

Examining the relationship between social cognition and neural synchrony during movies in children with and without autism

Lyons, KM^{a,b,*}, Stevenson, RA^{a,b}, Owen, AM^{a,b,c}, & Stojanoski, B^{a,b}

a The Brain and Mind Institute, Western University, London ON, N6A 5B7, Canada.

b The Department of Psychology, Western University, London ON, N6A 5B7, Canada.

c The Department of Physiology and Pharmacology, Western University, London ON, N6A 5B7, Canada.

Corresponding author

Kathleen Lyons

Email: klyons8@uwo.ca

The Brain and Mind Institute, Department of Psychology,
The University of Western Ontario, London, Ontario, N6C 5B7, Canada

1 **Abstract**

2 Children who have been diagnosed with autism spectrum disorder (ASD) often show a
3 marked deficit in measures of social cognition. In autistic adults, measures of social
4 cognition have been shown to relate to differences in brain synchronization (as measured
5 by fMRI) when individuals are processing naturalistic stimuli, such as movies. However,
6 whether children with social impairments, with or without a diagnosis of ASD, differ in
7 their neural responses to movies has not yet been investigated. In the current study, neural
8 synchrony, measured using fMRI, was examined in three groups of children aged 7 to 12,
9 who differed with respect to scores on a measure of autistic traits associated with social
10 impairment and whether or not they had been diagnosed with ASD. While watching the
11 movie ‘Despicable Me’, those diagnosed with ASD had significantly less neural
12 synchrony in areas that have been previously shown to be associated with social
13 cognition (e.g. areas related to ‘theory of mind’), and plot following (e.g. the lateral
14 prefrontal cortex), than those who did not have an ASD diagnosis. In contrast, two groups
15 who differed in their degree of social impairment, but did not have a diagnosis of ASD,
16 showed no significant differences in neural synchrony across the whole brain. These
17 results shed some light on how autistic traits may contribute to an individual’s conscious
18 experience of the world, and how, for children with ASD, that experience may differ
19 markedly from that of those without ASD.

20

21 **Keywords**

22 Autism spectrum disorder, neural synchrony, fMRI, development, Theory of Mind, social
23 impairment

24 **Introduction**

25 Autism spectrum disorder (ASD)¹ is a complex developmental condition
26 characterised by a variety of neurological and psychological features; however, the most
27 prominent feature of ASD is a marked deficit in ‘social cognition’. Social cognition refers
28 to understanding what other people believe, how they will react in situations, and why
29 they feel the way they do, and is a core element of successful human interactions. Autistic
30 individuals perform poorly on tasks that assess social cognition, such as face perception
31 (Spencer et al., 2011), perspective taking (Hamilton et al., 2009), and theory of mind
32 (ToM), or the ability to attribute mental states to oneself and others (Pedreño et al., 2017).
33 One of the most common tools to screen for deficits associated with ASD is the Social
34 Responsiveness Scale, which measures aspects of social awareness, communication, and
35 motivation (Constantino & Gruber, 2012).

36 The brains of autistic individuals often show differences when compared to those
37 of typically-developing individuals. These include structural abnormalities (Barnea-
38 Goraly et al., 2004; Brieber et al., 2007), functional differences during task-based fMRI
39 (Bölte et al., 2008; Gilbert et al., 2008; Just et al., 2007; Mason et al., 2008; Solomon et
40 al., 2009) and changes in resting-state functional connectivity (Cherkassky et al., 2006;
41 Kana et al., 2015; Monk et al., 2009; Weng et al., 2010). Many of the brain regions that
42 show differences in autistic individuals have been linked to ToM in healthy individuals,
43 including the temporal parietal junction (Saxe & Kanwisher, 2003; Saxe & Wexler,
44 2005), the medial prefrontal cortex (Hartwright et al., 2013; Krause et al., 2012; Völlm et

¹Abbreviations: ASD: Autism spectrum disorder; fMRI: functional magnetic resonance imaging; L-SRS: High SRS-2 score; SRS-2: Low SRS-2 score; H-SRS: Social responsiveness scale – revised; ToM: Theory of mind; Weschler Intelligence Scale for Children; WISC.

45 al., 2006), and the posterior superior temporal sulcus (Otsuka et al., 2009; Yang et al.,
46 2015).

47 Evidence has recently emerged that autistic adults process social information in
48 naturalistic, or ‘real-life’ contexts differently than typically-developing individuals.
49 Several studies have investigated social processing differences between those with and
50 without ASD by examining brain activity in response to watching movies (Bolton et al.,
51 2018; Byrge et al., 2015; Hasson et al., 2009; Salmi et al., 2013). Movie watching mimics
52 real-world experiences by requiring the viewer to integrate perceptual and cognitive
53 systems in order to follow the complexities of the plot. It is known that the brains of
54 healthy individuals become highly synchronized (or correlated) when viewing the same
55 movie (Hasson, Landesman, et al., 2008). This measure of synchronization across
56 different brains is termed *inter-subject correlation* and high levels of synchrony suggest
57 that individuals are experiencing the movie in much the same way. For example, Naci et
58 al. (2014) noted a high degree of synchrony in frontoparietal regions when healthy
59 individuals watched “Bang You’re Dead!” by Alfred Hitchcock and this was shown to
60 relate to how suspenseful and engaging viewers found the movie. The brains of autistic
61 adults have been shown to be less synchronized than those of typically-developing adults
62 during movie watching, and synchrony across individuals tends to be more variable
63 (Bolton et al., 2018; Byrge et al., 2015; Hasson et al., 2009; Salmi et al., 2013). However,
64 this has not been examined in autistic children.

65 Richardson et al. (2018) have shown that in typically-developing children, those
66 with poorer social cognition have reduced synchrony during movie watching in areas
67 known to be involved with ToM, suggesting that lower synchrony in these areas may also

68 be a feature of autistic children. In the current study, this question was investigated in
69 three groups of children who differed with respect to their social impairment scores and
70 whether or not they had been diagnosed with ASD. Specifically, a data-driven approach
71 was used to examine differences in the degree of inter-subject correlation during movie
72 watching in children aged 7 to 12, who had either been diagnosed with ASD, or did not
73 have ASD but their scores on the Social Responsiveness Scale – revised (SRS-2)
74 indicated social impairments related to autistic traits, or did not have ASD and had typical
75 SRS-2 scores for their age.

76 On the basis of the existing literature, it was predicted that group differences
77 would emerge in inter-subject correlation within brain networks associated with social
78 cognition. Specifically, it was hypothesized that brain activity within both frontoparietal
79 (Naci et al., 2014), and the ToM networks (Richardson et al., 2018) would be less
80 synchronized in children without ASD with higher social impairment scores than those
81 with lower social impairment scores. Furthermore, it was hypothesized that the brains of
82 children with ASD would be the least synchronized of all, based on their known
83 impairments in many aspects of social cognition.

84
85 **Methods**

86 *Dataset*

87 Data was analyzed from the Healthy Brain Network Biobank collected by the
88 Child Mind Institute (described in Alexander et al., 2017), which is an ongoing initiative
89 to collect neuroimaging, medical, and behavioural data on 10,000 participants between
90 the ages of 5 to 21. The Chesapeake Institutional Review Board approved this study.

91 Detailed information on the dataset can be found at

92 http://fcon_1000.projects.nitrc.org/indi/cmi_healthy_brain_network/

93 *Participants and data acquisition*

94 The Healthy Brain Network Biobank used a community-referred recruitment

95 model to generate a heterogeneous and transdiagnostic sample. Briefly, recruitment

96 involved advertising the study to community members, educators, local care providers,

97 and parents who were on email lists or at events. Potential participants were screened,

98 and were excluded if there were safety concerns, impairments that would interfere with

99 the study procedure (such as being nonverbal or having an IQ of less than 66), and/or

100 medical concerns that could potentially impact brain related findings (for a full

101 description, see Alexander et al., 2017). The study protocol included, where possible, the

102 acquisition of T1 weighted anatomical MRI scans and functional MRI data acquired

103 while the participants watched a ten-minute clip of ‘Despicable Me’ (from 1:02:09 to

104 1:12:09). All MRI data was collected on a 3T Siemens scanner using a Siemens 32-

105 channel head coil. Functional images were acquired with a gradient-echo planar imaging

106 pulse sequence (TR =800 ms, TE =30 ms, Flip Angle =31 degrees, whole brain coverage

107 60 slices, resolution 2.4 x 2.4 mm²). High-resolution T1-weighted MPRAGE structural

108 images were acquired in 224 sagittal (TR = 2500 ms, TE = 3.15 ms, resolution .8 x .8

109 mm²).

110 From this database, participants were included in the current analysis if they were

111 between the ages of 7-12 and both anatomical and functional MRI data had been

112 successfully acquired. Everyone included in the current study had written consent

113 obtained from their legal guardians and written assent obtained from the participant.

114 Participants were not excluded based on their handedness. All participants also had scores
115 on the Social Responsiveness Scale Revised (SRS-2), which is a measure of social
116 reciprocity and communication associated with deficits in ASD (Constantino & Gruber,
117 2012). Specifically, the SRS-2 assesses deficits associated with social awareness, social
118 cognition, social communication, social motivation, and restrictive interests and repetitive
119 behavior, and is rated by parents or caregivers of the child. A score of 59 or below
120 suggests that the child does not have social impairments associated with ASD. A score
121 above 59 is suggestive of impairments in social functioning.

