experience social withdrawal and isolation (American Psychiatric Association, 2013). The National Comorbidity Survey Replication estimated 9% prevalence of SAD in adolescents (Burstein et al., 2011), and SAD is diagnosed even more frequently in adolescents with neurodevelopmental disorders. Children

with ASD are particularly prone to social anxiety; estimates of comorbidity indicate 39.6% of youth with ASD have a comorbid anxiety disorder, with SAD the most common of all anxiety diagnoses (29.8%; van Steensel, Bogels, & Perrin, 2011). This estimate of SAD in ASD is triple that of typically developing peers (Burstein et al., 2011). However, current prevalence rates using the DSM-5 have yet to be established. Similarly, children with ADHD are also prone to anxiety and tend to worry about performance across multiple domains, including academics, athletics, and social behavior (see Tannock et al., 2009). A recent examination of comorbidity in children with ADHD found that 22.6% of children met criteria for SAD (Jarrett, Wolff, Dais, Cowart, & Ollendick, 2016). Research has also posited that children with a SLD might experience heightened social anxiety stemming from performance deficits in the classroom; struggles reading aloud or answering questions that result in teasing from peers or criticism from teachers is thought to manifest in a clinical fear of negative evaluation, which is recognized as a hallmark symptom of social anxiety (see Mineka & Zinbarg, 2006). Though there is not, to our knowledge, a specific estimate of comorbid SAD in children with SLD, research has found that an estimated 28.8% of children with SLD (Margari et al., 2013) also met criteria for an anxiety disorder. It is unsurprising, therefore, that the demonstrable comorbidity of anxiety within neurodevelopmental disorders has prompted a surge of clinical research.

Clinically, social anxiety is associated with a host of unfavorable outcomes in later childhood and adulthood. Children with SAD have been found to report lower quality friendships than peers with other anxiety diagnoses (Baker & Hudson, 2015). Dimensionally, social anxiety in neurotypical children was also found to predict decreased social functioning after a targeted psychosocial intervention (Wood, 2006). Within neurodevelopmental disorders, youth with comorbid SAD and ADHD are at a higher lifetime risk for developing major depressive disorder

and bipolar disorder later in life (Koyuncu et al., 2015). Although there is ample evidence of heightened anxiety in youth with neurodevelopmental disorders, research has only recently begun to venture into dimensional measurement and treatment of anxiety in this population (see Kerns & Kendall, 2013). This body of research paints a symptom profile wherein social anxiety might account for disheartening outcomes in children with neurodevelopmental disorders, but continued research is needed in this area to clearly delineate a relationship between social anxiety and adaptive/emotional functioning in this population.

Parent and child discrepancies in reported social anxiety

The field of clinical psychology has yet to converge on the classification of a definitively 'accurate' informant of psychopathology against which to compare other ratings (see Achenbach, McConaughy, & Howell, 1987). Thus, clinicians tend to rely on the inclusion of observer (i.e., parent, teacher, clinician, etc.) ratings of symptoms in addition to the child's self-report for both therapy and assessment. Extensive research has argued for the inclusion of multiple informants when collecting data in order to capture a more comprehensive picture of the child's behavioral and symptomatic presentation (De Los Reyes et al., 2015; Jansen, Bodden, Muris, van Doorn & Granic, 2017; Kraemer et al., 2003). With regard to social anxiety, specifically, children's insight may be limited by cognitive factors (i.e., difficulty understanding questions or abstract conceptualizations of anxiety; see Grills and Ollendick, 2003), and they are particularly susceptible to social desirability bias (Silverman & Ollendick, 2005). Research has examined convergence diagnostically (i.e., comparison of dichotomous clinical/nonclinical response patterns across reporters), but a dimensional approach to social anxiety paints a richer symptom profile and allows for more nuanced understanding of reporter differences (see Comer & Kendall, 2004). Jansen and colleagues (2017) reported higher concordance in reports of

dimensional anxiety between mothers and their children, as compared to child/father concordance, though ratings from both parents were argued to be important to the clinical conceptualization. Broadly, research integrating both child report and parent report is better suited to assessment, research, and treatment for social anxiety.

However, when informants report inconsistently on a certain symptom/behavior/pattern, this difference may also be useful to the broad clinical presentation (see De Los Reyes, 2011). Researchers disagree as to the directionality of parent/child informant discrepancy in reported social anxiety. Most studies have shown that parents report increased rates of social anxiety in their children compared to the child's self-report (Becker, Jensen-Doss, Kendall, Birmaher, & Ginsburg, 2016; Ferdinand, van der Ende, & Verhulst, 2006). However, other researchers have found that children report relatively higher symptoms of social anxiety than their parents (Cosi, Canals, Hernández-Martinez, & Vigil-Colet, 2010). This difference may be attributable to sampling differences; Becker and colleagues (2016) examined children participating in treatment for an anxiety disorder; and Ferdinand, van der Ende, and Verhulst interviewed children given a psychiatric diagnosis from an outpatient clinic. Meanwhile, Cosi and colleagues' (2010) sample identified children 'at risk' but without an official diagnosis. This distinction is supported by research which suggests that children with clinical levels of anxiety report lower symptoms than their parental informants, while non-anxious children self-report more symptoms of anxiety than do their parents (Rappaport, Pagliaccio, Pine, Klein, & Jarcho, 2017).

Research suggests age of youth does is not related to discrepancy between parent and child-reported social anxiety (Becker et al., 2016; Cosi et al., 2010, Hoffman & Chu, 2015). A meta analysis led by De Los Reyes (2015) corroborated the argument that discrepant reports of social anxiety between parents and children do not seem to reflect differences in the child's

developmental stage. However, this argument is contradicted by studies which have simultaneously found increased concordance (Berg-Nielsen, Vika, & Dahl, 2003; Choudhury, Pimental, & Kendall, 2003; Grills & Ollendick, 2003; Rapee Barrett, Dadds, & Evans, 1994) and decreased concordance with parent-report (Niditch & Varela, 2011) in self-reported social anxiety in older youth compared to younger children.

Social cognition in neurodevelopmental disorders

Social cognition is typically conceptualized as the mental processes which underlie reciprocal social interactions, including perception, interpretation, and response to others' intentions and behavior (Fiske, 1999). Deficits in social cognition are a hallmark characteristic of children with neurodevelopmental disorders. Children with ASD, for example, struggle particularly with recognizing facial expressions (e.g., Golan, Gordon, Fichman, & Keinan, 2018; Pelphrey et al., 2002), inferring others' mental states (i.e., Theory of Mind; see Bora & Pantelis, 2016), and attending to social cues in conversations (Chita-Tegmark, 2016). Children with ADHD, meanwhile, present with difficulties encoding social information (Matthys, Cuperus, & van Engeland, 1999; McQuade & Hoza, 2015) and interpreting (Ros & Graziano, 2018) social information, both of which are thought to stem from attention deficits (Nijmeijer et al., 2008). Meanwhile, there is less research looking at specific cognitive deficits in children with SLD, though research suggests overlap with both ASD and ADHD; children with SLD demonstrate difficulty with encoding social information, interpretation, and recognizing facial expressions (see Al-Yagon & Margalit, 2013).

Attention should also be given to social cognitive deficits which may be uniquely associated with SAD. Banerjee and Henderson (2001) found that neurotypical children with SAD demonstrated localized social cognitive deficits; increased social anxiety was associated

with poor performance on social cognitive tasks related to interpersonal interactions but not broader theory of mind abilities. In other words, these children were impaired in their interpretive abilities only when considering their own interactions with peers, in that they tended to overestimate/overweight negative evaluation of their peers (Banerjee & Henderson, 2001). Spence, Donovan, and Brechman-Toussaint (1999) also found that children with social anxiety presented with consistent social skill deficits relative to their non-anxious peers. Cartwright-Hatton, Tschernitz, and Gomersall (2005) elaborate on this work, conceptualizing this social deficit in children with social anxiety as 'perceptive' impairment, rather than a 'performance' impairment such that while independent observers could not discriminate between low and high socially anxious children based on social skill, those children with higher anxiety self-reported significantly lower social skill than their non-anxious peers. It should be noted, however, that the social cognitive deficits noted in SAD are distinct from those typical of children with neurodevelopmental disorders, and as such research is needed to continue examining how these deficits interact to affect functional outcomes in children.

Interaction between social anxiety and social cognitive impairment

Having established that both social cognitive deficits and impairing social anxiety are common among youth with neurodevelopmental disorders, several theories have been posited as to the relationship between these impairments. Researchers have theorized that social cognitive deficits lead to negative social interactions and the subsequent development or exacerbation of social anxiety (see Rapee & Spence, 2004). For example, socio-communicative impairments and reduced social motivation, which are hallmark characteristics of ASD, have been shown to increase susceptibility to bullying, peer victimization, and social isolation, (see Schroeder, Cappadocia, Bebko, & Weiss, 2014), which could exacerbate social anxiety symptoms in this

population. Alternatively, it has been posited that social anxiety and subsequent avoidance of social situations prevents positive and informative opportunities to engage with peers, preventing these individuals from learning from these social interactions and thus contributing to social cognitive impairment (see Clark, 2001). Both models emphasize the path to comorbid social anxiety and social cognitive deficits in children with neurodevelopmental disorders, though research has yet to parse out the directionality or interactive effects of both symptom profiles.

Impact of social anxiety and social cognitive deficits on functional outcome

Research has focused less on the clinical importance of discrepant parent and child reports of youth anxiety than discrepant parent teacher reports, but De Los Reyes (2011) makes a compelling theoretical argument for such an impact. Clinically, discrepant parent and child-reported anxiety prior to treatment has been shown to negatively impact parent-reported satisfaction with targeted therapy (Hoffman & Chu, 2015). Neurotypical children whose parents reported discrepantly higher anxiety prior to treatment were less likely to be diagnosis free after administration of CBT (Becker-Haimes, Jensen-Doss, Birmaher, Kendall, & Ginsburg, 2018). Additionally, Ferdinand, van der Ende, and Verhulst (2006) noted a particular uptick in police/judicial contact associated with parent/child discrepant reported social anxiety. Among youth with neurodevelopmental disorders, children with ASD have been studied, predominantly, as discrepant reporters of social anxiety. A recent meta-analysis of cross-sectional studies noted significant negative associations between self-reported social anxiety and self-reported social competence/clinician assessed social skill (Spain, Sin, Linder, McMahon, & Happe, 2018). Parent-reported social anxiety, though, was not associated with these domains (Spain et al. 2018).

