Reduced Language Lateralization in Autism and the Broader Autism Phenotype as Assessed with Robust Individual-Subjects Analyses

Olessia Jouravlev, Alexander J.E. Kell, Zachary Mineroff, Amanda J. Haskins, Dima Ayyash, Nancy Kanwisher, and Evelina Fedorenko

One of the few replicated functional brain differences between individuals with autism spectrum disorders (ASD) and neurotypical (NT) controls is reduced language lateralization. However, most prior reports relied on comparisons of group-level activation maps or functional markers that had not been validated at the individual-subject level, and/or used tasks that do not isolate language processing from other cognitive processes, complicating interpretation. Furthermore, few prior studies have examined functional responses in other brain networks, as needed to determine the spatial selectivity of the effect. Using functional magnetic resonance imaging (fMRI), we compared language lateralization between 28 adult ASD participants and carefully pairwise-matched controls, with the language regions defined individually using a well-validated language "localizer" task. Across two language comprehension paradigms, ASD participants showed less lateralized responses due to stronger right hemisphere activity. Furthermore, this effect did not stem from a ubiquitous reduction in lateralization of function across the brain: ASD participants did not differ from controls in the lateralization of two other large-scale networks—the Theory of Mind network and the Multiple Demand network. Finally, in an exploratory study, we tested whether reduced language lateralization may also be present in NT individuals with high autism-like traits. Indeed, autistic trait load in a large set of NT participants (n = 189) was associated with less lateralized language responses. These results suggest that reduced language lateralization is robustly associated with autism and, to some extent, with autism-like traits in the general population, and this lateralization reduction appears to be restricted to the language system. ***Autism Res*** *2020, 00: 1–16.* © 2020 International Society for Autism Research and Wiley Periodicals LLC

Lay Summary: How do brains of individuals with autism spectrum disorders (ASD) differ from those of neurotypical (NT) controls? One of the most consistently reported differences is the reduction of lateralization during language processing in individuals with ASD. However, most prior studies have used methods that made this finding difficult to interpret, and perhaps even artifactual. Using robust individual-level markers of lateralization, we found that indeed, ASD individuals show reduced lateralization for language due to stronger right-hemisphere activity. We further show that this reduction is not due to a general reduction of lateralization of function across the brain. Finally, we show that greater autistic trait load is associated with less lateralized language responses in the NT population. These results suggest that reduced language lateralization is robustly associated with autism and, to some extent, with autism-like traits in the general population.

Keywords: autism spectrum disorder (ASD); reduced language lateralization; language network; Theory of Mind network; Multiple Demand network; fMRI; individual differences

Introduction

Many differences in brain structure and function once thought to be hallmarks of autism [Gallagher et al., 2000; Hughes, 2009; Just, Cherkassky, Keller, & Minshew , 2004; Pierce, 2001] have turned out to be unreliable or artifactual [Deen & Pelphrey, 2012; Dufour et al., 2013; Hadjikhani, Joseph, Snyder, & Tager-Flusberg, 2007; He, Byrge, & Kennedy, 2020; Koldewyn et al., 2014; Moessnang et al., 2020]. One finding has withstood the test of time and replication: reduced lateralization during speech/language processing [Eyler, Pierce, & Courchesne, 2012; Herbert et al., 2002; Kleinhans, Müller, Cohen, & Courchesne, 2008; Knaus, Silver, Lindgren, Hadjikhani, & Tager-Flusberg, 2008; Knaus et al., 2010; see Lindell & Hudry, 2013 for a review; see Herringshaw, Ammons, DeRamus, & Kana, 2016 for a meta-analysis]. Phenotypically, deficits in language and

From the Brain and Cognitive Sciences Department, MIT, Cambridge, Massachusetts, USA (O.J., A.J.K., Z.M., A.J.H., D.A., N.K., E.F.); Department of Cognitive Science, Carleton University, Ottawa, Ontario, Canada (O.J.); Zuckerman Institute, Columbia University, New York, New York, USA (A.J.K.); Eberly Center, Carnegie Mellon University, Pittsburgh, Pennsylvania, USA (Z.M.); Department of Psychological and Brain Sciences, Dartmouth College, Hanover, New Hampshire, USA (A.J.H.); McGovern Institute for Brain Research, MIT, Cambridge, Massachusetts, USA (D.A., N.K., E.F.)

Received February 11, 2020; accepted for publication August 25, 2020

Address for correspondence and reprints: Olessia Jouravlev, Carleton University, 2209 Dunton Tower, 1125 Colonel By Drive, Ottawa, ON K1S 5B6, Canada. E-mail: olessia.jouravlev@carleton.ca

Published online 00 Month 2020 in Wiley Online Library (wileyonlinelibrary.com)
DOI: 10.1002/aur.2393

communication are a core feature of autism spectrum disorders (ASD) [Lord et al., 2012; Tager-Flusberg, Paul, & Lord, 2013; Volkmar, Lord, Bailey, Schultz, & Klin, 2004; Wilkinson, 1998]. Reduced language lateralization might therefore be a neural marker of communicative impairment in ASD.

Although reduced language lateralization appears to be consistent across paradigms and studies, the majority of prior work [cf. Kleinhans et al., 2008] has relied on comparisons of group-level activation maps. Because individuals vary in the precise locations of macro- and micro-anatomical areas [Amunts et al., 1999; Juch, Zimine, Seghier, Lazeyras, & Fasel, 2005; Tomaiuolo et al., 1999], functional activations do not align perfectly, especially in the higher-order association cortex [Fedorenko & Blank, 2020; Fischl et al., 2008; Frost & Goebel, 2012; Vázquez-Rodríguez et al., 2019]. This variability may further be greater in populations with neurodevelopmental disorders like autism [Coutanche, Thompson-Schill, & Schultz, 2011; Müller, Kleinhans, Kemmotsu, Pierce, & Courchesne, 2003; Pegado et al., 2020]. Apparent reduction in activity in some brain areas in ASD at the group level—which would translate into reduced lateralization if these were left hemisphere (LH) areas—may therefore simply reflect higher variability in the locations of the relevant functional regions.

Furthermore, the nature and scope of lateralization reduction in ASD remain poorly understood. *First*, the cause of the reduced lateralization is debated. Some report decreased activity in the LH [Eyler et al., 2012; Harris et al., 2006; Müller et al., 2003]; others increased activity in the right hemisphere (RH) [Anderson et al., 2010; Knaus et al., 2008; Takeuchi, Harada, Matsuzaki, Nishitani, & Mori, 2004; Tesink et al., 2009; Wang, Lee, Sigman, & Dapretto, 2006]; yet others find both [Boddaert et al., 2003; Kleinhans et al., 2008; Redcay & Courchesne, 2008]. Given that LH and RH language regions plausibly contribute differently to language processing [Lindell, 2006; Mitchell & Crow, 2005], understanding which hemisphere drives the lateralization differences is critical for interpretation. *Second*, many studies use language tasks that are not designed to isolate particular cognitive processes. For example, verbal-fluency tasks, where participants are presented with a cue (letter, category name, etc.) and are asked to generate as many associated words as possible [Kenworthy et al., 2013; Kleinhans et al., 2008] certainly engage linguistic resources—spanning several aspects of language [Bradshaw, Thompson, Wilson, Bishop, & Woodhead, 2017]—but also have an executive component [Thompson-Schill, D'Esposito, Aguirre, & Farah, 1997]. As a result, these tasks may activate multiple functionally distinct brain networks, complicating the interpretation of the lateralization differences. And *third*, most studies examine functional responses during a single task making it impossible to determine whether reduced lateralization is specific to that task or the brain areas / network(s) that the task targets, or whether it instead stems from an across-the-brain reduction in lateralization [Cardinale, Shih, Fishman, Ford, & Müller, 2013; Dawson, 1983; Fein, Humes, Kaplan, Lucci, & Waterhouse, 1984].

To illuminate the nature and scope of reduced language lateralization in autism, in Study 1, we used a well-validated language "localizer" [Fedorenko, Hsieh, Nieto-Castañón, Whitfield-Gabrieli, & Kanwisher, 2010] to identify language-responsive regions in each brain individually [for methodological, statistical, and theoretical advantages that functional localizers afford, see Brett, Johnsrude, & Owen, 2002; Fedorenko & Blank, 2020; Nieto-Castañon & Fedorenko, 2012; Saxe, Brett, & Kanwisher, 2006]. We then examined the responses of these language regions and their RH homologs in individuals with an ASD diagnosis and pairwise-matched controls during two language comprehension tasks to assess lateralization differences between the two groups.

To test whether lateralization reduction is restricted to the language network, we additionally examined two other networks: the network that supports social cognition, including Theory of Mind (ToM) [Saxe & Kanwisher, 2003] and the Multiple Demand (MD) network, which supports executive functions [Duncan, 2010, 2013]. These networks were chosen because (a) like the language network, they support high-level cognitive abilities; (b) like the language network, both social/emotional processes [e.g., Gainotti, 2019; Isik, Koldewyn, Beeler, & Kanwisher, 2017; Saxe & Powell, 2006] and at least some executive processes [e.g., d'Esposito et al., 1998; Nagel, Herting, Maxwell, Bruno, & Fair, 2013; Smith & Jonides, 1998; Thomason et al., 2009; Walter et al., 2003] have been argued to exhibit a hemispheric bias (with stronger responses in the right hemisphere); (c) like language and communication, both social/emotional cognition [e.g., Kana et al., 2015; Lombardo, Chakrabarti, Bullmore, & Baron-Cohen, 2011; White, Frith, Rellecke, Al-Noor, & Gilbert, 2014] and executive functions [e.g., Kana, Keller, Minshew, & Just, 2007; Kenworthy, Yerys, Anthony, & Wallace, 2008; Luna et al., 2002; Solomon et al., 2009] have been shown to be affected in autism; and iv) for both networks, robust individual-level "localizer" paradigms have been developed and extensively validated [ToM network: Dufour et al., 2013; Saxe & Kanwisher, 2003; MD network: Assem, Blank, Mineroff, Ademoglu, & Fedorenko, 2020; Fedorenko, Duncan, & Kanwisher, 2013].

Moreover, the emerging genetic picture of autism is complex, with numerous genes implicated and genetically mediated overlaps with autism-like traits present in the general population [Huguet, Ey, & Bourgeron, 2013; Miles, 2011; Talkowski, Minikel, & Gusella, 2014]. This

continuous (cf. categorical) construal of autism predicts that the same behavioral or neural features that show differences between individuals with ASD and neurotypical (NT) individuals should exhibit a relationship with autistic trait load in the NT population [e.g., Baron-Cohen et al., 2011; Lai, Lombardo, Auyeung, Chakrabarti, & Baron-Cohen, 2015]. In an exploratory Study 2, we therefore tested whether the strength of language lateralization relates to autistic trait load in a relatively large NT population (n = 189). (Although this continuous relationship should also characterize the ASD population, it may be more difficult to detect given the small sample size, and lower variability in both autistic trait load and lateralization. For completeness, we examined this relationship in the ASD participants, as well.)

