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Unimpaired Attentional Disengagement and Social Orienting in Children With Autism

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Abstract

Visual attention is often hypothesized to play a causal role in the development of autism spectrum disorder (ASD). Because attention shapes perception, learning, and social interaction, early deficits in attention could substantially affect the development of other perceptual and cognitive abilities. Here we test two key attentional phenomena thought to be disrupted in autism: attentional disengagement and social orienting. We find in a free-viewing paradigm that both phenomena are present in high-functioning children with ASD ($n = 44$, ages 5–12 years) and are identical in magnitude to those in age- and IQ-matched typical children ($n = 40$). Although these attentional processes may malfunction in other circumstances, our data indicate that high-functioning children with ASD do not suffer from across-the-board disruptions of either attentional disengagement or social orienting. Combined with mounting evidence that other attentional abilities are largely intact, it seems increasingly unlikely that disruptions of core attentional abilities lie at the root of ASD.

Keywords

ASD, autism, attention, social attention, social orienting, disengage, eye tracking, free viewing, face perception, vision

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A central puzzle of autism is how a highly heritable disorder can produce such a specific and uneven cognitive profile, characterized not only by the signature deficit in social communication, but also by other apparently unrelated features such as restricted interests (Szatmari et al., 2006) and the tendency to line up objects in a row (Turner, 1999). The most parsimonious theories of autism attempt to account for the full phenotype as resulting from a single, early-developing cognitive deficit. Featuring prominently among such theories is the idea that attention, our ability to selectively process a small subset of the sensory information impinging on our senses, is impaired in autism. Because attention is our window to the world, determining what we experience, respond to, and remember, an early-developing disorder of attention might have a far-reaching impact on cognitive development. Indeed, many have argued that the core clinical symptoms of autism spectrum disorder (ASD)—impairments in social interaction and communication—result

from differences in how people with autism attend to the world during infancy and childhood (Dawson et al., 2004; Keehn, Müller, & Townsend, 2013; Loveland & Landry, 1986; Maestro et al., 2002; Toth, Munson, Meltzoff, & Dawson, 2006). Here we test two of the most widely discussed attentional hypotheses of autism: that individuals with autism have atypical (a) attentional disengagement and (b) social orienting.

Impaired attentional disengagement is a prime suspect in the etiology of autism because it could neatly explain many of the diverse symptoms of ASD. Landry and Bryson (2004) reported a striking disengagement impairment in ASD: They found that it takes children with ASD more than 3 times as long as typically developing (TD)

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controls to disengage attention from a visual stimulus. They likened the attentional capacity of their 6-year-old children with autism to the “sticky” attention of TD 2- to 3-month-olds (Hood & Atkinson, 1993; Johnson, Posner, & Rothbart, 1991), which, if true, could provide a powerful account for the well-known tendencies of children with ASD to develop restricted interests (Szatmari et al., 2006), to fail to respond to their own name (Nadig et al., 2007), and to become fixated on single objects or tasks (Turner, 1999). The disengagement hypothesis features prominently in current theories of autism (Keehn et al., 2013; Menon, 2011).

However, the prominence of the theory has not been matched with equal empirical evidence. Studies that require attentional disengagement (those that instruct participants to make a saccade to new stimuli) have found equivalent or faster disengagement in ASD participants than in controls (Kelly, Walker, & Norbury, 2013; Kikuchi et al., 2011; van der Geest, Kemner, Camfferman, Verbanen, & van Engeland, 2001), suggesting that the fundamental mechanism of disengagement may be intact in ASD.¹ Studies using free-viewing paradigms have supported a disengagement deficit in ASD, but two issues weaken this conclusion. Most significant, these studies either tested children with low IQ (Landry & Bryson, 2004) or did not control for IQ because of the very young age of the participants (Elsabbagh et al., 2013; Zwaigenbaum et al., 2005). This confound is problematic because global developmental delay is associated with increased failure to disengage (Chawarska, Volkmar, & Klin, 2010). Studies using free-viewing paradigms in infant siblings of children with ASD have reported slow disengagement (Elsabbagh et al., 2009; Elsabbagh et al., 2013), but used stimuli that confound sensitivity to change with attentional disengagement (see the discussion section). Thus, in spite of the prominence of the attentional disengagement account of ASD, there is no clear answer to the basic question of whether disengagement deficits are present in children with ASD independently of global developmental delays. Here, we test this question using an unconstrained free-viewing paradigm, matching the conditions under which apparent disengagement deficits have been previously reported, but now testing high-functioning children with ASD and age- and IQ-matched TD children.

