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ORIGINAL PAPER

Explaining Variance in Social Symptoms of Children with Autism Spectrum Disorder

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Abstract

The social symptoms of autism spectrum disorder are likely influenced by multiple psychological processes, yet most previous studies have focused on a single social domain. In school-aged autistic children ($n = 49$), we compared the amount of variance in social symptoms uniquely explained by theory of mind (ToM), biological motion perception, empathy, social reward, and social anxiety. Parent-reported emotional contagion—the aspect of empathy in which one shares another's emotion—emerged as the most important predictor, explaining 11–14% of the variance in social symptoms, with higher levels of emotional contagion predicting lower social symptom severity. Our findings highlight the role of mutual emotional experiences in social-interactive success, as well as the limitations of standard measures of ToM and social processing in general.

Keywords Autism spectrum disorder · Theory of mind · Biological motion perception · Empathy · Social reward · Social anxiety

Introduction

Autism spectrum disorder (ASD) is defined by impaired social functioning, yet after decades of research, there is no clear consensus on the psychological basis for this impairment.[1] Social behavior depends on multiple cognitive and affective processes (Happé et al. 2017); accordingly, several constructs (e.g., theory of mind and social reward) have been extensively studied in the context of ASD, in some cases yielding converging evidence that a particular construct differs between the typically developing (TD) and autistic[2] populations at the group level. There is also ample research into the more clinically relevant issue of how individual differences in each construct individually relate to social symptoms (e.g., Altschuler et al. 2018; Klin et al. 2002; Sasson et al. 2020; Supekar et al. 2018), yet most of these studies focus on a single domain of social processing. Thus, the question remains: which variable, or set of variables, is *most important* for explaining social symptoms in ASD? The present study tackles this question by examining a range of social-cognitive, social-perceptual, and social-affective constructs and their relative contributions to explaining variance in social symptom severity in school-aged autistic children. We first discuss four constructs commonly posited as important deficits in ASD—theory of mind, biological motion perception, social reward, and empathy—followed by social

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[1] Throughout this paper, we use "social difficulties," "social impairment," "social dysfunction," and "social symptoms" interchangeably, with all four referring to behaviors (or lack of behaviors) that characterize the social dimension of the diagnostic criteria of ASD—for example, reduced initiation of social interactions, reduced social-emotional reciprocity, and abnormal nonverbal communication.

[2] We use identity-first language because this is preferred by many autistic self-advocates (Brown 2011).

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anxiety, a condition highly comorbid with ASD that may interfere with social processing during social interactions.

Theory of Mind

It is widely assumed that social competence hinges on one's ability to explain and predict others' behavior by representing their mental states (e.g., knowledge, beliefs, intentions, or emotions). This ability, known as *theory of mind* (ToM), encompasses multiple cognitive processes that may be differentially recruited depending on the situation and the type of mental state being represented (Molenberghs et al. 2016; Schaafsma et al. 2015). For example, it is common to distinguish between representing knowledge, beliefs, and intentions (*cognitive ToM*) and representing emotional states (*affective ToM*; Abu-Akel and Shamay-Tsoory 2011).

The presumed link between ToM and social competence is integral to the highly influential "mindblindness" theory, which places ToM impairment at the root of social difficulties in ASD (Baron-Cohen et al. 1985). In line with this theory, group-level comparisons often find deficits in ASD on both cognitive and affective ToM tasks (Chung et al. 2014). Even those autistic individuals who perform comparably to their TD peers on laboratory ToM tasks are often still impaired in *applied ToM*—that is, the ability to engage their ToM in real-world situations (Frith 1994; Peterson et al. 2009). Thus, impairment on some (though not all) ToM measures is a consistent finding in autism research (Tager-Flusberg 2007).

The mindblindness theory predicts not only that autistic individuals have ToM impairments, but that these impairments significantly account for their social symptoms (Baron-Cohen et al. 1985). Evidence for this prediction is surprisingly mixed (Sasson et al. 2020), though perhaps inconsistency should be expected given that ToM is multifaceted (Altschuler et al. 2018; Schaafsma et al. 2015; Warnell and Redcay 2019); the degree to which ToM contributes to social impairment may depend on which aspect is being considered. For example, a recent study of school-aged autistic children found that social symptoms were negatively associated with affective ToM but were unrelated to cognitive ToM (Altschuler et al. 2018). Another study found that applied ToM, but not performance on laboratory ToM tasks, mediated the relation between autistic symptoms and social functioning (Berenguer et al. 2018).

Taken together, such findings underscore the importance of employing multiple measures that tap distinct facets of ToM when assessing links between ToM and social symptoms. Furthermore, as we discuss below, ToM may relate to other constructs that play key roles in social impairment. It is therefore necessary to examine ToM variables alongside these other constructs before concluding that ToM uniquely explains social impairment in ASD.

Biological Motion Perception

Navigating the social world involves not only imputing unseen mental states onto others, but also perceiving and interpreting others' physical actions. TD individuals are highly sensitive to visual motion cues that evoke human actions (i.e., *biological motion*) and can recognize specific actions even from impoverished stimuli, such as point-light animations that depict the motion patterns of a walking figure (Blake and Shiffrar 2007; Johansson 1973). Autistic individuals often show disrupted biological motion perception (BMP; Van der Hallen et al. 2019; but see Cusack et al. 2015), leading some to propose this impairment and its neural signature as a hallmark of ASD (Kaiser and Pelphrey 2012; Pavlova 2012). However, while there is some evidence of a negative correlation between BMP performance and symptom severity in ASD (Blake et al. 2003), other studies have found no relation (Annaz et al. 2010; Nackaerts et al. 2012). Thus, though BMP impairment may be common in ASD, further study is needed to interrogate its direct relation to social dysfunction.

BMP may also be closely entwined with ToM, thus complicating the relation between each of these constructs and social functioning. It is often theorized that advanced social-cognitive abilities like ToM emerge from an earlier-developing understanding of intentional actions, which itself depends on BMP (Frith and Frith 1999). To our knowledge, there is no direct evidence of this link in ASD, but in TD adults and children, BMP is positively correlated with ToM performance (Miller and Saygin 2013; Rice et al. 2016). This raises the possibility that previously reported links between ToM and social impairment in ASD are largely explained by an earlier BMP deficit. Thus, it is of interest to examine both constructs in the same individuals to determine their independent associations with social symptoms.

Empathy

The capacity to experience others' emotions is often presumed necessary for prosocial behavior and moral understanding. Yet despite its supposed centrality to what it means to be human, empathy lacks a single, universally agreed-upon definition (Decety and Cowell 2014). This is likely because, much like ToM, the concept of empathy encompasses multiple processes. Also like ToM, it is common to distinguish between cognitive and affective aspects (Davis 1983; Fletcher-Watson and Bird 2020). *Cognitive empathy* involves recognizing and understanding others' emotions, often by means of perspective-taking, and is thus synonymous with affective ToM (discussed above).

