SHORT REPORT

Complex Interplay Between Cognitive Ability and Social Motivation in Predicting Social Skill: A Unique Role for Social Motivation in Children With Autism

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Impairment in social interaction is a core feature of autism spectrum disorder (ASD), but the factors which contribute to this social skill deficiency are poorly understood. Previous research has shown that cognitive ability can impact social skill development in ASD. Yet, children with ASD whose cognitive abilities are in the normal range nevertheless demonstrate deficits in social skill. More recently, the social motivation theory of ASD has emerged as a framework by which to understand how failure to seek social experiences may lead to social skill deficits. This study was designed to better understand the relationships between cognitive ability, social motivation, and social skill in a well-characterized cohort of children with ASD (n = 79), their unaffected siblings (n = 50), and unrelated neurotypical controls (n = 60). The following instruments were used: The Stanford-Binet intelligence quotient (IQ), the Social Responsiveness Scale's Social Motivation Subscale, and the Vineland Adaptive Behavior Scales' Socialization Standard Score. We found that lower cognitive ability contributed to diminished social skill, but did so universally in all children. In contrast, social motivation strongly predicted social skill only in children with ASD, such that those with the lowest social motivation exhibited the greatest social skill impairment. Notably, this relationship was observed across a large range of intellectual ability but was most pronounced in those with IQs ≥ 80. These findings establish a unique link between social motivation and social skill in ASD and support the hypothesis that low social motivation may impair social skill acquisition in this disorder, particularly in children without intellectual disability. Autism Res 2021, 14: 86-92. © 2020 International Society for Autism Research and Wiley Periodicals LLC.

Lay Summary: The relationships between cognitive ability, social motivation, and social skill are poorly understood. Here we report that cognitive ability predicts social skill in all children, whereas social motivation predicts social skill only in children with autism. These results establish a unique link between social motivation and social skill in autism, and suggest that low social motivation may impair social skill acquisition in this disorder, particularly in those without intellectual disability.

Keywords: autism spectrum disorder; children; cognitive dysfunction; intelligence tests; motivation; socialization; social skill

Introduction

Children with autism spectrum disorder (ASD) exhibit marked impairment in social skill that negatively impacts their social relationships [Carter, Davis, Klin, & Volkmar, 2005]. Previous research has shown that cognitive ability can influence social skill development in ASD, such that lower cognitive ability is associated with poorer social skill [Bölte & Poustka, 2002; Fombonne, 2003]. However, it is established that ASD is characterized by a wide range of cognitive functioning, extending well into the normal range of intellectual ability [Baio et al., 2018; Elsabbagh et al., 2012], and that impaired social skill

persists in individuals with ASD without intellectual disability [Shattuck et al., 2007]. This suggests that cognitive ability (i.e., IQ) cannot be the sole contributor to social skill deficiency in ASD.

The social motivation theory of ASD provides an alternative framework by which to understand the social skill deficits evident in this population. This theory maintains that low social motivation alters attention to social information and disrupts social experience seeking, thereby inducing a cascading effect of poor social learning [Chevallier, Kohls, Troiani, Brodkin, & Schultz, 2012]. While social skill deficiency is a presumed outcome of low social motivation in this theoretical framework,

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empirical studies linking social motivation to social skill in ASD are largely lacking [Neuhaus, Webb, & Bernier, 2019]. More broadly, no study has yet attempted to reconcile how cognitive ability and social motivation together impact social skill by concomitantly assessing these three constructs within the same study cohort.

To address these gaps in knowledge, we measured cognitive ability, social motivation, and social skill in a large, well-characterized pediatric cohort. The aims of this study were two-fold. First, we sought to investigate the roles that cognitive ability and social motivation play in predicting social skill in children with ASD, their unaffected siblings, and unrelated neurotypical (NT) controls. Second, we sought to determine whether these findings were universal (i.e., observed in all children), whether they were present in ASD families (i.e., restricted to children with ASD and their siblings, the latter due to potential "broader autism phenotype" effects [Piven, Palmer, Jacobi, Childress, & Arndt, 1997]), or were unique to children with ASD.