122 As part of this study, all participants completed a computerized version of the
123 Schedule for Affective Disorders and Schizophrenia - Children's version (KSADS) in
124 addition to the social responsiveness scale - revised (SRS-2). The KSADS is a semi-
125 structured diagnostic interview used to assess current and past psychopathology
126 according to the DSM-IV criteria, and is rated by a research clinician or social worker
127 (Alexander et al., 2017; Kaufman et al., 1997). Participants who were suspected to have
128 ASD were then assessed in person by a clinician. These participants were assessed using
129 the Autism Diagnostic Observation Schedule – 2nd edition (Lord, Rutter, DiLavore, Risi,
130 Gotham & Bishop, 2012) and the Autism Diagnostic Interview – Revised (Rutter, Le
131 Couteur & Lord, 2003) and those who met the relevant criteria were diagnosed with
132 ASD.

133 Participants were divided into three groups: The “Low SRS-2 score” (L-SRS)
134 group included those who had an SRS-2 score ≤ 59 ; the “High SRS-2 Score (H-SRS)”
135 group included participants who had an SRS-2 score of ≥ 60 (the ASD screener cut-off),
136 but were not diagnosed with ASD; and the Autism Spectrum Group (ASD) included

137 participants who were diagnosed with ASD by a clinician as part of the HBN protocol
138 (for details, see Table 1).

139 Because the groups differed with respect to sample size, age and sex, the L-SRS
140 and H-SRS groups were resampled to produce three demographically matched sub-
141 groups. Specifically, for each participant in the ASD group, an L-SRS and an H-SRS
142 individual who had the same sex and was closest in age (to the month) were selected for
143 inclusion where possible (see Table 1). This resulted in three groups of 28 participants,
144 ensuring sufficient power for acquiring reliable inter-subject correlation results (Pajula &
145 Tohka, 2016). The matched sample was used to statistically compare the groups in the
146 whole brain and network of interest analyses. All but one of the participants in the High
147 SRS-2 group were assessed in person by a clinician. All but one participant also
148 completed the Wechsler Intelligence Scale for Children (WISC; Wechsler, 2014).

149

150

151

152

153

154

155

156

157

158

159

160 **Table 1** Participant demographics. Means, standard deviations, and ranges of the ages,
 161 SRS-2 scores, and the WISC full scale IQ, as well as the number of females and males
 162 (F/M) are displayed for each group in the full and matched sample. The full sample of
 163 participants was used to create the matched groups. The matched sample was then used
 164 for all group comparisons (i.e. the whole brain analysis, the network of interest analysis,
 165 and the percentage of synchronized cortex). Only the pairwise cluster-based analysis used
 166 the full sample of participants.

Measure	Group			Test of group differences
	L-SRS	H-SRS	ASD	
<u>Full Sample:</u>				
N	64	34	28	
Age	9.9 (1.7) 7.1 to 12.9	9.2 (1.6) 7.1 to 11.8	9.4 (1.5) 7.1 to 12.5	$F_{(2,123)} = 1.99$, $p = .141$
Sex (F/M)	27/37	13/21	2/26	$X^2_{(2)} = 11.27$, $p = .003$
SRS-2 scores	49.6(4.8)* 39 to 59	67.5 (6.0)* 60 to 84	76.6 (10.5)* 62 to 90	$F_{(2,123)} = 180.98$, $p < .001$
WISC full scale IQ	103 (17) 61 to 143	98 (16) 53 to 135	93 (18) 56 to 129	$F_{(2,122)} = 3.77$, $p = .026$
<u>Matched Sample:</u>				
N	28	28	28	
Mean Age	9.4(1.5) 7.0 to 12.6	9.6 (1.6) 7.2 to 11.8	9.4 (1.5) 7.1 to 12.5	$F_{(2,81)} = .155$, $p = .857$
Sex (F/M)	3/25	7/21	2/26	$X^2_{(2)} = 4.08$, $p = .129$
SRS-2	48.6 (4.8)* 41 to 58	67.2 (5.7)* 60 to 84	76.6 (10.5)* 62 to 90	$F_{(2,81)} = 101.79$, $p < .001$
WISC full scale IQ	103 (18) 61 to 141	96 (15) 53 to 121	93 (18) 56 to 129	$F_{(2,80)} = 2.71$, $p = .073$

167

168 *MRI pre-processing*

169 For the current study, the MRI data were preprocessed and analyzed using the
 170 Automatic Analysis (AA) toolbox (Cusack et al., 2015), SPM8, and in-house MATLAB
 171 scripts. Pre-processing of functional data included motion correction (using six motion

172 parameters: left/right, anterior/posterior, superior/inferior, chin up/down, top of head
173 left/right, nose left/right), functional and structural scans were co-registered and
174 normalized to the Montreal Neurological Institute (MNI) template. Functional data were
175 then spatially smoothed using a Gaussian filter (8 mm kernel), and low-frequency noise
176 (e.g., drift) was removed by high-pass filtering with a threshold of 1/128 Hz. The data
177 was denoised using Bandpass filter regressors, with cerebrospinal fluid, white matter
178 signals, motion parameters, their lag-3 2nd-order volterra expansion (Friston et al., 2000),
179 and "spikes" (based on mean signal variance across volumes) as nuisance regressors.

180 *Exploratory whole brain synchronization*

181 To determine the degree of synchronization separately for each group, the degree
182 of inter-subject correlation across the whole brain was calculated using a leave-one-out
183 approach using the matched sample. That is, the pre-processed time course of every voxel
184 was correlated (Pearson and then Fisher z-transformed) between each participant and the
185 mean time course of every voxel from the rest of the group (N-1). A one-sample t-test
186 was calculated on the resulting individual brain-wide correlation values. Multiple
187 comparisons were corrected with a false discovery rate (FDR) of .05 to generate group
188 maps of significantly correlated voxels. To identify where in the brain inter-subject
189 correlation differences existed between the three groups, t-tests were performed on the
190 correlation values at each voxel derived for all of the individuals within each group.
191 Multiple comparisons were corrected with an FDR of .05.

192 *Network of interest inter-subject correlation*

193 The degree of synchronization within eight previously defined functional
194 networks was calculated. To address our specific hypotheses, a map for the ToM network

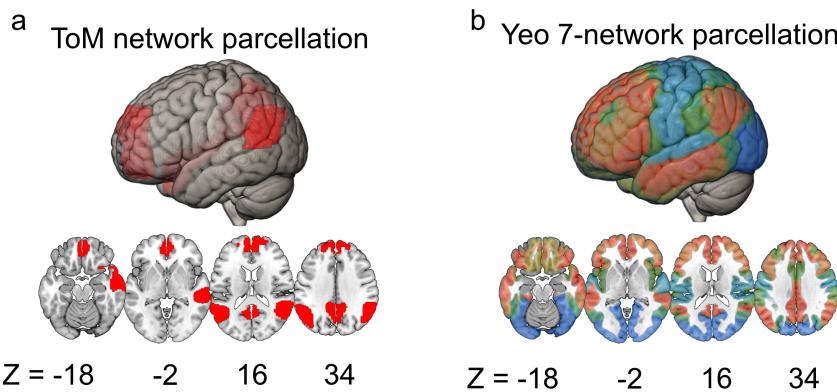
195 was used (Dufour et al., 2013) as well as the frontoparietal network from the Yeo et al.,
196 (2011) parcellation. Six additional networks (Visual, Dorsal Attention, Ventral Attention,
197 Somatomotor, Limbic, Default Mode Network) from Yeo et al. (2011) were also included
198 in an exploratory analysis to examine potential differences in other areas of the brain. The
199 8 network parcellations are displayed in Figure 1. Similar to the whole brain inter-subject
200 correlation analysis, the intra-group inter-subject correlation for each of these eight
201 networks was calculated using a leave-one-out approach using the matched sample.
202 Specifically, the time course of each network (based on the average time course of each
203 voxel within the network) for each participant was correlated with the average time
204 course of each network for the remaining participants in the group, minus that participant
205 (N-1). Finally, we used a general linear model to determine if group membership was a
206 significant predictor of intra-group synchronization across the 8 networks. The model
207 included inter-subject correlation values as the predicted variable and group as the
208 predictor variable. This was done separately for each network. The networks that showed
209 a significant effect of group were followed up with Welch t-tests (all results were FDR
210 corrected to .05).

211 To better understand the results from the intra-group analysis, the degree of inter-
212 group inter-subject correlation was then calculated, by taking the mean time course for
213 each individual in one group and correlating it with the mean of the two other groups.
214 This generated a correlation value that reflected how similar each participant's time
215 course was to the two other groups. For instance, we calculated how correlated each ASD
216 participant was to the mean of the other two groups. Finally, we calculated three separate
217 general linear models to determine if participants correlated significantly more with their

218 own group than the mean of the other two groups. The networks that showed a significant
219 effect of group were followed up with Welch t-tests (all results were FDR corrected to
220 .05).

221 *Percent synchronization across the cortex*

222 The percent of significant voxels across the cortex was calculated, for descriptive
223 purposes, to quantify the number of synchronized voxels common across all individuals
224 in each of the three matched sample groups. To calculate the total percentage of cortex
225 that was synchronized, the number of voxels that were significant per group were divided
226 by the total number of voxels in the brain. To calculate the total percentage of each
227 network that was synchronized, the number of voxels that were significant per group
228 were divided by the total number of voxels in the network of interest.