Methods

Participants

Participants gave informed consent in accordance with the requirements of MIT's Committee on the Use of Humans as Experimental Subjects (COUHES) and were paid for their time.

Study 1. Thirty-two individuals with a clinical ASD diagnosis participated. Four participants were not included in the analyses due to motion-related scanner artifacts, leaving 28 participants. All participants were native English speakers with normal hearing and vision (M_{age} = 26.7, SD = 6.8, range: 18–45; 7 females; 5 left-handed; in 4 of the left-handed participants, the language network was left-lateralized, and in the remaining participant, it was right-lateralized). All participants were administered the Autism Diagnostic Observation Schedule (ADOS) [Lord, Rutter, DiLavore, & Risi, 1999] and met the criteria for a clinical diagnosis: ADOS Social Communication Score M = 9.6, SD = 2.3, range: 7–15 (these summary statistics exclude two participants whose ADOS scores were lost). All participants were also administered the Autism Spectrum Quotient questionnaire (ASQ) [Baron-Cohen, Wheelwright, Skinner, Martin, & Clubley, 2001]: M = 31.7, SD = 8.4, range: 14–43. Finally, nonverbal IQ was measured with the Matrices subtest of the Kaufman Brief Intelligence Test (KBIT) [Kaufman, 1990]: M = 114.9, SD = 10.5, range 92–130; and verbal IQ was measured with the Verbal Knowledge and Riddles subtests of the KBIT: M = 115.2, SD = 12.7, range 84–135.

Twenty-eight native English speakers without a clinical ASD diagnosis or any other neurodevelopmental disorder were pairwise-matched to ASD participants on age (M = 26.1, SD = 5.4, range: 20–45; $t(27) = 0.39$, $P = 0.35$), sex, handedness (in all 5 left-handed participants, the language network was left-lateralized), nonverbal IQ (M = 118.1, SD = 10.6, range 94–130; $t(27) = -1.11$, $P = 0.14$), verbal IQ (M = 116.4, SD = 14.9, range 85–144; $t(27) = -0.32$, $P = 0.38$), fMRI acquisition sequence parameters, experimental parameters (the version of the localizer used for each network, as detailed below), and the amount of motion in the scanner during each task (Language localizer task: $t(27) = 1.14$, $P = 0.13$; ToM localizer task: $t(18) = 0.47$, $P = 0.32$; MD localizer task: $t(19) = -1.05$, $P = 0.15$; Table 1) [see Jenkinson, 1999, for details of the motion measure]. As expected, the ASQ scores of the control (or NT) group were reliably lower than the ASD group (M = 18.0, SD = 6.2, range 9–31; $t(27) = 6.98$, $P < 0.001$), and none of the NT participants scored at or above 32 (the threshold that has been argued to index "clinically significant levels of autistic traits," although this level is not meant to be diagnostic of an ASD) [Baron-Cohen et al., 2001].

(Exploratory) Study 2. We searched the Fedorenko lab's database for native English speakers with normal hearing and vision, no ASD diagnosis (or other neurodevelopmental disorders), who had completed the ASQ and the KBIT Matrices subtest (because the verbal and nonverbal KBIT scores are correlated, we typically only administer the nonverbal Matrices subset to our NT

TABLE 1. Matching the ASD and NT Participants (N = 28 in Each Group) for Study 1

	Group		
	ASDs	NTs	t test
Sex (male:female)	21:7	21:7	
Handedness (right:left)	23:5	23:5	
Age: mean (SD)	26.7 (6.8)	26.1 (5.4)	$t(54) = 0.39$
ADOS: mean (SD)	9.6 (2.3)	NA	
ASQ: mean (SD)	31.7 (8.4)	18.0 (6.2)	**$t(51) = 6.98$***
KBIT nonverbal: mean (SD)	114.9 (10.5)	118.1 (9.8)	$t(54) = 1.11$
KBIT verbal: mean (SD)	115.2 (12.7)	116.4 (14.9)	$t(54) = 0.32$
TVM—language localizer task: mean (SD)	0.14 (0.06)	0.12 (0.03)	$t(54) = 1.14$
TVM—ToM localizer task: mean (SD)	0.14 (0.05)	0.13 (0.04)	$t(43) = 0.47$
TVM—MD localizer task: mean (SD)	0.13 (0.05)	0.12 (0.03)	$t(36) = 1.05$

Note. Results of t tests significant at $P < 0.05$ are bolded and marked by an asterisk. TVM, total vector motion (in mm).

participants). These criteria yielded 189 individuals (M_{age} = 26.2, SD = 6.6, range: 19–61); 132 females; 14 left-handed (2 left-handed individuals had their language network left-lateralized; M_{ASQ} = 17.3, SD = 7.2, range: 3–42; M_{KBIT} = 119.3, SD = 11.9, range: 75–132). Eight participants (~4%) had an ASQ score of 32 or higher, indicating a high autistic trait load, as noted above.

Design, Materials, and Procedure

Study 1. All participants completed a language localizer [Fedorenko et al., 2010]. A subset of participants additionally completed a ToM localizer [Saxe & Kanwisher, 2003] (n = 18 in each group), and/or a MD system localizer [e.g., Blank, Kanwisher, & Fedorenko, 2014] (n = 19 in each group). (Not everyone completed all three localizers because this data set was pooled from across two projects differing in their goals.) Some participants completed additional tasks for unrelated studies. The scanning session lasted approximately 2 h.

Language localizer. Participants read sentences or nonword sequences in a blocked design. The Sentences > Nonwords contrast targets brain regions that selectively support high-level linguistic processing [Blank et al., 2014; Braga, Van Dijk, Polimeni, Eldaief, & Buckner, 2019; Fedorenko, Behr, & Kanwisher, 2011], including lexical-level and combinatorial—syntactic and semantic—processes [Bautista & Wilson, 2016; Fedorenko et al., 2010; Fedorenko, Nieto-Castanon, & Kanwisher, 2012; Fedorenko & Blank, 2020]. Two slightly different versions of the localizer were used (because the data were pooled from across two projects, as noted above), which have been previously established to elicit similar activations [Fedorenko et al., 2010]. In one version (*SNloc_ips189*), each trial started with 300 msec pretrial fixation, followed by a 12-word-long sentence/nonword sequence presented one word/nonword at a time for 350 msec each, followed by a probe word/nonword presented in blue for 1,000 msec. Participants pressed one of two buttons to indicate whether the probe appeared in the preceding stimulus. Each trial ended with 500 msec of fixation. In the other version (*SNloc_ips179*), each trial started with 100-msec pretrial fixation, followed by a 12-word-long sentence/nonword sequence presented at the rate of 450 msec per word/nonword, followed by a line drawing of a finger pressing a button presented for 400 msec. Participants pressed a button whenever they saw this drawing. Each trial ended with 100 msec of fixation. In both versions, each block consisted of three trials and lasted 18 sec. Each run consisted of 16 experimental blocks (8 per condition) and 5 fixation blocks (18 or 14 sec each for the two versions), for a total duration of 378 or 358 sec. Each participant performed two runs, with condition order counterbalanced across runs. Sixteen participants in each group (ASD, NT) performed the *SNloc_ips189* version of the localizer; the remaining 12 participants in each group performed the *SNloc_ips179* version.

ToM localizer. Participants read short stories. In the critical, False Belief, condition, each story described a protagonist who held a false belief. In the control, False Photo, condition, each story described an object (e.g., a photograph or a painting) depicting some state of the world that was no longer true. The False Belief > False Photo contrast targets brain regions that support ToM reasoning [Dodell-Feder, Koster-Hale, Bedny, & Saxe, 2011; Saxe & Kanwisher, 2003]. The stories were presented one at a time for 10 sec, centered on the screen, and followed by a True/False question presented for 4 sec. Participants pressed one of two buttons to indicate their response. Each run consisted of 10 trials (5 per condition), and 11 fixation blocks, for a total duration of 272 sec. Each participant performed two runs, with condition order counterbalanced across runs.

MD localizer task. Participants kept track of four (easy condition) or eight (hard condition) spatial locations in a 3 × 4 grid [Fedorenko et al., 2011]. The Hard > Easy contrast targets brain regions that support domain-general executive processes, like working memory and cognitive control [Duncan, 2010, 2013; Fedorenko et al., 2013]. The locations flashed up one or two at a time (for the easy and hard conditions, respectively), followed by the presentation of two sets of locations. Participants pressed one of two buttons to indicate which set of locations they just saw. Ech trial lasted 8 sec [see Fedorenko et al., 2011, for details]. Each block consisted of four trials and lasted 32 sec. Each run consisted of 12 experimental blocks (6 per condition) and 4 fixation blocks (16 s each), for a total duration of 448 sec. Each participant performed two runs, with condition order counterbalanced across runs.

Study 2. Participants completed one of six versions of the language localizer task (Table 2), with 165 of the 189 participants (87%) completing the versions used in Study 1. Detailed information on the procedure and timing of the different versions can be found in Mahowald and Fedorenko [2016].

fMRI Data Acquisition and Preprocessing

Structural and functional data were collected on the whole-body 3-Tesla Siemens Trio scanner with a 32-channel head coil at the Athinoula A. Martinos Imaging Center at the McGovern Institute for Brain Research at MIT. T1-weighted structural images were collected in 128 axial slices with 1-mm isotropic voxels (repetition time [TR] = 2,530 msec, echo time [TE]= 3.48 msec) (the anatomical image was not used in the analyses reported

Table 2. Information on Which Subsets of Participants in the Sample of 189 NT Participants Performed Which Version of the Language Localizer

Number of participants	Language localizer version	Conditions	Materials	Trials per block	Blocks per run/per condition per run
n = 153	SNloc_ips179	Sentences, Nonwords	12-Word-/nonword-long sequences	3	16/8
n = 12	SNloc_ips189	Sentence, Nonwords	12-Word-/nonword-long sequences	3	16/8
n = 13	SWNloc_ips168	Sentences, Wordlists, Nonwords	8-Word-/nonword-long sequences	5	12/4
n = 6	SWNloc_ips198	Sentences, Wordlists, Nonwords	12-Word-/nonword-long sequences	3	18/6
n = 1	SWJN_v1_ips252	Sentences, Wordlists, Jaberwocky, Nonwords	12-Word-/nonword-long sequences	5	16/4
n = 3	SWJN_v2_ips232	Sentences, Wordlists, Jaberwocky, Nonwords	8-Word-/nonword-long sequences	5	16/4
n = 1	SNloc_ips232	Sentences, Nonwords	8-Word-/nonword-long sequences	5	16/8

Note. Information on the procedure and timing details for the SNloc_ips179 and SNloc_ips189 is provided in "Methods" section. Information on the procedure and timing details for the other versions of the language localizer can be found in Mahowald and Fedorenko [2016], table 2].

here). Functional, blood oxygenation-level-dependent (BOLD) data were acquired using an echo planar imaging (EPI) sequence (with a 90° flip angle and using GRAPPA with an acceleration factor of 2), with the following acquisition parameters: thirty-one 4-mm thick near-axial slices, acquired in an interleaved order with a 10% distance factor; 2.1 mm × 2.1 mm in-plane resolution; field of view 200 mm in the phase-encoding anterior to posterior direction; matrix size 96 mm × 96 mm; TR 2,000 msec; TE 30 msec; 16 nonlinear iterations for spatial normalization with 7 × 9 × 7 basis functions. Prospective acquisition correction [Thesen, Heid, Mueller, & Schad, 2000] was used to adjust the positions of the gradients based on the participant's motion one TR back. The first 10 sec of each run were excluded to allow for steady-state magnetization.

