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Larger Receptive Field Size as a Mechanism Underlying Atypical Motion Perception in Autism Spectrum Disorder

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Abstract

Atypical visual motion perception has been widely observed in individuals with autism spectrum disorder (ASD). The pattern of results, however, has been inconsistent. Emerging mechanistic hypotheses seek to explain these variable patterns of atypical motion sensitivity, each uniquely predicting specific patterns of performance across varying stimulus conditions. Here, we investigated the integrity of two such fundamental mechanisms—response gain control and receptive field size. A total of 20 children and adolescents with ASD and 20 typically developing (TD) age- and IQ-matched controls performed a motion discrimination task. To adequately model group differences in both mechanisms of interest, we tested a range of 23 stimulus conditions varying in size and contrast. Results revealed a motion perception impairment in ASD that was specific to the smallest sized stimuli (1°), irrespective of stimulus contrast. Model analyses provided evidence for larger receptive field size in ASD as the mechanism that explains this size-specific reduction of motion sensitivity.

Keywords

motion perception, autism spectrum disorder, response gain control, receptive field size

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Autism spectrum disorder (ASD) has been associated with a wide range of sensory symptoms, including both increased and decreased behavioral responding to everyday sensory stimuli (American Psychiatric Association, 2013). These sensory atypicalities can impact individuals' daily functioning; however, little is known about their etiology. There is also strong evidence for more basic sensory atypicalities in ASD (Simmons et al., 2009). This includes motion processing—a fundamental visual ability. Most studies report impaired motion sensitivity in ASD (Koh, Milne, & Dobkins, 2010; Spencer et al., 2000; Takarae, Luna, Minshew, & Sweeney, 2008), but evidence exists for both intact (Bertone, Mottron, Jelenic, & Faubert, 2003) and even enhanced motion perception (Foss-Feig, Tadin, Schauder, & Cascio, 2013; Manning, Tibber, Charman, Dakin, & Pellicano, 2015). These studies have employed a variety of paradigms, which inherently target particular aspects of motion processing abilities. For example, studies have experimentally manipulated stimulus speed (Manning, Charman, & Pellicano, 2013), contrast (Foss-Feig et al., 2013), and duration (Robertson, Martin, Baker, & Baron-Cohen, 2012). A key outstanding question is to what degree these findings could be explained by abnormalities in core underlying mechanisms, as opposed to methodological differences or differences in study samples.

Several prominent hypotheses have emerged in regard to mechanisms underlying atypical visual perception in ASD. Each predicts specific patterns of motion sensitivity across variations in stimulus contrast and size (Fig. 1). One hypothesis suggests impairments in gain control

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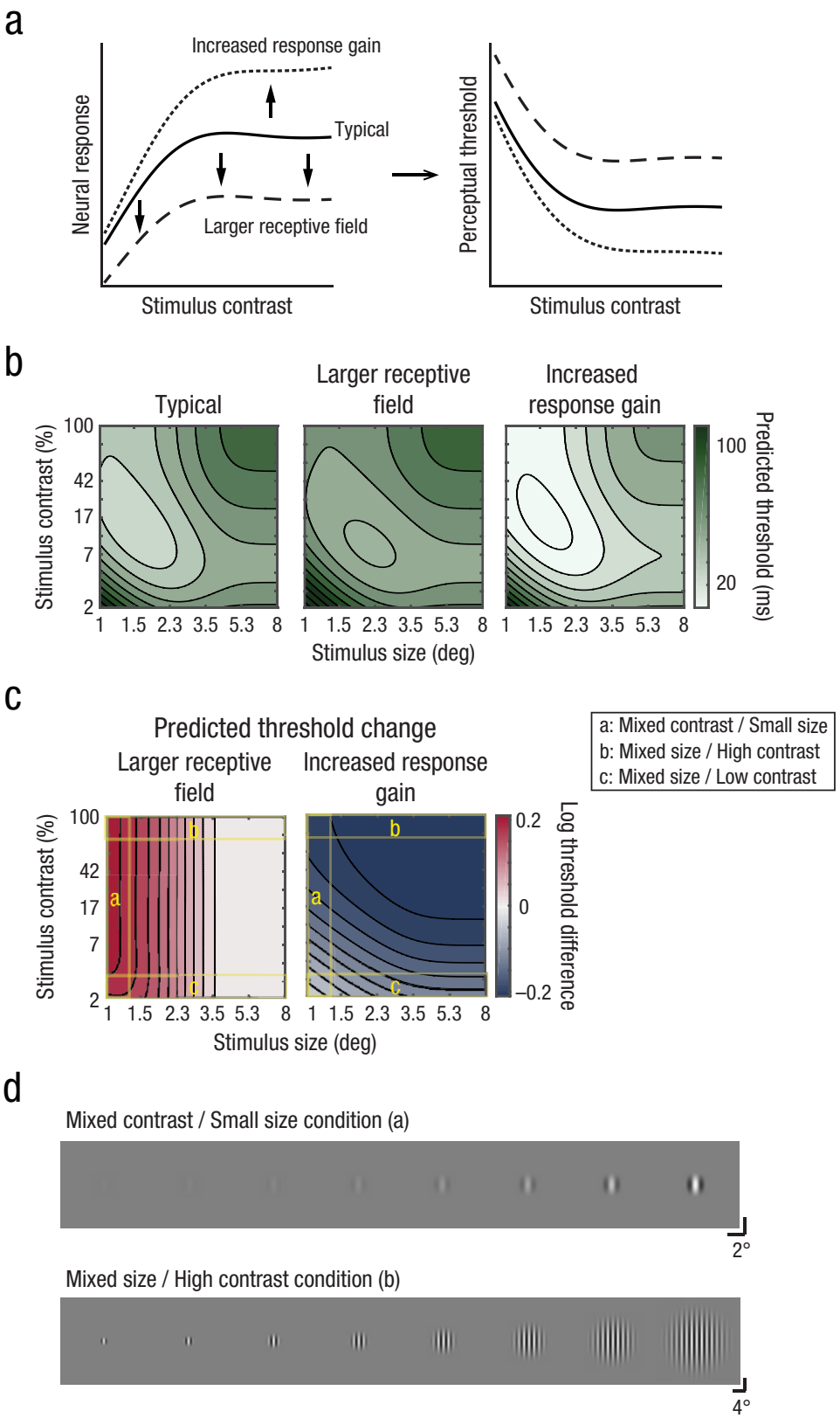

Fig. 1. (a) Predictions derived from impairments in gain control and receptive field size. Results are shown for a full range of contrasts, assuming a small stimulus size. Atypicalities in neural responses (left) result in reciprocal changes of perceptual thresholds (right). Solid line shows typical neural responses

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and perceptual thresholds over a range of stimulus contrasts. Dotted line illustrates effects of impaired response gain control, which increases neural responses and decreases perceptual thresholds, particularly at higher contrasts. Dashed line illustrates effects of atypically large receptive field size, which reduces neural responses across all contrast levels. (b) Perceptual consequences of changes in gain control and receptive field size. Left panel shows the typical pattern of results over a full range of stimulus sizes and contrasts, in which data suggest spatial summation at low contrast and spatial suppression at high contrast. The remaining panels depict selective changes in the excitatory receptive field size (center) and response gain parameters (right), and the resulting predicted thresholds. These predictions are derived from the main model used in this study. (c) Predicted thresholds change resulting from enlarged receptive field size (left) and increased response gain (right; specifically, by reduction in suppressive gain). Red and blue colors indicate increased and decreased thresholds, respectively, compared to typical results (as shown in the left panel of b). In sum, changes in gain control and receptive field size lead to markedly different patterns in motion sensitivity. (d) We selected three stimulus conditions—mixed-contrast/small-size, mixed-size/high-contrast, and mixed-size/low-contrast—to best test predictions derived from our two main hypotheses. All conditions included eight stimulus levels (note that the top and bottom panels are depicted here on different scales to better illustrate the stimuli).