Emotional contagion is the more purely affective phenomenon of experiencing another person's apparent emotion in oneself (Darwall 1998). The distinction between cognitive empathy/affective ToM and emotional contagion is supported by behavioral and neuroimaging studies (Dvash and Shamay-Tsoory 2014). Some also recognize a third component of *empathic concern* or compassion—that is, regard for others' wellbeing from a third-person perspective—which, though also affective in nature, appears to be distinct from emotional contagion (Jordan et al. 2016; Murphy 2019).

Despite these distinctions, studies of empathy in autism often rely on unidimensional measures. The widely used Empathy Quotient (Baron-Cohen and Wheelwright 2004) includes both cognitive and affective components, yet as Fletcher-Watson and Bird (2020) point out, most studies report only total scores. Conflating the cognitive and affective components of empathy is problematic not only for theoretical reasons. Though some researchers argue for the utility of undifferentiated empathy measures in the study and treatment of ASD (e.g., Russ et al. 2018), other researchers and autistic self-advocates have raised concerns that characterizing autistic individuals as lacking empathy—which is understood by the general public to include the sharing of others' emotions—could lead to a harmful, dehumanizing stereotype of autistic people as unfeeling (Cohen-Rottenberg 2011; Fletcher-Watson and Bird 2020; Nicolaidis et al. 2019). This stereotype is contradicted by testimonials from autistic individuals describing hypersensitivity to others' emotions (Smith 2009), as well as empirical evidence that autistic individuals do not differ from TD individuals on empathic concern or emotional contagion (Dziobek et al. 2008; Rueda et al. 2015), though this depends on how these constructs are measured. For example, Trimmer et al. (2017) found that the autistic group showed typical levels of physiological response to others' distress but dampened self-reported emotional responses relative to the TD group.

Regardless of whether there are group-level differences, individual differences in affective empathy among the autistic population may relate to differences in social impairment. A handful of studies suggests this may be the case for emotional contagion. For example, in one study of autistic children and adolescents, self-reported emotional contagion was positively correlated with peer engagement and prosocial behavior (Travis et al. 2001). Additionally, ASD symptom severity has been found to correlate negatively with young children's emotional responsivity to an experimenter's emotional display (Scambler et al. 2007) and to children and adolescents' contagious yawning and laughter (Helt et al. 2019). Further research is needed to probe the specificity of this relation while controlling for other social factors, particularly ToM.

Social Reward

The social motivation theory of ASD proposes that unlike TD individuals, autistic individuals do not experience social interactions as intrinsically rewarding (Chevallier, Kohls, et al. 2012). According to the theory, during infancy this reduced social reward manifests in reduced attention to social stimuli, leading to reduced opportunities for social learning, which in turn leads to deficits in social cognition, perception, and behavior. While the strongest evidence for this developmental cascade comes from studies showing atypical social orienting in autistic infants and young children (Dawson et al. 2004; Klin et al. 2009; Moore et al. 2018; Osterling et al. 2002), the social motivation theory also predicts continuing differences throughout the lifespan. Studies in older children, adolescents, and adults suggest that autistic individuals may be biased toward nonsocial stimuli, particularly those representing circumscribed interests, and away from social stimuli (Sasson et al. 2008, 2012; Unruh et al. 2016). Meanwhile, neuroimaging studies show altered functioning of the reward system in ASD, though this is not specific to social stimuli (see meta-analysis: Clements et al. 2018).

In a recent challenge to the social motivation theory, Jaswal and Akhtar (2019) argue against the common assumption that certain autistic behaviors, such as reduced eye contact and infrequent declarative pointing, necessarily reflect social disinterest. In addition to offering alternative explanations for these behavioral patterns, the authors highlight the potential negative consequences of mischaracterizing autistic individuals as lacking social motivation, a trait that, like empathy, many consider to be fundamentally human (Baumeister and Leary 1995). Thus, rather than decoding autistic behaviors through the lens of typical development, there is a compelling case to be made for measuring social reward via self-report—that is, allowing autistic individuals to directly convey their subjective experiences. Studies using self-reports thus far present a heterogeneous picture of social reward in ASD: some autistic individuals report experiencing little pleasure from social relationships (Chevallier, Grèzes, et al. 2012), yet others express satisfaction with their friendships or a strong desire for social connection (Calder et al. 2013; Jaswal and Akhtar 2019). The considerable variability in reports of social reward among the autistic population warrants further examination of whether this construct is reduced in ASD and how it relates to social symptoms. Furthermore, if impaired social reward is an early-developing feature of ASD, we might expect it to explain more unique variance in social symptoms compared to its downstream consequences of impaired social cognition or social perception (i.e., ToM or BMP).

Social Anxiety

Autistic individuals are much more likely than their TD peers to experience social anxiety (Spain et al. 2018). Beyond the high comorbidity between ASD and anxiety disorders in general (van Steensel et al. 2011), the negative peer interactions often experienced by autistic children and adolescents put them at an increased risk of developing social anxiety. In a self-perpetuating cycle, this anxiety may lead to increased social withdrawal and, in turn, more social impairment (Bellini 2006). Thus, while not considered to be a primary cause of social impairment in ASD, social anxiety may exacerbate existing difficulties by limiting autistic individuals' opportunities to learn from positive peer interactions.

Another way that social anxiety may impact social symptom severity is by interfering with social processing (Hope et al. 1990). For example, as mentioned above, many autistic individuals display intact ToM abilities on standard laboratory tasks—that is, removed from the context of a real-world social interaction (Barendse et al. 2018; Scheeren et al. 2013). If these individuals experience high anxiety during social interactions, their attentional resources may be diverted toward a perceived threat (negative evaluations of the self; Rapee and Heimberg 1997) and away from the task at hand (engaging effectively with a social partner), thus hindering their ability to apply their ToM skills within the interaction. As such, anxiety may moderate the relation between ToM (or some other social processing ability) and social symptoms, such that better ToM predicts better social functioning in low anxiety individuals but this relation is absent in those with high levels of social anxiety.

The Present Study

In a group of school-aged autistic children (full-scale IQ > 80), we evaluated the relative importance of each of the constructs reviewed above (specifically, cognitive, affective, and applied ToM; BMP; emotional contagion; social reward; and social anxiety) in explaining variance in social symptom severity, as measured by the Social Affect score from the Autism Diagnostic Observation Schedule (ADOS-2; Lord et al. 2012). To this end, we applied two complementary statistical approaches: (1) model comparison to determine the subset of variables that best explain variance in social symptoms, and (2) dominance analysis to determine the amount of variance in social symptoms uniquely explained by each variable. We hypothesized that social symptom severity would be best explained by a model that includes at least one ToM measure. Based on prior literature (Barendse et al. 2018; Berenguer et al. 2018; Scheeren et al. 2013), we further hypothesized that applied ToM (assessed via parent report) would be more important in explaining social symptoms compared to explicit laboratory tasks measuring verbal-cognitive and visual-affective ToM.[3] Our predictions regarding non-ToM variables were less defined, though we expected at least one non-ToM variable to emerge as important. Furthermore, based on the possibility that social anxiety interferes with social processing, we tested the hypothesis that social anxiety interacts with the other predictor variables in explaining social symptoms, as described in the previous section.