The concurrent presentation of social anxiety and social cognitive deficits may also interact and/or compound resulting functional impairment in youth with neurodevelopmental disorders. Functional impairment more broadly captures deficits in adaptive behavior; for example, social anxiety is associated with elevated rates of depression (Ohayon & Schatzberg, 2009), school withdrawal (Stein & Kean, 2000), and substance abuse (Sareen, Chartier, Paulus, & Stein, 2006). Research often measures cognitive ability or school performance in order to gauge adaptive behavior (see Pellecchia et al., 2016), but this unidimensional approach addresses functioning indirectly and fails to capture a behavioral index of such impairment. To our knowledge, research has not yet examined these compounded impairment profiles in individuals with neurodevelopmental disorders. Simonoff and colleagues (2008), in their examination of comorbidity in ASD, did not find an association between functional impairment (composite score of communication/daily living skills/ social behaviors) and any comorbid diagnoses; however, this was not a primary aim of their study. Continued research is clearly warranted to clarify the clinical impact of compounded social anxiety and social cognitive deficits in children with neurodevelopmental disorders.

Current Study

Heightened social anxiety and deficits in social cognition present more frequently in children with neurodevelopmental disorders, particularly youth with ASD, ADHD, and SLD, and these deficits have been shown to impact adaptive functioning in this population. However, social anxiety and social cognition have not yet been studied in tandem in children with neurodevelopmental disorders. Approaching social anxiety and social cognitive deficits dimensionally, per the Research Domain Criteria published by the National Institutes of Mental Health (Clark, Cuthbert, Lewis-Fernández, Narrow, & Reed, 2017), will allow for a

transdiagnostic analysis of these symptom profiles in children with ASD, ADHD, and SLD. Additionally, the inclusion of multiple informants when measuring these variables will paint a richer and more comprehensive picture of the pattern of deficits in this population. While recent work has begun to focus more explicitly on informant discrepancies (e.g., Becker-Haimes et al., 2018), scant research has examined these discrepancies in a population with neurodevelopmental disorders. Thus, findings from the present study might inform our understanding of how impairment profiles (i.e. social anxiety and social cognition) interact, which could subsequently inform mechanism-based intervention research addressing social impairment and adaptive functioning in these youth.

As such, the present study has two primary aims: first, to determine unique effects of both social cognitive impairment and social anxiety on functional impairment in children with neurodevelopmental disorders. It is hypothesized that parent-reported social cognition (SRS-2-SC) will account for unique variance in parent-reported adaptive functioning (VABS), above and beyond variance accounted for by parent-reported social anxiety (SA-Parent). Second, the current study will examine the degree to which SA-Parent, child-reported social anxiety (SA-Child), and informant discrepancies in reported social anxiety relate to the child's adaptive functioning (VABS). It is hypothesized that children and their parents will report significantly different symptoms of social anxiety; it also hypothesized that informant discrepancy will account for a significant amount of variance in the child's adaptive functioning, above and beyond variance accounted for by SA-Parent and SA-Child.

METHOD

Design

The study applied polynomial regression analyses and binary logistic regressions to data collected as part of the Virginia Tech Child Assessment Clinic Database. As the proposed study used previously-collected, deidentified data, it was exempt from review per the Institutional Review Board at the University of Alabama.

Participants

Participants in this study were referred to a university-affiliated clinic for assessment services. All participants consented for clinical data (i.e., surveys, clinician ratings, child reports, etc.) to be used for research purposes. Inclusion criteria for this study included 1) completion of four core measures and 2) a diagnosis provided by the clinic of either ADHD, ASD, or SLD. Diagnoses were provided by graduate-level clinicians under the supervision of licensed clinical psychologists, which took into consideration a comprehensive assessment targeting cognitive, behavioral and emotional symptoms. Ultimately, 99 children were identified for inclusion in this study (see Table 1 for demographic information). Participants were predominantly male (n = 64, 64.6%) and ranged in age from 7 – 17 years (M = 11.40, SD = 2.79). Participants' average FSIQ was 96.99 (SD = 19.45), captured by either the Wechsler Intelligence Scale for Children, Fourth edition (Wechsler et al., 2003); Wechsler Intelligence Scale for Children, Fifth Edition (Wechsler, 2014) or the Wechsler Adult Intelligence Scale, Fourth Edition (Wechsler, 2008).

Table 1. *Demographic Data*

N = 99 (unless otherwise noted)	**N (%)**		
Race			
White/Caucasian	84 (84.8%)		
Black/African American	2 (2.0%)		
Asian	1 (1.0%)		
Native American	1 (1.0%)		
Other	7 (7.1%)		
Ethnicity			
Hispanic	6 (6.1%)		
Non-Hispanic	93 (93.9%)		
Gender			
Male	64 (64.6%)		
Female	35 (35.4%)		
Diagnoses			
ASD	18 (18.2%)		
ADHD	85 (85.9%)		
SLD	24 (24.2%)		
		Skewness (*SE*)	**Kurtosis (*SE*)**
Mother Education		-1.03 (.24)	.38 (.48)
Graduated from Trade/Business School	1 (1.0%)		
Completed 9th grade	2 (2.0%)		
Complete 10th or 11th grade	13 (13.1%)		
Graduate from high school/GED	1 (1.0%)		
Attended college/ specialized training program	22 (22.2%)		
Graduated college	33 (33.3%)		
Completed Graduate School	27 (27.3%)		
Father Education (*n* = 83)		-.33 (.26)	-1.10 (.52)
Graduated from Trade/Business School	6 (6.1%)		
Completed 9th grade	1 (1.0%)		
Complete 10th or 11th grade	2 (2.0%)		
Graduate from high school/GED	20 (20.2%)		
Attended college/ specialized training program	17 (17.2%)		
Graduated college	16 (16.2%)		
Completed Graduate School	21 (21.2%)		

Table 1 continued. *Demographic Data*

	***M* (*SD*)**	**Range**	**Skewness (*SE*)**	**Kurtosis (*SE*)**
Age	11.40 (2.79)	7 - 17	.45 (.24)	-.63 (.48)
FSIQ[1]	96.08 (16.73)	50 - 136	-.33 (.24)	.18 (.48)
Family Income (n = 70)	$82,787 (48,668)	$3,500 – $220,000	.83 (.29)	.67 (.57)

Notes. 1. Full Scale IQ, as measured by WISC or WAIS

Measures

Screen for Child Anxiety and Related Emotional Disorders (SCARED-P and SCARED-C; Birmaher, Khetarpal, Cully, Brent, & McKenzie, 1995)

The SCARED is a 41-item instrument that includes both parent report (SCARED-P) and child self-report versions (SCARED-C). Items on the SCARED-C are identical in content to SCARED-P and have been altered only in their subject reference (i.e., "I…" instead of "My child…"). Each item is on a 3-point Likert scale (0 = not true, 1 = sometimes true, 2 = very true), and scores are totaled to create a single measure of anxiety (possible range: 0 – 74). The SCARED and its subscales have shown strong internal consistency, discriminant validity, and test-retest reliability (Birmaher et al., 1997). Subscales of the SCARED address panic symptoms, generalized anxiety, separation anxiety, social anxiety, and school avoidance, with suggested clinical cutoffs provided by Birmaher and colleagues (1995). The current study will use the social anxiety scale (SCARED-SA), specifically, to address self-reported (SA-Child) and parent-reported (SA-Parent) symptoms of social anxiety. The social anxiety subscale consists of seven items which asks questions such as "I don't *(My child doesn't)* like to be with people I don't *(my child doesn't)* know well" and "I am *(My child is)* shy". Items are totaled to create a composite measure of social anxiety (possible range: 0 – 14); scores above 8 "may indicate social anxiety disorder" (Birmaher et al., 1995).

Social Responsiveness Scale, Second Edition **(SRS-2; Constantino & Gruber, 2012)**

The SRS-2 is a 65-item parent-report questionnaire designed to measure social reciprocity in children. The school-age form of the SRS-2 demonstrates strong internal consistency ($\alpha = .95$) in children ages 4 – 18 (Constantino & Gruber, 2012). Subscales of the SRS-2 include social awareness, social cognition, social motivation, social communication, and restricted and repetitive behaviors. Items are rated on a 4-point Likert scale, (not true, sometimes true, often true, almost always true), and raw scores are totaled and then converted to age-based, standardized scores (T-scores), where greater scores reflect greater impairment. The present study will utilize the social cognition subscale (SRS-2-SC) as a measure of social cognitive impairment. The SRS-2-SC includes twelve items measuring ability to interpret social cues (e.g., "Has a sense of humor, understands jokes"; "Is able to understand the meaning of other people's tone of voice and facial expressions"). Previous research has used this subscale as a composite of social cognition for youth with ASD (see Aramaki et al., 2015; Rodgers et al., 2019), and the SRS-2-SC demonstrated sufficient internal validity in this sample ($\alpha = .86$).

Vineland Adaptive Behavior Scales, Third Edition **(VABS-III; Sparrow, Cicchetti, & Saulnier, 2016)**

The VABS-III is a 502-item parent-report questionnaire used to measure adaptive behavior within the domains of communication, daily living skills, and socialization in individuals aged 0 – 90+. Parents are instructed to begin at age-normed starting points, with basal and ceiling rules dictating the number of questions each informant answers. The VABS-III has been shown to have strong internal consistency ($\alpha = .94$ - $.99$), and discriminates children with developmental disabilities from their neurotypical peers (Mean functioning composite score = 14.93, 82.96, respectively; Sparrow, Cicchetti, & Saulnier, 2016). Respondents rate each item

on a 3-point Likert scale (never, sometimes, often). Items are totaled and standardized to create a composite score (VABS-III Adaptive Behavior Composite; VABS) which summarizes the child's performance across all domains (M = 100; SD = 15).