mechanisms in ASD, namely those underlying response gain (Foss-Feig et al., 2013; Rosenberg, Patterson, & Angelaki, 2015)—an inhibitory mechanism that prevents over-responding to high-contrast stimuli (Albrecht & Hamilton, 1982; Katzner, Busse, & Carandini, 2011). As contrast increases, neural responses first show rapid increases, but typically saturate at higher contrasts (Fig. 1a). This corresponds to decreasing perceptual thresholds that eventually asymptote with gradual increases in contrast (Fig. 1a). In a psychophysical study, we previously revealed enhanced sensitivity to visual motion in ASD at high, but not low, contrast (Foss-Feig et al., 2013). This contrast-dependent enhancement of motion perception in ASD is qualitatively consistent with impairments in response gain control, whereby neural responses are atypically increased at high contrast (Fig. 1a). Indeed, a recent computational study showed that the observed atypicality could be due to abnormalities in gain control (Rosenberg et al., 2015; namely a reduction in suppressive gain). This predicts large group differences in perceptual sensitivity at higher contrasts, irrespective of stimulus size (Figs. 1b, 1c).

A second mechanistic hypothesis concerns receptive field size. Schwarzkopf, Anderson, de Haas, White, and Rees (2014) found that individuals with ASD had atypically large population receptive fields in extrastriate visual areas. This included the middle temporal (MT) visual area, a region critical for motion processing (Born & Bradley, 2005). A recent finding of enhanced motion integration in ASD is consistent with the hypothesis of larger receptive field size in ASD (Manning et al., 2015; but see Schwarzkopf et al., 2014, for behavioral experiments). Larger receptive field size further predicts impaired visual sensitivity for smaller stimuli (Tadin & Lappin, 2005). This link between receptive field size and visual sensitivity is presumably due to reduced neural responses to stimuli significantly smaller than the receptive field, which shifts the contrast response function downward for such stimuli (Fig. 1a). Critically, this effect should be largely independent from stimulus contrast (Fig. 1a), yielding a pattern different from that predicted by the gain control hypothesis (Fig. 1c). Thus, the two hypotheses, implicating impaired response gain control and larger receptive field size, yield distinct testable predictions about motion perception differences in ASD.

Notably, both response gain control and receptive field size are affected by the excitation/inhibition (E/I) balance in the brain (Dorrn, Yuan, Barker, Schreiner, & Froemke, 2010; Katzner et al., 2011; Sillito, 1975; Vogels & Abbott, 2009). Given that a broad impairment in E/I imbalance has been proposed as a possible underlying cause of ASD (Rubenstein & Merzenich, 2003), testing these particular mechanistic hypotheses is of high importance (Heeger, Behrmann, & Dinstein, 2017). Here, we test the integrity of response gain control and receptive field size in children and adolescents with and without ASD in the context of motion perception. Unlike most perceptual studies of ASD that test a limited stimulus space, we used an approach that allowed us to manipulate both the size and contrast of moving gratings over a relatively large stimulus range (Fig. 1d), selected to best probe specific model predictions (Fig. 1c). Psychophysical sensitivity to these stimuli was fitted with a model that characterizes interactions between receptive field center and surround, with responses dependent both on stimulus contrast and size (Betts, Sekuler, & Bennett, 2012). This enabled us to test the mechanistic hypotheses. To our knowledge, this is the first study in ASD to both psychophysically test and computationally model either of these mechanisms.

Method

Participants

Participants were 20 children and adolescents with ASD (19 male) and 20 TD age- and IQ- matched controls (20 male). All participants were between 10 and 17 years old and had an IQ greater than 80, as measured by an abbreviated version of the Wechsler Intelligence Scale for Children (4th ed.; Wechsler, 2003) or Wechsler Adult Intelligence Scale (4th ed.; Wechsler, 2008). Groups were

matched on both age (ASD: M = 13.1, SD = 2.2; TD: M = 13.7, SD = 2.1), $t(18) = 0.99$, $p = .33$, and full-scale IQ (ASD: M = 107.5, SD = 13.7; TD: M = 113.4, SD = 15.8), $t(18) = 1.27$, $p = .21$. There were also no group differences on either verbal IQ (ASD: M = 110.3, SD = 15.2; TD: M = 116.1, SD = 16.5), $t(18) = 1.17$, $p = .25$, or performance IQ (ASD: M = 102.7, SD = 14.7; TD: M = 108.1, SD = 15.6), $t(18) = 1.13$, $p = .27$. Parents reported on their child's race/ethnicity and annual household income. In all, 90% identified as White/Caucasian (18 TD and 18 ASD), 5% as Black/African-American (2 TD), and 5% as more than one race (2 ASD). Annual household income was distributed as follows: 17.5% less than $50,000 (2 TD and 5 ASD), 22.5% $50,000 to $75,000 (5 TD and 4 ASD), 27.5% $75,000 to $100,000 (8 TD and 3 ASD), 25% $100,000 to $200,000 (3 TD and 7 ASD), 5% more than $200,000 (1 TD and 1 ASD). Income information for one TD participant was not reported. Of the participants with ASD, 10 were taking psychoactive medication (2 stimulants only, 5 other psychoactive [e.g., SSRIs] only, 3 both stimulant plus another psychoactive) at the time of the study. We did not observe any differences in psychophysical thresholds (all ps > .26) or model parameters (all ps > .13) between medicated and unmedicated participants with ASD.

The presence or absence of an ASD diagnosis was confirmed by research-reliable administration of the Autism Diagnostic Observation Schedule (ADOS; Lord, Rutter, DiLavore, & Risi, 2002). Diagnoses were further confirmed or ruled out via parent report, using the Autism Diagnostic Interview–Revised (ADI-R; Rutter, Le Couteur, & Lord, 2003) with parents of children with ASD and the Social Communication Questionnaire (Rutter, Bailey, & Lord, 2003) with parents of TD participants. Participants did not have any parent-reported genetic, neurological, or visual abnormalities. Visual acuity was confirmed using the Snellen eye chart; participants were required to have at least 20/40 corrected vision in both eyes. Relatively low spatial frequency of our stimuli (1 cycle/degree) ensures that this acuity cutoff is more than sufficient (20/40 Snellen letter corresponds to a 15 cycles/degree grating). In addition, TD participants did not have any parent-reported behavioral, learning, or psychiatric diagnoses and had no first- or second-degree relatives with ASD. Parents gave written informed consent, and participants gave assent; all were paid for participation. Procedures were approved by the university's Research Subjects Review Board.

General procedure

Participants completed three conditions (defined by the contrast or size of stimuli) in a single session lasting 1.5 hr. Each condition took approximately 20 min to complete with breaks given throughout. Condition order was randomized across participants. A chin rest was used to maintain a still and comfortable seated position. The stimuli were shown on a customized linear DLP projector (DepthQ WXGA 360 at 1280 × 720 resolution). The projector's color wheel, which presents three grayscale images per cycle at 120 Hz, was removed, yielding an effective frame rate of 360 Hz (2.67 ms frame duration). This allowed fine-grained control of stimulus motion and its duration (Glasser & Tadin, 2011, 2014). Viewing distance was 135 cm, with each pixel subtending 2 arcmin of visual angle. Participants were monitored by an experimenter throughout to encourage on-task behavior. In addition, several child-friendly supports were utilized to increase motivation and sustain attention throughout the session. Specifically, we presented the task instructions in the form of a story about a tiger moving behind the woods, which was paired with matching on-screen animations. An interactive visual schedule was used to track study progress.