Methods

This study was pre-registered through the Open Science Framework (https://osf.io/mkyuq). Deviations from the pre-registered analysis plan are described in the Supplementary Materials.

Participants and Procedure

All study procedures were approved by the institutional review board of a large, urban public university. Families of children with a prior diagnosis of ASD were recruited from the surrounding metropolitan area using the Interactive Autism Network (IAN), flyering at local events, and emailing relevant listservs. Data included in this study were collected as part of a larger study involving magnetic resonance imaging (MRI); as such, participation was limited to individuals without MRI contraindications and, to maximize the chance of success on our in-scanner tasks, without verbal or intellectual disability. Additional exclusionary criteria were diagnosis of epilepsy, post-traumatic stress disorder, oppositional defiant disorder, reactive attachment disorder, or conduct disorder. All participants were native English speakers.

Autistic children completed two behavioral sessions. At the first session, the ADOS-2 (Module 3) and the Kaufman Brief Intelligence Test, 2nd edition (KBIT-2; Kaufman and Kaufman 2004) were administered to confirm the child met criteria for ASD and had a full-scale IQ above 80. The remaining behavioral measures were administered at the second session. A subset of these children later completed one or two MRI sessions (data not included in the present study). At the start of each session, parents and children provided informed consent and assent, respectively.

Fifty-one autistic children were considered for inclusion in the current study. Two children were excluded from analyses due to full-scale IQ scores below 80, leaving a

[3] We refer to the constructs measured by the Strange Stories and Reading the Mind in the Eyes Test as verbal-cognitive and visual-affective ToM, respectively, to highlight the fact that these tasks differ not only in the type of mental state inference being assessed (cognitive vs. affective), but also in the modality of stimulus presentation (verbal vs. visual).

Table 1 Demographic characteristics

	ASD ($n=49$)	TD ($n=50$)	Overall ($n=99$)
Race			
White or Caucasian	35 (71%)	29 (58%)	64 (65%)
Black or African American	7 (14%)	13 (26%)	20 (20%)
Asian	3 (6%)	1 (2%)	4 (4%)
More than one race	4 (8%)	6 (12%)	10 (10%)
Did not wish to disclose	0 (0%)	1 (2%)	1 (1%)
Ethnicity			
Not Hispanic or Latino	47 (96%)	46 (92%)	93 (94%)
Hispanic or Latino	2 (4%)	2 (4%)	4 (4%)
Did not wish to disclose	0 (0%)	2 (4%)	2 (2%)

final sample of 49 autistic children (mean age at ADOS assessment: 11.48 ± 2.11 years, range: 7.11–14.86 years; 4 females). To characterize our autistic sample, we compared it to a gender-matched group of 50 TD children on all predictors (mean age: 11.45 ± 1.75 years, range: 7.51–14.46 years; 4 females); this group was selected from the aforementioned larger MRI study with the goal of minimizing group differences in age and full-scale IQ (Fig. 1; Supplementary Table S1). The TD children were not administered the ADOS and completed the other behavioral measures in a single session. Exclusionary criteria for the TD group included MRI contraindications, full-scale IQ below 80, diagnosis of any neurological or psychiatric disorders, or first-degree relatives with autism or schizophrenia.

Participant race and ethnicity are summarized in Table 1. In both the ASD and TD groups, most families were of high socioeconomic status. The highest level of education attained by either parent included a postgraduate degree (ASD: 55%, TD: 44%), some graduate school (ASD: 8%, TD: 10%), a college degree (ASD: 16%, TD: 16%), a technical or associate degree (ASD: 2%, TD: 2%), some college (ASD: 4%, TD: 8%), or high school (ASD: 0%, TD: 2%). A majority of families had an annual household income above $75,000 (ASD: 84%, TD: 76%). ASD and TD groups did not significantly differ in terms of race (Black vs. White vs. all other categories; $\chi^2(2)=2.42$, $p=0.30$), having a parent with a postgraduate degree ($\chi^2(1)=0.32$, $p=0.57$), or household income (greater vs. less than $75,000 per year; ($\chi^2(1)=0.49$, $p=0.48$).

Measures

Details on the scoring of each measure and example items from the questionnaires are provided in the Supplementary Materials.

We assessed *social symptom severity* via the Social Affect score from the ADOS-2, Module 3. All examiners administering the ADOS are research reliable. We converted raw algorithm scores to calibrated severity scores (CSS), since CSS are less influenced by demographic factors such as age and verbal ability when compared to raw scores (Hus et al. 2014).

We measured *verbal-cognitive ToM* using the Strange Stories task (White et al. 2009). After hearing prerecorded stories about social situations, children were asked to explain a character's behavior. We administered a subset of eight Mental stories assessing mental state inference and three Control stories assessing physical inference.

We measured *visual-affective ToM* via the Reading the Mind in the Eyes Test (RMET), in which children viewed a series of photographs of the eye region of various faces and chose from among four options the written phrase that best described the mental state expressed in each photograph (Baron-Cohen et al. 2001).

We measured *applied ToM* via the Theory of Mind Inventory (ToMI), in which parents evaluated their children on various applications of ToM in everyday situations (Hutchins et al. 2014). The ToMI consists of three empirically derived subscales corresponding to Early, Basic, and Advanced stages of typical ToM development.

We administered a *biological motion perception* task adapted from Miller and Saygin (2013). Children viewed a series of point-light walkers embedded in increasing levels of noise in the form of extraneous dots; their task was to indicate the direction in which the walker was facing (left or right). Details about stimulus presentation are provided in the Supplementary Materials.

Parents completed the Griffith Empathy Measure (GEM; Dadds et al. 2008), which includes two empirically derived subscales: Cognitive and Affective. As the Cognitive subscale somewhat overlaps in content with the ToMI Early subscale, we included only the Affective subscale. The Affective subscale assesses a child's propensity to share or mirror others' emotions, or *emotional contagion*.

Children completed the Social Reward Questionnaire (SRQ; Foulkes et al. 2014). The SRQ consists of six empirically derived subscales.[4] We included two subscales in the present analyses: Admiration and Prosocial Interactions. Our preregistered selection was guided by a previous study from our group[5] that found negative correlations between these subscales and ADOS Total Severity scores.

[4] We did not administer three items from the original SRQ that form the Sexual Relationships subscale. The omission of these items did not affect our subscales of interest.

[5] Sadikova, E., Kirby, L. A., Pecukonis, M., Warnell, K., & Redcay, E. (2017, May). Developmental relations between social reward, social cognition, and total severity in ASD. Poster presented at the International Meeting for Autism Research, San Francisco, CA.

To measure *social anxiety,* we administered both the parent and child versions of the Screen for Child Anxiety Related Emotional Disorders (SCARED; Birmaher et al. 1997). For the primary analyses, we used the Social Anxiety subscale from the parent report due to concerns that some children with ASD, particularly those with lower metacognitive ability, may underreport their social anxiety (Blakeley-Smith et al. 2012). Nevertheless, in a planned follow-up, we repeated the analyses with the Social Anxiety subscale from the child report. Except where noted, results from these follow-up analyses did not differ substantially from those using the parent report (Supplementary Materials).

