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Social network size relates to developmental neural sensitivity to biological motion

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ABSTRACT

The ability to perceive others' actions and goals from human motion (i.e., biological motion perception) is a critical component of social perception and may be linked to the development of real-world social relationships. Adult research demonstrates two key nodes of the brain's biological motion perception system—amygdala and posterior superior temporal sulcus (pSTS)—are linked to variability in social network properties. The relation between social perception and social network properties, however, has not yet been investigated in middle childhood—a time when individual differences in social experiences and social perception are growing. The aims of this study were to (1) replicate past work showing amygdala and pSTS sensitivity to biological motion in middle childhood; (2) examine age-related changes in the neural sensitivity for biological motion, and (3) determine whether neural sensitivity for biological motion relates to social network characteristics in children. Consistent with past work, we demonstrate a significant relation between social network size and neural sensitivity for biological motion in left pSTS, but do not find age-related change in biological motion perception. This finding offers evidence for the interplay between real-world social experiences and functional brain development and has important implications for understanding disorders of atypical social experience.

1. Introduction

Biological motion is a powerful cue that humans use to detect others, allowing them to navigate a complex social world and decode information about others' mental states (Frith and Frith, 1999). Even when viewing impoverished visual representations of biological motion (e.g., lights placed on the joints of a person walking), the human visual system can distinguish between biological and mechanical motion (Johansson, 1973). Further, individual differences in neural sensitivity to biological motion are related to real-world properties of social networks, such as one's diversity of roles within their social network (Dziura and Thompson, 2014). Thus, studying biological motion perception provides an important window into the typical and atypical development of social processing (Pelphrey and Carter, 2008). Of particular importance is understanding the relation between social perception and social network properties in middle childhood, as this is a time when individual differences in social experiences and social perception are rapidly growing.

Though infants are sensitive to biological motion soon after birth (Simion et al., 2008), neural and behavioral sensitivity to biological motion continues to develop into adolescence. Studies that increase task demands by adding noise dots to biological motion point light displays show continued improvement in detection ability between ages 6 and 14, at which time children reach adult-like accuracy (Freire et al., 2006; Hadad et al., 2011; Rice et al., 2016; but see Pavlova et al., 2001).

Evidence from non-human primates and human adults reveals that the neural systems supporting biological motion perception include orbitofrontal cortex, temporo-occipital cortex, nucleus accumbens, caudate nucleus, fusiform gyrus (FG), cerebellum (Bonda et al., 1996; Grossman et al., 2000), amygdala (Adolphs and Spezio, 2006; Bonda et al., 1996) and posterior superior temporal sulcus (STS; Grossman et al., 2005). Paralleling behavioral changes, neural sensitivity to biological motion may also change throughout childhood. Specifically, although the neural signatures of such perception emerge by 5 months (Lloyd-Fox et al., 2011), the pSTS in particular may show a protracted development in response to biological versus non-biological motion (Carter and Pelphrey, 2006; Lichtensteiger et al., 2008). However, this conclusion is based on only two studies with relatively small sample sizes, and thus neurodevelopmental changes in biological motion perception remain underexplored.

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Concurrent with these developmental changes in neural and behavioral sensitivity to biological motion are changes in children's social experience. In middle childhood, children begin spending less time with family and more time with same-aged peers and adults, resulting in more varied and complex social networks (Blyth, 1983; Bryant, 1985; Feiring and Lewis, 1991; Hartup, 1983; Parker et al., 2006). Further, between ages 6–12, children show great improvements in interpersonal skills and relationship quality (Parker et al., 2006). These developing individual differences in social networks may contribute to changes in responsivity to biological motion.

Evidence from adults suggests that individual differences in social experience do relate to individual differences in response to biological motion. Suggestively, two key nodes of the biological motion perception network—amygdala and pSTS—have been linked to variability in social network size. For example, in both nonhuman primates (Sallet et al., 2011) and human adults (Kanai et al., 2012), cortical thickness of the STS is correlated with social network size, defined as the total number of people with whom participants have regular contact. Social network size is also related to amygdala volume (nonhuman primates: Lewis and Barton, 2006; although cf. Joffe and Dunbar, 1997; human adults: Bickart et al., 2011), amygdala white matter microstructure (Hampton et al., 2016) and to resting state functional connectivity between the amygdala, STS, and FG (Bickart et al., 2012). Beyond these findings, *functional* sensitivity to biological motion in amygdala and pSTS is related to social network properties in human adults (Dziura and Thompson, 2014). Further, studies have found relations between biological motion perception and autistic traits in neurotypical adults (Miller and Saygin, 2013; Van Boxtel and Lu, 2013). These findings from nonhuman primates and human adults indicate that emerging differences in social experience during middle childhood may relate to concurrent developing neural sensitivity.