Stimuli, task, and experimental design

Drifting grating stimuli (1 cycle/degree, 4°/s, starting phase randomized) were created in MATLAB and Psychophysics Toolbox (Brainard, 1997; Pelli, 1997). Grating stimuli were presented in a spatially fixed two-dimensional raised cosine envelope, the radius of which defined the stimulus size. The participants' task was to discriminate its motion direction. For each trial, participants were instructed to fixate on the center of the screen. Fixation was facilitated by a shrinking fixation circle that appeared between trials (Foss-Feig et al., 2013). Specifically, the fixation sequence started with a 0.63° radius fixation circle that shrank to 0.1° radius over 250 ms and remained at that size for 360 ms and then disappeared. The stimulus, a moving grating with its contrast or size chosen pseudorandomly, appeared 300 ms later at the same location. Note that no dynamic stimuli appeared within 660 ms of stimulus onset. Participants indicated the perceived direction (left or right) of the stimulus by a key press. Auditory feedback followed correct responses. The subsequent trial sequence started 0.3 s after each response, with the subsequent stimulus appearing 1.21 s after each response. Although we did not record eye movements, our subjective observation is that the shrinking fixation circle sequence is effective at keeping children (and adults) looking at the center of the screen.

Task difficulty, manipulated by stimulus durations, was controlled using an adaptive paradigm (see Psychophysical Procedure). Stimulus duration was defined as the width at half height of a hybrid Gaussian temporal envelope (Tadin et al., 2011). Although this was not a reaction time task and participants did not receive instructions to respond as quickly as possible, we recorded

reaction time as a supplemental variable to analyze potential differences in how quickly participants respond to the trials. For example, a systematic difference in reaction times between the two groups of participants might be indicative of group differences in task strategy.

The experimental design included three conditions: mixed-contrast/small-size, mixed-size/high-contrast, and mixed-size/low-contrast. For the mixed-contrast/small-size condition (Fig. 1d), the stimulus radius was fixed at 1° with contrast spanning 8 levels, equally spaced from 2% to 99% in log space (contrast levels: 2%, 3.5%, 6%, 11%, 19%, 33%, 57%, and 99%). We used a logarithmic scale as for most visual stimuli, perceptual and neural responses tend to be proportional to the logarithm of stimulus intensity (i.e., they follow the Weber's law). Pilot testing confirmed that the lowest contrast level was visible to participants. For the mixed-size/high-contrast condition (Fig. 1d), all stimuli were set to a 99% contrast level and were presented at 8 sizes representing equal increases from 1° to 8° in log space (radius sizes: 1°, 1.3°, 1.8°, 2.4°, 3.2°, 4.4°, 5.9°, and 8°). The mixed-size/low-contrast condition was identical to the mixed-size/high-contrast condition, except all stimuli were set to a 2.3% contrast level. For each condition, participants completed a 96-trial practice block, followed by several 80-trial experimental blocks (5 blocks for mixed-contrast/small size, 4 for both mixed-size conditions). This difference in the number of blocks across conditions was due to differences in the number of free parameters in the model implemented in the adaptive stimulus presentation paradigm (see Psychophysical Procedure).

The selected stimulus space (detailed in the previous paragraph) was chosen to best test our mechanistic hypotheses (Fig. 1). Specifically, results consistent with the response gain control hypothesis should yield observable group differences at high contrast in the mixed-contrast/small-size condition and across all stimulus sizes in the mixed-size/high-contrast condition. On the other hand, results consistent with the receptive field size hypothesis should yield group differences across all stimulus contrasts in the mixed-contrast/small-size condition, and at the smallest size in the other two conditions.

Psychophysical procedure

Task difficulty was controlled by adjusting the stimulus duration to evaluate the minimum stimulus presentation duration for which each participant could reliably judge motion direction (i.e., duration thresholds; Tadin, Lappin, Gilroy, & Blake, 2003) at a given stimulus contrast and size level. Specifically, stimulus presentation was controlled using an adaptive psychophysical method, Functional Adaptive Sequential Testing (FAST; Vul, Bergsma, & MacLeod, 2010) that substantially increases efficiency in data collection (i.e., decreasing the number of trials by ~70%). Whereas other conventional adaptive psychophysical methods estimate the threshold at each contrast or size level independently, FAST estimates the full psychophysical function utilizing all data points, and, for each trial, selects the stimulus variable (e.g., stimulus duration) that increases the certainty about model parameters. The technique is shown to be effective in testing children and adolescents (Bogfjellmo, Bex, & Falkenberg, 2014) and therefore allowed us to collect the large amount of data necessary to most effectively test our hypotheses while still considering practical limitations of testing children with and without ASD.

For the mixed-contrast/small-size condition, we implemented the reciprocal of the Naka-Rushton function,

$$f(c) = R_{max}\frac{c^n}{c^n + c_{50}^n} + R_0, \quad (1)$$

where c is stimulus contrast. R_{max} determines the maximum response amplitude (and thus is related to the minimum duration threshold), c_{50} sets the semisaturation constant, n is the slope, and R_0 is the baseline response.

For the mixed-size/high- and low-contrast conditions, we implemented a descriptive function to characterize spatial suppression and summation patterns (Betts, Sekuler, & Bennett, 2009),

$$f(w) = p_1 w^{k_1} + p_2 w^{k_2}, \quad (2)$$

where w is stimulus size. The parameters p_1 and p_2 each determine the height of suppression and summation parts of the function, respectively, and k_1 and k_2 are the slopes (k_1 was fixed at -2; Betts et al., 2009). Note that Equations 1 and 2 were only used to increase the efficiency of data collection. Models fitted to this data are described at the end of the method section.

Psychophysical analysis

Although stimulus presentation was controlled with FAST, we estimated thresholds for each stimulus condition post hoc. This was done (a) to eliminate the possibility of biased threshold estimation in these types of adaptive techniques from erroneous responses in ASD (Dakin & Frith, 2005; Simmons et al., 2009) and (b) to allow statistical analyses that require independent estimates of data points. To obtain thresholds, a Weibull function (with a slope and threshold as free parameters) was fitted using Markov chain Monte Carlo (MCMC) techniques at each contrast and size level tested within each of the three conditions. Specifically, 10,000 samples for each parameter were obtained after burn-in using the JAGS (http://mcmc-jags.sourceforge.net/), an implementation of Gibbs

Sampler for Bayesian model analysis. Priors on the parameters were set to broad uniform distributions with their range large enough to cover all practically possible values. Threshold estimates for each condition were analyzed with 2 (group) × 8 (size or contrast) mixed model ANOVAs. Because we optimized our experimental design for estimating model parameters (detailed later) and keeping the total number of trials per subject low (to make the experiment feasible in our population), the resultant threshold estimates were noisy. We will consider this limitation when interpreting ANOVA results.