Functional data were preprocessed and analyzed using SPM5 and custom MATLAB scripts. The data were motion corrected, normalized to a common space functional template (Montreal Neurological Institute), resampled into 2-mm isotropic voxels, and high-pass filtered at 200 sec. The data were then smoothed with a 4-mm Gaussian filter. The effects were estimated using a general linear model (GLM) in which each experimental condition was modeled with a boxcar function (representing entire blocks/events) convolved with the canonical hemodynamic response function. The model also included first-order temporal derivatives of these effects, as well as nuisance regressors representing experimental runs and offline-estimated motion parameters. A constant scaling factor was applied to the BOLD signal from each individual participant and run in order to scale all data to percent signal change units.

Definition of Group-Constrained, Subject-Specific fROIs

For each participant, functional regions of inetrest (fROIs) were defined using the group-constrained subject-specific (GSS) approach [Fedorenko et al., 2010]. In this approach, a set of parcels (brain areas within which most individuals in prior studies showed activity for the relevant localizer contrast) is intersected with each individual participant's activation map for the same contrast. To define the language fROIs in the LH, we used parcels—derived from a probabilistic activation overlap map for the Sentences > Nonwords contrast for 220 participants—falling within inferior frontal gyrus (LIFG) and its orbital part (LIFGorb), middle frontal gyrus (LMFG), anterior and posterior temporal (LAntTemp and LPostTemp), and angular gyrus (LAngG). See Figure 1 for the images of the parcels. Furthermore, we defined the RH homologous fROIs using LH parcels mirror-projected onto the RH. The mirrored versions of the parcels are likely to encompass the RH homolog of the LH language network, despite possible hemispheric asymmetries in their precise locations. The probability maps and the masks are available for download from the Fedorenko Lab's website (https://evlab.mit.edu) or by request from EF.

For the ToM network, we focused on the right temporo-parietal junction (RTPJ), which has been shown to be most selective for mental-state attribution [Saxe & Powell, 2006] and its LH homolog. To define these fROIs, we used the parcels derived from a group-level representation for the False Belief > False Photo contrast in an independent group of 462 participants [Dufour et al., 2013].

To define the MD fROIs, following Fedorenko et al. [2013], we used 18 anatomical parcels across the two hemispheres [Tzourio-Mazoyer et al., 2002]:

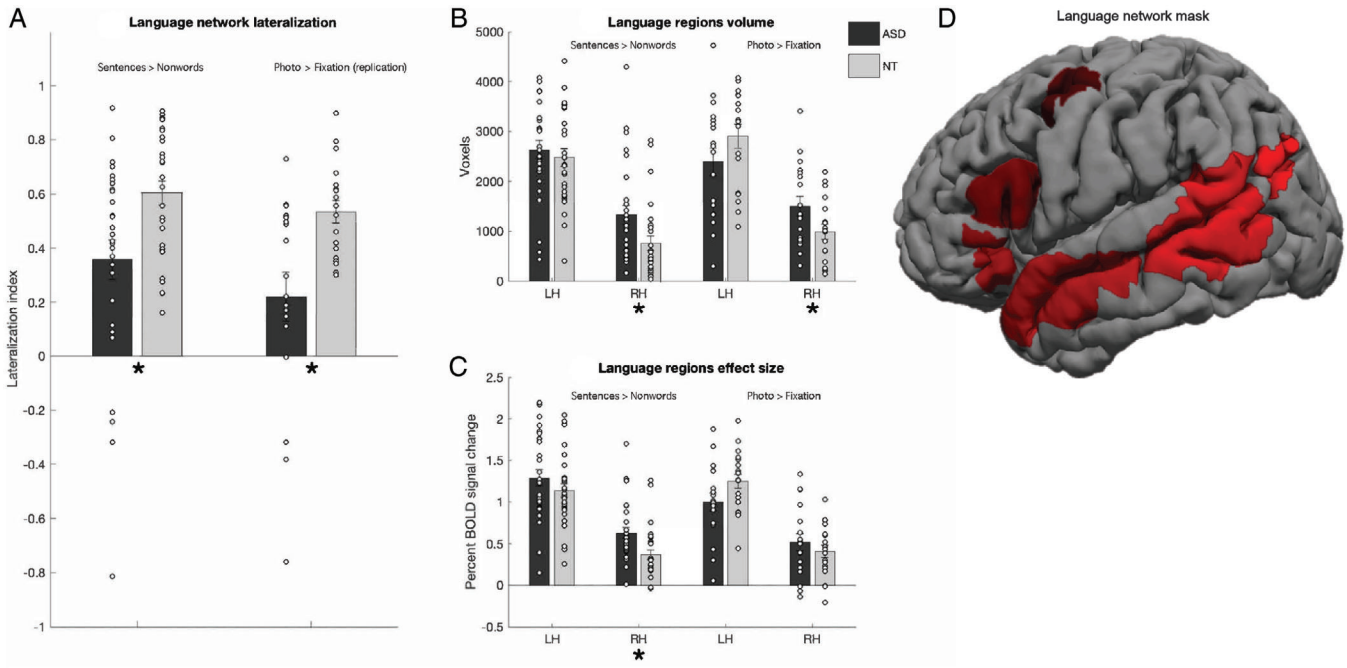

Figure 1. (A) Lateralization measures for the language network in individuals with ASD (n = 28, darker bars) versus NT controls (n = 28, lighter bars). (B) Region volumes of the language LH and RH networks. (C) Effect sizes of the language LH and RH networks. Significant group differences are marked by asterisks. (D) Language network mask.

opercular IFG (LIFGop and RIFGop), MFG (LMFG and RMFG), orbital MFG (LMFGorb and RMFGorb), insular cortex (LInsula and RInsula), precentral gyrus (LPrecG and RPrecG), supplementary and presupplementary motor areas (LSMA and RSMA), inferior parietal cortex (LParInf and RParInf), superior parietal cortex (LParSup and RParSup), and anterior cingulate cortex (LACC and RACC). The probability maps and the masks are available for download from the Fedorenko Lab's website (https://evlab.mit.edu) or by request from EF.

These parcels were used to extract functional *region volumes* and *effect sizes* in each network in each individual. To compute *region volumes*, we counted the number of voxels showing a significant effect (at the $P < 0.001$ whole-brain uncorrected threshold; this level was chosen based on prior work where we had observed that most participants show robust responses at this level for the contrasts used here) for the relevant localizer contrast within each relevant parcel. For example, for the language network, we counted the number of significant Sentences > Nonwords voxels within each of six LH and each of six RH language parcels. For the language and MD networks, these values were then summed across the regions within each hemisphere, to derive a single value per hemisphere per network per participant. The motivation for examining the networks holistically (cf. region by region) is that much evidence suggests that the regions within each network form a strongly functionally integrated system [Assem et al., 2020; Blank et al., 2014; Fedorenko & Blank, 2020; Mahowald & Fedorenko, 2016; Mineroff, Blank, Mahowald, & Fedorenko, 2018].

To compute *effect sizes*, we first defined subject-specific fROIs by selecting the top 10% of localizer-responsive voxels based on the t values for the relevant contrast and then extracted the responses of these fROIs to the relevant localizer contrast (in percent BOLD signal change). To ensure independence between the data used to define the fROIs vs. to extract effect-size measures [Kriegeskorte, Simmons, Bellgowan, & Baker, 2009], we used an across-runs cross-validation procedure [Nieto-Castañon & Fedorenko, 2012]. In particular, the first run was used to define the fROIs, and the second run to estimate the responses, the second run was used to define the fROIs, and the first run to estimate the responses, and finally, the estimates were averaged across the two left-out runs to derive a single value per participant per fROI. For the language and MD networks, these values were then averaged across the regions within each hemisphere, to derive a single value per hemisphere per network per participant.

For the language network, we additionally extracted the responses of the Sentences > Nonwords fROI to the False Photo stories from the ToM localizer (relative to fixation). As described in 2.2, the stories in the False Photo condition describe scenarios where a photograph (or some other medium) depicts a state of the world that is no longer true (to parallel the stories in the False Belief condition, where a conflict exists between reality and the

content of an individual's mind). These stories, like any sentence-level linguistic materials, are expected to elicit a strong response in the language regions (relative to the fixation baseline), thereby allowing us to examine potential differences in the lateralization of response. (We included only the False Photo condition in this analysis to avoid potential influences of the mental state content—present in the False Belief stories—on lateralization; although the language and the ToM network are functionally dissociable, they do exhibit some degree of synchronization [e.g., Paunov, Blank, & Fedorenko, 2019].)

Computing Lateralization

The critical measure for each network was the degree of *lateralization*. Following prior work [Binder et al., 1997; Seghier, Lazeyras, Pegna, Annoni, & Khateb, 2008], we used volume-based lateralization (cf. activation-strength-based lateralization) [see Mahowald & Fedorenko, 2016, for evidence that the two are correlated]. For each network, the number of activated voxels in the RH was subtracted from the number of activated voxels in the LH, and the resulting value was divided by the number of activated voxels across hemispheres. The obtained values could therefore range from 1 (exclusively LH activation) to −1 (exclusively RH activation), with 0 corresponding to bilateral activations. We have previously established that this measure is highly stable within individuals over time [Mahowald & Fedorenko, 2016].

Analyses

Study 1. We compared ASD individuals to pairwise-matched NT controls. To test for group differences in language lateralization, we used GLMs with *Group* (ASD vs. NT) as a predictor of lateralization in the language network identified using the Sentences > Nonwords contrast of the language localizer task. We also examined data fit under the null and alternative hypotheses by estimating Bayes factors (BF10) using Bayesian linear regressions [Wagenmakers, Wetzels, Borsboom, & Van Der Maas, 2011]. BF10 statistics—which reveal how many times the observed data are more likely under the alternative than the null hypothesis—were calculated using the JASP software package. We adopted the following interpretations of BF10 [Lee & Wagenmakers, 2013]: BF10 < 1 provides evidence for the null hypothesis, 1 < BF10 < 3 is considered anecdotal evidence for the alternative hypothesis, 3 < BF10 < 10—moderate evidence, and 10 < BF10—strong evidence.