RESULTS

Preliminary Analyses and Descriptive Data

All data analyses were completed using SPSS statistical software. Ninety-nine participants with a diagnosis of ASD, ADHD, and/or SLD were administered the SCARED-P and SCARED-C, a measure of cognitive ability (WISC or WAIS), SRS-2, and the VABS-III. Across all competed measures, two items (.05%) on the SCARED-C**,** 12 items on the SCARED-P (.30%), and 1 item on the SRS-2 (.02%) were imputed into the dataset using mean substitution of measure subscales. Reliability analyses of primary measures suggested sufficient internal consistency across all measures (α = .86 - .96; descriptive data are presented in Table 2). Skewness and kurtosis statistics were converted to *z*-scores by dividing by the standard error, where a *z*-score greater than 3.29 suggested significant nonnormality (Fox, 2015). Upon closer examination of all variables, only mother education was significantly skewed see Tables 1, 2).

Table 2. *Descriptive Data*

N = 99	α	***M* (*SD*)**	**% Clinical Range**	**Range**	**Skewness (*SE*)**	**Kurtosis (*SE*)**
SCARED-P Total	.943	21.95 (15.43)	36%	2 – 66	.96 (.24)	.30 (.48)
SA-Parent	.918	5.51 (4.46)	27%	0 – 14	.45 (.24)	-.95 (.48)
SCARED-C Total	.940	22.14 (15.47)	40%	1 – 61	.71 (.24)	.24 (-.26)
SA-Child	.866	5.46 (4.04)	32%	0 – 14	.31 (.24)	-1.06 (.48)
SRS-2 Total (T-Score)	.967	63.79 (13.51)	25%	41 – 90+	.27(.24)	-.94(.48)
SRS-2-SC (T-Score)	.858	62.16 (13.83)	-	39 – 90+	.20 (.24)	.83 (.29)
VABS	-	85.84(13.89)	-	55 – 125	.55 (.24)	.14 (.48)

Initial Pearson correlations between predictors (3 correlations), covariates, and VABS (8 correlations) showed age (r = -.29, p = .004) and FSIQ (r = .35, p < .001) were significantly

associated with adaptive functioning. Family income was also associated with VABS (r = -.25, p = .037), though adjustment for eleven planned correlations using Bonferroni's adjustment set α = .005, and this correlation was no longer significant. As a result, age and FSIQ were included, where relevant, as covariates in subsequent statistical analyses. Bivariate Pearson r correlations of predictor variables are shown in Table 3. SA-Parent was correlated with SA-Child (r = .28, p = .006), as well as SRS-2-SC (r = .38, p < .001) and VABS (r = -.39, p < .001); increased parent-report of social anxiety symptoms was associated with increased child-report of social anxiety symptoms, increased social cognitive impairment, and reduced adaptive functioning. SRS-2-SC was also significantly correlated with VABS (r = -.63, p < .001), such that increased social cognitive impairment was associated with reduced adaptive functioning. SA-Child was not correlated with SRS-2-SC, nor was it correlated with VABS.

Given established social cognitive impairments in ASD (Bora & Pantelis, 2016; Chita-Tegmark, 2016; Golan, Gordon, Fichman, & Keinan, 2018; Pelphrey et al., 2002), preliminary analyses were conducted to examine diagnostic group differences in social cognitive impairment and reported social anxiety. Significant group differences emerged, such that parents of children with ASD (n = 18) reported significantly greater social cognitive impairment on the SRS-2-SC than parents of youth without a diagnosis of ASD (t(78) = 4.72, p < .001). Parents of children with ASD also reported significantly more symptoms of social anxiety (t(78) = 3.44, p = .001), and significantly lower adaptive functioning (t(78) = -3.09, p < .001). Children with ASD did not self-report more symptoms of social anxiety than non-ASD youth (t(78) = -.67, p = .507). Subsequent analyses were conducted excluding participants with a diagnoses of ASD in order to ensure that model effects were not driven primarily by symptoms of ASD (e.g., social cognitive impairments).

Table 3. *Bivariate Pearson Correlations (r) of Predictor/Demographic Variables with VABS*

	SA-Child	SRS-2-SC	VABS
SA-Parent	.28**	.38***	-.39***
SA-Child		-.03	-.02
SRS-2-SC (T-score)			-.63***
Age	.05	.09	-.29**
FSIQ	-.10	-.18	.35***
Income	.02	-.26	.25
Mother Education	.01	-.33**	.10
Father Education	.13	-.29*	.39

Notes. Bonferroni correction for 11 correlations: *$p < .01$; **$p < .005$; ***$p < .01$

Power Analyses

A power analysis using G*Power (Faul, Erdfelder, Lang, & Buchner, 2007) suggested that a sample size of 55 participants was needed to confidently ($\alpha = .05$) discern a moderate effect size ($f^2 = .15$), with sufficient power (1-$\beta = .8$), when testing the unique contribution of a single predictor out of five total predictor variables used to address *Aim 1*. In regards to *Aim 2*, a second power analysis using G*Power (Faul et al., 2007) suggested that a sample size of 78 participants was needed to test the unique contribution of three predictors out of nine total predictor variables in the regression model under the same parameters. Exploration of individual predictors within the discrepancy model was not sufficiently powered; a sample size of 114 participants was needed to confidently ($\alpha = .05$) discern a moderate effect size ($f^2 = .15$), with sufficient power (1-$\beta = .8$), when testing a model with nine predictor variables.

Aim 1. Social cognition and social anxiety as predictors of adaptive functioning

To address Aim 1, parent-reported SCARED-SA scores were first mean-centered to limit multicollinearity in the regression model. An initial hierarchical regression was conducted to clarify the unique predictive weight of social cognitive impairment (SRS-2-SC) compared to SA-Parent on functional outcome (VABS). Initial examination of collinearity in the model showed tolerance values consistently above the recommended cutoff of .1 and Variance Inflation Factor

(VIF) values below the recommended cutoff of ten (Cohen, Cohen, West & Aiken, 2003), suggesting the mean-centered variables were sufficiently non-related to be included as separate predictors in the model. FSIQ and age were first entered into the model in Step 1 as demographic covariates; SA-Parent and the quadratic term of SA-Parent were entered as predictors in Step 2; and social cognitive impairment was entered in as a fifth predictor in Step 3. The overall model was significant (adj R^2 = .49; $F(5, 93) = 19.99$; $p < .001$), and the addition of social cognitive impairment into the model significantly increased the amount of variance accounted for in VABS (SRS-2-SC β = -.52, t = -6.56, p <.001; $R^2\Delta$ = .22, $F\Delta(1, 93) = 43.07$, $p < .001$; see Table 4). Specifically, greater social cognitive impairment predicted lower adaptive functioning (see Figure 1). Importantly, although participants with ASD exhibited greater social cognitive impairment than their peers with ADHD and/or SLD, exclusion of individuals with ASD (n = 18) from analyses produced a similar statistical model, where social cognitive impairment still accounted for unique variance in VABS ($R^2\Delta$= .22, $F\Delta$ = (1, 75) = 29.09, p <.001).

Table 4. *SA-Parent and Social Cognitive Impairment as Predictors of VABS*

	Adj. R^2	***F***	***p***	***β***	***t***	***p***	**Tol.**	**VIF.**
Step 1	.174	11.34	<.001					
Age				-.26	-2.87	.005	1.00	1.00
FSIQ				.33	3.61	<.001	1.00	1.00
Step 2[a] ($R^2\Delta$ = .10)	.265	9.83	<.001					
SA-Parent				-.34	-3.51	.001	.81	1.23
SA-Parent^2				.03	.30	.77	.83	1.20
Step 3[a] ($R^2\Delta$ = .22)	.492	19.99	<.001					
SRS-2-SC				-.52	-6.56	<.001	.83	1.20

Notes a. Steps 2 and 3 include model covariates and previously-entered predictors, though they have been omitted from the table for clarity.

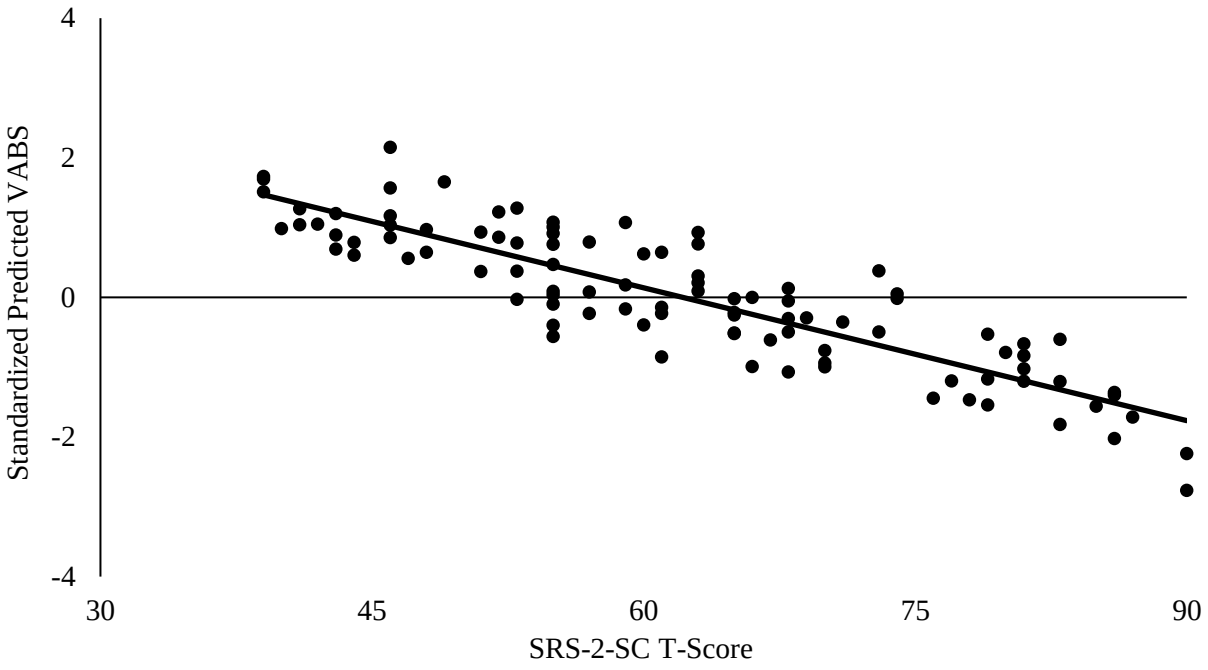

Figure 1. Predictive weight of social cognitive impairment regressed on VABS.