The purpose of the current study was to investigate the relation between social experience and neural sensitivity for biological motion during middle childhood. The aims of this study were to (1) replicate past work showing amygdala and posterior superior temporal sulcus (pSTS) sensitivity to biological motion in middle childhood; (2) examine age-related changes in the neural functional sensitivity for biological motion during middle childhood, and (3) determine whether neural sensitivity for biological motion relates to social network characteristics in children. We hypothesized neural sensitivity to biological motion would be seen in regions of the 'social brain' including the amygdala and pSTS and that this may increase with age. We also predicted that between ages 7 and 13, there would be an age-related increase in neural sensitivity to biological motion, and that sensitivity in regions previously identified as related to social network characteristics in adults (i.e., pSTS and amygdala) (Dziura and Thompson, 2014), would relate to metrics of social experience, specifically greater size, diversity, and/or complexity of children's social networks.

2. Methods

2.1. Participants

Participants were 51 children between the ages of 7 and 13 without a family history of autism or schizophrenia (as assessed via parent report). Participants had no history of psychiatric or psychological conditions or of neurological damage. Due to motion artifacts ($N = 10$) and failure to complete the scan ($N = 1$), 11 children were excluded from further analyses. Thus, the final sample comprised 40 children (*M* age: 10.37, *SD*: 1.89, 23 females). All participants provided informed consent and all protocols were approved by the University of Maryland Institutional Review Board.

2.2. Behavioral measures

Parents completed a version of the Social Network Index (Cohen et al., 1997; see Appendix A). The Social Network Index is a measure of a child's social network size (the number of people the child regularly sees or talks to), diversity (the number of social roles in which the child has regular contact with at least one person; e.g. sibling, friend, neighbor), and embeddedness (the number of social network domains in which the child is active; e.g. sports teams, clubs, religious groups). We included these different social network measures because they differentially predicted pSTS and amygdala activation in a past adult study (Dziura and Thompson, 2014). Modifications to the questionnaire were minimal and included deleting a question about employment and changing the questions on relationship status to be about a best friend. A limitation to the current study is that the Social Network Index is typically used as a self-report measure, and future research is needed to test the validity of this measure when used as a parent report. As with all parent report measures, other factors such as the quality of the parent-child relationship could influence the parent's responses on the Social Network Index. However, this was deemed unlikely given past evidence that parents of adolescents are 80% accurate regarding their knowledge about their child's daily experiences with peers (Updegraff et al., 2001).

2.3. fMRI Portion

2.3.1. Stimuli

Children were presented with biological motion and control scrambled motion visual displays. Displays were created using Matlab Psychophysics Toolbox Version 3 (PTB-3) and were based on coordinates from standard validated stimuli (Vanrie and Verfaillie, 2004). The biological motion displays were point-light depictions of an actor completing different actions such as walking or painting, each of which consisted of 13 points. To create scrambled motion versions of the biological motion displays, first, a bounding box was created of the same size and location as the figure in the biological motion display. The 13 points were then placed at a random x and y coordinate within the bounding box to create randomized starting positions for each point. The 13 points retained their original motion trajectories, but the randomized starting locations destroyed the illusion of a biological figure. For example, the point in the "walking" display that represented the left knee of the figure still moved in the backwards and forwards pattern of the knee, but it was given a different random start location to disrupt the appearance of a figure. In this way, the biological and scrambled displays contained the same amount and type of motion; however, the biological motion displays resembled human actions whereas the scrambled displays did not.

2.3.2. Scan procedure

During the scan, children viewed point-light displays of biological and scrambled motion, and to maintain attention, they completed a

one-back task, pressing a button whenever two identical videos occurred in a row. To ensure that children understood this task and were able to lie still, children first practiced a one-back task with different stimuli in a mock scanner. For the actual scan, stimuli were presented in two separate runs, each lasting six minutes and 10 s. Each run contained 12 blocks, alternating in a semi-random fashion between biological and scrambled motion stimuli (19.25 s per block). Each block contained seven 2 s videos. Six of these videos were unique, and one was a repeat for the one-back task. Between each video (within blocks), there was a 500 ms inter-stimulus interval (with a fixation cross), and between blocks, there was a 10 s fixation period.

For the two functional runs, data were collected using both 12-channel ($N = 19$) and 32-channel ($N = 21$) head coils on a Siemens 3T scanner (T2*-weighted echo-planer gradient-echo; 36 interleaved axial slices; TR: 2000 ms, TE: 24 ms, flip angle: 90°, pixel matrix: 64 × 64, voxel size: 3 mm^3). Separate analyses were conducted with head coil as a covariate, and these analyses did not lead to any changes in results. High-resolution anatomical images were also acquired (three-dimensional T1 magnetization-prepared rapid gradient-echo sequence; 176 contiguous sagittal slices; TR = 1900 ms, TE = 2.52 ms, flip angle = 9°; pixel matrix = 256 × 256, voxel size = 1 mm^3).

2.4. Data analysis

2.4.1. Social network index

Thirty-three participants' parents completed the Social Network Index about their child. Seven participants' parents completed a different social network measure, which did not yield the summary variables of interest and thus were not included in social network analyses. We assessed the normality of the distribution for the three social network measures (size, diversity, and embeddedness) using Shapiro-Wilk tests. To assess age-related changes, we then examined correlations between participant age and each social network measure.