229
230 **Figure 1** Network of interests used to parcellate the brain. a) ToM network parcellation. The ToM
231 network (displayed in red) included regions in the dorsal, ventral, and lateral medial prefrontal cortex,
232 bilateral temporal parietal junction, precuneus, and right superior temporal sulcus (Dufour et al.,
233 2013). b) The seven network parcellation from Yeo et al. (2011) contained 1) the frontoparietal
234 network (displayed in orange) which includes the lateral prefrontal cortex, medial cingulate,
235 intraparietal sulcus, and inferior temporal gyrus, 2) the visual network (displayed in dark blue) which
236 encompasses the visual cortex, 3) the somatomotor network (displayed in light blue) which includes
237 the motor cortex, premotor cortex, and postcentral gyrus, 4) the dorsal attention network (displayed in
238 dark green) which includes the frontal eye fields, precentral ventral frontal cortex, middle temporal
239 area, and intraparietal sulcus, 5) the ventral attention network (displayed in light green) which includes
240 the dorsal anterior prefrontal cortex, and anterior and posterior cingulate, 6) the limbic network
241 (displayed in mustard) which includes the temporal pole and orbital frontal cortex, 7) the default mode
242 network (displayed in red) which includes the dorsal medial prefrontal cortex, temporal parietal
243 junction, postcentral gyrus, precuneus, the superior temporal sulcus, the posterior cingulate cortex, and
244 retrosplenial cortex.

245

246 *Cluster-based inter-subject correlation analysis*

247 To explore the relationship between SRS-2 scores (as a continuous variable) and
248 neural synchrony, pairwise correlations were calculated between each participant and that
249 of every other participant in the ToM and frontoparietal networks. This was done by
250 calculating the mean time course (i.e. the mean activation across each voxel in the
251 network for each time point) in both networks for each participant, and then correlating it
252 with every other participant's mean time course. Because SRS-2 scores were skewed
253 (upwards) in the ASD and H-SRS groups, this analysis included all participants (N =
254 126), rather than the smaller matched groups. These pairwise correlations were then
255 plotted in a matrix by ranking each participant by their SRS-2 score (from low to high)
256 for descriptive purposes. Finally, a clustering analysis was conducted to determine
257 whether groups of participants could be identified based solely on their neural
258 synchronization, rather than group membership or SRS-2 scores. To do this, a k-means
259 clustering algorithm was used to group together participants using the time series of
260 neural activity in the ToM and frontoparietal networks. The MATLAB evalclusters
261 function was used to identify the optimal number of clusters based on the variance in the
262 data using the Calinski-Harabasz Index computed over 1000 iterations to minimize the
263 fitting parameter. Based on the groupings generated from this cluster analysis, a logistic
264 regression analysis was computed to investigate which factors (SRS-2 total and subscale
265 scores, age, sex, and social impairment group membership) best predicted the cluster-
266 generated groupings.

267

268

269

270 **Results**

271
272 There was a total of 267 eligible participants who met the inclusion criteria (see
273 Methods). Of this sample, 141 participants were removed because of excessive motion,
274 defined as large "spikes", or significant fluctuations in signal intensity (greater than 3
275 standard deviations of the mean), in at least 25% of the data.

276 There was a significant difference between the three groups in terms of SRS-2
277 scores ($F_{(2,81)} = 101.76, p < .001$) and post-hoc t-tests showed that the H-SRS group had
278 significantly higher scores than the L-SRS group ($t_{(51.9)} = 12.96, p < .001$) and had
279 significantly lower scores than the ASD group ($t_{(42.54)} = 4.12, p < .001$). There were no
280 significant differences between the groups on the WISC full scale IQ scores ($F_{(2,80)} =$
281 $2.71, p = .073$), or any of the WISC subscales except for working memory; ($F_{(2,80)} = 3.29,$
282 $p = .042$). The ASD group had significantly lower working memory scores compared to
283 the L-SRS group ($t_{(52.10)} = 2.35, p = .023$) but not the H-SRS group ($t_{(50.24)} = 1.05, p =$
284 $.30$).

285 Differences in correlated motion within each group were examined, in order to
286 ensure that this did not inflate the inter-subject correlation results. Correlated motion was
287 calculated separately for each group, by taking each participant's 6 motion parameters for
288 each frame and correlating the time course with that of the mean of the rest of the group
289 ($N-1$). No significant differences were found between the groups in their degree of
290 correlated motion ($F_{(2,81)} = .181, p = .835$).

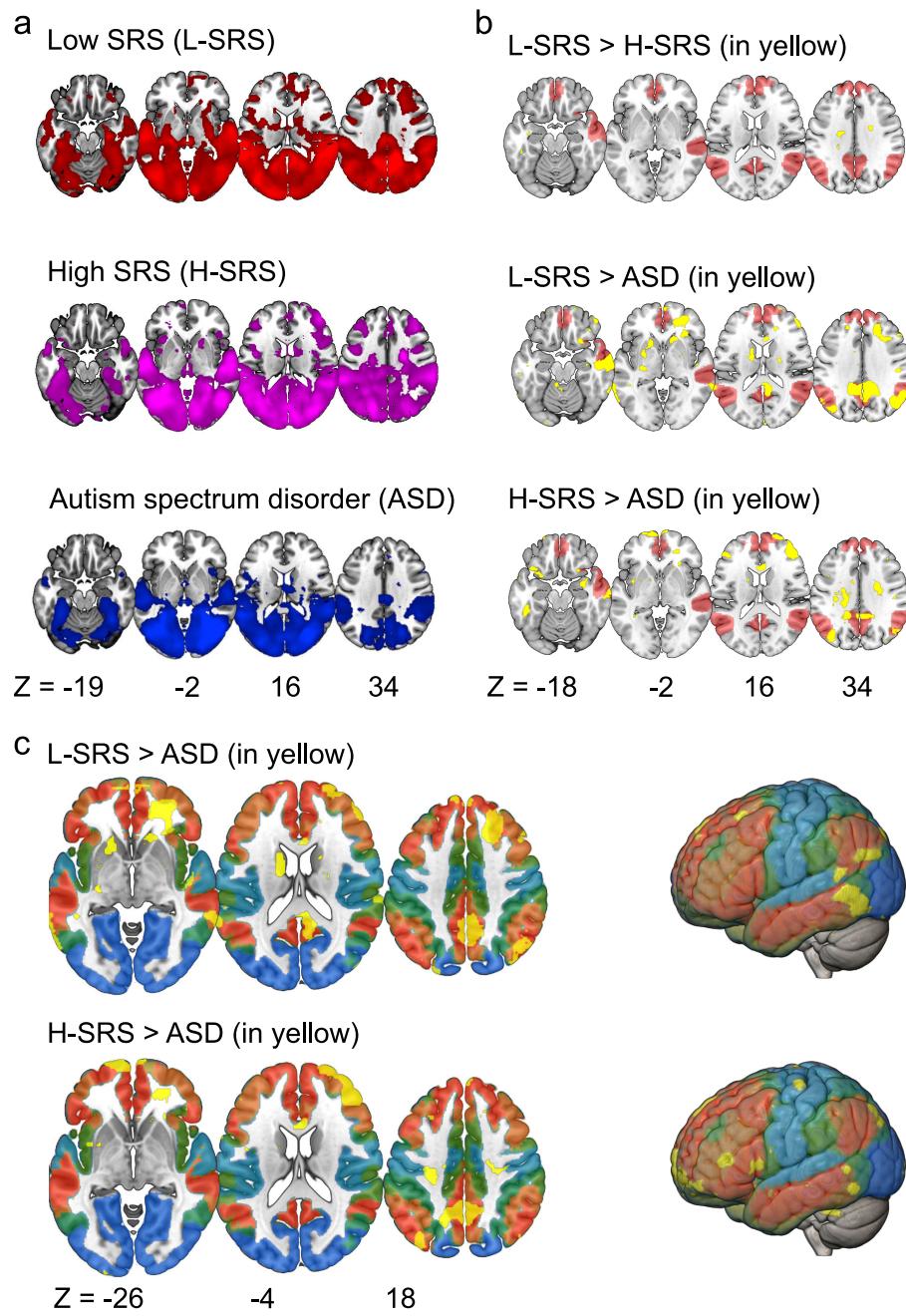
291 *Exploratory whole brain synchronization*

292 Whole brain synchronization was characterized in the three groups. All groups
293 showed significant synchronization in the auditory and visual areas (Figure 2a). In fact,

294 synchronization in these areas was stronger than in any other brain areas, replicating
295 previous inter-subject correlation findings during movie watching (Hasson et al., 2008).
296 The H-SRS and L-SRS groups also showed significant inter-subject correlation in areas
297 associated with ToM and executive processing, including parts of the right and left
298 temporal parietal junction, the precuneus, the intraparietal sulcus, the superior parietal
299 lobe, and portions of the medial and lateral prefrontal cortex. In contrast, the ASD group
300 had very little significant inter-subject correlation outside of visual and auditory areas
301 (see Figure 2a, bottom row).