Model analysis

Model fitting determined the extent to which perceptual performance differences were driven by differences in response gain control or receptive field size. We used a model that characterized the interactions between receptive field center and surround, whose responses are dependent on stimulus contrast and size (Betts et al., 2012). This model was fitted to average thresholds obtained from the psychophysical analysis separately for each group using the least-squares procedure. The model has been shown to be effective in characterizing changes in motion sensitivity at varying stimulus contrasts and sizes and also in explaining perceptual differences in special populations, such as aging adults (Betts et al., 2012). Specifically, the model assumes an excitatory center (E) and an inhibitory surround (I),

$$E(w) = 1 - e^{-\frac{(\frac{w}{\alpha})^2}{2}}, \tag{3}$$

$$I(w) = 1 - e^{-\frac{(\frac{w}{\beta})^2}{2}}, \tag{4}$$

where w denotes stimulus size, and α and β determine the size of the receptive field center and surround, respectively. The responses of the center and surround are also modulated by stimulus contrast, defined by the Naka-Rushton function,

$$K_e(c) = A_e \frac{c^{n_e}}{c^{n_e} + c_{50_e}^{n_e}}, \tag{5}$$

$$K_i(c) = A_i \frac{c^{n_i}}{c^{n_i} + c_{50_i}^{n_i}}, \tag{6}$$

where c denotes stimulus contrast, and parameters determining response gain (A_e, A_i), semisaturation point (c_{50_e}, c_{50_i}) and slope (n_e, n_i) for each of the excitatory and inhibitory fields. The overall response is determined by the ratio of the responses in the center and surround through divisive inhibition,

$$R(w,c) = \frac{K_e(c) \cdot E(w)}{1 + K_i(c) \cdot I(w)}, \tag{7}$$

which is then converted to thresholds,

$$T = \frac{Criterion}{R_0 + R}. \tag{8}$$

Criterion and R_0 scale the neural response to perceptual thresholds, and were fixed at 20 and 6, respectively, following Betts et al. (2012).

To explore the robustness of this modeling approach, we fitted three different versions of the group-level model. In the main model, we estimated the receptive field sizes (α, β) and response gain parameters (A_e, A_i) for each group (the key parameters of interest), while treating other free parameters (c_{50_e}, c_{50_i}, n_e, n_i) to be the same across groups. We consider this our main model because it directly targets the two key hypotheses motivating this study (and it is also the model with the fewest free parameters). Second, we fitted a less constrained model where all of the free parameters were allowed to vary between the groups. Finally, we fitted a version of the main model where excitatory receptive field size was allowed to vary with contrast—a property that is consistent with physiological data (Cavanaugh, Bair, & Movshon, 2002; Kapadia, Westheimer, & Gilbert, 1999; Sceniak, Ringach, Hawken, & Shapley, 1999). Here, the size of the excitatory receptive field is determined by a decreasing logistic function (as in Betts et al., 2012; Tadin & Lapin, 2005),

$$\alpha(c) = \frac{S}{1 + m \cdot e^{(-\frac{k}{c})}}, \tag{9}$$

where the parameters S, m, and k determine the receptive field size change with stimulus contrast.

To estimate confidence intervals for model parameters and to evaluate group differences, we used a bootstrap procedure (Efron & Tibshirani, 2003). For each iteration, we randomly resampled participants' thresholds in each group (with replacement). When a participant was selected, we used the full set of thresholds over all stimulus contrasts and sizes for that participant. We then fitted the models on the average thresholds calculated from the resampled data. The procedure was repeated 1,000 times. The 95% confidence intervals and p values were extracted from the resampled distributions. We computed difference distributions (TD – ASD) for each model parameter; p values were determined by the proportion of samples that "crossed" zero.

To relate the model parameters to ASD symptoms and to further confirm our results, we also fitted the model for each individual participant using a maximum likelihood

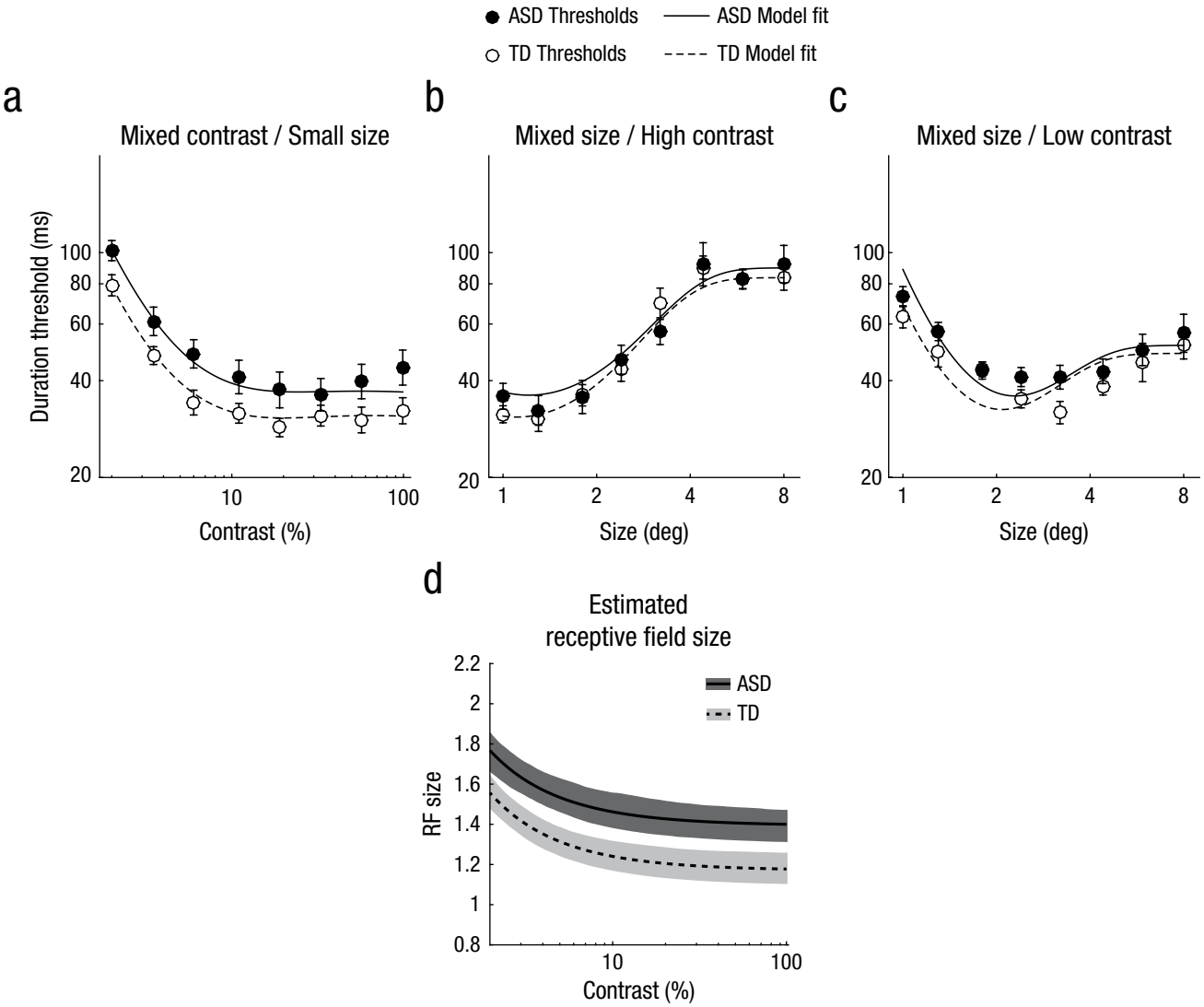

Fig. 2. Results from main experiments. (a–c) Perceptual thresholds across three experimental conditions for individuals with ASD and TD. Model fits to the psychophysical data from the main model are represented by the solid (ASD) and dashed (TD) lines. For the mixed-contrast/small-size condition (a), individuals with ASD showed higher thresholds (impaired motion sensitivity) across all contrast levels compared to those with TD. For the mixed-size/high-contrast condition (b) and the mixed-size/low-contrast condition (c), no group differences were observed. (d) Estimated excitatory receptive field size over a range of stimulus contrasts. For both ASD (solid line/dark gray) and TD (dashed line/light gray), estimated excitatory receptive field size decreased with increasing contrast. Lines represent the model estimate. Shaded regions indicate bootstrapped 68% confidence intervals. The excitatory receptive field size was significantly greater in ASD compared to TD.

estimation method. In this secondary analysis, we assumed that participants' performance at each contrast and size could be characterized by Weibull functions whose thresholds were determined by the full model with an identical slope for all stimulus levels. Group differences for estimated model parameters were tested using independent-samples *t* tests, providing a complementary way to test the robustness of our results. For parameters that showed group differences, estimated model parameters for each participant in the ASD group were used for the correlation analyses reported in the Results.