2.4.2. fMRI Analyses: biological > scrambled

2.4.2.1. Whole brain. We used the Analysis of Functional NeuroImages program (AFNI; Cox, 1996) to perform whole brain volume-based fMRI analyses. Preprocessing included slice time correction within each volume, registration (using affine transformation) of each functional volume to the first volume of the first functional run and co-registration of the structural image to the base functional volume. We then spatially normalized participants' data to the MNI pediatric template for ages 7.5–13.5 (Fonov et al., 2011). Data were then intensity normalized and smoothed using a Gaussian smoothing kernel (FWHM: 5 mm).

We ran Ordinary Least Squares regression analyses for the two runs concatenated with regressors for each of the two conditions (biological and scrambled) as well as nuisance regressors, which included baseline and linear, quadratic, and cubic trends as well as 12 motion regressors (i.e., the frame deviation at each volume for the six directions of translational and rotational motion and their derivatives). Individual runs with greater than 4 mm maximum motion in any direction were excluded, and we censored volumes that exceeded 1 mm motion (Siegel et al., 2014). We created regressors for each of the two conditions by convolving a gamma-variate basis function with the stimulus timing function with duration the length of one block (19.25 s) and an amplitude of 1. Contrasts were estimated for each condition of interest (biological vs. baseline and scrambled vs. baseline) and for the comparison of biological vs. scrambled conditions.

Coefficients and t-statistics for each contrast were incorporated in the group-level analyses using mixed effect models (3dMEMA) (Chen et al., 2013), modeling both within- and between-subject variance. For each contrast, we calculated an effect of group across all participants for each voxel using mixed effect models. We also conducted a whole-brain regression analysis with age. For all whole brain analyses, we applied a cluster-correction of 17 voxels, which maintains an overall alpha of $p < 0.05$ with a voxel-wise threshold of $p < 0.001$ (estimated from 1000 iterations of Monte Carlo Simulations using AFNI's updated 3dClustSim, see Eklund et al., 2016).

2.4.2.2. Region of interest (ROI): amygdala. For the volume-based amygdala ROI analysis, we used bilateral anatomical amygdala ROIs (Maldjian et al., 2003) in order to ensure independence. We extracted beta values—averaged across the ROI—for biological and scrambled motion from each participant for left and right ROIs. Using a paired *t*-test, we determined whether beta values for biological motion were significantly greater than beta values for scrambled motion within bilateral amygdala ROIs. We then conducted a Pearson's correlation between beta values for the contrast of biological > scrambled motion within each ROI and participant age to determine if the neural sensitivity to biological motion in the amygdala changed with age. The amygdala ROI analysis included 40 participants (33 of whom also had parent-reported social network questionnaires and thus were included in ROI brain-behavior correlation analyses).

2.4.2.3. Region of interest (ROI): pSTS. Given the difficulty in anatomically defining the region of the pSTS sensitive to biological motion, we instead functionally-defined this ROI (i.e., activation values for a run were extracted from an ROI defined by the other run). Further, because primary regions of interest were within sulci, we conducted surface-based (as opposed to volumetric) analyses. Surface-based analyses allow for smoothing on a flat surface map, thereby avoiding blurring between non-contiguous regions of cortex (e.g., two gyri; Jo et al., 2007). To create the pSTS ROI, we used Freesurfer's (version 5.1.0) automated pipeline (Fischl, 2012) and created cortical surface models from each participant's high-resolution anatomical image. These were subsequently inspected and corrected (if necessary) by trained research assistants. We then used the Analysis of Functional NeuroImages (AFNI; Cox, 1996) and surface mapping (SUMA) programs (Saad et al., 2004; Saad and Reynolds, 2012) to perform surface-based fMRI analyses. We used SUMA to create standard mesh surfaces (198,812 nodes per hemisphere) from the surfaces created by Freesurfer. After first performing the preprocessing steps noted in 2.4.2.1. *Whole-brain* (except for smoothing), we then aligned the surface volume to the functional data and then projected the timeseries to the surface (using a mean mapping function). On the surface, data were intensity normalized and smoothed using a Gaussian smoothing kernel (FWHM: 5 mm).