Methods

Participants

This research was approved by the Stanford Institutional Review Board and all participants and their families provided informed consent before initiation of study procedures. Children with a diagnostic history of ASD underwent a comprehensive diagnostic evaluation to determine the accuracy of their previous diagnosis based on Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision (DSM-IV-TR) criteria, which was confirmed with research diagnostic methods (i.e., Autism Diagnostic Interview-Revised [ADI-R] [Le Couteur et al., 1989; Lord, Rutter, & Le Couteur, 1994] and the Autism Diagnostic Observation Schedule [Gotham, Risi, Pickles, & Lord, 2007; Lord et al., 2000; Lord, Rutter, DiLavore, & Risi, 1999]). Expert clinical opinion and scores on the ADI-R were used to characterize children with ASD as having autistic disorder or pervasive developmental disorder-not otherwise specified (PDD-NOS). Detailed information on participant recruitment, eligibility criteria, and experimental procedures is provided elsewhere [Carson et al., 2015; Parker et al., 2014].

Briefly, all study participants were aged 3–12 years, prepubertal, and in good medical health. In the present study, participants were required to have completed the Social Responsiveness Scale (SRS), the Vineland Adaptive Behavior Scales, second edition (VABS-2), and the Stanford Binet Intelligence Test, fifth edition (SB-5). This yielded a total study sample of N=189 children: n=79 ASD (n=47 autistic disorder and n=32 PDD-NOS) and

n = 110 non-ASD (n = 50 unaffected sibling and n = 60 unrelated NT control) participants.

Social and Cognitive Assessments

Social motivation was measured using the Social Motivation Subscale raw score of the SRS [Constantino et al., 2003; Pine, Luby, Abbacchi, & Constantino, 2006]. This subscale is included in the norm-referenced SRS parent-report questionnaire that measures social motivation based on items assessing social avoidance or disinterest, where higher scores indicate greater impairment in social motivation. Hence, this study's measure of social motivation is "reverse-scored" and differs in direction from the measures of social skill and cognitive ability described below. Social skill was measured using the Socialization Standard Score from the VABS-2 [Sparrow, Cicchetti, & Balla, 2005]. This subscale is derived from the well-validated VABS-2 parent interview that measures social understanding, participation, engagement, as well as play and interaction with peers. Cognitive ability was determined using the full scale IQ from the SB-5 intelligence test [Roid & Pomplun, 2012], and is referred to as cognitive ability or IQ as appropriate.

Statistical Analysis

All analyses were performed using JMP14 Pro for Windows. To test whether social motivation predicted social skill, we used a General Linear Model (GLM) blocked by age, gender, and ethnicity (i.e., all other factors were tested after controlling for these variables). The ASD and non-ASD groups were nested (i.e., the ASD group included autistic disorder and PDD-NOS; the non-ASD group included unrelated NT controls and unaffected siblings). This approach first tests whether the ASD and non-ASD groups differ in general, and then tests whether the individual subgroups differ from this average. This approach limited our tests to only those that we were interested in, thereby increasing power and avoiding false discovery due to overtesting. Social motivation was included as a predictor in the model, and interacted with group and subgroup, to test whether the relationship between social motivation and social skill differed between group or subgroup. Cognitive ability (IQ) was also included as predictor, with the same interactions. This allowed us to test directly whether social motivation and cognitive ability predict social skill independently, or whether social motivation only appeared to predict social skill because of a mediating effect of cognitive ability.

The assumptions of GLM were tested *post hoc* [Grafen & Hails, 2002]. The social skill measure was square root transformed to meet assumptions of homogeneity of variance, normality of error, and linearity. The social motivation measure was square-root transformed to meet the

assumption of linearity. *Post hoc* planned contrasts tested the significance of slopes and were Bonferroni-corrected.

We repeated this analysis four times. Because the ASD group had a far lower range of IQ scores than the non-ASD group, we first excluded any participants with an IQ < 80 (i.e., any cognitively impaired children, thereby yielding n = 151). This ensured that IQ, group, and subgroup were not colinear, and thus enabled us to test their interactions. Given the lack of any interactions with IQ, we then removed interactions with IQ from the model (so that we could later include the full range of participants without risk of collinearity), and ran this simplified model excluding participants with an IQ < 80. We then ran the same analysis including all participants to ensure the results held in the full sample (N = 189). To test whether IQ was mediating the relationship between social motivation and social skill, we then ran the analysis excluding IQ as a covariate in this larger population (n = 189). Given the collinearity between IQ and Group, we then used the same model to test whether IQ predicted social motivation (n = 189). As before, we removed nonsignificant interactions to ensure that collinearity was not introducing Type II errors. As before, this analysis was also run excluding participants with an IQ < 80 to further control for collinearity, and to evaluate whether any result was being driven by the low IQ participants. Finally, as post hoc tests of these analyses, we performed path analyses separately for the ASD and non-ASD groups. The modeled path allowed cognitive ability to influence social skill directly, and also indirectly via social motivation.