Aim 2. Predictive value of discrepant reported social anxiety on VABS

To address Aim 2, a Pearson *r* correlation was used to first compare SA-Child and SA-Parent ratings, which were found to be significantly related (r = .28, p = .006). This association is slightly smaller than research examining parent/child dyad reports on the SCARED (r = .44; Becker et al., 2016), though Fisher's r to z transformations indicated that these associations were not significantly different (z = -1.65, p =.099). To further clarify initial patterns of informant discrepancies, the absolute value of the difference between SA-Parent and SA-Child was examined (M = 3.90, SD = 3.32). An independent samples *t*-test indicated that this difference between SA-Parent and SA-Child was significantly different from 0 (t(98) = 11.70, p < .001). Though this analysis does not provide information about directionality, these findings suggest

that parents and children reported significantly different levels of social anxiety symptoms, with an average dyad difference of approximately 4 units (SCARED-SA possible range: 0 – 14).

A Polynomial Regression Analysis was then utilized to evaluate the unique predictive weight of SA-Parent, SA-Child, and informant discrepancy on adaptive functioning (VABS), following the statistical procedure set forth by Laird and De Los Reyes (2013). Initial examination of collinearity in the model showed tolerance values consistently above the recommended cutoff of .1 and VIF values below the recommended cutoff of ten (Cohen et al., 2003), suggesting the mean-centered variables were sufficiently non-related to be included as separate predictors in the model. FSIQ and age were entered into the model in Step 1 as demographic covariates, then seven variables of social anxiety including parent- and child-report, parent- and child- report^2 in Step 2, and finally three interaction terms (SA-Child x SA-Parent; SA-Child^2 x SA-Parent SA-Child x SA-Parent^2) in Step 3. The overall model was significant (adj R^2 = .25, $F(9, 89) = 4.48$, $p < .001$). Examination of individual predictors revealed that age (β = -.26, t = -2.74, p = .007), FSIQ (β = .33, t = 3.58, p = .001), and SA-Parent (β = -.37, t = -3.76, p <.001) each significantly predicted VABS within their respective steps. As age decreased, and as FSIQ and SA-Parent increased, the model reflected an associated increase in parent-reported adaptive behavior. Although the addition of the three interaction terms into the model did not significantly increase the amount of variance accounted for in VABS ($R^2\Delta$ = .008, $F\Delta(3, 89) = .35$, p =.790; see Table 5), examination of the seven predictor variables capturing social anxiety in the final model indicated SA-Parent significantly predicted VABS (β = -.38, t = -2.60, p = .011), where increased parent-report of social anxiety was associated with decreased adaptive functioning (See Figure 2).

Table 5. *SA-Parent, SA-Child, and Informant Discrepancies as Predictors of VABS*

	Adj. R^2	*F*	*p*	*β*	*t*	*p*	Tol.	VIF.
Step 1	.174	11.34	<.001					
Age				-.26	-2.74	.007	1.00	1.00
FSIQ				.33	3.58	.001	1.00	1.00
Step 2[a] ($R^2\Delta$ = .12)	.268	7.00	<.001					
SA-Parent				-.37	-3.76	<.001	.77	1.30
SA-Child				.14	1.54	.128	.85	1.18
SA-Parent^2				.04	.36	.717	.80	1.24
SA-Child^2				-.06	-.68	.499	.87	1.15
Step 3[a] ($R^2\Delta$ = .01)	.252	4.48	<.001					
SA-Parent				-0.38	-2.60	.011	0.35	2.89
SA-Child				0.11	0.78	.437	0.38	2.66
SA-Parent^2				0.01	0.11	.916	0.75	1.33
SA-Child^2				-0.08	-.83	.410	0.85	1.18
SA-Parent x SA-Child				0.08	.75	.458	0.67	1.50
SA-Parent x SA-Child^2				0.02	.11	.915	0.34	3.00
SA-Child x SA-Parent^2				0.04	.26	.798	0.32	3.15

Notes a. Steps 2 and 3 include model covariates, though they have been omitted from the table for clarity.

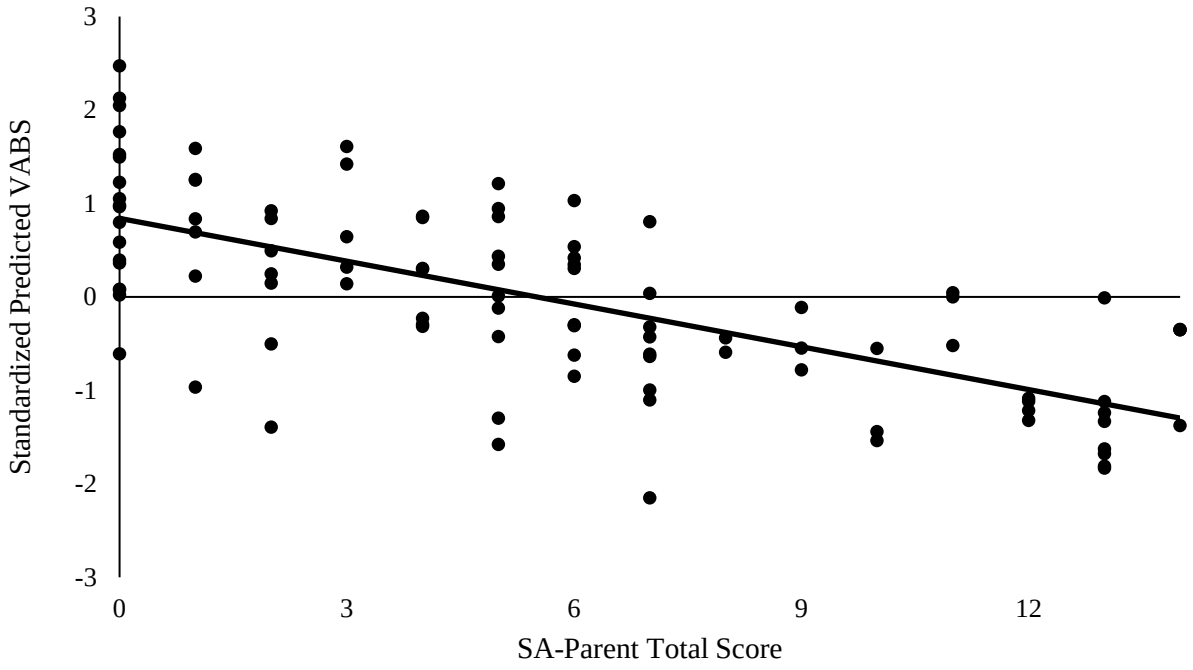

Figure 2. Predictive weight of SA-Parent regressed on VABS.

Supplemental Analyses

Power Analyses

Power analyses using G*Power (Faul et al., 2007) suggested that a sample size of 55 participants was needed to confidently (α = .05) discern a moderate effect size (f^2 = .15), with sufficient power (1-β = .8), when testing the unique contribution of a single predictor out of ten total predictor variables when examining the Children's Global Assessment Scale (CGAS) as the dependent variable. A sample size of 78 participants was needed to conduct a similarly powered regression when testing the unique contribution of three predictors out of eleven total predictor variables. However, a regression analysis exploring the relative weight of individual predictors within the discrepancy model was not sufficiently powered; a sample size of 123 participants was needed to confidently (α = .05) discern a moderate effect size (f^2 = .15), with sufficient power (1-β = .8), when testing a model with nine variables predicting CGAS. Finally, logistic regressions examining the Clinical Global Impression – Severity Scale (CGI) were all substantially underpowered; per the recommendation of Peduzzi and colleagues (1996) a sample size of 180 is needed to run a bivariate logistic regression model with nine predictors where the proportion of positive (i.e., clinical) cases is 50%, and a sample size of 200 is needed to run a similar model with ten predictors.

Children's Global Assessment Scale (CGAS; Shaffer et al., 1982)

The CGAS is a numeric scale used by clinicians to provide an objective rating of a patient's general functioning (Shaffer et al., 1982**)**. Seventy-nine participants were rated by their evaluating clinician using the CGAS scale, with ratings ranging from 1 to 100 where lower scores indicate greater impairment (*M* = 57.76; *SD* = 10.72). The primary assessing clinician provided a CGAS rating for each participant based on all data and assessment findings (e.g.,

parent- and child-reported surveys, behavioral analyses, cognitive assessment, etc.) Examination of the CGAS suggested that this variable was positively skewed (skewness = -1.03; *SE* = .25; *z* = 4.12), where a *z*-score greater than 3.29 suggests significant nonnormality (Fox, 2015). Preliminary correlations showed that the CGAS was significantly correlated with FSIQ (r = .31, p = .006), maternal education (r = .44, p < .001) and paternal education (r = .25, p = .024), so these demographic variables were included in subsequent regression analyses as covariates.

Social cognitive impairment and social anxiety as predictors of CGAS. The CGAS and primary predictor variables were first mean-centered in order to limit multicollinearity in the regression model. An initial hierarchical regression was conducted to clarify the unique predictive weight of social cognition (SRS-2-SC) compared to SA-Child, SA-Parent and informant discrepancy in social anxiety on general functioning (CGAS). Initial examination of collinearity in the model showed tolerance values consistently above the recommended cutoff of .1 and Variance Inflation Factor (VIF) values below the recommended cutoff of ten (Cohen et al., 2003), suggesting the mean-centered variables were sufficiently non-related to be included as separate predictors in the model. Maternal education, paternal education, and FSIQ were first entered into the model in Step 1 as demographic covariates; seven variables of social anxiety including SA-Child and SA-Parent, quadratic terms of SA-Child and SA-Parent, and three interaction terms were entered in Step 2; and social cognitive impairment was entered in as an eleventh predictor in Step 3. The overall model was significant (adj R^2 = .34; $F(11, 67) = 4.63$; p < .001), and the addition of social cognition into the model significantly increased the amount of variance accounted for in CGAS ($R^2\Delta$ = .03, $F\Delta(1, 67) = 3.97$, p =.050; see Table 6). These findings suggest that social cognitive impairment accounted for unique variance in global functioning, apart from that of SA-Parent, SA-Child, and discrepant reporting in social anxiety.