Analytic Approach

Data preparation and analyses were conducted using R (R Core Team 2019). Details about our treatment of missing data and evaluation of multicollinearity and potential outliers can be found in the Supplementary Materials.

Model Comparison

To determine which set of predictors best explains variance in social symptom severity, we conducted model comparison using Bayes factors (BF). BFs quantify evidence for or against a model (more precisely, the degree to which beliefs about a model should be updated in light of the data) in the form of likelihood ratios; for instance, a BF of 3/1 means the data are three times as likely under one hypothesis versus another. Higher BF values indicate a greater degree of evidence. Though there are no cut-points analogous to significance thresholds in frequentist statistics, we follow the widely used guidelines for interpretation of BFs from Lee and Wagenmakers (2013): 1–3 = anecdotal evidence (i.e., not worth reporting), 3–10 = moderate evidence, 10–100 = strong evidence, and > 100 = extreme evidence. Unlike frequentist approaches, the Bayesian framework allows for the quantification of evidence in favor of not only the alternative hypothesis (BF_{10}) but also the null hypothesis (BF_{01}, the reciprocal of BF_{10}). Furthermore, BFs enable the comparison of non-nested models, allowing us to evaluate all possible combinations among the set of predictors under consideration. In addition to our main measures, we included KBIT-2 verbal and non-verbal IQ scores and age as possible predictors.

From the Bayes Factor R package (Morey and Rouder 2018), we used the GeneralTestBF function with its default prior to conduct an all-possible-subsets regression over 10,000 Monte Carlo iterations, resulting in a BF_{10} for each possible model compared to the null (intercept-only) model (see Supplementary Materials for information on our use of a range of priors). For comparisons on the predictors between our autistic and TD participants, we conducted Bayes factor t-tests using the ttestBF function in the BayesFactor package with its default prior (Rouder et al. 2009).

Dominance Analysis

We determined the relative importance of each predictor via dominance analysis. This method overcomes the issue of collinearity among predictors—an issue that precludes the use of standardized regression coefficients as indicators of relative importance—by calculating each predictor's incremental validity (i.e., ΔR^2 when entered last in the model) within all possible sub-models containing that predictor (Budescu 1993; Nimon and Oswald 2013). These incremental validity values are then averaged to yield *general dominance* weights that can be interpreted as the portion of variance in the dependent variable attributable to each predictor independent of other predictors. A predictor is said to show *complete dominance* over another predictor if its incremental validity is higher across all possible sub-models.

Using the that R package (Nimon et al. 2013), we applied dominance analysis in two ways. First, to provide converging evidence with the model comparison, we applied dominance analysis to all predictors. Second, we assessed each of the models favored by the model comparison procedure described above in order to provide effect sizes (in the form of general dominance weights) for individual predictors within each model.

Results

Results are reported in three sections. In the first section, we characterize our autistic sample in comparison with a matched TD control sample on all predictors. Then, in the autistic group only, we address our research questions: 1) which variables best explain social symptom severity, and 2) does social anxiety interact with other variables in explaining social symptom severity?

Sample Characterization and Group Comparison

Distributions of each predictor for the autistic and TD groups are depicted in Fig. 1; descriptive statistics and between-group comparisons are summarized in Supplementary Table S1. By design, the two groups did not differ in age or full-scale IQ. We found moderate evidence ($BF_{01} \geq 3$) for the null hypothesis—i.e., no group difference—for these variables, as well as the Strange Stories Control condition and BMP. We found strong evidence that parents of the autistic children rated their children lower on all three ToMI subscales ($BF_{10} > 3*10^7$; large effects; Early: Hedge's $g = -2.13$; Basic: $g = -1.43$; Advanced: $g = -2.45$) and higher on social anxiety ($BF_{10} = 85$; medium effect;