Results

Demographic and phenotypic characteristics are summarized in Table 1. Several variables (e.g., age, sex, ethnicity) significantly differed between two or more analyzed groups. To eliminate the possibility that these

confounding effects could generate false positive or false negative results, we adopted the standard epidemiological approach to this problem and included these variables in the statistical models as blocking factors as described in the Methods section [Grafen & Hails, 2002].

In participants without cognitive impairment (IQ \geq 80), social skill was predicted by a group * social motivation interaction ($F_{1,135}=7.041$; P=0.0091). The subgroups (autistic disorder vs. PDD-NOS and unaffected siblings vs. unrelated NT controls) did not differ from these overall group differences ($F_{2,135}=0.6194$; P=0.5398). Furthermore, social skill was predicted by IQ in the same model ($F_{1,135}=4.5631$; P=0.0345), indicating that IQ and social motivation have independent additive effects on social skill. Neither of IQ's interactions with group ($F_{1,135}=0.9376$; P=0.3346) or subgroup ($F_{1,135}=0.0010$; P=0.9990) were significant, indicating that IQ had a consistent effect on social skill in all participants. These interactions were therefore removed from all further analyses.

Once these interactions were removed, we observed the same results; social skill was predicted by a group * social motivation interaction ($F_{1,138} = 6.910$; P = 0.0095; Fig. 1), such that social motivation did not predict social skill in the non-ASD group ($F_{1,138} = 1.466$; P = 0.2881), but did so in ASD group ($F_{1,138} = 10.67$; P = 0.0014). The subgroups (autistic disorder vs. PDD-NOS and unaffected siblings vs. unrelated NT controls) did not differ from these overall group differences ($F_{2,138} = 0.5885$; P = 0.5566). Furthermore, IQ showed a stronger effect in this simplified model ($F_{1,138} = 6.884$; P = 0.0097).

The same pattern was observed when participants with IQ < 80 were included, although the finding was slightly weaker for social motivation (group * social motivation interaction: $F_{1,176} = 4.540$; P = 0.0345), and stronger for IQ ($F_{1,176} = 22.24$; P < 0.001). The same pattern of results as before was also observed in the *post hoc* tests, and in

Table 1. Participant characteristics

			Sex*		Et	hnicity*					
	Group	N	F	М	Caucasian	Asian	0ther	Age (years)*	Full Scale IQ****	SRS Mot****	VABS-2 Soc****
	Control	60	20	40	6	44	10	7.13 ± 0.36^{b}	115.60 ± 1.19^{a}	3.60 ± 0.55^{c}	102.50 ± 1.56^{a}
Non-ASD	Sibling	50	23	27	18	24	8	$\textbf{7.73} \pm \textbf{0.39}^{ab}$	107.87 ± 1.88^a	3.80 ± 0.60^{c}	$\textbf{102.60} \pm \textbf{1.71}^{a}$
	Autistic	47	9	38	13	25	9	$\textbf{7.60} \pm \textbf{0.41}^{ab}$	$\textbf{71.58} \pm \textbf{3.51}^{c}$	14.68 ± 0.62^a	74.43 ± 1.76^{b}
ASD	PDD-NOS	32	8	24	4	22	6	9.10 ± 0.49^a	96.63 ± 4.18^{b}	11.63 ± 0.75^{b}	$\textbf{77.44} \pm \textbf{2.14}^{\text{b}}$

Note. Likelihood ratio tests were used to examine in this pediatric autism spectrum disorder (ASD) cohort whether the distribution of individuals in the four experimental groups (autistic disorder, Pervasive Developmental Disorder-Not Otherwise Specified [PDD-NOS], unaffected siblings, and unrelated neurotypical controls) differed by sex (F, female; M, Male) and ethnicity; weak significant effects were found for each (*P < 0.05). For age, full scale IQ (intelligence quotient; Stanford Binet—Fifth Edition), SRS Mot (Social Responsiveness Scale, Social Motivation subscale), and VABS-2 Soc (Vineland Adaptive Behavior Scales—Second Edition, Socialization Standard Score) differences between the four groups were tested using a simple one-way general linear model (*P < 0.05, ****P < 0.001). The values are reported as mean \pm standard error. Values with different letter superscripts (i.e., a, b, or c) within the same column differ significantly P < 0.05) per Tukey's post hoc test.