According to the social orienting hypothesis, a failure of individuals with autism to prioritize social information gives rise to the cognitive profile of ASD: Given that it is from other people that we learn much of what we know, a failure to preferentially attend to social stimuli could lead to far-reaching cognitive deficits (Klin, Jones, Schultz, & Volkmar, 2003; Mundy & Rebecca Neal, 2000; Schultz, 2005). The social orienting hypothesis has wide appeal for its parsimony and anecdotal agreement with

behaviors observed in autism, but evidence for the hypothesis is inconsistent. Some studies have reported that toddlers with ASD spend less time looking at faces than TD toddlers do (Chawarska & Shic, 2009), that toddlers (Chawarska et al., 2010) and adolescents (Kikuchi et al., 2011) with ASD are faster to disengage attention from a face, and that even when individuals with autism do look at faces, they show different patterns of eye fixation (Snow et al., 2011), focusing more on mouths than eyes relative to typical participants (Jones, Carr, & Klin, 2008). Yet other studies report that children and young adults with ASD prioritize social stimuli to the same degree as TD participants when searching a scene (Fletcher-Watson, Leekam, Findlay, & Stanton, 2008; New et al., 2010; Sheth et al., 2011) and that infants who later develop ASD exhibit the same attentional capture by faces as their TD counterparts, and even spend more time overall looking at faces than TD controls do (Elsabbagh et al., in press). Thus, it remains unclear whether social orienting is truly impaired in ASD, and whether social orienting impairments may have any role in the development of the signature characteristics of autism.

The goal of this study is to test two of the most parsimonious and widely embraced accounts of the etiology of autism: attentional disengagement and social orienting. First, do children with ASD have “sticky” attention, independently of global developmental delay? Second, do children with ASD fail to prioritize social stimuli when orienting attention? To answer these two questions, we tested a large group of well-characterized children with ASD ($n = 44$) and age- and IQ-matched TD children ($n = 40$) in a free-viewing paradigm.

Method

All experimental protocols were approved by the MIT Institutional Review Board.

Participants

Our participant pool comprised 44 children with ASD and 40 TD controls, matched on both chronological age and nonverbal IQ, measured by the second edition of the Kaufman Abbreviated Intelligence Test (Kaufman & Kaufman, 2004). Control participants were drawn from an original pool of 70 total TD children tested on this task; this original TD pool had an above-average mean nonverbal IQ, necessitating the exclusion of those with the highest IQ to equate the mean IQ across groups. We kept all ASD participants and removed the smallest number of TD participants necessary to yield an IQ match ($p > .1$). In the resulting groups, mean ages were 9.2 years for ASD children ($SD = 1.7$) and 8.6 years ($SD = 2.1$

for TD children (no significant difference; $p = .15$, Cohen's $d = 0.32$), and mean performance IQ was 108.8 for ASD ($SD = 16.2$) and 113.6 ($SD = 13.2$) for TD (no significant difference; $p = .14$, $d = -0.32$). Neither age nor IQ was correlated with our attentional measures, indicating that excluding participants to make the mean age or IQ more similar across groups would not alter our findings (see Figure S1 in the Supplemental Material available online). Analyzing the original group of 70 TD children yielded results consistent with those we report here. Six (14%) ASD participants were female and six (15%) TD participants were female. The same pattern of results was observed in each gender when analyzed separately.

Children with ASD had a diagnosis according to the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV; American Psychiatric Association, 2000) from a trained clinician and met the criteria for ASD or Autistic Disorder on the Autism Diagnostic Observation Schedule (ADOS) administered by a research-reliable psychologist (Lord et al., 2000; see Table S1 in the Supplemental Material for a summary of ADOS scores). Children with autism were recruited through the Simons Foundation and the Boston Autism Consortium. TD children were recruited from the local community. Potential participants were excluded if they had any history of birth or brain trauma, noncorrected

visual impairments, or a nonverbal IQ of less than 80. All participants had normal or corrected to normal vision. Participants received modest monetary compensation as well as small motivating prizes for their participation.