To determine whether presumed differences in language lateralization result from differences in the LH, the RH, or both, we submitted LH and RH *region volumes*, as well as *effect sizes* (in a separate analysis), as dependent variables to GLMs and Bayesian linear regressions with *Group* (ASD vs. NT) as a predictor. Effect sizes are generally highly correlated with volumes but show greater stability at the individual level given that they do not depend on the statistical-significance threshold [Mahowald & Fedorenko, 2016].

To assess the robustness of the potential language lateralization differences across different linguistic materials, we further examined the responses in the language fROIs (defined with the language localizer as described above) to the processing of the False Photo stories from the ToM localizer, as described above. The region volumes, effect sizes, and lateralization values were computed in the same way as for the Sentences > Nonwords contrast.

To test whether lateralization reduction is restricted to the language network, we submitted LH and RH *region volumes* and *effect sizes* as dependent variables to GLMs and Bayesian linear regressions with *Group* (ASD vs. NT) as a predictor of lateralization in the ToM and MD networks. We also computed bivariate and partial (controlling for age, sex, and nonverbal IQ) Pearson correlations between language and MD lateralization measures (networks where lateralization reduction was observed in individuals with ASD) across participants, to test whether these effects are driven by the same underlying factor.

Finally, to test whether language lateralization in our sample of individuals with ASD is related to autism severity, we computed bivariate and partial (controlling for age, sex, and non-verbal IQ) Pearson correlations between language lateralization measures and ASQ and ADOS scores.

(Exploratory) Study 2. To test whether language lateralization is related to autistic trait load in NT individuals, we computed bivariate and partial (controlling for age, sex, and nonverbal IQ) Pearson correlations between language lateralization measures and ASQ scores.

Results

Language Lateralization Is Reduced in ASD Individuals

Language activations were left-lateralized in both groups (Sentences > Nonwords: M_{ASD} = 0.36, SD = 0.06, $t(27)$ = 4.89, $P < 0.001$; M_{NT} = 0.61, SD = 0.04, $t(27)$ = 13.76, $P < 0.001$; False Photo > Fixation: M_{ASD} = 0.22, SD = 0.39, $t(17)$ = 2.41, P = 0.03; M_{NT} = 0.54, SD = 0.18, $t(17)$ = 12.70, $P < 0.001$). Critically, however, the degree of lateralization was significantly lower in ASD individuals (Sentences > Nonwords: $t(54)$ = 2.90, P = 0.005, BF10 = 7.71; False Photo > Fixation: $t(34)$ = 3.10, P = 0.004, BF10 = 10.46; Fig. 1A). (Note that one participant in the ASD group had a language lateralization value below the first quartile [i.e., could be considered an outlier] for the Sentences > Nonwords contrast. We reran the analysis without this participant and the

corresponding matched control. The results did not change. Language lateralization was significantly lower in the patient than in the control group, $t(52) = 2.94$, $P = 0.005$, BF10 = 7.73).

This group difference in language lateralization was primarily driven by differences in the RH activity (Fig 1B,C). In particular, for the Sentences > Nonwords contrast, LH region volumes and effect sizes were similar between the groups (volumes: $t(54) = 0.56$, $P = 0.58$, BF10 = 0.31; effect sizes: $t(54) = 1.19$, $P = 0.24$, BF10 = 0.49), but in the RH, ASD individuals exhibited larger volumes ($t(54) = -2.44$, $P = 0.018$, BF10 = 3.10) and effect sizes ($t(54) = -2.94$, $P = 0.005$, BF10 = 8.47) than NT controls. Similarly, for the False Photo > Fixation contrast, LH volumes were similar between the two groups ($t(34) = 1.56$, $P = 0.13$, BF10 = 0.82), but in the RH, ASD individuals exhibited somewhat larger volumes ($t(34) = -2.22$, $P = 0.03$, BF10 = 2.07), although no reliable group differences obtained for the effect sizes (LH: $t(34) = 1.83$, $P = 0.08$; BF10 = 1.15; RH: $t(34) = -0.91$, $P = 0.37$, BF10 = 0.44).

Reduced Language Lateralization in ASD Does Not Stem from Generalized Reduction in Lateralization across the Brain

ToM activations exhibited a trend toward right-lateralization in both groups ($M_{ASD} = -0.15$, SD = 0.37, $t(19) = -1.68$, $P = 0.11$; $M_{NT} = -0.12$, SD = 0.37, $t(19) = -1.37$, $P = 0.19$), in line with prior reports of stronger and more selective activations for some social/ToM contrasts in the RTPJ compared to the LTPJ [e.g., Isik et al., 2017; Saxe & Powell, 2006]. Replicating Dufour et al. [2013], we found no group difference in lateralization ($t(34) = 0.21$, $P = 0.84$, BF10 = 0.33; Figure 2A). Furthermore, also in line with Dufour et al. [2013, see also Moessnang et al., 2020], the groups did not reliably differ in the volumes or effect sizes in either the LH or RH (LH volumes: $t(34) = 0.74$, $P = 0.47$, BF10 = 0.40; LH effect sizes: $t(34) = 0.38$, $P = 0.71$, BF10 = 0.34; RH volumes: $t(34) = 0.03$, $P = 0.98$, BF10 = 0.32; RH effect sizes: $t(34) = 0.08$, $P = 0.94$, BF10 = 0.32; Fig. 2B,C).

MD activations did not show a laterality bias in ASD participants ($M_{ASD} = -0.02$, SD = 0.19, $t(18) = -0.47$, $P = 0.65$), and they were right-lateralized in the control group ($M_{NT} = -0.19$, SD = 0.25, $t(18) = -3.25$, $P = 0.01$). Lateralization of nonverbal executive function tasks to the right hemisphere has been previously reported in NT participants [e.g., d'Esposito et al., 1998; Nagel et al., 2013; Smith & Jonides, 1998; Thomason et al., 2009; Walter et al., 2003]. The degree of lateralization was lower in ASD individuals ($t(36) = 2.30$, $P = 0.03$; Figure 2A), but according to a Bayes factor (BF10 = 2.37), this evidence was weak. Furthermore, no reliable group differences obtained for the volumes or effect sizes in

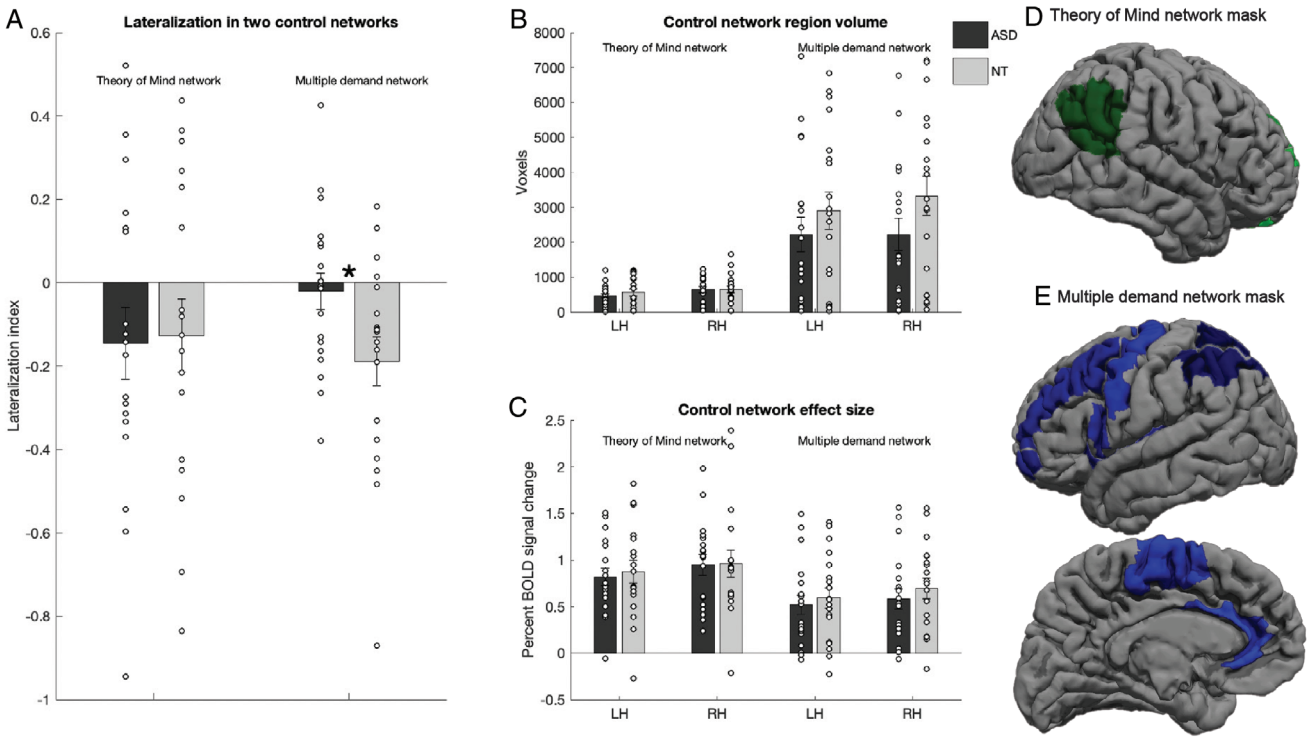

Figure 2. (A) Lateralization measures for the ToM and MD networks in individuals with ASD ($n = 28$, darker bars) versus NT controls ($n = 28$, lighter bars). (B) Region volumes of the ToM and MD LH and RH networks. (C) Effect sizes of the ToM and MD LH and RH networks. Significant group differences are marked by asterisks. (D) Theory of Mind network mask. (E) Multiple demand network mask.

either the LH or RH (LH region volumes: $t(36) = 0.92$, $P = 0.36$, BF10 = 0.44; LH effect sizes: $t(36) = 0.51$, $P = 0.62$, BF10 = 0.35; RH region volumes: $t(36) = 1.52$, $P = 0.14$, BF10 = 0.77; RH effect sizes: $t(36) = 0.71$, $P = 0.481$; BF10 = 0.39; Figure 2B,C).

To ensure that the lack of robust lateralization differences between ASD and NT participants in the ToM and MD networks is not due to lower power (fewer participants were included in these analyses compared to the analysis of the language network), we repeated the language latearlization analyses on the subsets of participants that were included in the ToM and MD analyses. The results showed reliably lower language lateralization even for these smaller samples (with the sample identical to the one used to examine MD lateralization: Sentences > Nonwords: $t(36) = 2.85$, $P = 0.007$, BF10 = 6.48; and with the sample identical to the one used to examine ToM lateralization: Sentences > Nonwords: $t(34) = 2.94$, $P = 0.006$, BF10 = 7.54), suggesting that real differences can be detected even with less power, and giving us more confidence for interpreting the lack of lateralization differences for the ToM and MD networks as meaningful.

Given that ASD individuals showed reduced lateralization in the language network and, to some extent, in the MD network, we asked whether the degree of this reduction was correlated between the two networks, which would suggest a shared underlying mechanism. The lateralization measures showed no reliable correlation ($r(17) = 0.09$, $P = 0.71$; partial $r(14) = 0.04$, $P = 0.88$), suggesting that these effects are independent.