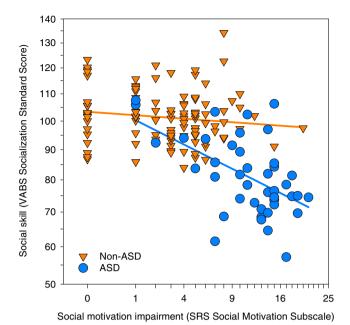


Figure 1. Social motivation impairment negatively predicts social skill in children with autism spectrum disorder (ASD; n=79), but not in non-ASD children (n=110). This relationship is plotted for children with normal cognitive abilities (intelligence quotient $[IQ] \geq 80$ as measured by the Stanford Binet), but this relationship holds whether IQ is included as a covariate in the model or removed altogether. Data were square-root transformed for analysis and are plotted as residuals from the Least-Squares-Line (i.e., correcting for other variables in the analysis). SRS, Social Responsiveness Scale; VABS-2, Vineland Adaptive Behavior Scales—second edition.

the comparison of the subgroups. When IQ was omitted as a covariate, this relationship was strengthened (group * social motivation interaction: $F_{1,177}$ = 8.2820; P = 0.0045), with the same pattern of results again observed in the *post hoc* tests, and in the subgroups.

Importantly, IQ and its interactions with group and subgroup did not predict social motivation. Once these were removed, IQ did predict social motivation in all groups ($F_{1,180} = 4.5784$; P = 0.0337). To evaluate whether this result was being driven by the low IQ participants, we reran the analysis excluding participants with IQ < 80, and accordingly, IQ no longer predicted social motivation in unimpaired children ($F_{1,142} = 0.4230$; P = 0.5165).

We used path analysis to visualize these relationships *post hoc.* Confirming the results above, in non-ASD participants, IQ predicted social skill directly; a significant indirect path via social motivation was not found (Fig. 2A). In ASD participants, IQ also predicted social skill directly. However, IQ also predicted social motivation, and social motivation directly predicted social skill (Fig. 2B).

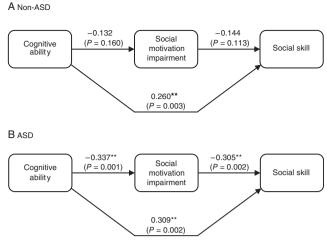


Figure 2. Post hoc path analysis confirms the role of cognitive ability in all children (A and B), and the role of social motivation impairment only in children with autism spectrum disorder (ASD; B), on social skill. Note that had social motivation impairment predicted social skill purely because both were correlated with cognitive ability, then the path between social motivation impairment and social skill would not have been significant for ASD. Thus, although cognitive ability impacts social motivation impairment, social motivation impairment itself impacts social skill independent of cognitive ability in children with ASD. Arrows show standardized path coefficients; P-values are provided in parentheses. Cognitive ability was measured by the Stanford Binet, social motivation impairment was measured by the Social Motivation Subscale (raw score) of the Social Responsiveness Scale, and social skill was measured by the Socialization Standard Score of the Vineland Adaptive Behavior Scales.

Discussion

The goal of this study was to investigate the relationships between cognitive ability, social motivation, and social skill in children with ASD, their unaffected siblings, and unrelated NT controls. Our findings indicate that lower cognitive ability contributed to diminished social skill in all children. In contrast, social motivation strongly predicted social skill only in children with ASD, such that those with the lowest social motivation exhibited the greatest social skill impairment. Notably, this relationship was observed across a large range of intellectual ability but was most pronounced in children with ASD without intellectual impairment (i.e., IQ ≥ 80). This suggests that in children with ASD who have intellectual functioning in the normal range, social motivation has a larger effect on social skill. Furthermore, post hoc path analysis confirmed that both cognitive ability and social motivation each significantly predict social skill in children with ASD. This indicates that while social motivation is predicted by cognitive ability, it also has an independent

and additive influence on social skill over that due to cognitive ability alone.