Results

Psychophysical results

The dependent variable of interest for these analyses is duration threshold (i.e., how long the stimulus needed to be visible for participants to accurately perceive motion direction), with higher values representing worse performance. For the mixed-contrast/small-size condition (Fig. 2a), both the ASD and TD groups showed differences in performance across contrast levels, $F(7, 266) = 66.4$, $p < .001$. Specifically, both groups showed decreasing thresholds

Table 1. Model Parameters

Group	Value	A_e	A_i	α	β	c_{50_e}	c_{50_i}	n_e	n_i
Autism spectrum disorder	Estimates	248.61	56.32	1.32	1.84	0.23	0.26	0.95	1.12
	95% CI	[242.68, 250.62]	[49.02, 67.91]	[1.19, 1.46]	[1.63, 2.05]	[0.13, 0.54]	[0.16, 0.72]	[0.74, 1.15]	[0.84, 1.35]
Typical development	Estimates	249.16	54.32	1.2	1.72				
	95% CI	[246.61, 253.56]	[48.2, 67.11]	[1.06, 1.33]	[1.54, 1.91]				
	p value	.42	.26	.009	.12				

from low to mid contrasts, and a plateau at high contrasts. There was a significant main effect of group, $F(1, 38) = 5.49$, $p = .02$, with participants in the ASD group performing worse than the TD group across all contrast levels. No significant interaction, $F(7, 266) = 0.46$, $p = .86$, was observed. As detailed later, this pattern of results is consistent with typically functioning gain control mechanisms and atypical receptive field size.

For the mixed-size/high-contrast condition (Fig. 2b), both groups performed worse with increasing stimulus size, $F(7, 266) = 72.26$, $p < .001$, showing a typical pattern of spatial suppression (Tadin, 2015; Tadin et al., 2003). No significant effect of group, $F(1, 38) = 0.04$, $p = .85$, nor an interaction, $F(7, 266) = 0.94$, $p = .48$, was observed. For the mixed-size/low-contrast condition (Fig. 2c), both groups showed improving performance with increasing stimulus size, $F(7, 266) = 19.05$, $p < .001$, consistent with spatial summation (Anderson & Burr, 1991; Tadin et al., 2003). There was no overall effect of group, $F(1, 38) = 1.94$, $p = .17$, and no significant interaction, $F(7, 266) = 0.60$, $p = .76$.

Taking all results together, we show that individuals with ASD have worse sensitivity to small moving stimuli, irrespective of stimulus contrast. Note that this should also lead to interactions in the mixed-size conditions. However, as our experimental approach is optimized for estimating model parameters and underpowered for estimating single thresholds (see the method section), we are also underpowered for detecting interactions that depend on deficits that are restricted to the smallest sizes.

Model results

The pattern of behavioral results (Figs. 2a–2c) is inconsistent with the general prediction from the response gain control hypothesis. However, it appears to support a receptive field size difference in ASD. These informal observations were confirmed by quantitative model analyses. To preview the results, all four analyses provided statistically significant evidence for larger excitatory receptive field size in ASD.

The main model provided a good fit to the data, $R^2 = .93$, $\chi^2(34, N = 46) = 3.66$, $p > .999$. However, in the mixed-size/low-contrast condition, the model had a tendency to underestimate the stimulus size at which the maximum sensitivity should occur in both ASD and TD (Fig. 2c). This is largely due to the fact that the main model does not allow the receptive field size to change with contrast, a consideration that has been shown to better characterize performance at low contrast (Tadin & Lappin, 2005). In fact, the version of the model where excitatory receptive field size changes with contrast improves the fit by better fitting the mixed-size/low-contrast condition (increasing R^2 for this condition from .43 to .82 and overall R^2 to .96)—a condition where we found no group differences.

The main model revealed that groups significantly differed only on the excitatory receptive field size parameter ($p = .009$), whereby the ASD group showed a significantly larger excitatory receptive field size (ASD = 1.32°, TD = 1.20°; Table 1). Neither the inhibitory receptive field size parameter nor the gain control parameters were different between groups (Table 1). Fitting a full model where all free parameters were estimated separately for each group did not change the results. Namely, the only significant parameter difference between the two groups was excitatory receptive field size ($p = .042$). Furthermore, when excitatory receptive field size was allowed to vary with stimulus contrast, estimated receptive field size from the model parameters was still significantly larger in ASD. Notably, this group difference was significant across all contrasts ($.008 < p < .011$; Fig. 2d). In this model, we also found a significant group difference in the inhibitory receptive field size estimate ($p = .031$). Finally, we fitted the main model for each participant and compared the model parameters between TD and ASD groups. This analysis again revealed a significant group difference only in excitatory receptive field size, $t(38) = 2.85$, $p = .007$; inhibitory receptive field size was not significant, $t(38) = 1.60$, $p = .12$.

In sum, for all four analyses, we find significant group differences in excitatory receptive field size, with larger receptive field size in ASD. We also find inconclusive evidence that this atypical enlargement might also extend to estimates of inhibitory receptive field size. Overall, our results indicating that excitatory receptive field size is

larger in ASD are consistent with Schwarzkopf et al. (2014), who found a group difference in excitatory center size but not in sizes of inhibitory surrounds. In the context of our study, this difference in excitatory receptive field size can explain reduced visual sensitivity to small moving stimuli in ASD (Fig. 2a).

Relationships of perceptual findings and clinical variables. To investigate possible relationships between our data and ASD symptoms, we correlated the excitatory receptive field size parameter and motion sensitivity for small stimuli (average threshold from mixed-contrast/small-size condition) with ADOS severity (Gotham, Pickles, & Lord, 2009; Hus & Lord, 2014) and ADI-R total scores in the ASD group. Excitatory receptive field size was not related to symptoms, ADI-R total score: $r(18) = -.04$, $p = .88$; ADOS severity score: $r(18) = -.19$, $p = .41$. Although worse motion sensitivity for small stimuli was weakly related to higher symptoms as measured by the ADI-R total score, $r(18) = .45$, $p = .05$, no relationship was found with the ADOS severity score, $r(18) = .34$, $p = .14$. In addition, these two correlations were not significantly different using Fisher's *r*-to-*z* transformation ($z = .36$, $p = .72$), further suggesting that motion sensitivity and ASD symptoms are not related, consistent with other reports (e.g., Foss-Feig et al., 2013).

Control experiment and additional analyses

Our findings show that individuals with ASD have worse sensitivity to small moving stimuli, which can be characterized by larger excitatory receptive fields. However, several additional analyses and a control experiment were conducted to rule out alternative explanations of our results.