Reduced Language Lateralization Relates to Autistic Trait Load

In 189 NT participants, ASQ scores correlated significantly with the degree of language lateralization, with higher autistic trait load associated with less lateralized responses ($r(187) = -0.15$, $P = 0.05$, partial $r(184) = -0.16$, $P = 0.03$; Figure 3B). A further exploratory examination of the correlations between the lateralization measure and ASQ subscale scores revealed that the observed relationship is primarily driven by the communicative abilities subscale ($r(187) = -0.18$, $P = 0.05$, partial $r(185) = -0.20$, P(corrected) = 0.03; P values are corrected using the Bonferroni method to account for multiple comparisons, i.e., $n = 5$ subscales). Scores on the other subscales—tapping social skills, imagination, attention switching, or attention to detail—did not reliably correlate with language lateralization (all $r(187) < |0.11|$, all p(corrected) > 0.14; all partial $r(187) < |0.12|$, all p(corrected) > 0.6).

For completeness, we also examined the relationships between language lateralization and ASQ/ADOS scores in the ASD participants keeping in mind that correlations in small samples should be interpreted with caution [Schönbrodt & Perugini, 2013]. Neither ASQ scores nor ADOS scores correlated with the degree of lateralization (ASQ: $r(26) = -0.09$, $P = 0.64$; partial $r(23) = 0.03$; $P = 0.89$; Fig. 3A; ADOS: $r(24) = -0.08$, $P = 0.70$; partial $r(21) = 0.05$; $P = 0.82$), possibly due to insufficient variability in the ASQ and ADOS scores in this population [Hedge, Powell, & Sumner, 2018].

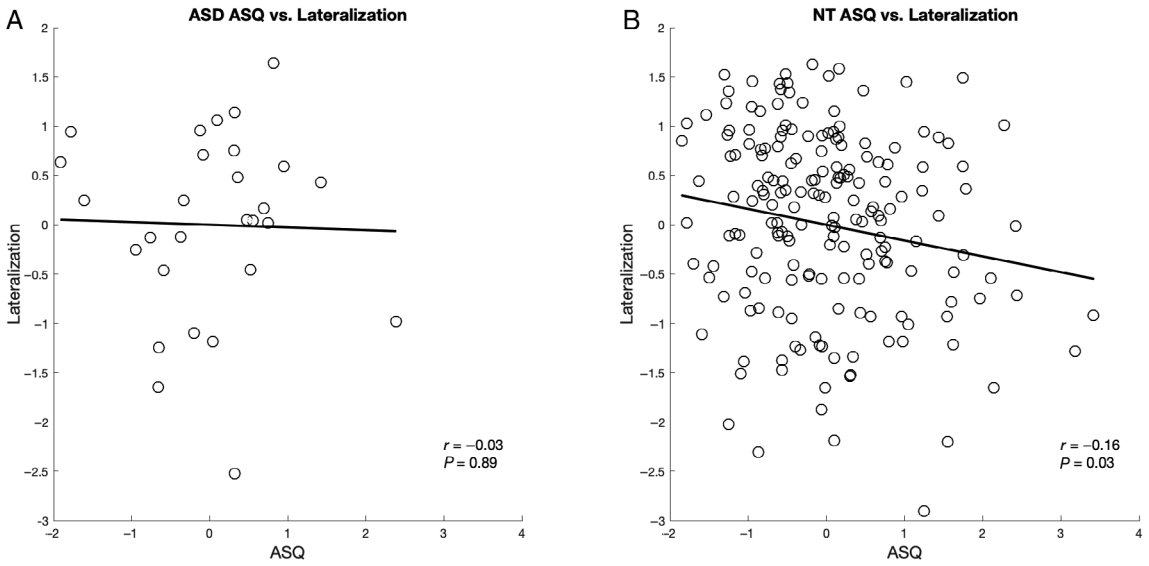

Figure 3. Reduced language lateralization and the presence of autistic traits. (A) A correlation between the language lateralization measure and autism severity, as measured by ASQ scores, in individuals with ASD ($n = 28$). (B) A correlation between the language lateralization measure and autistic trait load, as measured by the ASQ scores, in NTs ($n = 189$). The data points are standardized residual values, controlling for age, sex, and nonverbal IQ.

Discussion

Perhaps the most consistent finding from cognitive neuroscience of autism is reduced lateralization of neural activity during language tasks [Herringshaw et al., 2016; Lindell & Hudry, 2013]. However, as discussed in the Introduction, most prior studies have relied on comparisons of group-level maps, complicating interpretation, and left the nature and scope of this effect ambiguous. Here, using a robust individual-subjects functional localization approach [Fedorenko et al., 2010], we found reduced lateralization during language comprehension—across two paradigms—in individual participants. Furthermore, we established that this reduced lateralization (a) results from increased RH activity (cf. decreased LH activity or both); (b) occurs within the language-selective network (given that the language localizer effectively isolates this network from nearby functionally distinct networks) [Braga et al., 2019; Fedorenko & Blank, 2020]; (c) is not due to a global lateralization reduction (given that two other lateralized networks did not show a similar reduction). Finally, in a more exploratory investigation of NT individuals, we found some evidence for reduced language lateralization in individuals with high autistic trait load. Below, we discuss several issues that our results bear on, and highlight some open questions and challenges for future work.

Increased RH Activity During Language Processing in ASD

We found stronger RH responses during language processing in ASD individuals compared to controls, leading to more bilateral responses. Is the LH bias for speech absent/reduced in autism and other developmental disorders at birth, or is the LH bias ubiquitous at birth, with the RH playing a gradually stronger role in individuals who experience speech/language difficulties [Bishop, 2013]?

Some have proposed that increased RH activity stems from an aberrant brain development trajectory in autism [Courchesne et al., 2001; Redcay & Courchesne, 2008]. In particular, whereas typically developing brains grow at a relatively constant rate [Toga & Thompson, 2003], brains of ASD individuals exhibit initial rapid growth, followed by a premature arrest of growth [Courchesne, 2004; Pardo & Eberhart, 2007]. Because the RH matures earlier than the LH [Chiron et al., 1997; Geschwind, Miller, DeCarli, & Carmelli, 2002], this difference in the growth rate at different brain development stages may lead to an overdeveloped RH and underdeveloped LH in ASD [Courchesne, 2004]. Furthermore, genes related to language and cognitive development, like *FOXP2* [Fisher & Scharff, 2009; MacDermot et al., 2005] or *CNTNAP2* [Alarcón et al., 2008; Scott-Van Zeeland et al., 2010], are differentially expressed in the left versus right embryonic perisylvian cortex [Sun et al., 2005]. Dysregulation of these genes in autism [Muhle, Trentacoste, & Rapin, 2004; cf. Newbury et al., 2002] could potentially lead to increased RH engagement during language processing. Others have advanced an experiential account whereby speech/language difficulties in ASD individuals result in overtaxing the specialized LH mechanisms and the consequent recruitment of less specialized homologous RH areas [Mason, Williams, Kana, Minshew, & Just, 2008; Tesink et al., 2009].

Regardless of the origin of increased RH activity in autism, we can ask whether it leads to better language outcomes, or is maladaptive. There are both theoretical and empirical reasons in support of the latter possibility. On the theoretical side, RH language regions have been argued to be less functionally specialized for language processing [Gotts et al., 2013; Lindell, 2006], instead supporting visual semantic processing and/or visual imagery [Joseph, 1988; Roland & Friberg, 1985]. Indeed, some individuals with ASD describe themselves as "visual thinkers," who translate linguistic representations into mental images to achieve comprehension [Grandin, 2006]. Some have further proposed that RH language regions are less engaged in linguistic prediction [Federmeier & Kutas, 1999; Federmeier, Wlotko, & Meyer, 2008]. And empirically, in aphasia research, the recruitment of RH language regions (cf. the intact LH language regions) following stroke has been argued to be maladaptive [Barwood et al., 2011; Hamilton et al., 2010; Turkeltaub et al., 2012].

Our data also suggestively point to the maladaptive nature of the RH engagement: NT individuals with more bilateral language responses reported greater communication difficulties, and a similar trend was present in ASD individuals, although the sample is too small to draw meaningful conclusions. So, greater RH activity does not appear to alleviate language/communicative difficulties.

Evidence Against Global Lateralization Reduction in ASD

Some have argued that reduced lateralization in autism extends beyond language processing [Cardinale et al., 2013; Dawson, 1983; Fein et al., 1984; Postema et al., 2019]. To test this idea, in addition to the language network, we examined activity in two other networks that are right-lateralized in NT individuals: the ToM mentalizing network [Saxe & Kanwisher, 2003] and the domain-general MD executive control network [Duncan, 2010]. We found no evidence for decreased lateralization of the ToM activity (although it remains to be examined whether group differences in lateralization of the ToM activity would emerge if a more visual-processing based social cognitive task (e.g., facial/emotion recognition) was used), and only weak evidence for decreased lateralization of the MD activity in ASD

individuals [see also Dufour et al., 2013; Gilbert, Regier, Kay, & Ivry, 2006]. These results argue against a generally more functionally symmetrical brain in ASD individuals: reduced lateralization appears to be most pronounced—and perhaps restricted to—the language network [see also Nielsen et al., 2014, for similar conclusions drawn from resting state fMRI].

Reduced Language Lateralization in NT Individuals with High Autistic Trait Load

Autism-like traits are present to some degree in many individuals who do not have a clinical ASD diagnosis. In our exploratory Study 2, we observed reduced language lateralization in NT individuals with a higher autistic trait load (although note that the size of this correlation was only moderate). These results demonstrate that reduced language lateralization may extend to NT individuals with autism-like characteristics, in line with a continuum model of underlying genetic risk [Gaugler et al., 2014; Geschwind, 2011; Robinson et al., 2016; Wing, 1988]. Given the relatively moderate size of the observed correlation, further examination of the relationship between autistic trait load and language lateralization in NT individuals is needed.

Limitations and Future Directions

Functional brain-imaging (and many behavioral) investigations of ASD are characterized by small samples, which is problematic given the well-documented heterogeneity of this population. Furthermore, most studies, including ours, only include verbal individuals with ASD with average or above average intelligence [cf. Tager-Flusberg & Kasari, 2013]. Yet lower-functioning ASD individuals, some of whom never acquire functional linguistic skills [Maljaars, Noens, Scholte, & van Berckelaer-Onnes, 2012], may hold critical clues as to the nature and neural basis of the linguistic/communicative impairment in ASD.

Another challenge—relevant to probing the *functional importance* of reduced language lateralization—is the lack of robust and validated behavioral measures of linguistic processing that are not confounded by executive demands, like commonly used vocabulary and grammar assessments that strongly correlate with measures of nonverbal IQ [Beck & Black, 1986; Hodapp & Gerken, 1999]. Efforts to develop robust language-selective measures will be critical in understanding how more bilateral language processing affects behavioral outcomes. Investigations of the stability of reduced language lateralization within individuals across development will also be important to further inform its functional significance.