302 Next, whole brain contrasts were conducted (Figure 2b) to examine whether the
303 magnitude of synchronization differed between the three groups. When the L-SRS group
304 was contrasted to the H-SRS group, only tiny areas of difference were observed after
305 multiple comparisons corrections, in the inferior temporal gyrus (MNI coordinates x, y, z
306 = -46, -37, -17, $t_{(54)} = 4.61$, $p_{\text{corrected}} = .030$), and white matter (see Figure 2b, top row).
307 The L-SRS group showed significantly greater inter-subject correlation than the ASD
308 group (Figure 2b, middle row) in the bilateral temporal parietal junction (MNI
309 coordinates (left) = -57, -61, 30, $t_{(54)} = 3.97$, $p_{\text{corrected}} = .011$, MNI coordinates (right) = 49,
310 -67, 31, $t_{(54)} = 5.07$, $p_{\text{corrected}} = .002$), precuneus (MNI coordinates = 4, -51, 41, $t_{(54)} = 4.14$,
311 $p_{\text{corrected}} = .009$), right superior temporal sulcus (MNI coordinates = 60, -11, -16, $t_{(54)} =$
312 4.47, $p_{\text{corrected}} = .005$), right hippocampus (MNI coordinates = 32, -14, -19, $t_{(54)} = 3.52$,
313 $p_{\text{corrected}} = .026$), and in regions of the lateral (MNI coordinates = 39, 54, -9, $t_{(54)} = 3.61$,
314 $p_{\text{corrected}} = .022$), and the right medial prefrontal cortex (MNI coordinates = 22, 42, 37, $t_{(54)} =$
315 3.89, $p_{\text{corrected}} = .014$). The H-SRS group had significantly greater synchronization than
316 the ASD group in the precuneus (MNI coordinates = -3, -55, 45, $t_{(54)} = 4.00$, $p_{\text{corrected}} =$

317 .017), right hippocampus (MNI coordinates = 28, -5, -21, $t_{(54)} = 3.37$, $p_{\text{corrected}} = .043$), and
318 in regions of the lateral (MNI coordinates = 46, 44, 12, $t_{(54)} = 6.30$, $p_{\text{corrected}} < .001$), and
319 medial prefrontal cortex (MNI coordinates = -5, 65, -7, $t_{(54)} = 4.27$, $p_{\text{corrected}} = .012$)
320 (Figure 2b, bottom row).

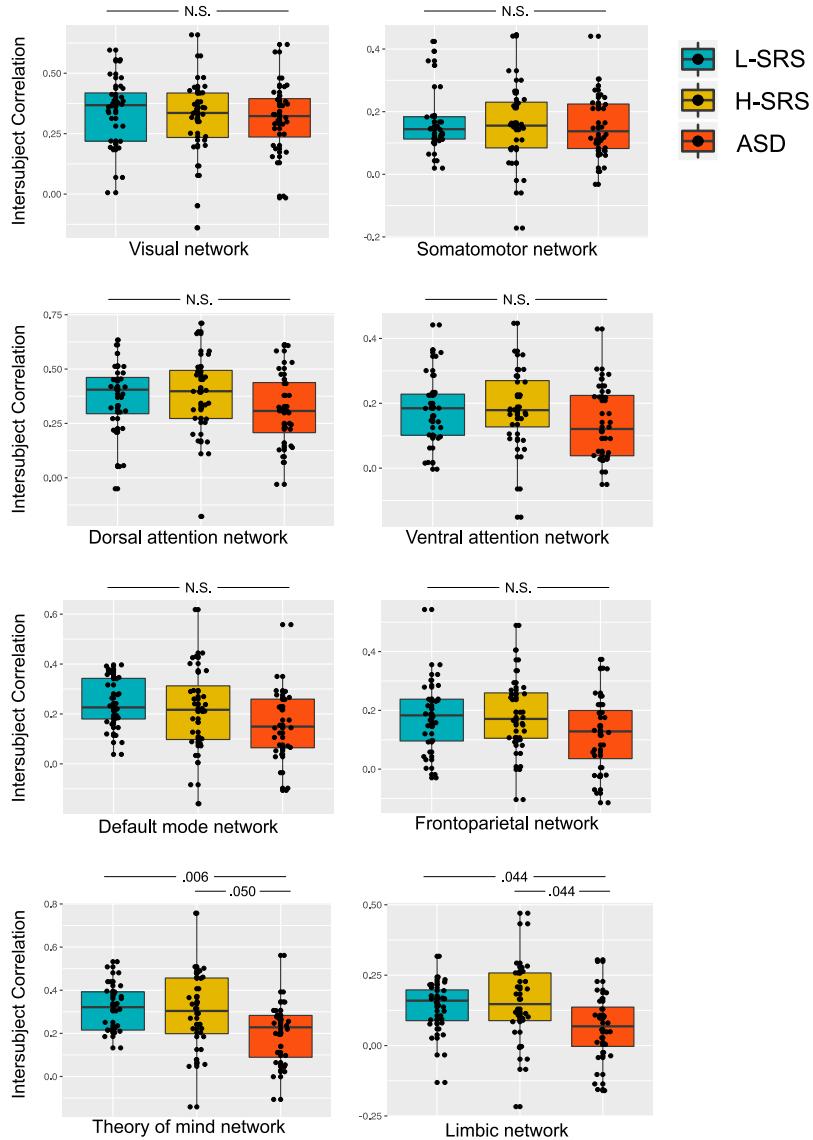


321

322 **Figure 2** Exploratory whole brain inter-subject correlation analysis. **a)** Voxels displayed in red
323 showed significant inter-subject correlation during movie watching in the L-SRS group. Voxels
324 displayed in violet showed significant inter-subject correlation in the H-SRS group. Voxels displayed
325 in blue showed significant inter-subject correlation in the ASD group. All p values were FDR
326 corrected to an alpha of .05. **b)** Whole brain contrasts were calculated by conducting one-tailed t-tests
327 on the inter-subject correlation values between each group (p values corrected to an FDR of .05).
328 Voxels displayed in yellow showed significantly greater inter-subject correlation values based on this
329 contrast, voxels displayed in red show the ToM network parcellation. **c)** Voxels displayed in yellow
330 showed significantly greater inter-subject correlation values based on the same contrast displayed in **b**,
331 overlaid on top of the Yeo et al. (2011) 7-network parcellation. (Frontoparietal = orange, Visual =
332 dark blue, Somatomotor = light blue, Dorsal attention = dark green, Ventral attention = light green,
333 Limbic = mustard, Default mode = red).

334
335 *Network based synchronization*

336 Group differences in the magnitude of intra-group synchronization revealed a
337 main effect of group in the ToM ($F_{(2,81)} = 4.94, p = .009$) and the limbic ($F_{(2,81)} = 3.93, p$
338 = .023) networks (Figure 3), but not in any of the others examined, including the
339 frontoparietal network ($F_{(2,81)} = 2.02, p = .140$, Cohen's d ranged from .037 to .476).
340 Post-hoc analyses of neural synchronization revealed that the ASD group had
341 significantly lower inter-subject correlation values compared to the L-SRS group within
342 the ToM ($t_{(50.11)} = 3.50, p_{\text{corrected}} = .006$, Cohen's d = .934) and limbic networks ($t_{(50.00)} =$
343 $2.48, p_{\text{corrected}} = .044$, Cohen's d = .664). They also had significantly lower inter-subject
344 correlation values compared to the H-SRS group in the limbic network ($t_{(50.21)} = 2.18,$
345 $p_{\text{corrected}} = .044$, Cohen's d = .631), although differences in inter-subject correlation just
346 failed to meet the corrected alpha level in the ToM network ($t_{(52.33)} = 2.36, p_{\text{corrected}} =$
347 $.0504$, Cohen's d = .584). Moreover, no significant differences in inter-subject correlation
348 were observed between the L-SRS and H-SRS groups within the ToM ($t_{(45.72)} = .488,$
349 $p_{\text{corrected}} = .628$, Cohen's d = .130) or limbic networks ($t_{(45.21)} = .417, p_{\text{corrected}} = .628$,
350 Cohen's d = .111).



351

352 **Figure 3** Intra-group network of interest analysis. Mean inter-subject correlation, based on the leave
353 one out correlation analysis conducted separately for each group, is displayed as dots for each
354 participant in the eight networks. Boxplots indicate the median inter-subject correlation value and
355 interquartile range for each group (blue = L-SRS, yellow = H-SRS, red = ASD). The ASD group had
356 significantly lower inter-subject correlation in the limbic and ToM networks compared to the L-SRS
357 group. The ASD group also had significantly lower inter-subject correlation in the limbic network
358 compared to the H-SRS group, while in the ToM network this difference narrowly missed statistical
359 significance (corrected p value = .0504). The groups did not differ significantly in any of the six other
360 networks.