Comparison with a more conventional staircase experimental design. One surprising finding in our study is the striking difference in the pattern of psychophysical results in comparison to Foss-Feig et al. (2013), despite the use of the same task and similar stimulus configurations. Slight differences included stimulus size range (1° to 8° in our task and 1° to 6° in Foss-Feig et al.), number of stimulus sizes (8 in our task and 3 in Foss-Feig et al.), and presentation display (projector in our study and CRT monitor in Foss-Feig et al.). However, the main difference between the two studies was the use of different adaptive threshold estimation procedures, with Foss-Feig et al. using a more conventional staircase design. To rule out the possibility that procedural differences affected the results, we repeated the mixed-size/high-contrast condition using a QUEST staircase design (Watson & Pelli, 1983), the staircase method used in the previous study. We also eliminated other experimental design differences, matching the range and number of stimulus sizes, the type of display and the number of participants, and even using the same experimenter to run the participants (i.e., we conducted an exact replication). Participants were 15 children and adolescents with ASD and 17 controls matched on age (ASD: $M = 13.8$, $SD = 1.7$; TD: $M = 13.8$, $SD = 2.3$), $t(30) = 0.07$, $p = .94$, and IQ (ASD: $M = 107.3$, $SD = 16.8$; TD: $M = 111.8$, $SD = 15.5$), $t(30) = 0.78$, $p = .44$. Results from this control experiment again revealed no group differences across all tested sizes at high contrast, $F(1, 30) = 0.80$, $p = .38$ (Fig. 3a). This matches our primary results and indicates that the differences between the present results and those by Foss-Feig et al. are unlikely to have been caused by methodological variations. In addition, analyses of thresholds for the three similar stimulus sizes across the different methods from our sample reveals that performance was not affected by methodological approach differences (all Fs < 1 for effects of method and method by size interaction for both TD and ASD; all ps > .34; Fig. 3b).

It is worth noting that the inconsistency between the Foss-Feig et al. (2013) result and our follow-up task result was driven primarily by differences in performance across the two ASD groups. The thresholds in the TD groups across the two sites were comparable, $F(1, 32) = 3.27$, $p = .08$. However, ASD participants in our current control experiment performed much worse compared to those in Foss-Feig et al., $F(1, 28) = 9.08$, $p = .005$, but at a similar level to both TD groups (Figure 3a). It is important to note that the two groups with ASD did not differ in age, $t(28) = 1.3$, $p = .20$, gender composition, $\chi^2(1, N = 30) = 1.03$, $p = .30$, IQ, $t(28) = 1.7$, $p = .10$, or diagnostic severity, ADI-R summary score: $t(28) = 0.63$, $p = .53$; ADOS severity score: $t(28) = 1.50$, $p = .15$. Together, this suggests that these individuals might not be distinguishable in terms of behavioral symptoms, but rather in terms of underlying neural mechanisms.

Effect of stimulus context on perceptual performance. Because group differences were seen in the mixed-contrast/small-size condition, but not in either of the mixed-size conditions, it is possible that the stimulus context (e.g., varying contrast in one condition and size in the other) differentially affected performance in ASD compared to TD. One way to directly test this is to compare thresholds at identical stimulus levels across different conditions. The smallest-size (1°), high-contrast (99%) stimulus was presented in both the mixed-contrast/small-size and mixed-size/high-contrast conditions, allowing us to perform this analysis (Figure 3c, circles). Paired *t* tests using the threshold estimates at this stimulus level across the two conditions revealed no differences based on stimulus context for the control group, $t(19) = 0.44$, $p = .66$,

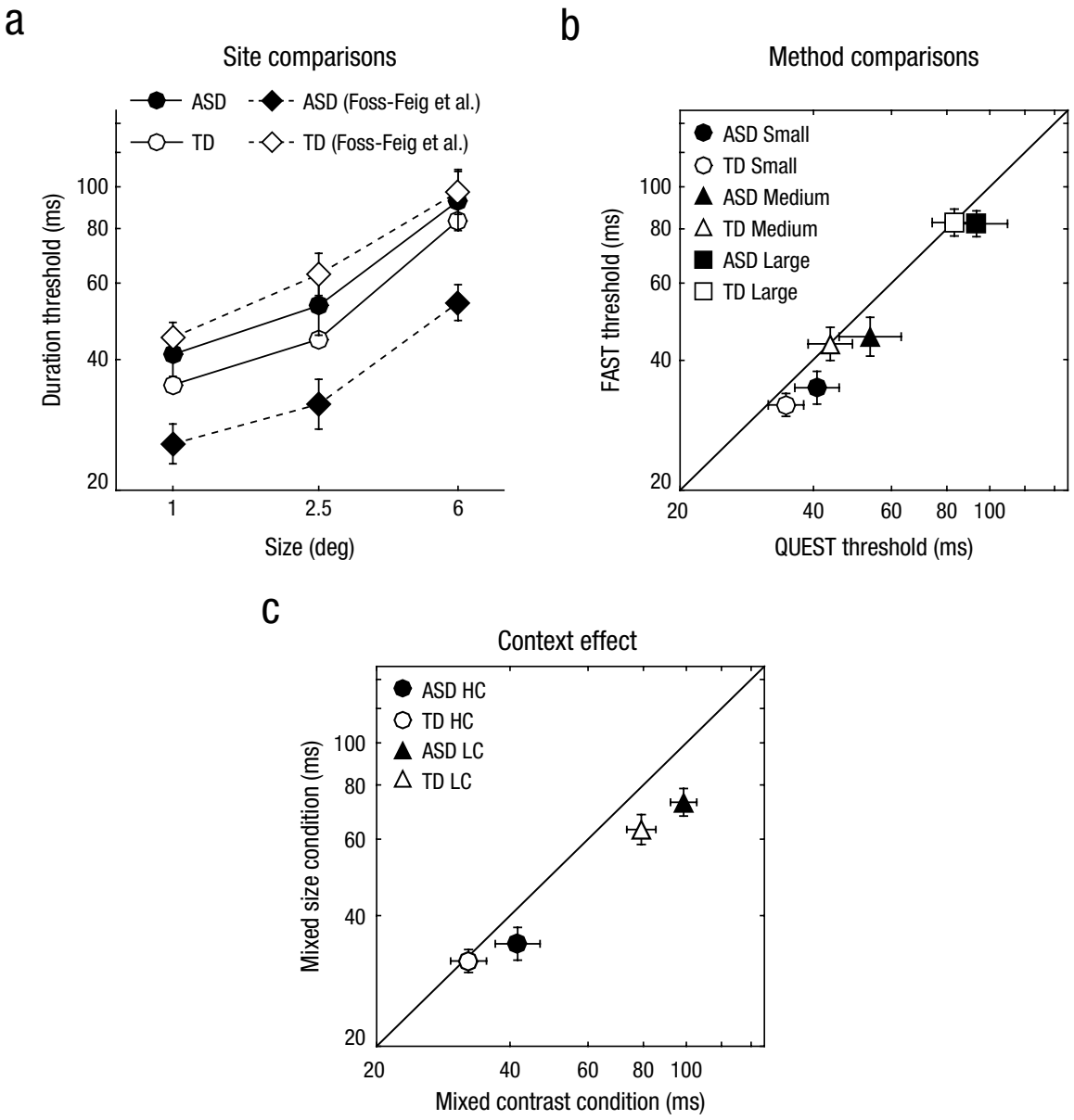

Fig. 3. Control experiment and supplemental analyses. (a) Exact replication of Foss-Feig et al. (2013), testing three stimulus sizes at high-contrast using the QUEST procedure. Results from Foss-Feig et al. are plotted for comparison (diamonds and dashed lines). Our results show no group difference in motion sensitivity, consistent with our results shown in Figure 3b. The ASD group from Foss-Feig et al. shows enhanced performance relative to the other three groups. See the results section for more details. (b) Comparison of results using different adaptive presentation methods (FAST versus QUEST). Data largely fall near the unity line, indicating no difference between the two procedures (all *p*s > .34). (c) Effects of stimulus context. As in panel b, data points on the unity line indicate no effects of stimulus context. Data show that for identical (high contrast, one size) stimuli, thresholds were largely unaffected by stimulus context, with ASD performing slightly worse in the mixed-contrast/small-size condition. Both groups show the predicted pattern of worse performance in the mixed-contrast/small-size condition where stimuli were presented at 2% contrast compared to the mixed-size/low-contrast condition where stimuli were presented at 2.3% contrast.