Finally, evidence for reduced language lateralization has been previously provided for diverse developmental disorders, including those that affect linguistic functions, like dyslexia and specific language impairment [e.g., De Guibert et al., 2011; Wehner, Ahlfors, & Mody, 2007], but also those that do not typically affect language/communication, like schizophrenia, attention-deficit hyperactivity disorder, and epilepsy [e.g., Hale et al., 2005; Oertel-Knöchel & Linden, 2011; Yuan et al., 2006]. Whether patterns of atypical lateralization for language differ across these disorders, and whether they result from the same underlying mechanisms remains to be determined.

Acknowledgments

We would like to acknowledge the Athinoula A. Martinos Imaging Center at McGovern Institute for Brain Research at MIT, and the support team (Steve Shannon and Atsushi Takahashi). This work was supported by a grant from the Simons Foundation to the Simons Center for the Social Brain at MIT to E.F. and N.K. E.F. was additionally supported by NIH awards R00-HD057522 and R01-DC016607. We also thank Matt Siegelman and Hannah Small for help with the figures, and Rebecca Saxe and members of the Saxe Lab, Kanwisher Lab, and Fedorenko Lab for helpful discussions. Human subjects oversight and approval was provided by MIT's Committee on the Use of Humans as Experimental Subjects (COUHES).

Conflict of Interest

The authors declare no conflict of interest.

References

Alarcón, M., Abrahams, B. S., Stone, J. L., Duvall, J. A., Perederiy, J. V., Bomar, J. M., … Nelson, S. F. (2008). Linkage, association, and gene-expression analyses identify CNTNAP2 as an autism-susceptibility gene. The American Journal of Human Genetics, 82(1), 150–159.

Amunts, K., Schleicher, A., Bürgel, U., Mohlberg, H., Uylings, H. B. M., & Zilles, K. (1999). Broca's region revisited: Cytoarchitecture and intersubject variability. Journal of Comparative Neurology, 412(2), 319–341.

Anderson, J. S., Lange, N., Froehlich, A., Dubray, M. B., Druzgal, T. J., Froimowitz, M. P., … Lainhart, J. E. (2010). Decreased left posterior insular activity during auditory language in autism. American Journal of Neuroradiology, 31(1), 131–139.

Assem, M., Blank, I., Mineroff, Z., Ademoglu, A., & Fedorenko, E. (2020). Activity in the fronto-parietal multiple-demand network is robustly associated with individual differences in working memory and fluid intelligence. Cortex, 131, 1–16.

Baron-Cohen, S., Lombardo, M. V., Auyeung, B., Ashwin, E., Chakrabarti, B., & Knickmeyer, R. (2011). Why are autism spectrum conditions more prevalent in males? PLoS Biology, 9(6), e1001081.

Baron-Cohen, S., Wheelwright, S., Skinner, R., Martin, J., & Clubley, E. (2001). The autism-spectrum quotient (AQ): Evidence from asperger syndrome/high-functioning autism, malesand females, scientists and mathematicians. Journal of Autism and Developmental Disorders, 31(1), 5–17.

Barwood, C. H., Murdoch, B. E., Whelan, B. M., Lloyd, D., Riek, S., JD, O. S., … Wong, A. (2011). Improved language performance subsequent to low-frequency rTMS in patients with chronic non-fluent aphasia post-stroke. European Journal of Neurology, 18(7), 935–943.

Bautista, A., & Wilson, S. M. (2016). Neural responses to grammatically and lexically degraded speech. Language, Cognition and Neuroscience, 31(4), 567–574.

Beck, F. W., & Black, F. L. (1986). Comparison of PPVT—R and WISC—R in a mild/moderate handicapped sample. Perceptual and Motor Skills, 62(3), 891–894.

Binder, J. R., Frost, J., Hammeke, T., Cox, R. W., Rao, S. M., & Prieto, T. (1997). Human brain language areas identified by functional magnetic resonance imaging. The Journal of Neuroscience, 17(1), 353–362.

Bishop, D. V. (2013). Cerebral asymmetry and language development: Cause, correlate, or consequence? Science, 340(6138), 1230531.

Blank, I., Kanwisher, N., & Fedorenko, E. (2014). A functional dissociation between language and multiple-demand systems revealed in patterns of BOLD signal fluctuations. Journal of Neurophysiology, 112, 1105–1118.

Boddaert, N., Belin, P., Chabane, N., Poline, J. B., Barthélémy, C., Mouren-Simeoni, M. C., … Zilbovicius, M. (2003). Perception of complex sounds: Abnormal pattern of cortical activation in autism. American Journal of Psychiatry, 160(11), 2057–2060.

Bradshaw, A. R., Thompson, P. A., Wilson, A. C., Bishop, D. V., & Woodhead, Z. V. (2017). Measuring language lateralisation with different language tasks: A systematic review. PeerJ, 5, e3929.

Braga, R. M., Van Dijk, K. R., Polimeni, J. R., Eldaief, M. C., & Buckner, R. L. (2019). Parallel distributed networks resolved at high resolution reveal close juxtaposition of distinct regions. Journal of Neurophysiology, 121(4), 1513–1534.

Brett, M., Johnsrude, I. S., & Owen, A. M. (2002). The problem of functional localization in the human brain. Nature Reviews Neuroscience, 3(3), 243–249.

Cardinale, R. C., Shih, P., Fishman, I., Ford, L. M., & Müller, R.-A. (2013). Pervasive Rightward Asymmetry Shifts of Functional Networks in Autism Spectrum Disorder. JAMA Psychiatry, 70(9), 975–982.

Chiron, C., Jambaque, I., Nabbout, R., Lounes, R., Syrota, A., & Dulac, O. (1997). The right brain hemisphere is dominant in human infants. Brain, 120(6), 1057–1065.

Courchesne, E. (2004). Brain development in autism: Early overgrowth followed by premature arrest of growth. Mental Retardation and Developmental Disabilities Research Reviews, 10 (2), 106–111.

Courchesne, E., Karns, C. M., Davis, H. R., Ziccardi, R., Carper, R. A., Tigue, Z. D., … Courchesne, R. Y. (2001). Unusual brain growth patterns in early life in patients with autistic disorder: An MRI study. Neurology, 57(2), 245–254.

Coutanche, M. N., Thompson-Schill, S. L., & Schultz, R. T. (2011). Multi-voxel pattern analysis of fMRI data predicts clinical symptom severity. NeuroImage, 57(1), 113–123.

Dawson, G. (1983). Lateralized brain dysfunction in autism: Evidence from the Halstead-Reitan neuropsychological battery. Journal of Autism and Developmental Disorders, 13(3), 269–286.

De Guibert, C., Maumet, C., Jannin, P., Ferré, J. C., Tréguier, C., Barillot, C., … Biraben, A. (2011). Abnormal functional lateralization and activity of language brain areas in typical specific language impairment (developmental dysphasia). Brain, 134(10), 3044–3058.

Deen, B., & Pelphrey, K. (2012). Perspective: Brain scans need a rethink. Nature, 491(7422), S20–S20.

d'Esposito, M., Aguirre, G. K., Zarahn, E., Ballard, D., Shin, R. K., & Lease, J. (1998). Functional MRI studies of spatial and nonspatial working memory. Cognitive Brain Research, 7(1), 1–13.

Dodell-Feder, D., Koster-Hale, J., Bedny, M., & Saxe, R. (2011). fMRI item analysis in a theory of mind task. NeuroImage, 55 (2), 705–712.

Dufour, N., Redcay, E., Young, L., Mavros, P. L., Moran, J. M., Triantafyllou, C., … Saxe, R. (2013). Similar Brain Activation during False Belief Tasks in a Large Sample of Adults with and without Autism. PLoS One, 8(9), e75468.

Duncan, J. (2010). The multiple-demand (MD) system of the primate brain: Mental programs for intelligent behaviour. Trends in Cognitive Science, 14, 172–179.

Duncan, J. (2013). The structure of cognition: Attentional episodes in mind and brain. Neuron, 80, 35–50.

Eyler, L. T., Pierce, K., & Courchesne, E. (2012). A failure of left temporal cortex to specialize for language is an early emerging and fundamental property of autism. Brain, 135(3), 949–960.

Federmeier, K. D., & Kutas, M. (1999). Right words and left words: Electrophysiological evidence for hemispheric differences in meaning processing. Cognitive Brain Research, 8(3), 373–392.

Federmeier, K. D., Wlotko, E. W., & Meyer, A. M. (2008). What's "right" in language comprehension: Event-related potentials reveal right hemisphere language capabilities. Language and linguistics compass, 2(1), 1–17.

Fedorenko, E., Behr, M. K., & Kanwisher, N. (2011). Functional specificity for high-level linguistic processing in the human brain. Proceedings of the National Academy of Sciences of the United States of America, 108(39), 16428–16433.

Fedorenko, E., & Blank, I. (2020). Broca's area is not a natural kind. Trends in Cognitive Sciences, 24(4), 270–284.

Fedorenko, E., Duncan, J., & Kanwisher, N. (2013). Broad domain generality in focal regions of frontal and parietal cortex. Proceedings of the National Academy of Sciences of the United States of America, 110, 16616–16621.

Fedorenko, E., Hsieh, P.-J., Nieto-Castañón, A., Whitfield-Gabrieli, S., & Kanwisher, N. (2010). New method for fMRI investigations of language: Defining ROIs functionally in individual subjects. Journal of Neurophysiology, 104, 1177–1194.