Regression analyses were conducted in the same way described above (2.4.2.1. *Whole Brain*), except rather than concatenating runs, we ran regressions for each run separately. For each participant, pSTS ROIs were created using the contrast of biological > scrambled motion. Two trained research assistants identified clusters of activation on each participant's pSTS for each run and each hemisphere. Masks of these

ROIs were then used to extract data from the opposite run (i.e., ROIs created for run 1 were used to extract beta values from run 2 and vice versa, and then both betas were averaged for a single value), ensuring that the creation and use of the ROIs were independent (Kriegeskorte et al., 2009). The ROIs included all significant surface nodes within a 9-node distance along the surface's mesh from the node of peak activation. Within-participant pSTS ROIs generally overlapped between runs, though we observed wide variability (left hemisphere: 23.9 ± 27.0, range: 0–89.2% overlap; right hemisphere: 27.3 ± 30.4, range: 0–100% overlap). However, despite the large variability in ROI overlap, biological > scrambled beta values were significantly correlated (left pSTS: r(30) = 0.45, p < 0.01; right pSTS: r(30) = 0.53, p < 0.01). Using a paired *t*-test, we determined whether beta values for biological motion were significantly greater than those for scrambled motion within bilateral pSTS. We then conducted a Pearson's correlation between beta values for the contrast of biological > scrambled motion within each ROI and participant age to investigate age-related changes in pSTS neural sensitivity to biological motion.

One participant was excluded for not having the useable high-resolution anatomical image necessary for surface-based analyses, and seven participants were excluded for not having two useable runs (as both functional runs were required for the independent selection of ROIs). Thus, the surface-based pSTS ROI analysis included 32 participants (26 of whom also had parent-reported social network questionnaires and thus were included in ROI brain-behavior correlation analyses).

2.4.3. fMRI Analyses: brain-behavior correlations

2.4.3.1. Whole brain. We conducted a whole-brain mixed-effect linear model (3dMEMA; Chen et al., 2013) with social network scores to assess relations between social network scores and participants' neural activation to biological > scrambled motion. We ran a separate regression for each of the three social network measures (size, diversity, and embeddedness) as whole-brain mean-centered regressors. Again, these whole-brain correlations were corrected for multiple comparisons using AFNI's 3dClustSim, maintaining an overall alpha of 0.05 with a voxel-wise threshold of $p < 0.001$ (k = 17, 540 mm^3).

2.4.3.2. Region of interest: pSTS and amygdala. We conducted separate partial correlation analyses (controlling for age) to determine if biological > scrambled beta values for the pSTS and amygdala were related to children's social network characteristics (size, diversity, and embeddedness). These were corrected for multiple comparisons using Bonferroni correction.

3. Results

3.1. Behavioral analyses

The range of values for social network size was 12–114, the range for social network diversity was 5–10, and the range for social network embeddedness was 2–7. Shapiro-Wilk tests for normality suggested that none of the three social network measures (size, diversity, and embeddedness) were from normally distributed populations (*ps* < 0.05). We log-transformed the social network variables; however, Shapiro-Wilk tests for normality still suggested that social network diversity and embeddedness were each from a non-normally distributed population (*ps* < 0.05). Thus, nonparametric correlation coefficients (Spearman's rho: r_s) are reported for all correlations with social network measures. All three measures of social networks were significantly correlated with one another (*ps* < 0.01), and no measures of social network were significantly correlated with participant age (*ps* > 0.05).

3.2. fMRI Analyses: biological > scrambled

3.2.1. Whole brain

As predicted, there was significant bilateral pSTS activation, in addition to activation in other areas of the 'social brain' (Adolphs, 2009; Brothers et al., 1990) including orbitofrontal cortex, fusiform gyrus, and inferior frontal gyrus ($p < 0.001$, $\alpha = 0.05$, k = 17 voxels) to the contrast of biological > scrambled motion (Fig. 1A, Table 1). There was no significant relation between whole-brain activation to biological > scrambled motion and age ($p < 0.001$, $\alpha = 0.05$, k = 17 voxels).

3.2.2. Region of interest (ROI): pSTS and amygdala

The neural response to biological motion in an independently defined amygdala region was significantly greater than the neural response to scrambled motion (left: $t(39) = 3.22$, $p < 0.01$; right: $t(39) = 3.18$, $p < 0.01$) (Fig. 1B), and this neural sensitivity did not vary as a function of age (left: $r(38) = 0.13$, $p = 0.43$, right: $r(38) = 0.14$, $p = 0.38$).

The neural response to biological motion in independently-defined pSTS was significantly greater than the neural response to scrambled motion (left: $t(31) = 5.14$, $p < 0.001$; right: $t(31) = 3.37$, $p < 0.01$) (Fig. 1B). Neural sensitivity to biological motion within bilateral pSTS did not vary as a function of age (left: $r(30) = -0.07$, $p = 0.72$, right: $r(30) = -0.08$, $p = 0.67$).

3.3. fMRI Analyses: brain-behavior correlations

3.3.1. Whole brain

There were no significant correlations between neural sensitivity to biological motion and social network size, diversity or embeddedness at the whole-brain level ($p < 0.001$, $\alpha = 0.05$, k = 17 voxels).