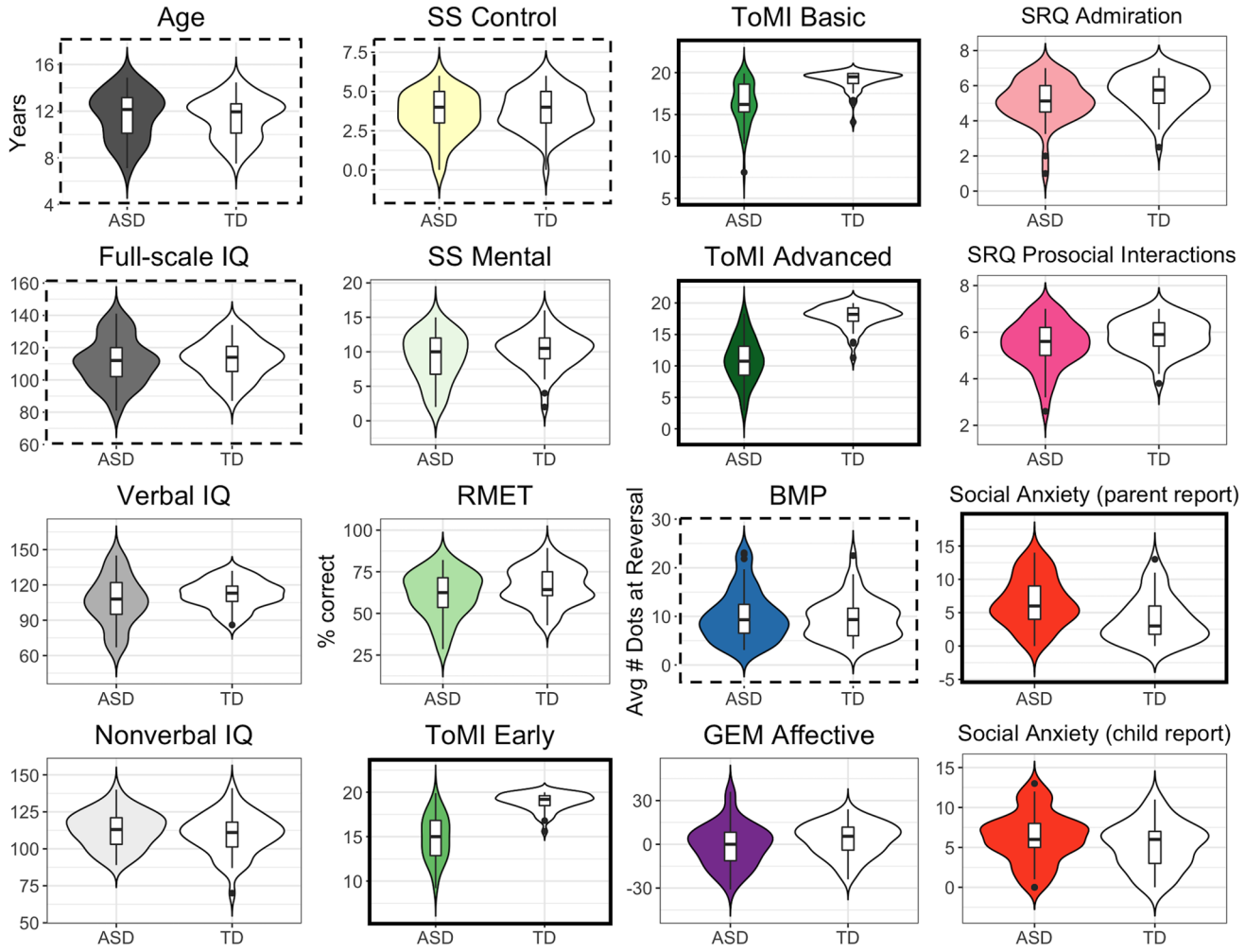

Fig. 1 Distributions of ASD compared with TD groups on all variables. Solid bold outlines indicate strong evidence for group differences ($BF_{10} > 10$). Dashed outlines indicate moderate evidence for no group differences ($BF_{01} \geq 3$). *SS Control* Strange Stories Control condition; *SS Mental* Strange Stories Mental condition; *RMET* Reading the Mind in the Eyes Test; *ToMI* Theory of Mind Inventory; *BMP* biological motion perception; *GEM Affective* Griffith Empathy Measure, Affective subscale (emotional contagion); *SRQ* Social Reward Questionnaire

$g = 0.73$). For the remaining variables, evidence regarding group differences was inconclusive. Zero-order correlations are reported in Supplementary Tables S2.1–2.3.

Question 1: Which Variables Best Explain Social Symptom Severity in ASD?

Model Comparison

Figure 2 depicts the six best models, that is, those with the highest BF_{10}. Each of these models yielded a BF_{10} between 3 and 6, indicating moderate evidence against the null model (see Supplementary Tables S3.1–3.3 for robustness to changes in the prior scale). All other models yielded a BF_{10} less than 3, indicating only anecdotal evidence, and thus are not reported here. The model with the highest BF_{10} (5.75) included only emotional contagion (as measured by the GEM Affective scale); this variable was included in each of the six best models, whereas BMP and verbal IQ were each included in three of the six best models. Against our hypothesis that applied ToM would be more important than explicit laboratory measures of verbal-cognitive or visual-affective ToM, none of the applied ToM variables (the three ToMI subscales) were included in any of the best models, whereas verbal-cognitive ToM was included in one of the six best models (model 6). Except for age in model 5, all effects were negative—that is, increases in the predictors were associated with decreases in social symptom severity (see Supplementary Table S4 for standardized beta coefficients).

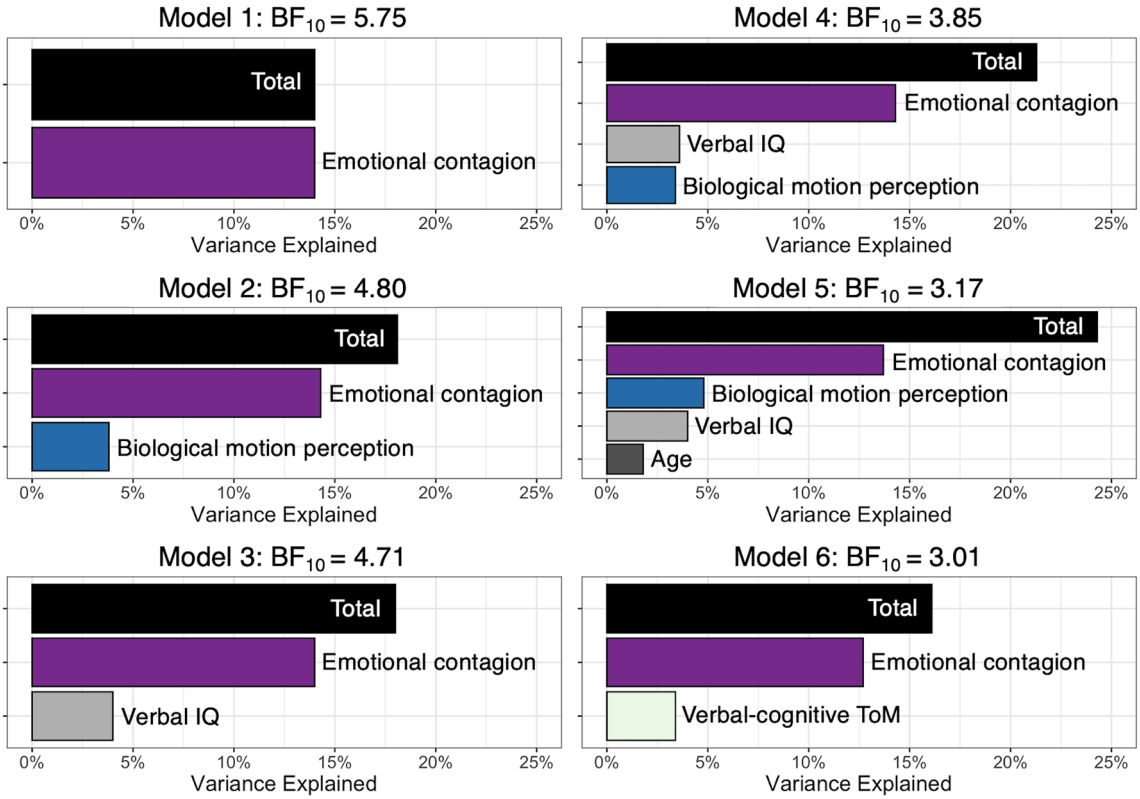

Fig. 2 Percentage of variance in social symptom severity in autistic participants uniquely explained by each predictor (general dominance weights) within the top six models. BF_{10} = Bayes factor in favor of each model against the null (intercept-only) model

In addition to comparing each model against the null model, one may also compare any two models by dividing their BFs. For example, comparison of the top two models yields a BF of 1.2. As we found only anecdotal evidence favoring model 1 over each of the other top five models (all BFs < 2), we considered all six as the "best models" in subsequent analyses.

Dominance Analysis

We first applied dominance analysis to all predictors (Fig. 3, Supplementary Table S5). Converging with the above model comparison, in terms of general dominance (GD) weights, the five most important predictors were the same as those included in the six best models: emotional contagion explained the most variance (GD = 0.112), followed by BMP (GD = 0.046), age (GD = 0.027), verbal-cognitive ToM (Strange Stories Mental condition; GD = 0.023), and verbal IQ (GD = 0.020). Furthermore, emotional contagion demonstrated complete dominance; that is, across all possible sub-models, emotional contagion explained more unique variance than each of the other predictors.