but a small and marginally significant effect in ASD, $t(19) = 2.10$, $p = .050$, with worse performance in the mixed-contrast/small-size condition compared to the mixed-size/high-contrast condition. Comparing changes in thresholds between the two conditions, we did not find a significant group difference between TD and ASD groups, $t(38) = 1.59$, $p = .12$.

At low contrast, stimuli were similar, yet not identical across the tasks; in the mixed-contrast/small-size condition the lowest contrast was 2% and in the mixed-size/low-contrast condition stimuli were presented at 2.3% contrast. With this limitation in mind, group means showed the expected pattern, with higher thresholds for 2% contrast compared to 2.3% contrast, across both groups

(Figure 3c, triangles); paired t tests revealed differences between these contrast levels in both groups: ASD, $t(19) = 4.73$, $p < .001$; TD, $t(19) = 2.80$, $p = .01$. Similar to high-contrast results, we did not find a significant group difference when we compared changes in thresholds between the two conditions, $t(38) = 0.97$, $p = .34$, a result arguing against differential effects of context.

To further explore possible effects of stimulus context, we performed a more fine-grained analysis to test whether variations in stimulus contrast or size from the previous trial affected performance on the subsequent trial. This analysis was motivated by results with neurotypical subjects showing that previous trials affect behavioral performance even when trials are fully independent (Abrahamyan, Silva, Dakin, Carandini, & Gardner, 2016). This analysis revealed that both groups, across both tasks, were equally likely to choose correctly on a trial regardless of the preceding visual stimulus, mixed-contrast/small-size: $F(1, 38) = 0.45$, $p = .51$; mixed-size/high-contrast: $F(1, 38) = 3.08$, $p = .087$. In addition, analyses of reaction time data revealed no group differences in reaction time, mixed-contrast/small-size mean reaction time: ASD = 0.47 s, TD = 0.52 s, $F(1, 38) = 0.97$, $p = .33$; mixed-size/high-contrast mean reaction time: ASD = 0.49 s, TD = 0.51 s, $F(1, 38) = 0.209$, $p = .65$, suggesting that all tasks required similar levels of effort across both groups, at least by this gross measure of task difficulty. Thus, at the task level there is some evidence, albeit weak, for an effect of stimulus context in ASD; however, these differences are not substantial enough to account for impaired performance in ASD with small sized stimuli across a wide range of contrast levels.

Discussion

In individuals with ASD, we aimed to investigate the integrity of two fundamental neural mechanisms—response gain control and receptive field size—using psychophysical and computational approaches. Results revealed impairment in motion processing in ASD that was specific to small stimulus sizes, across all contrast levels. Computational modeling showed that this pattern of results could be explained by increased receptive field size in ASD. These findings complement a recent fMRI study showing larger population receptive fields in adults with ASD (Schwarzkopf et al., 2014). However, results are inconsistent with the prediction from the impaired response gain control hypothesis and with the previously reported finding of enhanced motion sensitivity in children with ASD (Foss-Feig et al., 2013). Our results add to the existing literature on atypical motion processing in ASD, while linking perceptual deficits with possible impairments in underlying mechanisms. In the context of existing literature, this study, as detailed later, raises the possibility of subgroups within ASD that may be characterized by impairments in distinct mechanisms.

Our psychophysical results revealed selective motion perception deficits in ASD for the smallest stimulus size (radius = 1°). This finding provides a level of specificity to the motion perception deficits in ASD. Namely, for brief stimulus presentations, deficits exist only for small stimulus sizes (Sysoeva et al., 2017). In fact, Bertone et al. (2003) showed intact motion perception in ASD using a stimulus size of 5°, corroborating our findings of intact motion perception at larger stimulus sizes. However, other studies with similar stimulus configurations show impaired motion perception at larger sizes (Koh et al., 2010; Takarae et al., 2008), suggesting some heterogeneity among individuals with ASD. Differences in language delay have been proposed as a possible explanation of heterogeneity in motion perception abilities in ASD. Specifically, Takarae et al. (2008) divided their ASD group based on history of language delay, and revealed impaired versus intact motion sensitivity in those with versus without a language delay, respectively. However, exploratory analyses with our data do not support differences in motion sensitivity based on language delay. We split the participants into those with ($n = 9$) and without language delay ($n = 11$) as described in Takarae et al. (2008). There were no group differences or group by stimulus interactions in all three tasks tested in this study (all ps > .32). Furthermore, it remains unclear why or how these perceptual abilities and language development might be linked.

Another possibility underlying this discrepancy relates to subgroups characterized by different mechanistic impairments. An unexpected aspect of our results was the markedly different motion sensitivity in ASD compared to Foss-Feig et al. (2013). They showed enhanced motion perception at high, but not low contrast, using a paradigm that differed methodologically in only very subtle ways to our mixed-size conditions. As a control experiment, we conducted an exact replication of the Foss-Feig et al. methods for the mixed-size/high-contrast condition; the results were consistent with our main findings but not with Foss-Feig et al. Namely, the ASD group performed worse whereas the TD groups performed similarly across sites. This rules out the effects of methodological differences, and instead suggests that the inconsistency across studies likely arises from variability in individuals with ASD themselves (Heeger et al., 2017). Rosenberg et al. (2015) have previously shown that the enhanced motion sensitivity at high contrast in ASD observed in Foss-Feig et al. can be explained by reduced suppressive gain in a model that implements divisive normalization. Similarly, reduction in A_i (Equation 6) in our model, which represents the gain in the inhibitory surround field, would yield similar enhancement in motion sensitivity as in Foss-Feig et al. However, reduction in this parameter was not found in our study. Such observations, together with different patterns of motion sensitivity in ASD not explained by methodological

differences, suggest that subgroups may exist within ASD, whose atypicality in motion perception is characterized by distinct mechanisms.

Our results revealed larger excitatory receptive field size in ASD, which could provide a mechanistic explanation for impaired motion sensitivity with small stimuli across different contrast levels. Our receptive field size estimates are roughly in the middle of two often-cited estimates of foveal receptive fields in area MT of macaques (Albright & Desimone, 1987; Raiguel, Van Hulle, Xiao, Marcar, & Orban, 1995), and receptive field sizes in macaque tend to be similar to those in humans (Kastner et al., 2001). Previous studies that utilized our motion paradigm also implicate area MT as a potential neural locus (see Tadin, 2015, for review). However, our methods most likely estimate the size of the "perceptive" region, which we believe reflect population responses—responses that will reflect contributions of multiple brain areas. Here, we speculate that MT is a strong contributor among the several visual processing areas that are likely involved.