3.3.2. Region of interest: pSTS and amygdala

Controlling for age and correcting for multiple comparisons, social network size was significantly correlated with neural sensitivity for biological motion in left pSTS ($r_s(23) = 0.51$, $p = 0.004$) (Fig. 2). Neither social network diversity nor social network embeddedness was significantly related to neural sensitivity for biological motion in left pSTS. The magnitude of correlation with left pSTS activity, however, did not statistically differ across the three social network measures (*ps* > 0.05). Social network size was not significantly related to neural sensitivity for biological motion in right pSTS ($r_s(23) = 0.24$, $p = 0.24$) or bilateral amygdala (left: $r_s(23) = 0.07$, $p = 0.71$, right: $r_s(23) = 0.04$, $p = 0.81$); however, the magnitude of the correlation coefficients for left vs. right pSTS did not statistically differ ($p > 0.05$). As with left pSTS, neither social network diversity nor social network embeddedness was significantly related to neural sensitivity for biological motion in right pSTS or bilateral amygdala (*ps* > 0.05). Table 2 depicts all correlation values for ROIs and social network measures. Using a cutoff of 2 standard deviations, there were no outliers on

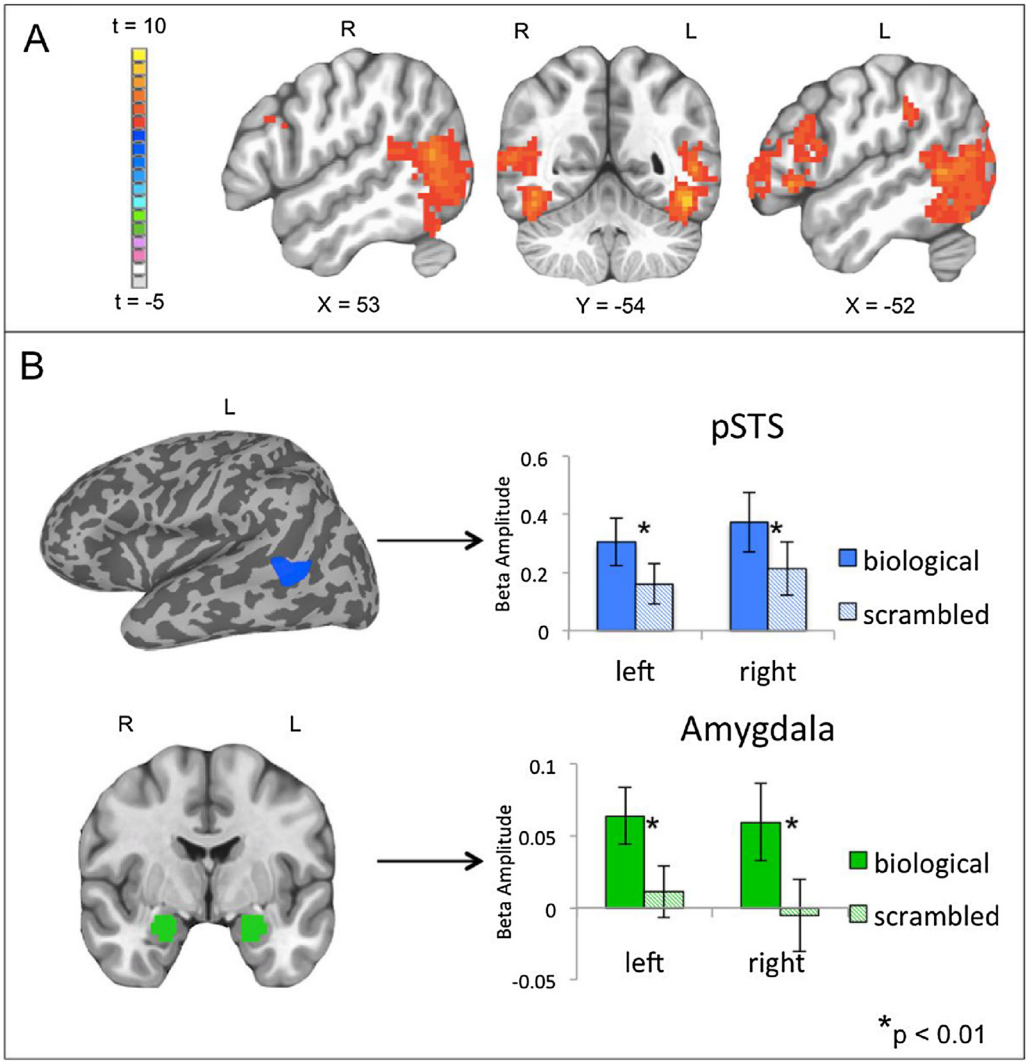

Fig. 1. A) Whole-brain activation Biological > Scrambled motion ($p < 0.001$, $\alpha = 0.05$, k = 17 voxels). B) Region of interest (ROI) analysis. A functionally defined pSTS ROI from an individual participant and structurally defined amygdala ROIs are displayed on the left. Beta amplitude plots are displayed on the right.

Table 1
Whole-brain Biological > Scrambled peak t values, coordinates, and number of voxels ($p < 0.001$, $\alpha = 0.05$, k = 17 voxels). *Local peaks that are part of the larger fusiform gyrus/pSTS clusters of activation.