The present study replicates and extends recent research reporting a relationship between social motivation and social skill in children with ASD [Neuhaus et al., 2019]. Our study also included a non-ASD comparison group, thereby enabling us to determine that the relationship between social motivation and social skill is unique to ASD, and that it varies as a function of cognitive ability. Specifically, cognitive ability drives part of the relationship between social motivation and social skill in children with ASD who have lower cognitive ability, but contributes little to this relationship in children without intellectual disability. This finding speaks to the notion that there are likely distinct subtypes of ASD, and has potentially important clinical implications. For example, this knowledge can be used to fine-tune behavioral interventions aimed at improving social skill in this population. In particular, clinicians may find it useful for individuals without intellectual disability to work on improving social motivation prior to acquiring social skills. For individuals with intellectual disability, it may be important to focus on social motivation in the context of other treatment targets, such as social orienting and learning, while using techniques shown to be valid in this population, such as scaffolding and repetition [Sukhodolsky & Butter, 2007].

The present study found no differences between unaffected siblings and unrelated NT controls in the relationship between social motivation and social skill. Although past studies have reported social functioning impairments in relatives of ASD probands [Constantino et al., 2006; Pisula & Ziegart-Sadowska, 2015; Piven et al., 1997], neither social motivation nor social skill differed between unaffected siblings and NT controls in the present study, nor have these two groups differed in other behavioral or biological variables of interest [Carson et al., 2015; Parker et al., 2014]. Thus, whether low social motivation is related to poor social skill in unaffected siblings awaits investigation in a study cohort confirmed to include relatives with the "broader autism phenotype."

There are certain limitations of the present study that merit comment. First, participants' behavioral characteristics were evaluated using parent report measures designed to assess developmental delay or disorder. These measures were therefore subjective in nature and could have generated "floor" or "ceiling" effects when administered to both ASD and non-ASD participants. Second, the Social Motivation Subscale of the SRS is clinically derived, does not comprehensively capture individual differences in social motivation [Frazier et al., 2014; Phillips et al., 2019], and does not assess manifestations of social motivation such as social orienting and social seeking/liking [Chevallier et al., 2012]. Moreover, specific traits used

to measure social motivation can potentially have alternative explanations (e.g., gaze avoidance following eye contact could be due to anxiety rather than a lack of social interest) [Jaswal & Akhtar, 2019]. Finally, the SRS's Social Motivation Subscale does not enable differentiation of social motivational impairment from a more global motivational deficit [Clements et al., 2018]. There is thus growing scientific interest in developing a more sensitive and specific measure of this construct [Phillips et al., 2019]. Future work should confirm the present study's findings using a more refined measure of social motivation, as well as more objective behavioral tasks that directly measure social motivation as well as social skill in ASD and non-ASD participants. Third, although we endeavored to include as many female participants in this study as possible, ASD is male-biased [Maenner et al., 2020], and our study cohort composition roughly reflects its prevalence. Consequently, we were not powered to detect sex differences in the relationship between social motivation and social skill in this sample. Fourth, the present study relied on a single cross-sectional assessment of social motivation, cognitive ability, and social skill in children aged 3-12 years. Longitudinal assessment of these measures within individuals is now needed to more fully understand how social motivation and cognitive ability affect social skill acquisition during this developmental period. Finally, we acknowledge that although cognitive ability and social motivation are likely to be key drivers of social skill acquisition in ASD, they are likely not to be the only ones. Follow up research that includes additional potential drivers of social skill development such as social communication and language, theory of mind, social attention, and emotion regulation will provide a more nuanced picture of social skill acquisition in children with ASD.

In summary, our findings indicate that low cognitive ability contributes to diminished social skill in all participants. In contrast, low social motivation contributes to social skill deficiency only in children with ASD. Path analysis confirmed that both cognitive ability and social motivation each contribute to social skill in children with ASD, and that while social motivation is predicted by cognitive ability, social motivation has an independent and additive influence on social skill in ASD over that due to cognitive ability alone. These findings support the hypothesis that low social motivation impairs social skill acquisition in this disorder, particularly in children without intellectual disability.

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