Fedorenko, E., Nieto-Castanon, A., & Kanwisher, N. (2012). Lexical and syntactic representations in the brain: An fMRI

investigation with multi-voxel pattern analyses. Neuropsychologia, 50(4), 499–513.
Fein, D., Humes, M., Kaplan, E., Lucci, D., & Waterhouse, L. (1984). The question of left hemisphere dysfunction in infantile autism. Psychological Bulletin, 95(2), 258–281.
Fischl, B., Rajendran, N., Busa, E., Augustinack, J., Hinds, O., Yeo, B. T., … Zilles, K. (2008). Cortical folding patterns and predicting cytoarchitecture. Cerebral Cortex, 18(8), 1973–1980.
Fisher, S. E., & Scharff, C. (2009). FOXP2 as a molecular window into speech and language. Trends in Genetics, 25(4), 166–177.
Frost, M. A., & Goebel, R. (2012). Measuring structural-functional correspondence: Spatial variability of specialised brain regions after macro-anatomical alignment. NeuroImage, 59(2), 1369–1381.
Gainotti, G. (2019). The role of the right hemisphere in emotional and behavioral disorders of patients with frontotemporal lobar degeneration: An updated review. Frontiers in Aging Neuroscience, 11, 55.
Gallagher, H. L., Happé, F., Brunswick, N., Fletcher, P. C., Frith, U., & Frith, C. D. (2000). Reading the mind in cartoons and stories: An fMRI study of "theory of mind" in verbal and nonverbal tasks. Neuropsychologia, 38(1), 11–21.
Gaugler, T., Klei, L., Sanders, S. J., Bodea, C. A., Goldberg, A. P., Lee, A. B., … Buxbaum, J. D. (2014). Most genetic risk for autism resides with common variation. Nature Genetics, 46 (8), 881–885.
Geschwind, D. H. (2011). Genetics of autism spectrum disorders. Trends in Cognitive Sciences, 15(9), 409–416.
Geschwind, D. H., Miller, B. L., DeCarli, C., & Carmelli, D. (2002). Heritability of lobar brain volumes in twins supports genetic models of cerebral laterality and handedness. Proceedings of the National Academy of Sciences of the United States of America, 99(5), 3176–3181. https://doi.org/10.1073/pnas.052494999
Gilbert, A. L., Regier, T., Kay, P., & Ivry, R. B. (2006). Whorf hypothesis is supported in the right visual field but not the left. Proceedings of the National Academy of Sciences of the United States of America, 103(2), 489–494.
Gotts, S. J., Jo, H. J., Wallace, G. L., Saad, Z. S., Cox, R. W., & Martin, A. (2013). Two distinct forms of functional lateralization in the human brain. Proceedings of the National Academy of Sciences of the United States of America, 110(36), E3435–E3444.
Grandin, T. (2006). Thinking in pictures: And other reports from my life with autism. New York: Vintage Books.
Hadjikhani, N., Joseph, R. H., Snyder, J., & Tager-Flusberg, H. (2007). Abnormal activation of the social brain during face perception in autism. Human Brain Mapping, 28(5), 441–449.
Hale, T. S., McCracken, J. T., McGough, J. J., Smalley, S. L., Phillips, J. M., & Zaidel, E. (2005). Impaired linguistic processing and atypical brain laterality in adults with ADHD. Clinical Neuroscience Research, 5(5–6), 255–263.
Hamilton, R. H., Sanders, L., Benson, J., Faseyitan, O., Norise, C., Naeser, M., … Coslett, H. B. (2010). Stimulating conversation: enhancement of elicited propositional speech in a patient with chronic non-fluent aphasia following transcranial magnetic stimulation. Brain and Language, 113 (1), 45–50.
Harris, G. J., Chabris, C. F., Clark, J., Urban, T., Aharon, I., Steele, S., … Tager-Flusberg, H. (2006). Brain activation during semantic processing in autism spectrum disorders via functional magnetic resonance imaging. Brain and Cognition, 61(1), 54–68.
He, Y., Byrge, L., & Kennedy, D. P. (2020). Nonreplication of functional connectivity differences in autism spectrum disorder across multiple sites and denoising strategies. Human Brain Mapping, 41(5), 1334–1350.
Hedge, C., Powell, G., & Sumner, P. (2018). The reliability paradox: Why robust cognitive tasks do not produce reliable individual differences. Behavior Research Methods, 50(3), 1166–1186.
Herbert, M. R., Harris, G. J., Adrien, K. T., Ziegler, D. A., Makris, N., Kennedy, D. N., … Caviness, V. S. (2002). Abnormal asymmetry in language association cortex in autism. Annals of Neurology, 52(5), 588–596.
Herringshaw, A. J., Ammons, C. J., DeRamus, T. P., & Kana, R. K. (2016). Hemispheric differences in language processing in autism spectrum disorders: A meta-analysis of neuroimaging studies. Autism Research, 9(10), 1046–1057.
Hodapp, A. F., & Gerken, K. C. (1999). Correlations between scores for Peabody picture vocabulary test—III and the Wechsler intelligence scale for children—III. Psychological Reports, 84(Suppl. 3), 1139–1142.
Hughes, J. R. (2009). Update on autism: A review of 1300 reports published in 2008. Epilepsy & Behavior, 16(4), 569–589.
Huguet, G., Ey, E., & Bourgeron, T. (2013). The genetic landscapes of autism spectrum disorders. Annual Review of Genomics and Human Genetics, 14(1), 191–213.
Isik, L., Koldewyn, K., Beeler, D., & Kanwisher, N. (2017). Perceiving social interactions in the posterior superior temporal sulcus. Proceedings of the National Academy of Sciences, 114 (43), E9145–E9152.
Jenkinson, M. (1999). Measuring transformation error by RMS deviation. FMRIB Technical Report, TR99(MJ1), 1–4.
Joseph, R. (1988). The right cerebral hemisphere: Emotion, music, visual-spatial skills, body-image, dreams, and awareness. Journal of Clinical Psychology, 44(5), 630–673.
Juch, H., Zimine, I., Seghier, M. L., Lazeyras, F., & Fasel, J. H. D. (2005). Anatomical variability of the lateral frontal lobe surface: Implication for intersubject variability in language neuroimaging. NeuroImage, 24(2), 504–514.
Just, M. A., Cherkassky, V. L., Keller, T. A., & Minshew, N. J. (2004). Cortical activation and synchronization during sentence comprehension in high-functioning autism: Evidence of underconnectivity. Brain, 127(8), 1811–1821.
Kana, R. K., Keller, T. A., Minshew, N. J., & Just, M. A. (2007). Inhibitory control in high-functioning Autism: Decreased activation and underconnectivity in inhibition networks. Biological Psychiatry, 62(3), 198–206.
Kana, R. K., Maximo, J. O., Williams, D. L., Keller, T. A., Schipul, S. E., Cherkassky, V. L., … Just, M. A. (2015). Aberrant functioning of the theory-of-mind network in children and adolescents with autism. Molecular Autism, 6(1), 59.
Kaufman, A. S. (1990). Kaufman brief intelligence test: KBIT. Circle Pines, MN: AGS, American Guidance Service.

Kenworthy, L., Wallace, G. L., Birn, R., Milleville, S. C., Case, L. K., Bandettini, P. A., & Martin, A. (2013). Aberrant neural mediation of verbal fluency in autism spectrum disorders. Brain and Cognition, 83(2), 218–226.

Kenworthy, L., Yerys, B. E., Anthony, L. G., & Wallace, G. L. (2008). Understanding executive control in autism spectrum disorders in the lab and in the real world. Neuropsychology Review, 18(4), 320–338.

Kleinhans, N. M., Müller, R. A., Cohen, D. N., & Courchesne, E. (2008). Atypical functional lateralization of language in autism spectrum disorders. Brain Research, 1221, 115–125.

Knaus, T. A., Silver, A. M., Kennedy, M., Lindgren, K. A., Dominick, K. C., Siegel, J., & Tager-Flusberg, H. (2010). Language laterality in autism spectrum disorder and typical controls: A functional, volumetric, and diffusion tensor MRI study. Brain and Language, 112(2), 113–120.

Knaus, T. A., Silver, A. M., Lindgren, K. A., Hadjikhani, N., & Tager-Flusberg, H. (2008). fMRI activation during a language task in adolescents with ASD. Journal of the International Neuropsychological Society, 14(6), 967–979.

Koldewyn, K., Yendiki, A., Weigelt, S., Gweon, H., Julian, J., Richardson, H., … Kanwisher, N. (2014). Differences in the right inferior longitudinal fasciculus but no general disruption of white matter tracts in children with autism spectrum disorder. Proceedings of the National Academy of Sciences of the United States of America, 111(5), 1981–1986.

Kriegeskorte, N., Simmons, W. K., Bellgowan, P. S., & Baker, C. I. (2009). Circular analysis in systems neuroscience: the dangers of double dipping. Nature Neuroscience, 12(5), 535–540.

Lai, M. C., Lombardo, M. V., Auyeung, B., Chakrabarti, B., & Baron-Cohen, S. (2015). Sex/gender differences and autism: setting the scene for future research. Journal of the American Academy of Child & Adolescent Psychiatry, 54(1), 11–24.

Lee, M. D., & Wagenmakers, E. J. (2013). Bayesian cognitive modeling: A practical course. Cambridge: Cambridge University Press.

Lindell, A. K. (2006). In your right mind: Right hemisphere contributions to language processing and production. Neuropsychology Review, 16(3), 131–148.

Lindell, A. K., & Hudry, K. (2013). Atypicalities in cortical structure, handedness, and functional lateralization for language in autism spectrum disorders. Neuropsychology Review, 23, 257–270.

Lombardo, M. V., Chakrabarti, B., Bullmore, E. T., & Baron-Cohen, S. (2011). Specialization of right temporo-parietal junction for mentalizing and its relation to social impairments in autism. NeuroImage, 56(3), 1832–1838.

Lord, C., Petkova, E., Hus, V., Gan, W., Lu, F., Martin, D. M., … Risi, S. (2012). A multisite study of the clinical diagnosis of different autism spectrum disorders. Archives of General Psychiatry, 69(3), 306–313.

Lord, C., Rutter, M., DiLavore, P. C., & Risi, S. (1999). Autism Diagnostic Observation Scale-WPS (ADOS-WPS). Los Angeles, CA: Western Psychological Services.

Luna, B., Minshew, N. J., Garver, K. E., Lazar, N. A., Thulborn, K. R., Eddy, W. F., & Sweeney, J. A. (2002). Neocortical system abnormalities in autism: An fMRI study of spatial working memory. Neurology, 59(6), 834–840.

MacDermot, K. D., Bonora, E., Sykes, N., Coupe, A.-M., Lai, C. S. L., Vernes, S. C., … Fisher, S. E. (2005). Identification of FOXP2 truncation as a novel cause of developmental speech and language deficits. The American Journal of Human Genetics, 76(6), 1074–1080.

Mahowald, K., & Fedorenko, E. (2016). Reliable individual-level neural markers of high-level language processing: A necessary precursor for relating neural variability to behavioral and genetic variability. NeuroImage, 139, 74–93.

Maljaars, J., Noens, I., Scholte, E., & van Berckelaer-Onnes, I. (2012). Language in low-functioning children with autistic disorder: Differences between receptive and expressive skills and concurrent predictors of language. Journal of Autism and Developmental Disorders, 42(10), 2181–2191.

Mason, R. A., Williams, D. L., Kana, R. K., Minshew, N., & Just, M. A. (2008). Theory of mind disruption and recruitment of the right hemisphere during narrative comprehension in autism. Neuropsychologia, 46(1), 269–280.

Miles, J. H. (2011). Autism spectrum disorders-a genetics review. Genetics in Medicine: Official Journal of the American College of Medical Genetics, 13(4), 278–294.

Mineroff, Z., Blank, I. A., Mahowald, K., & Fedorenko, E. (2018). A robust dissociation among the language, multiple demand, and default mode networks: evidence from inter-region correlations in effect size. Neuropsychologia, 119, 501–511.

Mitchell, R. L. C., & Crow, T. J. (2005). Right hemisphere language functions and schizophrenia: The forgotten hemisphere? Brain, 128, 963–978.