Stimuli and task design

Stimuli were presented on a 17-inch LCD monitor with a built-in eye-tracking camera (Tobii T120 eye tracker; Tobii Technology, Stockholm, Sweden). Participants viewed the stimuli from a chin rest positioned 52 cm from the screen. We measured attentional disengagement and social orienting using the "gap-overlap" paradigm (Reulen, 1984a, 1984b; Reuter-Lorenz, Hughes, & Fendrich, 1991; Saslow, 1967). Figure 1 depicts the series of events in each trial: Each trial began with a 1-s presentation of a blank white screen, followed by the appearance of a color photograph of a face or object in the center of the screen (7 degrees \times 7 degrees). After a delay of 1 or 2 s, a peripheral color photograph appeared at 14 degrees eccentricity, randomly to the left or right of fixation. Variability (1 or 2 s) in the peripheral stimulus onset time kept participants from anticipating exactly when it would appear and making anticipatory saccades. Children were instructed to simply "look at the pictures in whatever way you want to, but pay attention to the screen the whole time." Each child

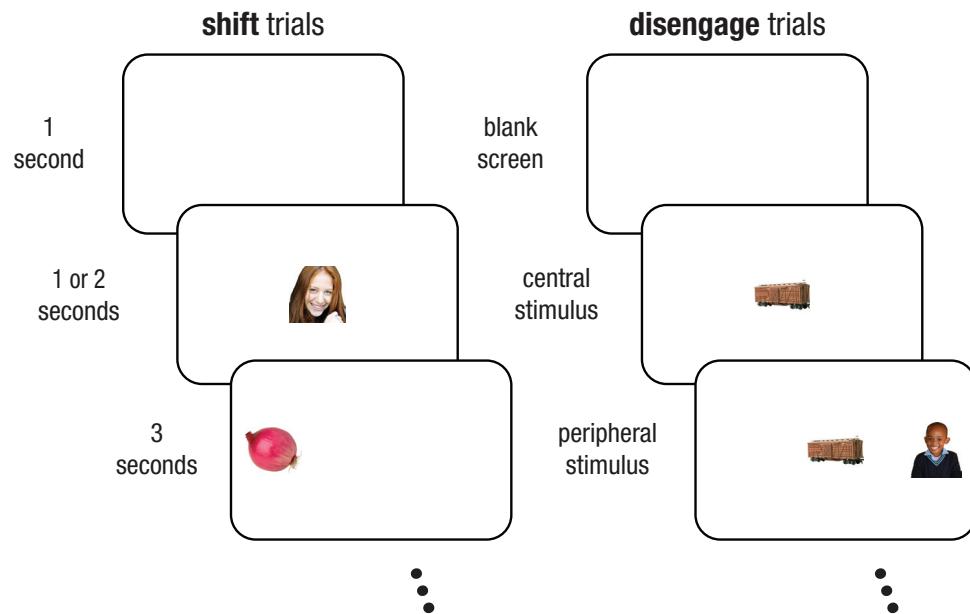


Fig. 1. Series of events in each trial. A stimulus appeared at the center of the screen first for 1 or 2 s, and then a second stimulus appeared at 14 degrees in the periphery. In "shift" trials, the central stimulus disappeared at the onset of the peripheral stimulus, so no "disengagement" was required. In "disengage" trials, the central stimulus remained on-screen for the entire trial duration, requiring participants to disengage attention from the central stimulus to move their eyes to the periphery. Both the central and peripheral stimuli were either social images (faces) or nonsocial images (fruits, vegetables, or trains), manipulated independently.

completed two runs of 64 trials each, for a total of 64 shift trials and 64 disengage trials.

Four factors were manipulated orthogonally in a $2 \times 2 \times 2 \times 2$ design. First, each trial was either a “shift” trial or a “disengage” trial: In shift trials, the central stimulus disappeared at the onset time of the peripheral stimulus, allowing participants to freely shift attention to the periphery. In disengage trials, the central stimulus remained on-screen until the end of the trial; saccading to the peripheral stimulus necessitated disengaging attention from the central stimulus. Second, the central stimulus was either social (a face) or nonsocial (a fruit, vegetable, or train). Third, the peripheral stimulus was either social or nonsocial. Stimuli were never repeated; each child saw a total of 256 unique photographs over the course of the experiment. We chose these stimulus categories to provide a broad sampling of the kinds of images that might elicit attentional differences between ASD and TD children. For example, vehicles may be of particular interest to some children with autism; the inclusion of trains provided a strong test of whether stimuli of interest could interfere with disengagement in children with ASD. Presenting unique images on every trial eliminated potential confounds stemming from stimulus repetition, for example by priming (Maljkovic & Nakayama, 1994). Finally, as noted earlier, the second stimulus appeared after the first stimulus had been displayed for either 1 s or 2 s. All trials were randomly interleaved within a run, but trials were presented in the same order for every participant to avoid any potential influence of differences in stimulus order across groups.