Specifically, higher T-scores (reflecting higher social cognitive impairment) predicted worse global functioning (see Figure 3). Importantly, although participants with ASD exhibited greater social cognitive impairment than their peers with ADHD and/or SLD, exclusion of individuals with ASD produced very similar statistical models, where social cognition still accounted for unique variance in CGAS ($R^2\Delta$= .04, $F\Delta$ = (1, 51) = 4.35, p =.042).

Table 6. *SA-Parent, SA-Child, and Social Cognitive Impairment as Predictors of CGAS*

n = 79	**Adj. R^2**	***F***	***p***	***β***	***t***	***p***	**Tol.**	**VIF.**
Step 1	.210	7.91	<.001					
Mother Education				0.38	3.20	.002	0.73	1.37
Father Education				0.02	0.20	.846	0.74	1.36
FSIQ				0.23	2.21	.030	0.95	1.05
Step 2[a] ($R^2\Delta$ = .16)	.310	4.50	<.001					
SA-Parent				-0.05	-0.28	.779	0.33	3.03
SA-Child				0.21	1.29	.202	0.35	2.90
SA-Parent^2				-0.04	-0.34	.737	0.75	1.34
SA-Child^2				-0.15	-1.40	.169	0.79	1.26
SA-Parent x SA-Child				-0.14	-1.08	.283	0.55	1.82
SA-Parent x SA-Child^2				-0.24	-1.38	.171	0.30	3.40
SA-Child x SA-Parent^2				-.01	-.03	.979	0.30	3.33
Step 3[a] ($R^2\Delta$ = .03)	.339	4.63	<.001					
SRS-2-SC				-0.23	-2.00	.050	0.64	1.55

Notes a. Steps 2 and 3 include model covariates and previously-entered predictors, though they have been omitted from the table for clarity

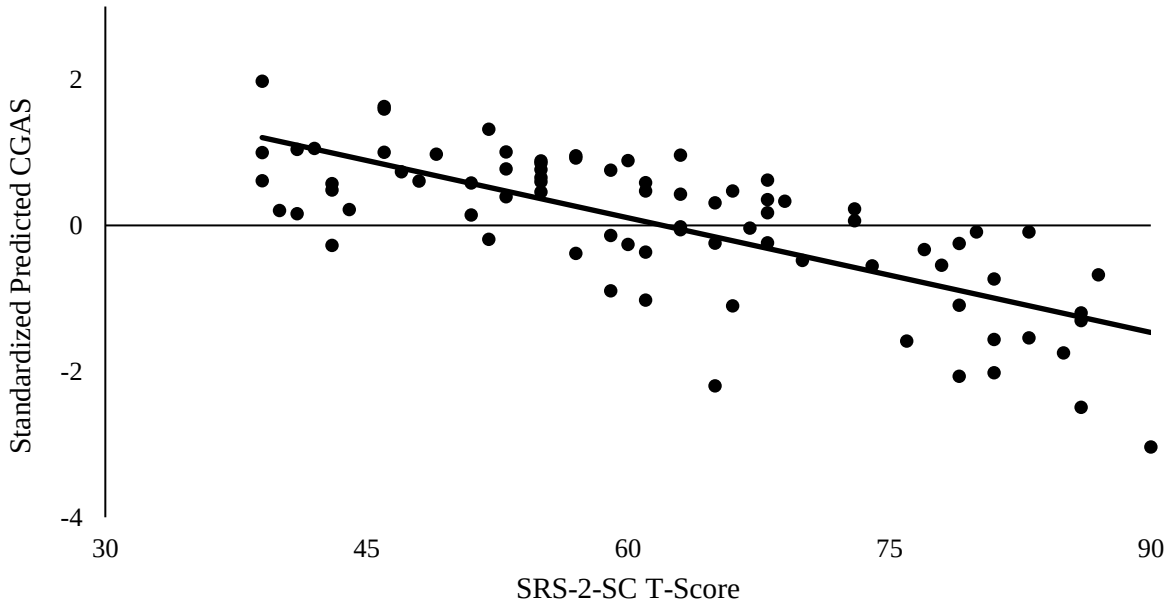

Figure 3. Predictive weight of social cognitive impairment regressed on CGAS.

Predictive value of discrepant reported social anxiety on CGAS. A Polynomial Regression Analysis was then utilized to evaluate the unique predictive weight of SA-Parent, SA-Child, and informant discrepancy in reported social anxiety on clinician-rated impairment (CGAS), following the statistical procedure set forth by Laird and De Los Reyes (2013). Initial examination of collinearity in the model showed tolerance values consistently above the recommended cutoff of .1 and Variance Inflation Factor (VIF) values below the recommended cutoff of ten (Cohen et al., 2003), suggesting the mean-centered variables were sufficiently non-related to be included as separate predictors in the model. Maternal education, paternal education, and FSIQ were first entered into the model in Step 1 as demographic covariates; in Step 2 four variables capturing social anxiety (SA-Parent, SA-Child, and respective quadratic terms) were entered into the model; finally, in Step 3, three interaction terms (SA-Parent x SA-Child; SA-Child^2 x SA-Parent, and SA-Child x SA-Parent^2) were entered into the model. The

overall model was significant (adj R^2 = .31; $F(10, 68) = 4.50$; p <.001); however, the addition of the three interaction terms into the model did not significantly increase the amount of variance accounted for in CGAS ($R^2\Delta$ = .05, $F\Delta(3, 68) = 1.90$, p =.137; see Table 7). These findings suggest that the interaction between parent- and child-reported social anxiety is not uniquely associated with variance in clinician-report of global functioning in this population of children with neurodevelopmental disorders. Additionally, examination of the final model suggests that none of the seven predictor variables accounted for a significant amount of variance in global functioning.

Though nonsignificant, several predictors in the final model were relatively large compared to others in the model; SA-Child (β = .21, $t(1, 78) = 1.29$, p = .202), the quadratic effect of SA-Child (β = -.15, $t(1, 78) = -1.39$, p = .169), and the interaction between SA-Parent and the quadratic effect of SA-Child (β = -.24, $t(1, 78) = -1.38$, p = .171) each explained considerable, though not statistically significant, variance in overall functioning. Cohen's f^2 values were calculated for each predictor: Cohen's $f^2 = \frac{sr_i^2}{1-R^2_{full}}$ where sr_i^2 is the squared semipartial correlation coefficient for the identified predictor and R^2_{full} is the squared multiple correlation coefficient for the full model. The identified Cohen's f^2 values (SA-Child = .024, SA-Child^2 = .029, SA-Parent x SA-Child^2 = .028) are considered small effects (Cohen, 1988), but given the relatively tiny effects noted in research examining moderation, the effect size of the interaction (SA-Parent x SA-Child^2) might actually be considered a large effect in the context of similar research (Aguinis, Beaty, Boik, & Pierce, 2005). Further examination of these effect sizes indicated that sample sizes of 329 (SA-Child), 273 (SA-Child^2), and 283 (SA-Parent x SA-Child^2) would be needed in order to achieve 80 percent power in these regression analyses (Faul et al., 2007). Considering these effect sizes, and given the demonstrably underpowered nature of

this analysis, attention should be drawn to these predictors as well. The quadratic effect of SA-Child provides a better estimate of the relationship between SA-Child and global functioning than the linear effect, and this quadratic term reflects that both depressed and elevated child ratings of social anxiety symptoms are associated with decreased global functioning (Figure 4). The interaction was interpreted with SA-Parent as the moderator in order to plot the SA-Child quadratic effect. As shown in Figure 5, the quadratic effect is negative for high levels of SA-Parent and neutral for low levels of SA-Parent, suggesting that congruence in elevated reports of social anxiety is associated with the lowest levels of global functioning.

Table 7. *SA-Parent, SA-Child, and Informant Discrepancies as Predictors of CGAS*

n = 79	**Adj. R^2**	***F***	***p***	***β***	***t***	***p***	**Tol.**	**VIF.**
Step 1	.210	7.91	<.001					
Mother Education				0.38	3.20	.002	0.73	1.38
Father Education				0.02	0.20	.846	0.74	1.36
FSIQ				0.23	2.21	.030	0.95	1.05
Step 2[a] ($R^2\Delta$ = .11)	.283	5.41	<.001					
SA-Parent				-0.24	-2.19	.032	0.76	1.31
SA-Child				0.17	1.58	.119	0.77	1.30
SA-Parent^2				-0.08	-0.76	.453	0.79	1.26
SA-Child^2				-0.18	-1.65	.104	0.81	1.23
Step 3[a] ($R^2\Delta$ = .05)	.310	4.50	<.001					
SA-Parent				-0.05	-0.28	.779	0.33	3.03
SA-Child				0.21	1.29	.202	0.35	2.89
SA-Parent^2				-0.04	-0.34	.737	0.75	1.34
SA-Child^2				-0.15	-1.39	.169	0.79	1.26
SA-Parent x SA-Child				-0.14	-1.08	.283	0.55	1.82
SA-Parent x SA-Child^2				-0.24	-1.38	.171	0.29	3.40
SA-Child x SA-Parent^2				-0.01	-0.03	.979	0.30	3.33

Notes a. Steps 2 and 3 include model covariates, though they have been omitted from the table for clarity