361

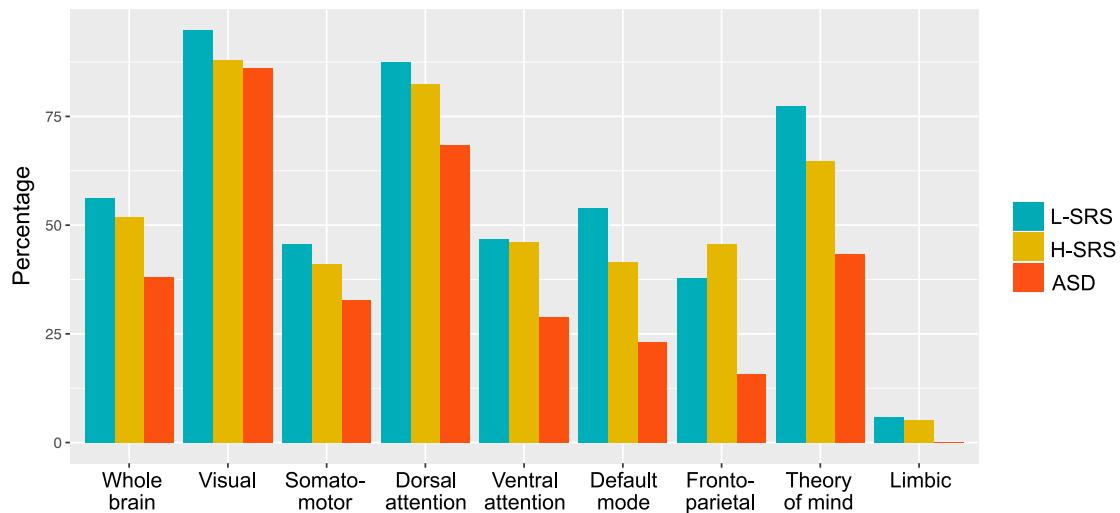
362 An inter-group inter-subject correlation network analysis was performed to

363 investigate whether individuals in one group had significantly greater neural

364 synchronization with their own group than that of the other two groups. The results
365 revealed that the degree of inter-subject correlation was not significantly different
366 between any of the groups in any of the examined networks, including the frontoparietal
367 and ToM networks.

368 *Percent synchronization across the cortex*

369 When looking at the percentage of synchronized voxels across the whole brain,
370 the ASD group had nearly one-third less (38%) than the L-SRS (56%) and H-SRS (52%)
371 groups (see Figure 4). The percentage of significant voxels in each of the eight networks
372 of interest was also calculated (see Figure 4). The difference in percentage across the
373 whole brain between the groups was not accounted for by less synchronization in any one
374 network; rather, the ASD group had fewer synchronized voxels in every network,
375 including in the ToM and frontoparietal networks.



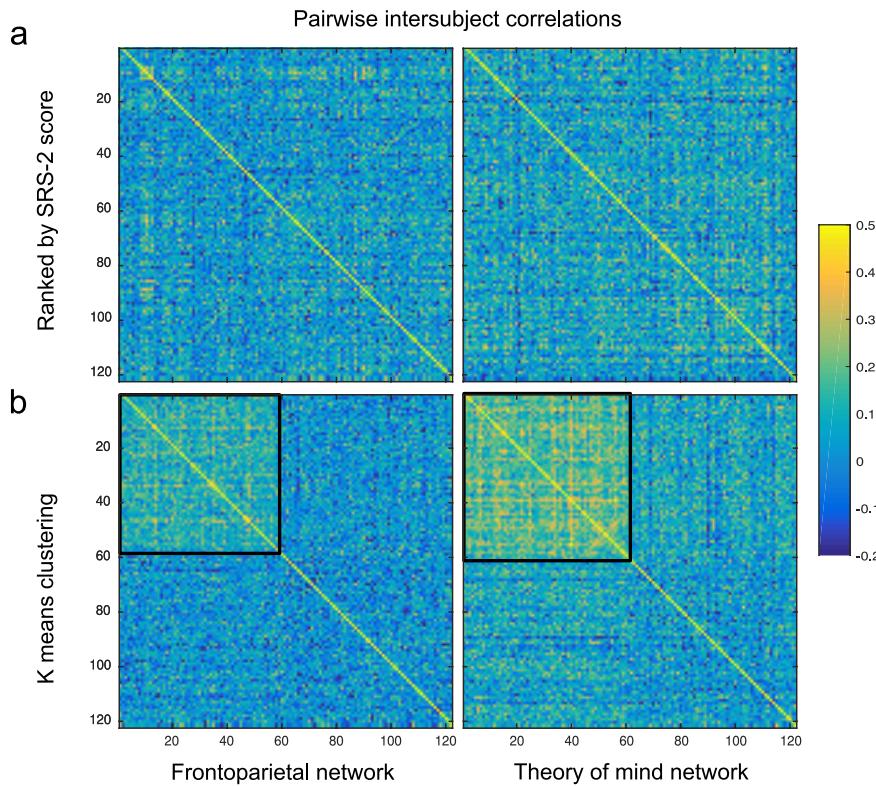
376
377 **Figure 4** Percentage of correlated voxels. The percent of significant voxels across the cortex was
378 calculated, for descriptive purposes, to quantify the number of synchronized voxels common across all
379 individuals in each of the three matched sample groups. This was calculated by dividing the number of
380 voxels with significant inter-subject correlation by the total number of voxels in the whole brain or
381 network for each group separately (blue = L-SRS, yellow = H-SRS, red = ASD).
382
383
384
385

386

387

388 *Cluster-based inter-subject correlation analysis*

389 To explore whether SRS-2 scores predicted inter-subject correlation values when
390 used as a continuous measure (instead of a categorical variable), pairwise inter-subject
391 correlations were calculated between each participant (N=126) in the frontoparietal and
392 ToM networks. The entire sample was used so that the SRS-2 scores were normally
393 distributed and to increase statistical power. Pairwise correlations were conducted to
394 reduce any influence the groupings may have had on the mean time course originally
395 used to calculate inter-subject correlation. For instance, if those with low SRS-2 scores
396 and those with high SRS-2 scores both correlated with their own group to a similar
397 degree, but the pattern of activations was different, using these groupings would
398 obfuscate any differences. For descriptive purposes, the matrix of pairwise correlation
399 values was plotted by ranking each participant by their SRS-2 score, from low to high
400 (see Figure 5a). A k-means clustering analysis was conducted on the pairwise correlations
401 to explore potential factors that predicted groups of participants who have the most
402 similar degree of synchrony in these two networks. The best fit was achieved by dividing
403 the data into two clusters in both the frontoparietal and ToM networks; cluster 1 included
404 individuals with similar neural responses to the movie (large positive correlations) and
405 cluster 2 included individuals with unrelated neural responses to the movie (Figure b).
406 Moreover, there was also a large overlap between the participants who were in cluster 1
407 in the ToM and frontoparietal networks. Specifically, of the 58 participants who had high
408 similarity in the ToM network (cluster 1), 45 of them also had high similarity in the
409 frontoparietal network.



410

411 **Figure 5** Pairwise inter-subject correlations. Yellow squares indicate a higher positive correlation (i.e.
412 high similarity in time series), blue squares indicated a low or negative correlation (i.e. low similarity
413 in time series). **a)** Pairwise correlations in time series in the frontoparietal and ToM networks between
414 each pair of participants are ordered by SRS-2 scores (from low to high). **b)** Pairwise correlations in
415 time series are ordered based on the K-means analysis in the frontoparietal and ToM networks. Black
416 boxes show cluster 1 (the high similarity group) for each network.
417

418

418 Logistic regression was run to determine whether the probability of being in
419 cluster 1 versus cluster 2 could be predicted by age, sex, full scale IQ, SRS-2 total and
420 subscales, or group membership (i.e., L-SRS, H-SRS and ASD). None of these factors
421 significantly predicted cluster membership in the frontoparietal network. However, in the
422 ToM network, group membership significantly predicted cluster membership. Cluster 1
423 comprised 35 participants (60%) in the L-SRS group, 17 individuals (29%) from the H-
424 SRS groups, and 6 individuals (10%) who were diagnosed with ASD. In contrast, cluster
425 2 consisted of 29 individuals (45%) from the L-SRS group, 17 individuals (20%) from

426 the H-SRS group, and 22 individuals (35%) diagnosed with ASD. There were
427 significantly more participants from the ASD group in cluster 2 than in cluster 1 in the
428 ToM network ($\chi^2_{(1)} = 7.5$, $p = .006$), while there was no significant difference in the
429 number of H-SRS participants between the two clusters ($\chi^2_{(1)} = .11$, $p = .73$), and
430 although there were more L-SRS participants in cluster 1, this difference did not reach
431 significance ($\chi^2_{(1)} = 3.25$, $p = .072$).

432
433 **Discussion**
434

435 In the current study, a group of ASD participants had significantly less neural
436 synchronization when watching a movie compared to the L-SRS and H-SRS groups
437 across the whole brain, including the ToM and limbic networks, as well as the lateral and
438 medial prefrontal cortex. These regions have been shown previously to be associated with
439 elements of ‘plot following’ during movie watching (Hasson, Furman, et al., 2008;
440 Hasson, Landesman, et al., 2008; Naci et al., 2014; Nguyen et al., 2019), suggesting that
441 the children in the ASD group were experiencing the movie qualitatively differently than
442 the participants in the other two groups. These results, in particular the fact that the ToM
443 network was less synchronized in the ASD group, are intriguing given that regions within
444 this network are associated with social cognition (Dufour et al., 2013; Mills et al., 2014;
445 Richardson et al., 2018; Rilling et al., 2004), which is known to be affected in ASD
446 (Hamilton et al., 2009; Pedreño et al., 2017; Spencer et al., 2011). While aspects of social
447 cognition are usually discussed in the context of inter-personal relationships, they are also
448 essential components of movie-watching, allowing one to become immersed in the plot
449 by taking the perspective of the characters appropriately, understanding their motives,
450 and following their verbal and nonverbal communication cues. Yeshurun et al. (2017)

451 have reported previously that manipulating an individual's understanding of a plot
452 reduces neural synchrony in ToM regions, including the precuneus, temporal parietal
453 junction, and medial prefrontal cortex. Thus, these findings support the idea that autistic
454 children process social stimuli in a distinct way, as they have different neural responses in
455 the ToM network during a movie, when compared to children without ASD.