Region	Hemisphere	Peak t	Peak x	Peak y	Peak z	# Voxels
fusiform gyrus/pSTS	Left	4.61	−46.5	−46.5	−26.0	1157
fusiform gyrus/pSTS	Right	5.48	46.5	−58.5	−23.0	874
posterior superior temporal sulcus*	Left	6.22	−58.5	−70.5	10.0	
posterior superior temporal sulcus*	Right	5.95	55.5	−61.5	10	
inferior frontal gyrus	Left	4.38	−52.5	52.5	−8.0	447
caudate	Left	5.24	−10.5	1.5	16.0	107
inferior occipital gyrus	Right	4.17	22.5	−106.5	−5.0	101
inferior parietal lobule	Left	4.12	−67.5	−37.5	31.0	92
inferior frontal gyrus	Right	3.64	61.5	22.5	28.0	34
middle temporal gyrus	Left	4.07	−52.5	−79.5	28.0	31
hippocampus	Right	3.97	22.5	−22.5	−8.0	30
thalamus	Left	5.35	−7.5	−13.5	10.0	27

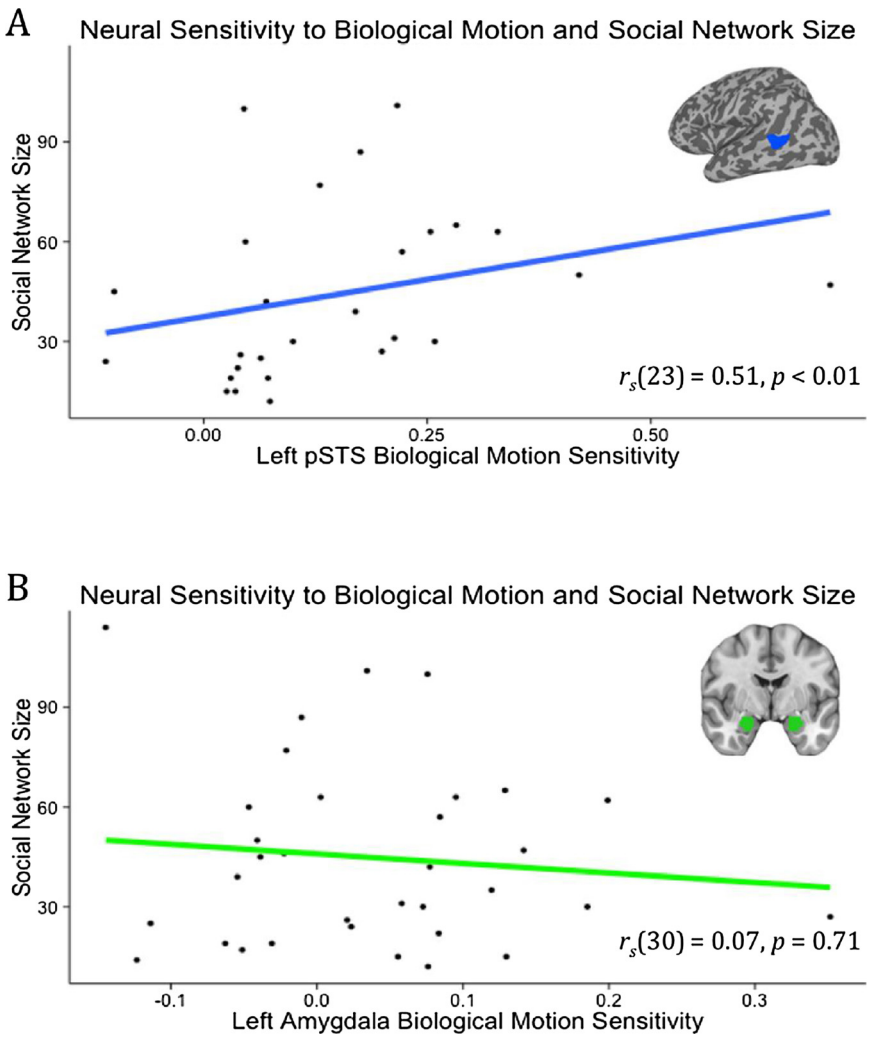

Fig. 2. A) Social network size was significantly correlated with neural sensitivity for biological motion (i.e., biological > scrambled beta amplitude) in left pSTS, controlling for age. An individual's left pSTS ROI is displayed as an example. B) Social network size was not significantly correlated with biological > scrambled beta amplitude in the left or right amygdala. Biological > scrambled beta amplitudes for the left anatomical amygdala ROI are displayed.

Table 2
Correlations (r_s) between ROIs and social network measures, controlling for age.

	Left pSTS	Right pSTS	Left amygdala	Right amygdala
Social network size	0.51 ($p = 0.004$)	0.24 ($p = 0.24$)	0.07 ($p = 0.71$)	0.04 ($p = 0.81$)
Social network diversity	0.11 ($p = 0.60$)	−0.06 ($p = 0.79$)	0.06 ($p = 0.73$)	0.19 ($p = 0.28$)
Social network embeddedness	0.15 ($p = 0.45$)	−0.04 ($p = 0.87$)	0.07 ($p = 0.69$)	0.05 ($p = 0.80$)

measures of embeddedness and diversity, but there were two outliers in social network size. We repeated the analysis excluding these two individuals and found the same pattern of results: a significant correlation with left pSTS activation ($r_s(21) = 0.49$, $p = 0.008$) and no relations with other regions.