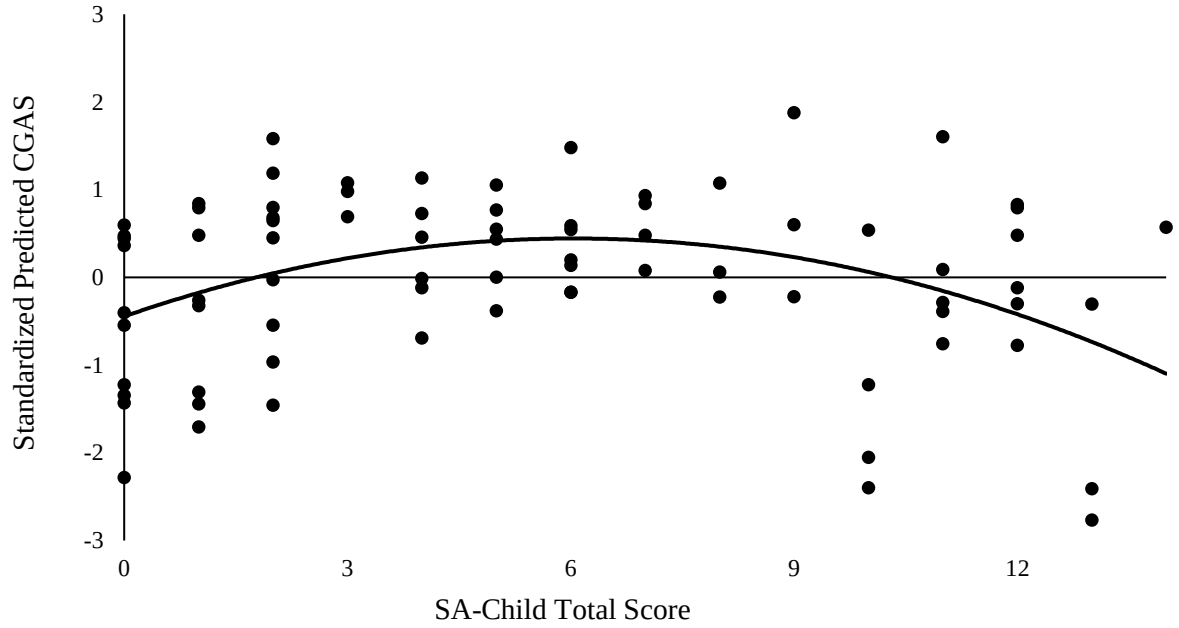

Figure 4. Quadratic effect of SA-Child regressed on CGAS.

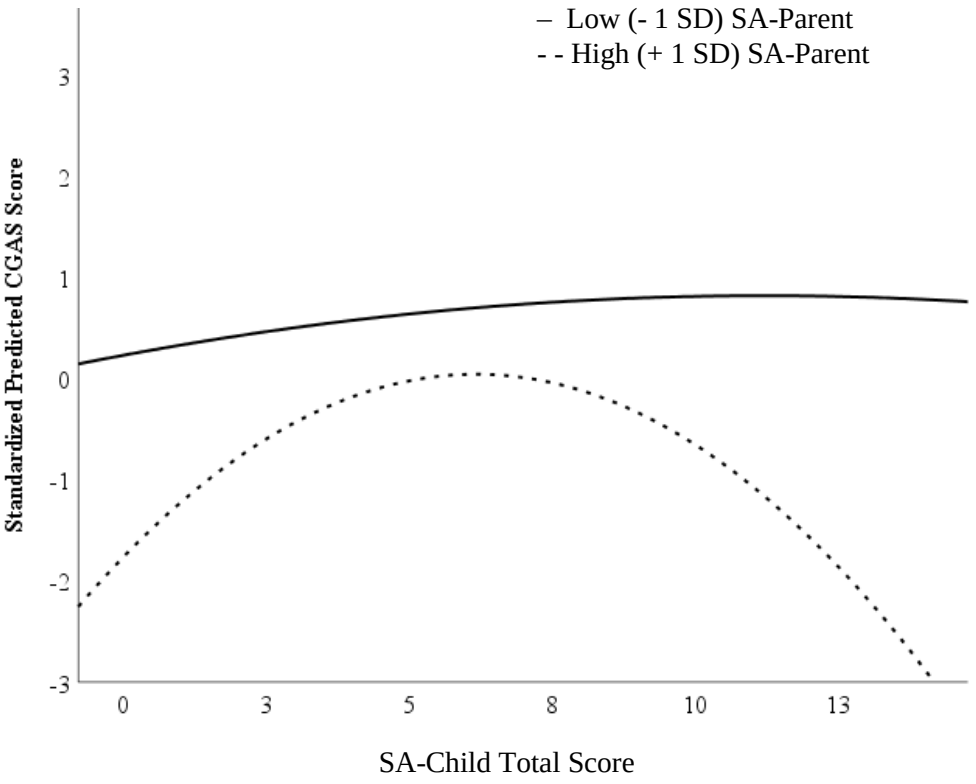

Figure 5. Predicted values of CGAS as a function of SA-Child at high and low values of SA-Parent.

Clinical Global Impression – Severity Scale (CGI; Guy, 1976)

The clinician-rated CGI uses a 7-point Likert scale to capture the severity of an individuals' illness at the time of assessment, relative to the clinician's past experience with patients who have the same diagnosis (Guy, 1976). Scores on this measure range from 1 (normal; not at all ill) to 7 (among the most extremely ill patients), and scores above 3 suggest moderate clinical impairment (see Appendix 1). The primary assessing clinician provided a CGI rating for each participant based on all available data and assessment findings, and this rating was subsequently discussed with the assessment team and supervising psychologist in order to reach a consensus CGI rating which was ultimately entered into the dataset. Participants in the current study were rated as mild to moderately ill, on average (n = 79; M = 3.68; SD = .86). The CGI is used often as a measurement of functioning in intervention studies (e.g., Kent et al., 2014; Mankad et al., 2015; Voigt et al., 2014) and variance in this measure may also be explained by social anxiety and social cognition. CGI ratings were divided into either minimal to no clinical impairment (CGI = 0 – 3, n = 36, 45% of sample), or moderate to severe clinical impairment (CGI = 4 – 7, n = 43; 54% of sample). Preliminary correlations showed that CGI was correlated with maternal education (r = -.35, p = .001) and paternal education (r = -.26, p = .022), so both variables were included in the first step of subsequent regression analyses as covariates.

Social cognitive impairment and social anxiety as predictors of CGI. A bivariate logistic regression was performed to determine the unique influence of social cognitive impairment on clinician-reported CGI (see Table 8). Maternal education and paternal education were first entered into the model as covariates, and this first step of the logistic regression model trended towards statistical significance ($\chi^2(2) = 5.96$, p = .051) and explained 9.7% (Nagelkerke R^2) of the variance in CGI, correctly classifying 58.2% of cases. Seven variables capturing social

anxiety (SA-Child and SA-Parent, quadratic terms of SA-Child and SA-Parent, and three interaction terms) were then entered into the model in the second step. This second logistic regression model was statistically significant ($\chi^2(9) = 27.634$, $p = .001$), and the adjusted model explained 39.5% (Nagelkerke R^2) of the variance in CGI, correctly classifying 75.9% of cases. Finally, social cognitive impairment was entered into the model in a third and final step, and the final model was also statistically significant ($\chi^2(10) = 38.64$, $p < .001$). The third and final model explained 51.7% (Nagelkerke R^2) of the variance in CGI and correctly classified 75.9% of cases. The third step of this model was explained a significant amount of variance in predicted rating of moderate clinical impairment ($\chi^2(1) = 11.01$, $p = .001$), suggesting social cognitive impairment significantly predicted group membership (i.e., clinician rating of moderate vs. minimal impairment). The odds ratio of 1.09 indicates that when social anxiety, mother education, and father education are held constant, the odds of a rating of moderate-to-severe clinical impairment on the CGI increase by a factor of 1.09 with every unit increase in T-score. In other words, a T-score of 65, indicating clinical threshold of impairment on the SRS-2, is 3.64 times more likely to be associated with a CGI rating of moderate clinical impairment than is a T-score of 50, (fifteen units less than T = 60; $1.09^{15} = 3.64$). In this model, higher T-scores reflecting higher social cognitive impairment predicted with increasing likelihood membership in the group with clinically impairing CGI scores (See Figure 6). Importantly, although participants with ASD exhibited greater social cognitive impairment than their peers with ADHD and/or SLD, exclusion of individuals with ASD produced very similar statistical models, where social cognition still accounted for unique variance in CGI score ($\chi^2(10) = 5.54$, $p = .018$, Odds Ratio = 1.07).

Table 8. *Binary Logistic Regression of Social Anxiety and Social Cognition as Predictors of CGI*

n = 79	Nagelkerke R^2	χ^2	*p*	*B*	*SE*	*Wald*	*p*	Odds Ratio
Step 1	.097	5.96	.051					
Mother Education				-0.39	.23	2.92	0.088	.68
Father Education				-0.08	.17	.22	0.636	.92
Step 2[a]	.395	21.68	.003					
SA-Parent				-0.18	.11	2.51	.113	.84
SA-Child				-0.27	.13	4.61	0.32	.76
SA-Parent^2				0.00	.02	0.01	.936	1.00
SA-Child^2				0.08	.03	9.07	.003	1.08
SA-Parent x SA-Child				-0.02	.02	0.89	.346	.98
SA-Parent x SA-Child^2				0.02	.01	6.47	.011	1.02
SA-Child x SA-Parent^2				0.00	.00	0.82	.364	1.00
Step 3[a]	.517	11.01	.001					
Social Cognition (T-score)				.09	.03	8.87	.003	1.09

Notes a. Steps 2 and 3 include model covariates and previously-entered predictors, though they have been omitted from the table for clarity