Next we applied dominance analysis to each of the six best models separately (except for model 1, in which emotional contagion was the only predictor; Fig. 2 and Supplementary Table S6). Again, emotional contagion showed complete dominance within each of the models and had GD weights ranging

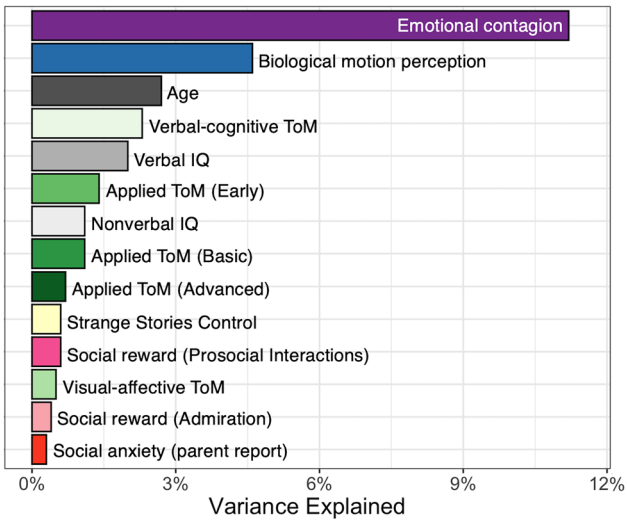

Fig. 3 Percentage of variance in social symptom severity in autistic participants uniquely explained by each predictor (general dominance weights). All predictors together explained 29.5% of the variance

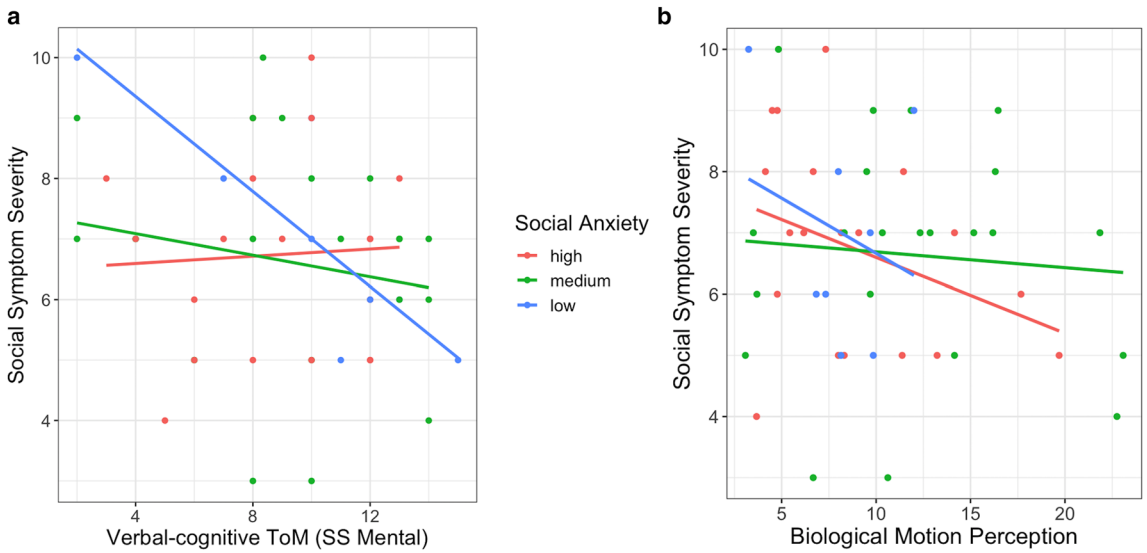

Fig. 4 Interactions between parent-reported social anxiety and **a** verbal-cognitive ToM and **b** biological motion perception in explaining social symptom severity in autistic participants. We defined high social anxiety as a raw score of 8 or above; according to the Screen for Child Anxiety Related Emotional Disorders (SCARED) scoring guide, these scores may indicate social anxiety disorder. We defined low social anxiety as a raw score of 3 (approximately one standard deviation below the mean of our sample) or below. SS Mental = Strange Stories Mental condition

from 0.127 to 0.143. In sum, whether considered alongside all potential predictors or within the context of specific models, emotional contagion emerged as the most important variable in terms of explaining unique variance in social symptom severity. Furthermore, although the inclusion of verbal-cognitive ToM in one of the six best models is somewhat consistent with our hypothesis that at least one ToM measure would be included in the best model, the dominance analysis for this model indicates that verbal-cognitive ToM explains only 3.4% of variance in social symptoms, compared to emotional contagion explaining 12.7%.

Question 2: Does Social Anxiety Interact with Other Variables in Explaining Social Symptom Severity in ASD?

We also applied Bayes factor model comparison to models containing interactions between social anxiety and each predictor included in the best models (i.e., the five predictors included in Fig. 2; Supplementary Table S7). We found moderate evidence ($BF_{10} = 4.33$) for a model containing interactions between social anxiety and verbal-cognitive ToM and BMP, respectively, as well as main effects of emotional contagion, BMP, verbal IQ, verbal-cognitive ToM, and social anxiety (see Supplementary Tables S7.1-S7.3 for robustness to changes in the prior scale). However, these results did not hold when we replaced the parent report with the child report version of the same measure of social anxiety. Nevertheless, to explore the interaction between parent-reported social anxiety and verbal-cognitive ToM and BMP, respectively, we plotted these two variables with individual data points separated by low, medium, and high levels of social anxiety (Fig. 4). The resulting pattern for verbal-cognitive ToM is consistent with our hypothesis: social symptom severity is negatively related to verbal-cognitive ToM for individuals with low but not high levels of social anxiety. The pattern for BMP is less straightforward, and both patterns should be interpreted with caution given our modest sample size, particularly within the low social anxiety group.

Discussion

In school-aged autistic children, we examined the relative importance of several social-cognitive, social-perceptual, and social-affective constructs in explaining variance in social symptom severity. Across multiple models, parent-reported emotional contagion emerged as the most important predictor, explaining around 14% of the variance in social symptom severity. In this section, we first discuss the implications of this result, then reflect on our findings related to ToM and other constructs.

Emotional Contagion was the Most Important Predictor of Social Symptom Severity

Our most robust finding is a moderate negative association between emotional contagion (measured by the GEM

Affective subscale) and social symptom severity. Even when accounting for all other predictors in our study, emotional contagion explained more than twice the amount of variance in social symptoms as any other predictor. At face value, our findings affirm the theory that empathy—specifically, the capacity to share emotions with others[6]—is significantly associated with social behavior in ASD, consistent with previous studies (Helt et al. 2019; Scambler et al. 2007; Travis et al. 2001). Importantly, however, we did not find evidence of a difference between the autistic and TD groups on this measure, suggesting that emotional contagion is not pervasively impaired in ASD; rather, it appears to vary comparably among autistic and TD individuals.

Research over the last few decades supports the intuition that "catching" others' emotions is a frequent and powerful component of social connection (Hatfield et al. 1993). Widely cited studies have shown that emotional contagion affects the moods and behaviors of people in laboratory settings and real-world social networks (Barsade 2002; Fowler and Christakis 2008; Kramer et al. 2014), and neuroimaging studies have been able to detect the phenomenon in patterns of brain activity (Anders et al. 2011; Singer et al. 2004; Wicker et al. 2003). Perhaps most relevant to the present study, it has been suggested that occupying a "shared space of affect" (Anders et al. 2011) supports ToM; that is, we understand others' minds by first simulating their emotions and other mental states from our own perspective (Gallese and Goldman 1998; Niedenthal 2007). Under this embodied simulation account, shared emotions enable shared understanding, thereby facilitating social interaction (Nummenmaa et al. 2012). Within this framework, it is entirely plausible that the degree to which an autistic individual experiences emotional contagion affects their degree of social symptoms.