4. Discussion

This study investigated the relation between social experience and neural sensitivity for biological motion during middle childhood. Consistent with past work in humans and primates linking social network measures to the structure (Kanai et al., 2012; Sallet et al., 2011) and function (Dziura and Thompson, 2014) of the STS, we demonstrated a significant relation between social network size and neural sensitivity for biological motion in left pSTS. To our knowledge, this is the first study to find this relation in middle childhood.

At the group level, we found significant bilateral pSTS activation to the contrast of biological > scrambled motion, in addition to activation in other areas of the 'social brain'. These findings are consistent with the limited body of research that has investigated biological motion sensitivity from infancy through adulthood (e.g., Anderson et al., 2013;

Bonda et al., 1996; Carter and Pelphrey, 2006; Grossman et al., 2000; Hirai and Hiraki, 2005; Lichtensteiger et al., 2008; Lloyd-Fox et al., 2011; Pavlova, 2012; Reid et al., 2006).

In contrast to our predictions, neural sensitivity to biological motion within bilateral pSTS did not vary as function of age within this middle childhood age range. However, the previous fMRI study investigating age-related neural changes in biological motion processing during middle childhood (7–11 years) had only 9 participants (Carter and Pelphrey, 2006). The current study had greater power to detect an effect of age if present (n = 40) but did not find a significant relation with age. Further, a prior EEG study with 50 children (aged 7–14 years) indicated no developmental changes in the amplitude of the N2 component in response to biological motion (Hirai et al., 2009). Thus, while still very limited, the current evidence suggests no significant age-related differences in the magnitude of pSTS activation in response to biological motion between middle childhood and early adolescence. Future studies should employ standardized study procedures and utilize longitudinal designs with larger sample sizes in order to clarify these conflicting findings.

Additionally, while prior research with adults (Dziura and Thompson, 2014) found that right amygdala and right pSTS sensitivity to biological motion was related to multiple social network characteristics, we only found a relation between left pSTS and social network size. Limited variability in reported social network diversity and embeddedness scores may have restricted the ability to find significant correlations with these measures. One interesting question that can not be addressed in the current study is whether that limited variability is due to qualitative differences in children's social network characteristics compared to adults (e.g., less diversity may arise from children engaging in more similar activities such as in being in school, living with family members, etc.) or whether this is simply due to a restricted range in our current sample. It is also important to note that our correlation between social network size and left pSTS was not statistically different from correlations between social network metrics and right pSTS or bilateral amygdala sensitivity to biological motion. Thus, we hesitate to make any strong claims about specificity of this relation to left pSTS during middle childhood. Future research will be needed to clarify whether discrepancies between our study and previous work reflect true developmental differences or other factors.

While we found evidence that social network characteristics are correlated with neural sensitivity to biological motion in pSTS, the developmental mechanism linking these measures remains open. We argue for a bidirectional relation in which, first, social perception (e.g., interpreting eye gaze and body movements) acts as a foundational social skill for building a larger social network, and second, the resultant larger social network results in more social-perceptual experience, which then tunes the brain's social perceptual system, improving social perception. This possibility fits with theories that emphasize the importance of the reciprocal relation between experience and brain development (Gottlieb, 1991, 2007; Johnson, 2011).

Supporting the first link of this model, evidence suggests that social perceptual ability influences social skills that affect one's social experience. Enhanced social perceptual ability in infancy predicts more advanced theory of mind in early childhood (Brooks and Meltzoff, 2015; Wellman et al., 2008), and this relation persists in middle childhood (Rice et al., 2016) and adulthood (Miller and Saygin, 2013). And increased theory of mind is related to competence in peer interaction and popularity (McElwain and Volling, 2002; Peterson and Siegal, 2002; Slaughter et al., 2002). Thus, increasing social perceptual abilities may expand children's social networks. Future studies would benefit from additional measures of peer relationships (e.g. sociometrics; Asher and Dodge, 1986; Marsden 1990) beyond parent report.

The second link of our developmental model is that the social perceptual experience influences the brain's biological motion processing system. Evidence for this link comes from studies in which participants were trained to discriminate biological motion embedded in noise (Grossman et al., 2004; Herrington et al., 2011) and showed improved behavioral performance and increased sensitivity for biological (compared to scrambled) motion in STS and FG following training. Although that training was specific, a broader mechanism may be at play in the current study: children may gain social perceptual experience via interacting with others in their social network, which tunes the brain's social perceptual system. Ultimately, future studies should track the development of social perceptual brain networks and social network size longitudinally.