Moessnang, C., Baumeister, S., Tillmann, J., Goyard, D., Charman, T., Ambrosino, S., … Crawley, D. (2020). Social brain activation during mentalizing in a large autism cohort: the Longitudinal European Autism Project. Molecular Autism, 11(1), 1–17.

Muhle, R., Trentacoste, S. V., & Rapin, I. (2004). The genetics of autism. Pediatrics, 113(5), e472–e486.

Müller, R. A., Kleinhans, N., Kemmotsu, N., Pierce, K., & Courchesne, E. (2003). Abnormal variability and distribution of functional maps in autism: An fMRI study of visuomotor learning. American Journal of Psychiatry, 160(10), 1847–1862.

Nagel, B. J., Herting, M. M., Maxwell, E. C., Bruno, R., & Fair, D. (2013). Hemispheric lateralization of verbal and spatial working memory during adolescence. Brain and Cognition, 82(1), 58–68.

Newbury, D. F., Bonora, E., Lamb, J. A., Fisher, S. E., Lai, C. S., Baird, G., … Bolton, P. F. (2002). FOXP2 is not a major susceptibility gene for autism or specific language impairment. The American Journal of Human Genetics, 70(5), 1318–1327.

Nielsen, J. A., Zielinski, B. A., Fletcher, P., Alexander, A. L., Lange, N., Bigler, E. D., … Anderson, J. S. (2014). Abnormal lateralization of functional connectivity between language and default mode regions in autism. Molecular Autism, 5 (1), 8.

Nieto-Castañon, A., & Fedorenko, E. (2012). Subject-specific functional localizers increase sensitivity and functional resolution of multi-subject analyses. NeuroImage, 63, 1646–1669.

Oertel-Knöchel, V., & Linden, D. E. J. (2011). Cerebral asymmetry in schizophrenia. The Neuroscientist, 17(5), 456–467.

Pardo, C. A., & Eberhart, C. G. (2007). The neurobiology of autism. Brain Pathology, 17(4), 434–447.

Paunov, A. M., Blank, I. A., & Fedorenko, E. (2019). Functionally distinct language and Theory of Mind networks are synchronized at rest and during language comprehension. Journal of Neurophysiology, 121(4), 1244–1265.

Pegado, F., Hendriks, M. H., Amelynck, S., Daniels, N., Steyaert, J., Boets, B., & de Beeck, H. O. (2020). Adults with high functioning autism display idiosyncratic behavioral patterns, neural representations and connectivity of the "Voice Area" while judging the appropriateness of emotional vocal reactions. Cortex, 125, 90–108.

Pierce, K. (2001). Face processing occurs outside the fusiform "face area" in autism: Evidence from functional MRI. Brain, 124(10), 2059–2073.

Postema, M. C., Van Rooij, D., Anagnostou, E., Arango, C., Auzias, G., Behrmann, M., … Deruelle, C. (2019). Altered structural brain asymmetry in autism spectrum disorder in a study of 54 datasets. Nature Communications, 10(1), 1–12.

Redcay, E., & Courchesne, E. (2008). Deviant functional magnetic resonance imaging patterns of brain activity to speech in 2-3-year-old children with autism spectrum disorder. Biological Psychiatry, 64(7), 589–598.

Robinson, E. B., St Pourcain, B., Anttila, V., Kosmicki, J. A., Bulik-Sullivan, B., Grove, J., … Daly, M. J. (2016). Genetic risk for autism spectrum disorders and neuropsychiatric variation in the general population. Nature Genetics, 48(5), 552–555.

Roland, P. E., & Friberg, L. (1985). Localization of cortical areas activated by thinking. Journal of Neurophysiology, 53(5), 1219–1243.

Saxe, R., Brett, M., & Kanwisher, N. (2006). Divide and conquer: A defense of functional localizers. NeuroImage, 30(4), 1088–1096.

Saxe, R., & Kanwisher, N. (2003). People thinking about thinking people: the role of the temporo-parietal junction in "theory of mind". NeuroImage, 19(4), 1835–1842.

Saxe, R., & Powell, L. J. (2006). It's the thought that counts: Specific brain regions for one component of theory of mind. Psychological Science, 17(8), 692–699.

Schönbrodt, F. D., & Perugini, M. (2013). At what sample size do correlations stabilize? Journal of Research in Personality, 47 (5), 609–612.

Scott-Van Zeeland, A. A., Abrahams, B. S., Alvarez-Retuerto, A. I., Sonnenblick, L. I., Rudie, J. D., Ghahremani, D., … Bookheimer, S. Y. (2010). Altered functional connectivity in frontal lobe circuits is associated with variation in the autism risk gene CNTNAP2. Science Translational Medicine, 2(56), 5680.

Seghier, M. L., Lazeyras, F., Pegna, A. J., Annoni, J. M., & Khateb, A. (2008). Group analysis and the subject factor in functional magnetic resonance imaging: Analysis of fifty right-handed healthy subjects in a semantic language task. Human Brain Mapping, 29(4), 461–477.

Smith, E. E., & Jonides, J. (1998). Neuroimaging analyses of human working memory. Proceedings of the National Academy of Sciences, 95(20), 12061–12068.

Solomon, M., Ozonoff, S. J., Ursu, S., Ravizza, S., Cummings, N., Ly, S., & Carter, C. S. (2009). The neural substrates of cognitive control deficits in autism spectrum disorders. Neuropsychologia, 47(12), 2515–2526.

Sun, T., Patoine, C., Abu-Khalil, A., Visvader, J., Sum, E., Cherry, T. J., … Walsh, C. A. (2005). Early asymmetry of gene transcription in embryonic human left and right cerebral cortex. Science, 308(5729), 1794–1798.

Tager-Flusberg, H., & Kasari, C. (2013). Minimally verbal school-aged children with autism spectrum disorder: The neglected end of the spectrum. Autism Research, 6(6), 468–478.

Tager-Flusberg, H., Paul, R., & Lord, C. (2013). Language and communication in autism. In Handbook of autism and pervasive developmental disorders (pp. 335–364). Hoboken, NJ: John Wiley & Sons.

Takeuchi, M., Harada, M., Matsuzaki, K., Nishitani, H., & Mori, K. (2004). Difference of signal change by a language task on autistic patients using functional MRI. Journal of Medical Investigation, 51(1–2), 59–62.

Talkowski, M. E., Minikel, E. V., & Gusella, J. F. (2014). Autism spectrum disorder genetics: Diverse genes with diverse clinical outcomes. Harvard Review of Psychiatry, 22(2), 65–75.

Tesink, C. M., Buitelaar, J. K., Petersson, K. M., Van der Gaag, R. J., Kan, C. C., Tendolkar, I., & Hagoort, P. (2009). Neural correlates of pragmatic language comprehension in autism spectrum disorders. Brain, 132(7), 1941–1952.

Thesen, S., Heid, O., Mueller, E., & Schad, L. R. (2000). Prospective acquisition correction for head motion with image-based tracking for real-time fMRI. Magnetic Resonance in Medicine, 44, 457–465.

Thomason, M. E., Race, E., Burrows, B., Whitfield-Gabrieli, S., Glover, G. H., & Gabrieli, J. D. (2009). Development of spatial and verbal working memory capacity in the human brain. Journal of Cognitive Neuroscience, 21(2), 316–332.

Thompson-Schill, S. L., D'Esposito, M., Aguirre, G. K., & Farah, M. J. (1997). Role of left inferior prefrontal cortex in retrieval of semantic knowledge: a reevaluation. Proceedings of the National Academy of Sciences of the United States of America, 94(26), 14792–14797.

Toga, A. W., & Thompson, P. M. (2003). Mapping brain asymmetry. Nature Reviews Neuroscience, 4(1), 37–48.

Tomaiuolo, F., MacDonald, J. D., Caramanos, Z., Posner, G., Chiavaras, M., Evans, A. C., & Petrides, M. (1999). Morphology, morphometry and probability mapping of the pars opercularis of the inferior frontal gyrus: An in vivo MRI analysis. European Journal of Neuroscience, 11(9), 3033–3046.

Turkeltaub, P. E., Coslett, H. B., Thomas, A. L., Faseyitan, O., Benson, J., Norise, C., & Hamilton, R. H. (2012). The right hemisphere is not unitary in its role in aphasia recovery. Cortex, 48(9), 1179–1186.

Tzourio-Mazoyer, N., Landeau, B., Papathanassiou, D., Crivello, F., Etard, O., Delcroix, N., … Joliot, M. (2002). Automated anatomical labeling of activations in SPM using a macroscopic anatomical parcellation of the MNI MRI single-subject brain. NeuroImage, 15, 273–289.

Vázquez-Rodríguez, B., Suárez, L. E., Markello, R. D., Shafiei, G., Paquola, C., Hagmann, P., … Misic, B. (2019). Gradients of structure–function tethering across neocortex. Proceedings of the National Academy of Sciences of the United States of America, 116(42), 21219–21227.

Volkmar, F. R., Lord, C., Bailey, A., Schultz, R. T., & Klin, A. (2004). Autism and pervasive developmental disorders. Journal of Child Psychology and Psychiatry and Allied Disciplines, 45, 135–170.

Wagenmakers, E. J., Wetzels, R., Borsboom, D., & Van Der Maas, H. L. (2011). Why psychologists must change the way they analyze their data: The case of psi: Comment on Bem. Journal of Personality and Social Psychology, 100(3), 426–432.

Walter, H., Bretschneider, V., Grön, G., Zurowski, B., Wunderlich, A. P., Tomczak, R., & Spitzer, M. (2003). Evidence for quantitative domain dominance for verbal and spatial working memory in frontal and parietal cortex. Cortex, 39(4–5), 897–911.

Wang, A. T., Lee, S. S., Sigman, M., & Dapretto, M. (2006). Neural basis of irony comprehension in children with autism: The role of prosody and context. Brain, 129(4), 932–943.

Wehner, D. T., Ahlfors, S. P., & Mody, M. (2007). Effects of phonological contrast on auditory word discrimination in children with and without reading disability: A magnetoencephalography (MEG) study. Neuropsychologia, 45(14), 3251–3262.

White, S. J., Frith, U., Rellecke, J., Al-Noor, Z., & Gilbert, S. J. (2014). Autistic adolescents show atypical activation of the brain's mentalizing system even without a prior history of mentalizing problems. Neuropsychologia, 56(1), 17–25.

Wilkinson, K. M. (1998). Profiles of language and communication skills in autism. Mental Retardation and Developmental Disabilities Research Reviews, 4, 73–79.

Wing, L. (1988). The continuum of autistic characteristics. In Diagnosis and assessment in autism (pp. 91–110). Boston, MA: Springer.

Yuan, W., Szaflarski, J. P., Schmithorst, V. J., Schapiro, M., Byars, A. W., Strawsburg, R. H., & Holland, S. K. (2006). fMRI shows atypical language lateralization in pediatric epilepsy patients. Epilepsia, 47(3), 593–600.