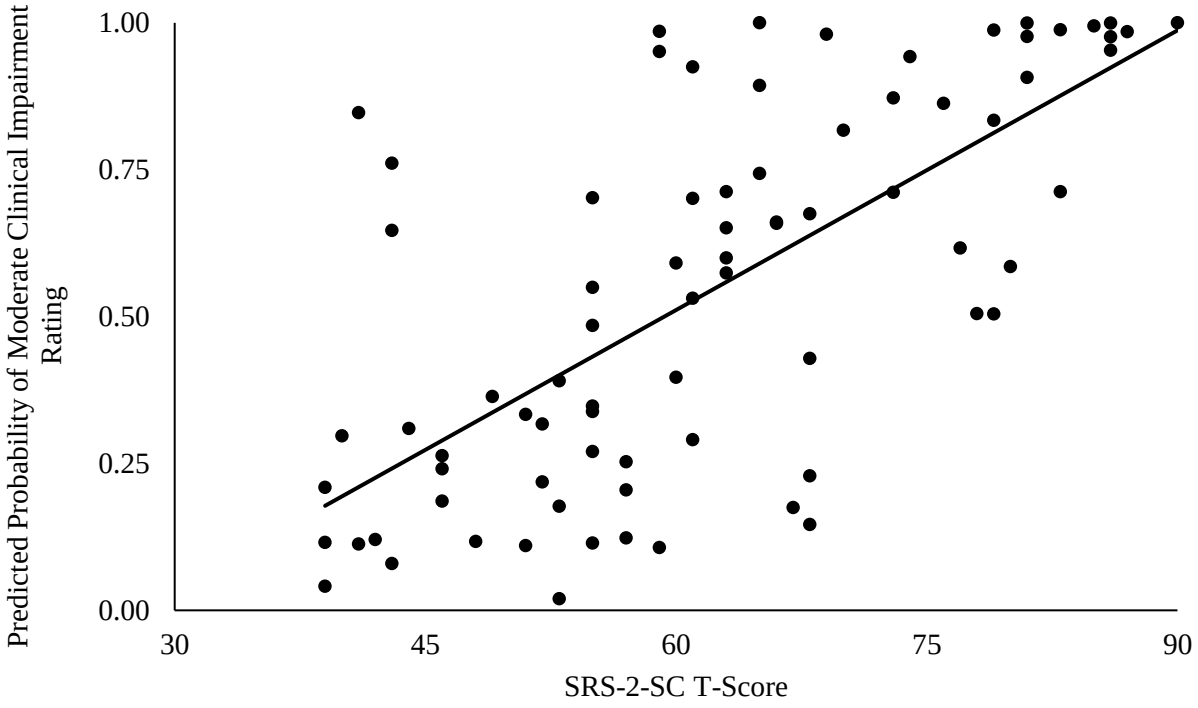

Figure 6. Predictive weight of social cognitive impairment regressed on probability of CGI moderate impairment rating.

Predictive value of discrepant reported social anxiety on CGI. A second bivariate logistic regression was performed to examine the extent to which discrepancies in SA-Parent and SA-Child accounted for variance in CGI (see Table 9). The overall model was significant ($\chi^2(9) = 27.634$, $p = .001$). Maternal education and paternal education were again entered into the model as covariates, followed by four variables capturing social anxiety (SA-Parent, SA-Child, and respective quadratic terns) in the second step. This second logistic regression model was statistically significant ($\chi^2(6) = 17.18$, $p = .024$), and the adjusted model explained 26.1% (Nagelkerke R^2) of the variance in CGI, correctly classifying 69.6% of cases. Finally, social cognitive impairment was entered into the model in a third and final step, and this final model was also statistically significant ($\chi^2(9) = 27.63$, $p = .001$), explaining 39.5% (Nagelkerke R^2) of the variance in CGI and correctly classifying 75.9% of cases. Examination of individual predictor variables showed SA-Child, the quadratic effect of SA-Child (Figure 7), and the interaction between SA-Parent and the quadratic effect of SA-Child (Figure 8) significantly predicted group membership. Examination of SA-Child as a predictor revealed an odds ratio of .76 suggesting that, when other measures of social anxiety, mother education, and father education were held constant, the odds of a rating of moderate-to-severe clinical impairment on the CGI decreases by a factor of 1.3 (or increases by a factor of .76) with every unit increase in SA-Child (i.e., SA-Child score of 4 is 1.3 times less likely than an SA-Child score of 3 to be associated with a CGI rating of moderate clinical impairment). More informative, though, is the significant quadratic term of SA-Child, which indicated that both low and high reports of social anxiety were associated with increased likelihood of a clinician rating of moderate clinical impairment on the CGI (see Figure 7). The interaction was interpreted with SA-Parent as the moderator in order to plot the quadratic effect of SA-Child. As shown in Figure 8, the quadratic

effect is positive for high levels of SA-Parent and slightly negative for low levels of SA-Parent, suggesting that congruence in elevated reports of social anxiety and discrepancy when SA-Parent is elevated are both associated with the highest likelihood of clinician-rated impairment on the CGI. Per this interaction, lower ratings of SA-Parent were less predictive of clinician-rated impairment, regardless of SA-Child values.

Table 9. *Binary Logistic Regression of SA-Parent, SA-Child, and Informant Discrepancies as Predictors of CGI*

n = 79	**Nagelkerke R^2**	χ^2	*p*	*B*	*SE*	*Wald*	*p*	**Odds Ratio**
Step 1	.097	5.96	.051					
Mother Education				-0.39	.23	2.92	0.088	.68
Father Education				-0.08	.17	.223	0.636	.92
Step 2[a]	.261	11.22	.024					
SA-Parent				0.07	.07	1.23	0.176	1.08
SA- Child				-0.10	.07	1.75	0.119	.91
SA-Parent^2				0.00	.01	0.00	1.00	1.00
SA-Child^2				0.05	.02	7.61	0.006	1.05
Step 3[a]	.395	10.46	.015					
SA-Parent				-.18	.11	2.51	.113	.84
SA- Child				-.27	.13	4.61	.032	.76
SA-Parent^2				.00	.02	.01	.936	1.00
SA-Child^2				.08	.03	9.07	.003	1.08
SA-Parent x SA-Child				-0.02	.02	.89	.346	.98
SA-Parent x SA-Child^2				0.01	.01	6.47	.011	1.02
SA-Child x SA-Parent^2				0.00	.00	.82	.364	1.00

Notes. a. Steps 2 and 3 include model covariates, though they have been omitted from the table for clarity

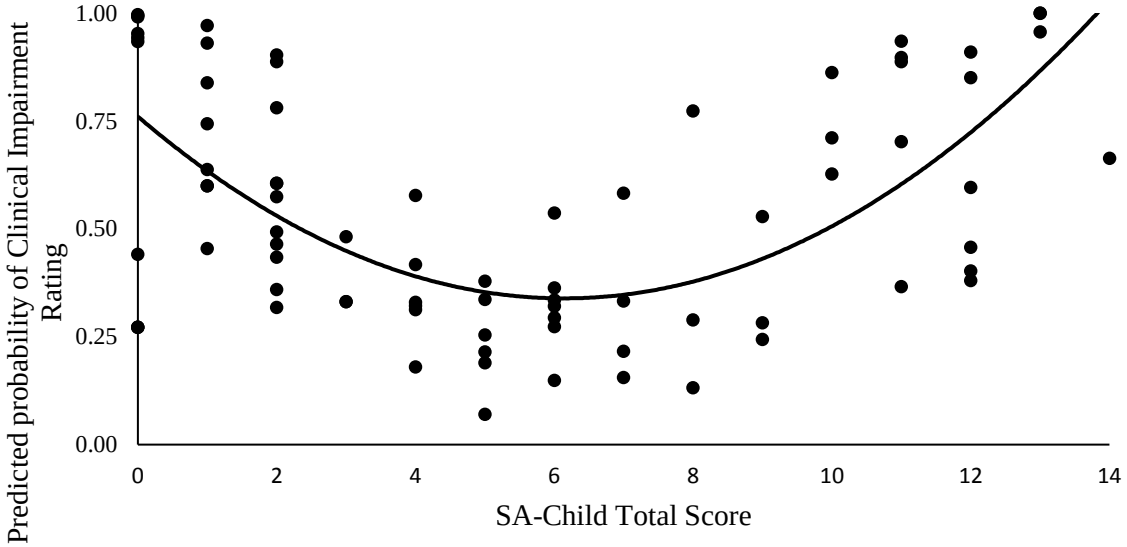

Figure 7. SA-Child regressed on probability of CGI moderate clinical impairment rating.

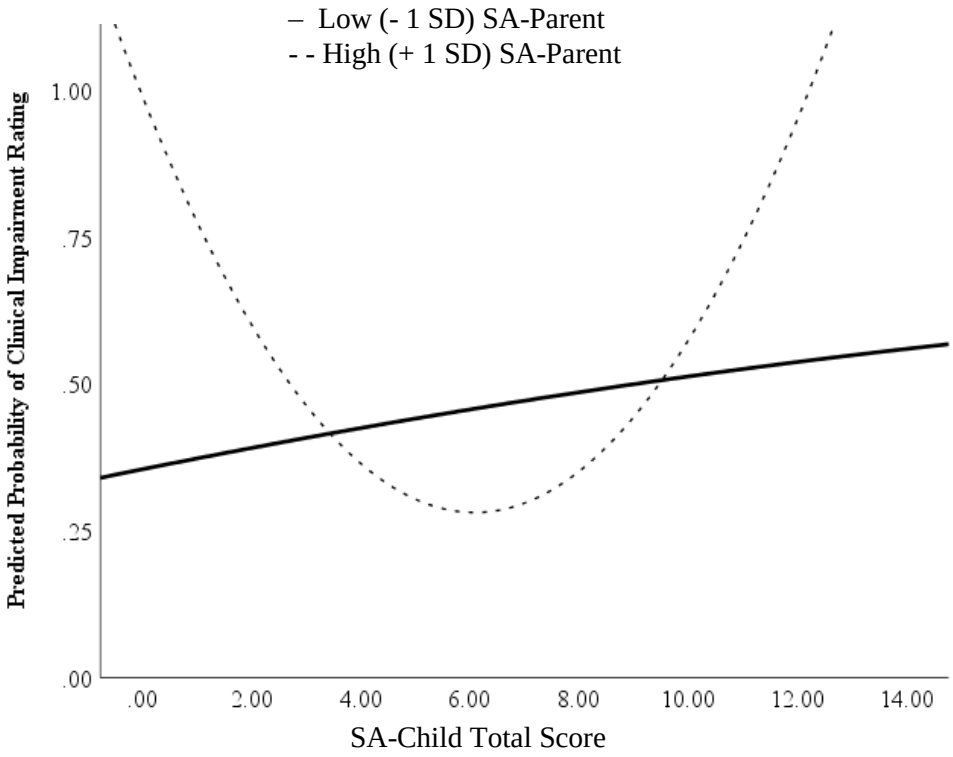

Figure 8. Predicted rating of moderate clinical impairment as a function of SA-Child at high and low values of SA-Parent.