The current study has implications for greater understanding of disorders of atypical social experience, such as autism spectrum disorder (ASD). ASD is characterized by social impairments such as reduced attention to social-perceptual stimuli (e.g., biological motion) (Klin et al., 2009). These differences in social perception are also seen within the typical population, as those with high levels of autistic-like traits have poorer performance on biological motion tasks (Miller and Saygin, 2013; Van Boxtel and Lu, 2013). This reduced social attention may lead to cascading effects that impact social development, and eventually, the ability to form meaningful relationships. Thus, ASD may be an extreme case of low social perceptual ability (Klin et al., 2009), disruption in neural circuitry supporting social perception (e.g., Kaiser et al., 2010), and limited social experience (e.g., half of adults with ASD report having no friends; Howlin et al., 2004).

5. Conclusions

In sum, this study was the first to demonstrate a significant correlation between social network size and neural sensitivity to biological motion in middle childhood. Findings suggest that social perceptual ability and social experience may reciprocally influence one another; however, future longitudinal research will be needed to determine the time course and directionality of this relation throughout development.

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Appendix A. Parent-Reported Social Network Index

Instructions: This questionnaire is concerned with how many people your child sees or talks to on a regular basis including family, friends, workmates, neighbors, etc. Please read and answer each question carefully. Answer follow-up questions where appropriate.

1. How many siblings does your child have? (If you don't have any siblings, check '0' and skip to Q2.)
___0 ___1 ___2 ___3 ___4 ___5 ___6 ___7 or more

1a. How many of your child's siblings does your child live with?
___0 ___1 ___2 ___3 ___4 ___5 ___6 ___7 or more

1b. Please list the initials of everyone in 1a:
_____

2. Are both of your child's parents living? (If neither is living, check '0' and skip to question 3.)
___ (0) neither ___ (1) one parent only ___ (2) both

2a. Does your child live with both of his/her parents or speak with them once every 2 weeks?
___ (0) neither ___ (1) one parent only ___ (2) both

3. How many other relatives (other than parents and siblings) does your child feel close to? (If '0', check that space and skip to Q4.)
___0 ___1 ___2 ___3 ___4 ___5 ___6 ___7 or more

3a. How many of these relatives does your child see or talk to on the phone at least once every 2 weeks?
___0 ___1 ___2 ___3 ___4 ___5 ___6 ___7 or more

3b. Please list the initials of everyone in 3a:
_____
_____

4. Is there someone that your child considers his or her best friend?
___ (1) currently has a best friend
___ (2) does not currently have a best friend
___ (3) never has had a best friend

4a. Please list this person's initials (choose **one** if your child has multiple), if applicable:
_____

5. How many close friends does your child have other than the person in Q4? (meaning people that he/she feels at ease with, can talk to about private matters, and can call on for help)
___0 ___1 ___2 ___3 ___4 ___5 ___6 ___7 or more

5a. How many of these friends does your child see or talk to at least once every 2 weeks?
___0 ___1 ___2 ___3 ___4 ___5 ___6 ___7 or more

5b. Please list the initials of everyone in 5a:
_____
_____

6. Does your child belong to a church, temple, or other religious group? (If not, check 'no' and skip to Q7.)
____ no ____ yes

6a. How many members of your church or religious group does your child talk to at least once every 2 weeks? (This includes at group meetings and services.)
___0 ___1 ___2 ___3 ___4 ___5 ___6 ___7 or more

6b. Please list the initials of everyone in 6a:
_____
_____

7. Does your child attend school with others on a regular basis? (If not, check 'no' and skip to Q8)
____ no ____ yes

7a. How many fellow students does your child talk to at least once every 2 weeks?

___0 ___1 ___2 ___3 ___4 ___5 ___6 ___7 or more

7b. Approximately how large is your child's class _____

7c. Please list the initials of everyone in 7a (if less than 7):
_____

8. How many of your neighbors does your child visit or talk to at least once every 2 weeks?
___0 ___1 ___2 ___3 ___4 ___5 ___6 ___7 or more

8a. Please list the initials of everyone in Q8 (if less than 7):
_____

9. Is your child currently involved in regular volunteer work? (If not, check 'no' and skip to Q10).
____ no ____ yes

9a. How many people involved in this volunteer work does your child talk to at least once every 2 weeks?
___0 ___1 ___2 ___3 ___4 ___5 ___6 ___7 or more

9b. Please list the initials of everyone in 9a (if less than 7):
_____
_____

10. Does your child belong to any groups outside of school that meet at least once every 2 weeks? Examples include social clubs, recreational groups, sports teams, student government, professional organizations, Girl or Boy Scouts, groups concerned with community service, etc. (If your child doesn't belong to any such groups, check 'no' and skip the section below.)
____ no ____ yes

Consider those groups in which your child talks to a fellow group member at least once every 2 weeks. Please provide the following information for each such group: the name or type of group and the total number of members in that group *that your child talks to at least once every 2 weeks.*

List **Name of group** and **Approximate number of group members** child talks to at least once every 2 weeks

---
1.

2.

3.

4.

5.

6.

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