However, we must also consider that the GEM is a parent-report measure and not a direct measure of emotional responding. As such, it is perhaps more indicative of a child's *display* of emotions than of the child's actual experience of emotional contagion. Several studies suggest that autistic people express emotions atypically, particularly in terms of reduced displays of positive affect during social interaction (Capps et al. 1993; Kasari et al. 1990; Snow et al. 1987) or more "flat" expressions in general (Stagg et al. 2014; but see Begeer et al. 2008; Faso et al. 2014). Meanwhile, autistic people have shown typical responses to others' emotions in terms of brain activity (Bird et al. 2010) as well as skin conductance and facial electromyography (Trimmer et al. 2017), though these findings were in the context of pain or distress; less is known about autistic physiological responses to others' positive affect. Still, given the potential for discrepancy between actual and displayed emotion, and findings that TD people have difficulty interpreting autistic people's mental states (Edey et al. 2016; Sheppard et al. 2016), in the current study we cannot assume that parents' ratings of emotional contagion—or, for that matter, clinicians' judgments of "shared enjoyment in the interaction," a component of the ADOS Social Affect score—accurately reflected our participants' emotional experiences. In other words, our finding may reflect concordance between non-autistic perceptions of autistic children's behavior rather than a relation between social symptoms and intrinsic emotional contagion.

More specifically, it is worth noting the overlap between items on the GEM Affective subscale (e.g., "My child acts happy when another person is acting happy") and two components of the ADOS Social Affect score: 1) the above-mentioned shared enjoyment, defined as "the participant's ability to *indicate* pleasure to the examiner," and 2) "facial expressions directed to examiner." Thus, the relation between these measures might be driven by clinicians picking up on the same emotional display tendencies as parents. Studies using different social outcome measures—for example, ratings from peer interaction partners that do not explicitly ask about emotional expression—are needed to further establish the link between emotional contagion and social impairment.

Evidence that TD people (and some autistic people themselves[7]) misinterpret autistic emotions should caution against exclusively relying on subjective reports to measure empathy (Fletcher-Watson and Bird 2020). However, such misinterpretation is not a mere confound, but instead may be integral to the nature of social dysfunction. If TD people do not perceive an autistic person to be sharing their emotional space, they are likely to experience the autistic person as an atypical social partner regardless of the ground truth about his or her emotional state. ASD has long been conceptualized as a "lack of intersubjective engagement *by autistic individuals*" (Hobson and Lee 1998; emphasis added), but there has been growing recognition in recent years of the role that TD individuals play in these intersubjective breakdowns (Jaswal and Akhtar 2019; Morrison et al. 2019, 2020). This shift

[6] We emphasize the specificity of our measure of emotional contagion within the larger construct of empathy. Whereas another aspect, cognitive empathy/affective ToM, is captured by the RMET and the ToMI (particularly the Early subscale, which includes several items pertaining to emotion recognition and understanding), our study lacks a measure of empathic concern.

[7] Self-reports are also imperfect measures of emotional contagion in autistic individuals, who are more likely than TD individuals to experience alexithymia—that is, difficulty interpreting and verbalizing one's own emotional state (Bird & Viding 2014). The study by Trimmer et al. (2017) illustrates this: autistic individuals under-reported their affective responses to distressing videos despite exhibiting typical levels of physiological arousal and facial affect.

toward understanding social difficulties as emerging from misaligned interactions between autistic and TD individuals is reflected in concepts like the "double empathy problem" (Milton 2012) and "interpersonal mismatch" (Bolis et al. 2017). Beyond reconceptualizing the nature of autism, these ideas implicate TD individuals as potential targets for intervention (Bottema-Beutel 2017; Morrison et al. 2019, 2020). In the context of emotional contagion, in addition to helping autistic people recognize and respond to affective cues, practitioners could assist their families, friends, and peers in learning to recognize the idiosyncratic ways in which autistic people might express emotion, thereby increasing the potential for emotional resonance.

ToM did not Predict Social Symptom Severity

Given the ubiquity of the mindblindness theory, it is notable that none of the ToM variables emerged as important predictors of social symptom severity. Altogether, the ToM variables accounted for only 6% of the variance in social symptoms, similar to another recent study in which social cognition accounted for 6% of the variance in social skills in autistic adults after accounting for other cognitive skills (Sasson et al. 2020). Further undermining the mindblindness theory, we found no substantial evidence of group differences in either verbal-cognitive or visual-affective ToM. An important caveat to these negative findings is that our autistic sample consisted mainly of individuals with average to above-average verbal and intellectual abilities. Such abilities are known to relate to performance on ToM tasks (Baker et al. 2014; Happé 1995); thus, sampling from this relatively narrow range of the autism spectrum may have precluded us from detecting ToM deficits similar to those found in some previous studies. Nevertheless, our results are in accord with other findings and theoretical positions challenging the view that ToM impairment is the dominant source of social difficulties for all autistic individuals (Bottema-Beutel 2017; De Jaegher 2013).

Rather than concluding that ToM is irrelevant to social impairment in ASD, we suspect that our negative findings reflect the limitations of commonly used measures (Livingston et al. 2019). There is no strong consensus on which tasks are best suited to measuring the various aspects of ToM, particularly affective ToM. For example, some have questioned whether the RMET is a valid measure of affective ToM, arguing that it is instead a test of facial emotion recognition (Quesque and Rossetti 2020). Emotion recognition can be dissociated from ToM in clinical samples (Fairchild et al. 2009; Freedman et al. 2013; O'Nions et al. 2014) and may be impaired in ASD due to alexithymia rather than impaired ToM (Oakley et al. 2016). Other tasks that require participants to reason about emotions expressed within complex social situations, such as the Theory-of-Mind Test (TOM Test; Muris et al. 1999) or the Movie for the Assessment of Social Cognition (MASC; Dziobek et al. 2006), may be more valid measures of the affective ToM abilities that are most relevant to real-world interactions. Supporting this notion, Altschuler et al. (2018) found that affective ToM, indexed by performance on the TOM Test and Social Attribution Task (Klin 2000), was negatively associated with social symptom severity measured by the ADOS Social Affect score.

Additionally, a major limitation of tasks like the Strange Stories and RMET is that they involve explicitly reflecting on artificial social stimuli, rather than the spontaneous mental state inferences required by real-world interaction (Redcay and Schilbach 2019; Schilbach et al. 2013). Illustrating this discrepancy, recent neuroimaging studies of TD children have found differences in social processing when observing versus interacting (Alkire et al. 2018; Warnell et al. 2018), and autistic individuals who perform well on explicit ToM tasks are nevertheless impaired on spontaneous measures of ToM (Abell et al. 2000; Klin 2000; Senju et al. 2009). Spontaneous ToM measures—that is, those that do not explicitly prompt the participant to apply ToM reasoning—may therefore better predict social symptom severity compared to explicit measures. Furthermore, social interaction often elicits anxiety in autistic individuals (Spain et al. 2018), and this anxiety may interfere with social-cognitive processes that are intact in more observational contexts. Consistent with this idea, for the handful of autistic children low in social anxiety, we observed the expected negative relation between verbal-cognitive ToM (i.e., the Strange Stories) and social symptoms, yet no such relation was present for children with higher levels of social anxiety. We speculate that social anxiety may have hindered these children's ability to apply ToM skills when interacting with the clinician during the ADOS. Further research using larger samples is needed to confirm and explicate this dynamic between social anxiety and social cognition. In the meantime, our findings suggest that when trying to account for social difficulties in ASD, ToM should not be considered as an isolated ability, divorced from its application within social interactions.