456 It is also interesting that participants in the ASD group had significantly less
457 synchrony in the lateral prefrontal cortex, a region within the frontoparietal network,
458 when compared to those in the other two groups. Understanding a complex narrative
459 (such as a movie's plot) requires a viewer to remember previous events, pay attention to
460 what is currently happening, make predictions about the future consequences of current
461 events, and integrate this information over time, all of which depends on frontoparietal
462 executive processing (Naci et al., 2014). In previous studies, reduced synchrony in this
463 network has been associated with 'losing the plot' during deep sedation (Naci et al.,
464 2018), and in patients with severe brain damage (Naci et al., 2014). Thus, this decrease in
465 inter-subject correlation in the lateral prefrontal cortex may suggest that participants in
466 the ASD group are also failing to grasp elements of the plot in the way that the other
467 participants do.

468 Despite finding that inter-subject correlation was reduced in prefrontal regions
469 using a whole brain analysis, no differences in the degree of inter-subject correlation
470 were found in the frontoparietal network when a network of interest analysis was used.
471 One potential reason is that the parcellation used for the frontoparietal network was based
472 on adult data and may not accurately capture this network in children. Previous work has
473 shown that the frontoparietal network continues to develop into early adulthood (Baum et

474 al., 2017; Peters et al., 2016), and so the parcellation masks from Yeo et al. (2011) may
475 have led us to average neural activity from regions that are not yet fully integrated in
476 children.

477 While not part of our hypotheses, it is interesting that the ASD group showed less
478 inter-subject correlation in the right hippocampus in the whole brain analysis as well as in
479 the limbic network, when examined using the parcellation by Yeo et al. (2011). Similar
480 findings have been reported in autistic adults watching movies (Byrge et al., 2015).
481 Moreover, Chen et al. (2017) found that, in healthy adults, the degree of inter-subject
482 correlation within the hippocampus during movie watching predicted events that were
483 later recalled, although this has not been examined during development. Nevertheless,
484 long-term memory deficits have been reported in ASD; specifically, autistic individuals
485 perform worse on episodic, but not semantic, memory tasks (Crane & Goddard, 2008;
486 Lind, 2010)

487 Contrary to our hypothesis, no meaningful differences in neural synchrony were
488 found between the L-SRS and H-SRS groups. This contrasts with the results of
489 Richardson et al., (2018) who found that social cognition in typically-developing children
490 was related to the degree of inter-subject correlation within the ToM network during
491 movie-watching. One potential reason for this difference is that Richardson et al., (2018)
492 calculated inter-subject correlation based on how similar each child's time course was to
493 a group of adults watching the same movie, whereas in the current study, inter-subject
494 correlation was calculated by correlating each participant's time course to the mean of
495 their own group. Moreover, the measure of social cognition used by Richardson et al.
496 (2018) focused specifically on comprehension of a social narrative, which has many

497 things in common with how people follow the plot of a movie. It is perhaps not surprising
498 then, that the two things correlated. In the current study, a measure of social impairment
499 was used – the SRS-2, which measures an individual's motivation to engage in social
500 interactions, their use of social communication, and their ability to understand social cues
501 (Constantino & Gruber, 2012). Thus, while the H-SRS and L-SRS groups differed in
502 terms of their social impairments as measured by the SRS-2 scale, these mechanisms may
503 be unrelated, or only moderately related, to those that are involved in plot following.
504 Moreover, it is also possible that creating categorical groups based on the SRS-2 scores
505 may have obscured subtle differences in individuals with differing levels of social
506 impairment. To investigate this possibility, the exploratory pairwise correlation analysis
507 was conducted, which found that SRS-2 scores as a continuous measure did not predict
508 whether participants had similar patterns of neural activity in the ToM or frontoparietal
509 networks. Taken together, these results suggest that it is only when social impairment is
510 in the clinical range, as is seen in ASD, that differences in conscious processing of
511 naturalistic stimuli emerge.

512 As a group, autistic participants had less inter-subject correlation compared to
513 those without ASD, but these differences did not apply uniformly to each individual. The
514 clustering analysis indicated that the majority of ASD participants had low similarity in
515 their time courses compared to all other participants. However, six out of 28 of those
516 diagnosed with ASD clustered with the 'high similarity' group (comprising about 10% of
517 the group) according to their synchronization in the ToM network. Using a similar
518 clustering analysis, Byrge et al., (2015) found that in a sample of 17 high functioning
519 autistic adults, five showed idiosyncratic patterns of inter-subject correlation compared to

520 typically-developing individuals, while the other 12 clustered with the control group.
521 Moreover, they found that these five individuals were significantly worse than the control
522 group and the other 12 ASD participants, when asked to explain elements of a movie plot.
523 Together, these findings suggest that lower synchronization during movie-watching may
524 be common, but not a uniform characteristic of either autistic children or adults. Indeed,
525 heterogeneity in clinical features, cognitive profiles, and differing genetic and
526 environmental risk factors has plagued research in ASD (Betancur, 2011; Jeste &
527 Geschwind, 2014; Lenroot & Yeung, 2013). For example, within the neuroimaging
528 literature, some studies have reported underconnectivity across the brains of autistic
529 individuals (Cherkassky et al., 2006; Di Martino et al., 2014; von dem Hagen et al.,
530 2013), while others find hyperconnectivity (Supekar et al., 2013; Uddin et al., 2010,
531 2013).

532 Finally, it is important to keep in mind the exploratory nature of the current study
533 when interpreting these findings. This is a step towards a better understanding of how
534 children with and without ASD process naturalistic stimuli, but replication and further
535 investigation is needed to better understand the nature of the differences observed. For
536 instance, one potential mechanism underlying our results could be that participants in the
537 ASD group had more variable neural responses to the movie. However, it would be
538 valuable for future studies to directly examine if more variable neural responses to
539 movies are driving reduced neural synchronization in those diagnosed with ASD.
540 Additionally, a major limitation of this study is that no memory test, or measure of how
541 well the movie clip was understood, was collected. A behavioral measure of movie
542 comprehension may help to explain the nature of the neural differences observed in this

543 study. It is possible that individuals were attending to different features of the movie,
544 which has been shown to influence the degree of neural synchrony (Nguyen et al., 2019),
545 although previous work has confirmed that movies similar to ‘Despicable me’ maintain
546 the viewers’ attention (Hasson, Landesman, et al., 2008; Naci et al., 2015). It is also
547 unlikely that participants were asleep during the movie, as most of the visual network was
548 synchronized across the three groups during the movie, which is not observed when
549 individuals are sedated (Naci et al., 2018).

550

551 **Conclusion**

552 In sum, the current results suggest that autistic children, as a group, process
553 movies in a unique way compared to those without ASD. Interestingly, a minority of
554 these children had time courses that were highly correlated with a group of children
555 without ASD in the ToM network. Future research should investigate factors that underlie
556 this heterogeneity, as this may be one avenue to better understand how autistic
557 individuals process the world around them.

558

559 **Author contributions**

560 Conceptualization, writing–reviewing and editing, K.M.L., R.A.S, A.M.O, B.S.;
561 Methodology, Formal analysis, K.M.L., B.S.; writing–original draft preparation, K.M.L.,
562 B.S., A.M.O; funding acquisition, A.M.O., B.S

563

564 **Acknowledgements**

565 The authors thank the Child and Mind Institute for designing and collecting data for the
566 Healthy Brain Biobank.

567

568 **Funding**

569 This research was funded by the Canada Excellence Research Chairs Program (#215063),
570 the Canadian Institutes of Health Research (#408004), and the Natural Sciences and
571 Engineering Research Council of Canada (#390057).

572

573 **Conflict of interest**

574 The authors have no conflicts of interest to declare.

575

576 **References**

577 Alexander, L. M., Escalera, J., Ai, L., Andreotti, C., Febre, K., Mangone, A., ... & Litke,
578 S. (2017). The Healthy Brain Network Biobank: An open resource for
579 transdiagnostic research in pediatric mental health and learning disorders.
580 *Scientific data*, 4, 170181. <https://doi.org/10.1038/sdata.2017.181>.