Broadly, larger receptive fields have several implications for perception and clinical features of ASD. Notably, larger receptive fields can facilitate the integration of local information (Anton-Erxleben & Carrasco, 2013). Receptive fields at each visual processing stage pool inputs from lower level visual areas, with larger receptive fields being more suitable for integrating visual information, and potentially increasing our ability to detect the presence of moving stimuli (Niebergall, Khayat, Treue, & Martinez-Trujillo, 2011). In fact, a recent motion coherence study showed enhanced motion integration abilities in ASD (Manning et al., 2015). Although this finding might appear in contrast to impaired motion coherence abilities in ASD commonly observed in the literature (e.g., Koldewyn, Whitney, & Rivera, 2010; Milne et al., 2002; Milne et al., 2006; Pellicano, Gibson, Maybery, Durkin, & Badcock, 2005; Robertson et al., 2012), these studies typically require segregation of "noise" dots from "signal" dots (Manning et al., 2015), which may, in fact, be more difficult with larger receptive fields. Specifically, when the stimulus consists of a coherent signal (i.e., no added noise), integration over a larger area within the receptive fields is beneficial. However, when the stimulus contains both signal and added noise (e.g., a commonly used motion coherence task), segregation becomes a better strategy (Braddick, 1993). There is evidence that motion segregation, at least in part, may rely on feedback connections from higher-order areas (Raudies & Neumann, 2010). This is notable because ASD has been characterized by reduced feedback connectivity (Kana, Libero, & Moore, 2011), which may interfere with segregation, but leave the process of integration intact or even enhanced. Another factor to consider is stimulus duration. Robertson and colleagues (2012) reported elevated motion coherence thresholds in ASD for short presentation times (200 ms), but not for longer durations (1,500 ms). Although our stimuli were brief (~100 ms), stimulus duration cannot explain our pattern of results, as elevated thresholds were only observed for a subset of stimuli. Rather, it appears that duration is a factor in the motion coherence measurements.

Receptive field size is often linked with sensory acuity, where smaller receptive fields are known to support finer spatial resolution. This is relevant as there are reports of enhanced visual acuity (Ashwin, Ashwin, Rhydderch, Howells, & Baron-Cohen, 2009) and theories of detailed-focused processing in ASD (Dakin & Frith, 2005; Fitch, Fein, & Eigsti, 2015; Happe & Frith, 2006; Lopez & Leekam, 2003). These accounts predict sharper spatial selectivity, which may result from smaller receptive fields across visual processing regions, but most notably V1, a region with relatively small receptive fields. Although our study was not designed to estimate small receptive fields that underlie fine visual acuity, it is worth noting that enhanced visual acuity reports in ASD have been questioned. Schwarzkopf et al. (2014) did not find any V1 receptive field size differences in ASD and initial accounts of enhanced visual acuity at the perceptual level have since been critiqued (Bach & Dakin, 2009; Crewther & Sutherland, 2009) and empirically contradicted (Bolte et al., 2012). Alternatively, several additional higher cognitive factors (e.g., attention, memory) may contribute to an ASD preference for details over the gestalt (Beversdorf et al., 2000; Mottron, Dawson, Soulieres, Hubert, & Burack, 2006; Van der Hallen, Evers, Brewaeys, Van den Noortgate, & Wagemans, 2015). It is likely that an interaction among several of these factors lead to the detailed-focused processing patterns associated with ASD.

Possible alternative accounts for our findings may involve differences in fixational eye movements or attentional impairments in ASD. Although we did not measure eye movements, participants were monitored by an experimenter and no gross deviations in fixation behavior were observed. Moreover, given that our stimuli were very brief, saccades and even fast reflexive eye movements are not a concern (Glasser & Tadin, 2014). However, neither observation rules out smaller systematic deviations in central fixation. For our motion task, fixation away from center should actually improve performance for high-contrast stimuli (Nyquist, Lappin, Zhang, & Tadin, 2016; Tadin et al., 2003), and because stimulus sizes were intermixed, it would do so across all stimulus sizes, which is inconsistent with our findings. Taken together, we believe that eye movements cannot explain our findings. Atypical attentional deployment in ASD (Ohta et al., 2012; Robertson, Kravitz, Freyberg, Baron-Cohen, & Baker, 2013; Schwarzkopf et al., 2014) cannot be completely ruled out as an explanation for our findings. Attention has been shown to decrease receptive

field size (de Haas, Schwarzkopf, Anderson, & Rees, 2014); however, attention-modulated increases in receptive field size have also been observed (Sprague & Serences, 2013). In addition, the extent to which individuals with ASD exhibit attentional differences in simple sensory discrimination tasks remains unclear (Haigh et al., 2016). Critically, attentional differences should also affect our estimates of response gain control (Carrasco, 2011; Hillyard, Vogel, & Luck, 1998), which we did not observe in the present study. Thus, it is unlikely that group differences in attention can fully explain our findings. However, additional studies that carefully and explicitly manipulate attention are necessary to fully understand effects of attention on receptive field size and motion perception in ASD.

Sensory hyper- and hyposensitivity are common symptoms of ASD that are assumed to result at least in part from neural hyper- and hypo- responding, respectively. Our data, implicating larger receptive field size in ASD, suggests decreased neural sensitivity to small moving objects (Figure 1a), which in turn could be related to symptoms of sensory *hyposensitivity*. On the other hand, deficits in response gain control, observed in other studies (Foss-Feig et al., 2013; Pei, Baldassi, & Norcia, 2012; Rosenberg et al., 2015), would predict increased neural responding (Figure 1a), which in turn could be related to symptoms of sensory *hypersensitivity*. Thus, differences in these mechanisms not only have implications for differences in perceptual sensitivity, but may also contribute to differences in sensory symptoms that impact individuals day to day. Given that both of these mechanisms have been linked to the E/I balance (Foss-Feig et al., 2013; Pei et al., 2012; Schwarzkopf et al., 2014), E/I imbalance may be an underlying impairment in ASD that manifests through different mechanisms that in turn lead to different symptom presentations (Robertson, Ratai, & Kanwisher, 2016). If this is indeed the case, heterogeneity in sensory sensitivity might account for differences between our sample and that of Foss-Feig et al. (2013).

In sum, we found evidence for reduced sensitivity selective to small moving stimuli in ASD that could be characterized by increased visual receptive field size. Our findings begin to uncover various task-based (methodological) and participant-based (mechanistic) sources that likely contribute to the large variability in motion sensitivity in ASD reported in the literature (Heeger et al., 2017). The findings were made possible by the use of advanced and well-designed psychophysical paradigms that can be easily implemented in children and special populations. The model analysis added to the study by allowing us to test different mechanistic accounts and compare the findings with existing theories in the literature. Differences in receptive field size are at such a basic level that they affect how visual information is processed and thus how the visual world is perceived. In turn, these differences likely contribute to many of the sensory, cognitive, and behavioral differences observed in ASD. Future studies should build on the present findings to further uncover the nature of heterogeneity in visual perception in ASD, and to investigate how these basic perceptual processes influence core behavioral symptoms of ASD.

Author Contributions

K. B. Schauder and W. J. Park contributed equally as joint first authors. D. Tadin and L. Bennetto contributed equally as joint senior authors. All authors contributed to the study concept and design. Testing, data collection, data analysis, and interpretation were performed by K. B. Schauder and W. J. Park under the supervision of D. Tadin and L. Bennetto. K. B. Schauder and W. J. Park drafted the manuscript, and D. Tadin and L. Bennetto provided critical revisions. All authors approved the final version of the manuscript for submission.

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Declaration of Conflicting Interests

The authors declared that they had no conflicts of interest with respect to their authorship or the publication of this article.

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