Though not socially interactive itself, the ToMI is an indirect measure of real-world ToM application. As expected, our autistic participants scored markedly lower on this parent-report measure than their TD peers, but surprisingly, these scores did not predict social symptom severity. One possible explanation is that parents underestimate their autistic children's ToM abilities. This is suggested by a recent study in which autistic individuals accurately predicted how their family members would rate them on a set of skills (often at odds with how they rated themselves), yet their family members perceived them to have limited perspective-taking abilities (Heasman and Gillespie 2018). An alternative explanation for the lack of association between

ToMI ratings and social symptom severity is that the ADOS Social Affect score may not reflect the specific difficulties that result from ToM impairment. To address this potential limitation, more fine-tuned measures are needed to capture ToM-related behavior within social interactions.

BMP and Social Reward also did not Predict Social Symptom Severity

We found neither BMP nor social reward to explain a meaningful amount of variance in social symptoms. As discussed earlier, we were interested in comparing the relative contributions of BMP and ToM given previous links between these constructs (Miller and Saygin 2013; Rice et al. 2016). In line with BMP impairment being the earlier deficit, BMP explained more unique variance than any single ToM variable and was more consistently included in the best models, yet it still accounted for only around 4% of the variance in social symptoms. Moreover, we found no difference between the autistic and TD groups on the BMP task, consistent with previous work showing intact action perception in ASD (Cusack et al. 2015; Murphy et al. 2009).

The similar BMP performance between our autistic and TD groups may be explained by the properties of our task and sample. A recent meta-analysis found decreased performance in ASD across all BMP paradigms tested, but this decrease was especially pronounced for tasks involving emotion recognition (Todorova et al. 2019). In another study, autistic people were less able to use one agent's communicative action to predict another agent's action (von der Lühe et al. 2016). Thus, social impairment in ASD may relate to difficulties with integrating perceptual cues with higher-level social information, as opposed to simple action perception as measured by tasks such as ours. Furthermore, BMP deficits are larger in children compared to adolescents and adults, suggesting that autistic individuals are delayed on BMP but eventually catch up to TD performance levels (Todorova et al. 2019). As our sample includes early adolescents, our results may reflect this developmental trajectory; indeed, BMP performance was positively correlated with age in our autistic participants ($r = 0.51$; Table S2.1).

In contrast with the social motivation theory, we found only anecdotal evidence that autistic individuals experience less social reward than their TD peers. Consistent with previous self-reports of social reward in ASD (reviewed in Jaswal and Akhtar 2019), scores on each of the two SRQ subscales varied widely, with the autistic group's distributions largely overlapping with those of the TD group; only a few autistic children reported particularly low levels of social reward. Despite this variability, we found no evidence that social reward deficits are associated with social dysfunction. Instead of social reward deficits driving social impairment, it may be that some autistic individuals dislike social situations as a consequence of repeated unsuccessful attempts to navigate them (Drew 2017; Rentenbach and Prislovsky 2012; as cited in Jaswal and Akhtar 2019).

However, the conclusions we can draw from this negative finding are again constrained by our choice of measures. As others have pointed out, social motivation is a loosely defined construct, of which social reward is only one component—namely, the hedonic response to social experiences (i.e., "liking"; Keifer et al. 2019;). Our study cannot speak to the claim that other social motivational processes such as orienting and "wanting" are altered in ASD and explain social symptoms (Chevallier et al. 2012). The SRQ is also limited by its self-report nature. Neuroimaging studies have revealed group-level differences in reward processing in ASD (Clements et al. 2018), differences that may not be consciously accessible but may nevertheless affect behavior (Yankowitz and Clements 2019). Still, in terms of lived experience, our findings support the view that many autistic individuals are capable of enjoying social interactions if given the opportunity. Finally, though social reward did not predict social symptoms exhibited during the ADOS, it may play a role in autistic people's tendencies to seek out and maintain real-world relationships.

Limitations and Conclusions

Some general caveats apply to the interpretation of our findings. As noted above, our autistic sample comprised verbal, non-intellectually-disabled children; additionally, most of these children were Caucasian males from high-SES families. While not unusual in the context of psychological research on autism, this sample is not representative of the wider autistic population, limiting the generalizability of our results. Furthermore, despite the breadth of constructs we included as potential predictors, we readily acknowledge that these are not exhaustive of all factors that may influence social symptoms. This is evident in the large amount of unexplained variance (over 70%) even in the model with all predictors included. Our aim was to compare constructs within the social domain, and though our inclusion of verbal IQ, non-verbal IQ, and the Strange Stories Control condition likely captured some domain-general factors, more fine-grained measures of neurocognitive skills may be necessary to better explain variance in social symptom severity (Sasson et al. 2020).

Lastly, we emphasized above that our predictor variables captured limited aspects of their respective constructs; the same can be said of our outcome measure. That is, the social symptoms indexed by the ADOS Social Affect score may not reflect the complexities of real-world social functioning. While the ADOS is well validated as a diagnostic tool, other measures may better capture variability in social impairments in ASD (Anagnostou et al. 2015). In particular, we encourage further investigation of our research questions using more ecologically valid measures of social behavior and perceptions thereof, such

as peer or self-reports following naturalistic social interactions (Morrison et al. 2019, 2020; Usher et al. 2018).

Despite these limitations, the present study has clear implications for future research. Our primary finding that parent-perceived emotional contagion uniquely predicted social symptom severity should encourage further study of how emotions are shared and communicated between autistic and TD individuals. Beyond this, two general themes emerge from our discussion above. First, when autistic and non-autistic groups differ on a particular construct, it is tempting to assume that this construct meaningfully contributes to social impairment in ASD. This assumption is challenged by our findings of pronounced group differences in applied ToM, which did not predict social symptoms, and no group difference in emotional contagion, the dominant predictor of social symptoms of ASD in our study. Second, the cognitive and affective mechanisms underlying social functioning are likely sensitive to interactive contexts and transcend the individual. Therefore, studies—and ultimately, interventions—that take into account the interactional nature of social impairment may be more fruitful than those solely focused on deficits within the autistic person.

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Author contributions DA conceived of the present study and methods, analyzed the data, and wrote the manuscript. ER served in an advisory role throughout the research and is the principal investigator of the larger study from which these data are derived. KRW and LAK contributed to material development, and KRW and DA oversaw scoring procedures. LAK conducted the majority of assessments to confirm ASD diagnoses. KRW, LAK, DM, and DA assisted with data collection. KRW, LAK, DM, and ER critically reviewed and contributed to the preregistration of the study and the final manuscript.

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