581 Barnea-Goraly, N., Kwon, H., Menon, V., Eliez, S., Lotspeich, L., & Reiss, A. L. (2004).
582 White matter structure in autism: Preliminary evidence from diffusion tensor
583 imaging. *Biological Psychiatry*, 55(3), 323–326.
584 <https://doi.org/10.1016/j.biopsych.2003.10.022>

585 Baum, G. L., Ceric, R., Roalf, D. R., Betzel, R. F., Moore, T. M., Shinohara, R. T., Kahn,
586 A. E., Vandekar, S. N., Rupert, P. E., Quarmley, M., Cook, P. A., Elliott, M. A.,
587 Ruparel, K., Gur, R. E., Gur, R. C., Bassett, D. S., & Satterthwaite, T. D. (2017).
588 Modular Segregation of Structural Brain Networks Supports the Development of
589 Executive Function in Youth. *Current Biology*, 27(11), 1561-1572.e8.
590 <https://doi.org/10.1016/j.cub.2017.04.051>

591 Betancur, C. (2011). Etiological heterogeneity in autism spectrum disorders: More than
592 100 genetic and genomic disorders and still counting. *Brain Research*, 1380, 42–
593 77. <https://doi.org/10.1016/j.brainres.2010.11.078>

594 Bölte, S., Hubl, D., Dierks, T., Holtmann, M., & Poustka, F. (2008). An fMRI-study of
595 locally oriented perception in autism: Altered early visual processing of the block
596 design test. *Journal of Neural Transmission*, 115(3), 545–552.
597 <https://doi.org/10.1007/s00702-007-0850-1>

598 Bolton, T. A. W., Jochaut, D., Giraud, A.-L., & Ville, D. V. D. (2018). Brain dynamics in
599 ASD during movie-watching show idiosyncratic functional integration and
600 segregation. *Human Brain Mapping*, 39(6), 2391–2404.
601 <https://doi.org/10.1002/hbm.24009>

602 Brieber, S., Neufang, S., Bruning, N., Kamp-Becker, I., Remschmidt, H., Herpertz-
603 Dahlmann, B., Fink, G. R., & Konrad, K. (2007). Structural brain abnormalities in
604 adolescents with autism spectrum disorder and patients with attention
605 deficit/hyperactivity disorder. *Journal of Child Psychology and Psychiatry*,
606 48(12), 1251–1258. <https://doi.org/10.1111/j.1469-7610.2007.01799.x>

607 Byrge, L., Dubois, J., Tyszka, J. M., Adolphs, R., & Kennedy, D. P. (2015). Idiosyncratic
608 Brain Activation Patterns Are Associated with Poor Social Comprehension in
609 Autism. *Journal of Neuroscience*, 35(14), 5837–5850.
610 <https://doi.org/10.1523/JNEUROSCI.5182-14.2015>

611 Chen, J., Leong, Y. C., Honey, C. J., Yong, C. H., Norman, K. A., & Hasson, U. (2017).
612 Shared memories reveal shared structure in neural activity across individuals.
613 *Nature Neuroscience*, 20(1), 115–125. <https://doi.org/10.1038/nn.4450>

614 Cherkassky, V. L., Kana, R. K., Keller, T. A., & Just, M. A. (2006). Functional
615 connectivity in a baseline resting-state network in autism. *NeuroReport*, 17(16),
616 1687–1690. <https://doi.org/10.1097/01.wnr.0000239956.45448.4c>

617 Constantino, J. N., & Gruber, C. P. (2012). *Social Responsiveness Scale—Second Edition*
618 (*SRS-2*). Western Psychological Services.

619 Crane, L., & Goddard, L. (2008). Episodic and Semantic Autobiographical Memory in
620 Adults with Autism Spectrum Disorders. *Journal of Autism and Developmental
621 Disorders*, 38(3), 498–506. <https://doi.org/10.1007/s10803-007-0420-2>

622 Cusack, R., Vicente-Grabovetsky, A., Mitchell, D. J., Wild, C. J., Auer, T., Linke, A. C.,
623 & Peelle, J. E. (2015). Automatic analysis (aa): Efficient neuroimaging workflows
624 and parallel processing using Matlab and XML. *Frontiers in Neuroinformatics*, 8.
625 <https://doi.org/10.3389/fninf.2014.00090>

626 Di Martino, A., Yan, C.-G., Li, Q., Denio, E., Castellanos, F. X., Alaerts, K., Anderson, J.
627 S., Assaf, M., Bookheimer, S. Y., Dapretto, M., Deen, B., Delmonte, S., Dinstein,
628 I., Ertl-Wagner, B., Fair, D. A., Gallagher, L., Kennedy, D. P., Keown, C. L.,
629 Keysers, C., ... Milham, M. P. (2014). The autism brain imaging data exchange:
630 Towards a large-scale evaluation of the intrinsic brain architecture in autism.
631 *Molecular Psychiatry*, 19(6), 659–667. <https://doi.org/10.1038/mp.2013.78>

632 Dufour, N., Redcay, E., Young, L., Mavros, P. L., Moran, J. M., Triantafyllou, C.,
633 Gabrieli, J. D. E., & Saxe, R. (2013). Similar Brain Activation during False Belief
634 Tasks in a Large Sample of Adults with and without Autism. *PLoS ONE*, 8(9).
635 <https://doi.org/10.1371/journal.pone.0075468>

636 Friston, K. J., Mechelli, A., Turner, R., & Price, C. J. (2000). Nonlinear responses in
637 fMRI: the Balloon model, Volterra kernels, and other hemodynamics.
638 *NeuroImage*, 12(4), 466-477. <https://doi.org/10.1006/nimg.2000.0630>

639 Gilbert, S. J., Bird, G., Brindley, R., Frith, C. D., & Burgess, P. W. (2008). Atypical
640 recruitment of medial prefrontal cortex in autism spectrum disorders: An fMRI

641 study of two executive function tasks. *Neuropsychologia*, 46(9), 2281–2291.

642 <https://doi.org/10.1016/j.neuropsychologia.2008.03.025>

643 Hamilton, A. F., Brindley, R., & Frith, U. (2009). Visual perspective taking impairment
644 in children with autistic spectrum disorder. *Cognition*, 113(1), 37–44.
645 <https://doi.org/10.1016/j.cognition.2009.07.007>

646 Hartwright, C. E., Apperly, I. A., & Hansen, P. C. (2013). Representation, Control, or
647 Reasoning? Distinct Functions for Theory of Mind within the Medial Prefrontal
648 Cortex. *Journal of Cognitive Neuroscience*, 26(4), 683–698.
649 https://doi.org/10.1162/jocn_a_00520

650 Hasson, U., Avidan, G., Gelbard, H., Vallines, I., Harel, M., Minshew, N., & Behrmann,
651 M. (2009). Shared and idiosyncratic cortical activation patterns in autism revealed
652 under continuous real-life viewing conditions. *Autism Research*, 2(4), 220–231.
653 <https://doi.org/10.1002/aur.89>

654 Hasson, U., Furman, O., Clark, D., Dudai, Y., & Davachi, L. (2008). Enhanced
655 Intersubject Correlations during Movie Viewing Correlate with Successful
656 Episodic Encoding. *Neuron*, 57(3), 452–462.
657 <https://doi.org/10.1016/j.neuron.2007.12.009>

658 Hasson, U., Landesman, O., Knappmeyer, B., Vallines, I., Rubin, N., & Heeger, D. J.
659 (2008). Neurocinematics: The Neuroscience of Film. *Projections*, 2(1), 1–26.
660 <https://doi.org/10.3167/proj.2008.020102>

661 Jeste, S. S., & Geschwind, D. H. (2014). Disentangling the heterogeneity of autism
662 spectrum disorder through genetic findings. *Nature Reviews. Neurology*, 10(2),
663 74–81. <https://doi.org/10.1038/nrneurol.2013.278>

664 Just, M. A., Cherkassky, V. L., Keller, T. A., Kana, R. K., & Minshew, N. J. (2007).
665 Functional and Anatomical Cortical Underconnectivity in Autism: Evidence from
666 an fMRI Study of an Executive Function Task and Corpus Callosum
667 Morphometry. *Cerebral Cortex*, 17(4), 951–961.
668 <https://doi.org/10.1093/cercor/bhl006>

669 Kana, R. K., Maximo, J. O., Williams, D. L., Keller, T. A., Schipul, S. E., Cherkassky, V.
670 L., Minshew, N. J., & Just, M. A. (2015). Aberrant functioning of the theory-of-
671 mind network in children and adolescents with autism. *Molecular Autism*, 6(1),
672 59. <https://doi.org/10.1186/s13229-015-0052-x>

673 Kaufman, J., Birmaher, B., Brent, D., Rao, U. M. A., Flynn, C., Moreci, P., ... & Ryan, N.
674 (1997). Schedule for affective disorders and schizophrenia for school-age
675 children-recent and lifetime version (K-SADS-PL): Initial reliability and validity
676 data. *Journal of the American Academy of Child & Adolescent Psychiatry*, 36(7),
677 980-988. <https://doi.org/10.1097/00004583-199707000-00021>.