DISCUSSION

Examination of parent-rated social anxiety and social cognitive impairment indicated that both accounted for significant variance in child's adaptive functioning. However, social cognitive impairment accounted for unique variance in adaptive functioning apart from parent-reported social anxiety. Social cognitive impairment was also more strongly associated with adaptive behavior than any other predictor variable, and the predictive model including social cognition accounted for variance above and beyond that accounted for by parent-reported social anxiety and identified covariates. Interestingly, age was found to be a negative predictor of adaptive functioning, echoing prior literature which has found age-related declines in adaptive functioning in youth with ASD, specifically (Pugliese et al., 2015). Additionally, supplemental analyses showed social cognitive impairment significantly predicted clinician-rating of global functioning (CGAS), as well as clinician-rating of clinical impairment (CGI). Although participants with ASD exhibited greater social cognitive impairment than their peers with ADHD and/or SLD, exclusion of individuals with ASD produced very similar statistical models, where social cognitive impairment still accounted for unique variance in all three measured outcomes. These findings suggest that social cognition as a construct carries substantial weight when considering functional outcomes in youth with neurodevelopmental disorders. The predictive value of social cognition can and should be leveraged in future research examining and intervening on broader impairment in youth with ASD, ADHD, and SLD. In sum, findings support the first hypothesis, providing evidence for the unique contribution of social cognitive impairment to adaptive functioning in youth with neurodevelopmental disorders.

Findings from Aim 2 support *Hypotheses 2,* indicating SA-Parent and SA-Child were associated, and yet significantly different from one another. Without speaking to directionality, findings suggest that parent/children dyad reports of social anxiety tended to differ by a score of approximately 4 points on the SCARED-SA subscale. This finding echoes previous literature which has found parents disagree with their children when reporting symptoms of social anxiety (Becker et al., 2016; Cosi et al., 2010). Regarding *Hypothesis 3,* findings revealed the interaction between SA-Parent and SA-Child was not uniquely associated with variance in the child's adaptive behavior. This finding may be related to the chosen outcome, in that VABS may be more distally related to social anxiety than the present study was able to capture in planned statistical analyses. It is also possible that the VABS, though widely used in anxiety research and well-validated in a variety of youth demographics (Rappaport et al., 2017), may not be an ideal outcome for research examining informant discrepancies. Additionally, parent- and self-report may not vary sufficiently on the seven-item SCARED-SA subscale to identify the impact of differential response patterns on measured outcomes. Research examining informant discrepancies in the SCARED echoes this nonsignificant finding of informant discrepancy as a predictor of youth functioning (Becker-Haimes et al., 2018).

However, supplemental analyses suggested that child ratings of social anxiety significantly predicted clinician ratings of global functioning and clinical impairment (i.e., CGAS and CGI) in a curvilinear model, as did the interaction between the quadratic-report of child-reported social anxiety and parent-reported social anxiety. A similar curvilinear relationship has been found between child-reported social anxiety and aggression, where youth with ASD who reported relatively high and relatively low social anxiety exhibited the highest levels of aggression within the sample (Pugliese, White, White, & Ollendick; 2013). This

curvilinear relationship between child-report and outcome is thought to demonstrate dual points of impairment derived from social anxiety, such that low levels of social anxiety may reflect disinhibition while high levels of social anxiety may reflect internalizing symptoms, both of which affect measured outcomes. This curvilinear relationship has not been demonstrated outside of ASD, and future research is needed to confidently assert this model in a population of youth with neurodevelopmental disorders. In sum, evidence from supplemental analyses builds upon previous findings suggesting that discrepant informant reports are related to functional outcome (De Los Reyes, 2011). Future research should continue to explore these variable relationships using alternative and more comprehensive measures in order to clarify the impact of discrepant reporting on a range of outcomes and subsequently inform diagnostic and treatment research for youth with neurodevelopmental disorders.

Limitations

There are a number of limiting factors to consider when conceptualizing this study. The selected measurement of social cognition does not reflect standard use of the SRS-2, which is typically use as a screening instrument for ASD. However, previous research has examined this subscale as an independent measure of social cognition (see Aramaki et al., 2015; Rodgers et al., 2019), and preliminary analyses of the SRS-2-SC suggest strong internal reliability ($\alpha = .86$), which supports the use of this subscale as a reliable assessment of social cognition within this sample. Nevertheless, the use of this parent-report scale may not fully capture social cognitive abilities and thus results must be interpreted with caution and with particular attention paid to the items within this subscale. Additionally data collected from parent/child dyads did not examine particular differences in maternal compared to paternal discrepancy in reported social anxiety. This study focused instead on the report of the primary caregiver, limiting the extent to which

conclusions can be drawn about particular parent/child dynamics. As dyadic discrepancies have been found to differ (Jansen et al., 2018), future research parsing apart differential patterns of mother/child and father/child discrepancies would help to clarify the relationship between social anxiety and functional outcome in youth with neurodevelopmental disorders. Furthermore, unmeasured variables such as social functioning and executive functioning might may account for additional variance in adaptive functioning, which diminishes the power of all proposed statistical analyses used to address Aim 1. Supplemental analyses using logistic regression were also substantially underpowered, limiting the interpretive value of these findings, in particular. Finally, this sample reflects a homogenous population of primarily Caucasian youth whose families had access to this specialty assessment clinic, limiting the generalizability of findings from this study to a broader population of youth with neurodevelopmental disorders.

Implications and Future Directions

Previous literature has established clear patterns of symptoms in youth with neurodevelopmental disorders, but research has yet to clearly examine how impairment in particular domains such as social anxiety and social cognition might interact and subsequently impact functional outcomes in this population. Furthermore, the inclusion of reporter discrepancies as an additional variable further clarifies the path to functional outcome in youth with neurodevelopmental disorders. While parent-reported adaptive functioning was not associated with informant discrepancy, both measures of clinician-rated functioning/impairment were, underscoring the prevailing argument for the inclusion of multiple informants and analysis of informant discrepancies as they relate to identified outcomes (De Los Reyes et al., 2015). This study builds on current literature, depicting an increasingly nuanced profile of social impairment

that transcends diagnostic groups, and findings speak to the importance of studying dimensional symptom impairment, as emphasized in the NIMH's RDoc Initiative (Clark et al., 2017).

The results from this study speak most directly to the relative importance of social cognitive impairment as a significant predictor of child functioning, rated by both parents and clinicians. Given that the relationship between social cognition and child functioning was not driven by symptoms of ASD, such a strong association certainly merits continued attention in both assessment and treatment research. Continued investigation of the influence of social cognitive impairment on other domains of functioning, as well as the potential wide-spanning treatment gains of interventions targeting social cognitive impairment in youth with neurodevelopmental disorders is certainly warranted. Moreover, this study's dimensional, rather than diagnostic, approach to measuring clinical symptoms also allows for application of these findings across neurodevelopmental disorders. These findings encourage the development of mechanism-based interventions addressing social cognitive impairment in the interest of impacting both proximal (i.e., social skill) and distal (i.e., adaptive functioning) outcomes in youth with neurodevelopmental disorders.

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APPENDIX 1

CGI Severity Scale, modified from Guy, 1976

Scoring guidelines for CGI

The CGI Impairment rating should be assigned when the evaluation is complete, and be assigned jointly by the lead clinician and supervisor. Ratings will be based on all available data including interviews, observations, and parent/child reports.

CGI: S (Severity)

- Every subject receives a severity rating of 1 to 7
- Higher scores reflect more severe impairment, or worse functioning
- Any ASD diagnosis automatically cannot get a lower rating than a 3 (i.e., only 3 through 7)

0	NOT ASSESSED
1	NORMAL No apparent mental health problems, not ill at all
2	BORDERLINE MENTALLY ILL Some mental health problem or diagnosis is present but functioning is not directly impaired as a result
3	MILDLY ILL Affected but functioning reasonably well Example: ASD diagnosis, but very minimal impairment – able to function pretty well with minimal accommodations
4	MODERATELY ILL Some adaptive, appropriate functioning but some pretty major problems as well (e.g., obsessions that get in the way) Example: child can function reasonably well in activities of daily living (e.g., school attendance, family functioning) but may require some accommodations from teachers or parents to do so (such as driving child to school to avoid taking the bus)
5	MARKEDLY ILL Minimal functioning, impairment across domains Example: child has considerable difficulty with activities of daily living, such as attending school regularly or getting along with family, due to severity of behavior problems/symptoms
6	SEVERELY ILL Little independence in any domain Example: child has been, within the past year, at risk for hospitalization
7	AMONG THE MOST EXTREMELY ILL PATIENTS Unable to function, even with supports, in all environments Example: child may not have been hospitalized for behavior problems, but there is considerable cause for concern due to safety of child or others due to child's behavior and hospitalization is probable/likely

APPENDIX 2

UA Documentation of IRB Exemption

Crimson

Grace (Grace Lee) Simmons <glsimmons@crimson.ua.edu>

IRB Protocol Approved: 19-01-1930, Simmons, Grace
1 message

eprotocol@fa.ua.edu <eprotocol@fa.ua.edu> Mon, Apr 22, 2019 at 12:40 PM
To: glsimmons@crimson.ua.edu, swwhite1@ua.edu

IRB has approved the protocol with the following details.

Protocol ID: 19-01-1930
Principal Investigator: Simmons, Grace
Department: Psychology
Protocol Title: Social Anxiety and Social Cognition in Youth with Neurodevelopmental Disorders
Review Type: EXEMPT
Approval Date: April 17, 2019