678 Krause, L., Enticott, P. G., Zangen, A., & Fitzgerald, P. B. (2012). The role of medial
679 prefrontal cortex in theory of mind: A deep rTMS study. *Behavioural Brain
680 Research*, 228(1), 87–90. <https://doi.org/10.1016/j.bbr.2011.11.037>

681 Lenroot, R. K., & Yeung, P. K. (2013). Heterogeneity within Autism Spectrum
682 Disorders: What have We Learned from Neuroimaging Studies? *Frontiers in
683 Human Neuroscience*, 7, 733. <https://doi.org/10.3389/fnhum.2013.00733>

684 Lind, S. E. (2010). Memory and the self in autism: A review and theoretical framework.
685 *Autism*, 14(5), 430–456. <https://doi.org/10.1177/1362361309358700>

686 Lord, C., Rutter, M., DiLavore, P., Risi, S., Gotham, K., & Bishop, S. (2012). Autism
687 diagnostic observation schedule–2nd edition (ADOS-2). *Los Angeles, CA:*
688 *Western Psychological Corporation.*

689 Mason, R. A., Williams, D. L., Kana, R. K., Minshew, N., & Just, M. A. (2008). Theory
690 of Mind disruption and recruitment of the right hemisphere during narrative
691 comprehension in autism. *Neuropsychologia*, 46(1), 269–280.
692 <https://doi.org/10.1016/j.neuropsychologia.2007.07.018>

693 Mills, K. L., Lalonde, F., Clasen, L. S., Giedd, J. N., & Blakemore, S.J. (2014).
694 Developmental changes in the structure of the social brain in late childhood and
695 adolescence. *Social Cognitive and Affective Neuroscience*, 9(1), 123–131.
696 <https://doi.org/10.1093/scan/nss113>

697 Monk, C. S., Peltier, S. J., Wiggins, J. L., Weng, S.-J., Carrasco, M., Risi, S., & Lord, C.
698 (2009). Abnormalities of intrinsic functional connectivity in autism spectrum
699 disorders,. *NeuroImage*, 47(2), 764–772.
700 <https://doi.org/10.1016/j.neuroimage.2009.04.069>

701 Naci, L., Cusack, R., Anello, M., & Owen, A. M. (2014). A common neural code for
702 similar conscious experiences in different individuals. *Proceedings of the
703 National Academy of Sciences*, 111(39), 14277–14282.
704 <https://doi.org/10.1073/pnas.1407007111>

705 Naci, L., Haugg, A., MacDonald, A., Anello, M., Houldin, E., Naqshbandi, S., Gonzalez-
706 Lara, L. E., Arango, M., Harle, C., Cusack, R., & Owen, A. M. (2018). Functional
707 diversity of brain networks supports consciousness and verbal intelligence.
708 *Scientific Reports*, 8. <https://doi.org/10.1038/s41598-018-31525-z>

709 Nguyen, M., Vanderwal, T., & Hasson, U. (2019). Shared understanding of narratives is
710 correlated with shared neural responses. *NeuroImage*, 184, 161–170.
711 <https://doi.org/10.1016/j.neuroimage.2018.09.010>

712 Otsuka, Y., Osaka, N., Ikeda, T., & Osaka, M. (2009). Individual differences in the
713 theory of mind and superior temporal sulcus. *Neuroscience Letters*, 463(2), 150–
714 153. <https://doi.org/10.1016/j.neulet.2009.07.064>

715 Pajula, J., & Tohka, J. (2016). How Many Is Enough? Effect of Sample Size in Inter-
716 Subject Correlation Analysis of fMRI. *Computational Intelligence and*
717 *Neuroscience*. <https://doi.org/10.1155/2016/2094601>

718 Pedreño, C., Pousa, E., Navarro, J. B., Pàmias, M., & Obiols, J. E. (2017). Exploring the
719 Components of Advanced Theory of Mind in Autism Spectrum Disorder. *Journal*
720 *of Autism and Developmental Disorders*, 47(8), 2401–2409.
721 <https://doi.org/10.1007/s10803-017-3156-7>

722 Peters, S., Van Duijvenvoorde, A. C. K., Koolschijn, P. C. M. P., & Crone, E. A. (2016).
723 Longitudinal development of frontoparietal activity during feedback learning:
724 Contributions of age, performance, working memory and cortical thickness.
725 *Developmental Cognitive Neuroscience*, 19, 211–222.
726 <https://doi.org/10.1016/j.dcn.2016.04.004>

727 Richardson, H., Lisandrelli, G., Riobueno-Naylor, A., & Saxe, R. (2018). Development
728 of the social brain from age three to twelve years. *Nature Communications*, 9(1),
729 1027. <https://doi.org/10.1038/s41467-018-03399-2>

730 Rilling, J. K., Sanfey, A. G., Aronson, J. A., Nystrom, L. E., & Cohen, J. D. (2004). The
731 neural correlates of theory of mind within interpersonal interactions. *NeuroImage*,
732 22(4), 1694–1703. <https://doi.org/10.1016/j.neuroimage.2004.04.015>

733 Rutter, M., Le Couteur, A., & Lord, C. (2003). Autism diagnostic interview-revised. *Los
734 Angeles, CA: Western Psychological Services*, 29, 30.

735 Salmi, J., Roine, U., Glerean, E., Lahnakoski, J., Nieminen-von Wendt, T., Tani, P.,
736 Leppämäki, S., Nummenmaa, L., Jääskeläinen, I. P., Carlson, S., Rintahaka, P., &
737 Sams, M. (2013). The brains of high functioning autistic individuals do not
738 synchronize with those of others. *NeuroImage: Clinical*, 3, 489–497.
739 <https://doi.org/10.1016/j.nicl.2013.10.011>

740 Saxe, R., & Kanwisher, N. (2003). People thinking about thinking people: The role of the
741 temporo-parietal junction in “theory of mind.” *NeuroImage*, 19(4), 1835–1842.
742 [https://doi.org/10.1016/S1053-8119\(03\)00230-1](https://doi.org/10.1016/S1053-8119(03)00230-1)

743 Saxe, R., & Wexler, A. (2005). Making sense of another mind: The role of the right
744 temporo-parietal junction. *Neuropsychologia*, 43(10), 1391–1399.
745 <https://doi.org/10.1016/j.neuropsychologia.2005.02.013>

746 Solomon, M., Ozonoff, S. J., Ursu, S., Ravizza, S., Cummings, N., Ly, S., & Carter, C. S.
747 (2009). The neural substrates of cognitive control deficits in autism spectrum
748 disorders. *Neuropsychologia*, 47(12), 2515–2526.
749 <https://doi.org/10.1016/j.neuropsychologia.2009.04.019>

750 Spencer, M. D., Holt, R. J., Chura, L. R., Suckling, J., Calder, A. J., Bullmore, E. T., &
751 Baron-Cohen, S. (2011). A novel functional brain imaging endophenotype of

752 autism: The neural response to facial expression of emotion. *Translational*
753 *Psychiatry*, 1(7), e19. <https://doi.org/10.1038/tp.2011.18>

754 Supekar, K., Uddin, L. Q., Khouzam, A., Phillips, J., Gaillard, W. D., Kenworthy, L. E.,
755 Yerys, B. E., Vaidya, C. J., & Menon, V. (2013). Brain Hyperconnectivity in
756 Children with Autism and its Links to Social Deficits. *Cell Reports*, 5(3), 738–
757 747. <https://doi.org/10.1016/j.celrep.2013.10.001>

758 Yeo, T. H., Krienen, F. M., Sepulcre, J., Sabuncu, M. R., Lashkari, D., Hollinshead, M.,
759 Roffman, J. L., Smoller, J. W., Zöllei, L., Polimeni, J. R., Fischl, B., Liu, H., &
760 Buckner, R. L. (2011). The organization of the human cerebral cortex estimated
761 by intrinsic functional connectivity. *Journal of Neurophysiology*, 106(3), 1125–
762 1165. <https://doi.org/10.1152/jn.00338.2011>

763 Uddin, L. Q., Supekar, K., & Menon, V. (2010). Typical and atypical development of
764 functional human brain networks: Insights from resting-state fMRI. *Frontiers in*
765 *Systems Neuroscience*, 4, 21. <https://doi.org/10.3389/fnsys.2010.00021>

766 Uddin, L. Q., Supekar, K., & Menon, V. (2013). Reconceptualizing functional brain
767 connectivity in autism from a developmental perspective. *Frontiers in Human*
768 *Neuroscience*, 7, 458. <https://doi.org/10.3389/fnhum.2013.00458>

769 Völlm, B. A., Taylor, A. N. W., Richardson, P., Corcoran, R., Stirling, J., McKie, S.,
770 Deakin, J. F. W., & Elliott, R. (2006). Neuronal correlates of theory of mind and
771 empathy: A functional magnetic resonance imaging study in a nonverbal task.
772 *NeuroImage*, 29(1), 90–98. <https://doi.org/10.1016/j.neuroimage.2005.07.022>

773 von dem Hagen, E. A. H., Stoyanova, R. S., Baron-Cohen, S., & Calder, A. J. (2013).
774 Reduced functional connectivity within and between ‘social’ resting state

775 networks in autism spectrum conditions. *Social Cognitive and Affective*
776 *Neuroscience*, 8(6), 694–701. <https://doi.org/10.1093/scan/nss053>

777 Wechsler, D. (2014). The Wechsler intelligence scale for children—fifth edition. *San*
778 *Antonio, TX: The Psychological Corporation.*

779 Weng, S.-J., Wiggins, J. L., Peltier, S. J., Carrasco, M., Risi, S., Lord, C., & Monk, C. S.
780 (2010). Alterations of resting state functional connectivity in the default network
781 in adolescents with autism spectrum disorders. *Brain Research*, 1313, 202–214.
782 <https://doi.org/10.1016/j.brainres.2009.11.057>

783 Yang, D. Y.-J., Rosenblau, G., Keifer, C., & Pelphrey, K. A. (2015). An integrative
784 neural model of social perception, action observation, and theory of mind.
785 *Neuroscience & Biobehavioral Reviews*, 51, 263–275.
786 <https://doi.org/10.1016/j.neubiorev.2015.01.020>

787 Yeshurun, Y., Swanson, S., Simony, E., Chen, J., Lazaridi, C., Honey, C. J., & Hasson,
788 U. (2017). Same Story, Different Story: The Neural Representation of Interpretive
789 Frameworks. *Psychological Science*, 28(3), 307–319.
790 <https://doi.org/10.1177/0956797616682029>