Eye tracking

Gaze position was recorded at 120 Hz using a Tobii T120 eye tracker. Prior to the start of each run, participants completed a brief calibration procedure to ensure accurate tracking. Left and right eye gaze positions were recorded separately, but for subsequent analyses, to improve the quality of the data, the left and right eye positions were averaged to determine gaze position. Most studies of attentional disengagement in ASD have used less precise eye-tracking measures, for example, electro-oculography or video recording. The use of an infrared eye-tracking system in this study yielded richer data than those produced in many previous studies, allowing us to examine not only when disengagement occurred, but also whether eye gaze patterns to individual stimuli differed between groups (see Figure S3).

Saccadic reaction time analysis

To measure attentional disengagement cost, we measured saccadic reaction times (SRTs), defined as the time

between the onset of the peripheral stimulus and the first eye gaze measurement that fell within the peripheral stimulus. We included only trials that met three quality criteria: (a) No more than 25% of eye gaze measurements were missing (not properly read from the eye tracker) in that trial, (b) the child was looking at the central stimulus location during the last 250 ms prior to the peripheral stimulus onset, and (c) the child made an eye movement to the peripheral stimulus within 2 s after its onset. The number of trials thrown out because of these quality criteria did not differ across groups (ASD: an average of 19.9% of trials per child, $SD = 15.2\%$ across participants, TD: 16.0% of trials, $SD = 14.8\%$; $p = .09$, $d = 0.26$). If fewer than half of a participant’s trials were usable, the child was excluded from the analysis (47 ASD and 43 TD children were originally analyzed and 3 from each group were excluded based on these criteria). In the participants retained for analysis, the number of discarded trials did not differ across conditions in our tests of disengagement or social orienting, $F(1, 82) = 0.22$, $p = .64$, $\eta_p^2 = .003$, for disengagement, $F(1, 82) = 2.40$, $p = .13$, $\eta_p^2 = .028$, for social orienting, and there was no group difference in the distribution of discarded trials across conditions, $F(1, 82) = 0.076$, $p = .78$, $\eta_p^2 = .001$, for the Group \times Condition interaction in disengagement, $F(1, 82) = 0.019$, $p = .89$, $\eta_p^2 < .001$, for the Group \times Condition interaction in social orienting. In all, 45.5% of ASD children and 45.0% of TD children had at least one trial excluded from the analysis based on the third (long SRT) criterion (no group difference; $p = .50$, $d = 0.059$). Among participants who had one or more trials excluded because of long SRTs, the mean number of excluded trials was 1.45 in ASDs and 1.67 in TDs (no group difference; $p = .27$, $d = -0.12$).

Disengagement costs, that is, the extra time that it took participants to orient to the peripheral stimulus when the central stimulus remained on-screen, were computed as the mean SRT from disengage trials minus the mean SRT from shift trials. Error bars on the mean SRTs and disengagement costs were obtained through a bootstrapping analysis (Efron, 1981): On each of 5,000 iterations, the data were resampled with replacement across participants and the group statistic was recomputed; error bars show ± 1 standard deviation of the bootstrapped distribution. Similarly, significance tests were conducted using permutations tests (Pitman, 1937), which characterized the null distribution by randomly assigning the group labels on each of 5,000 iterations. We used this nonparametric significance test for our planned comparisons to avoid assuming normality of the SRT distribution in our sample. ANOVA analysis showed the same pattern of results. Effect sizes for tests of differences are reported as Cohen’s d and partial eta-squared, and effect sizes for tests of association are reported as Pearson’s r .

Results

Saccadic reaction times

Our key dependent measure was SRT: How long after the peripheral stimulus appeared did viewers take to bring their eyes onto that peripheral stimulus? We conducted a repeated measures ANOVA with SRT as the dependent measure; trial type (shift vs. disengage), central stimulus duration (1 or 2 s), central stimulus type (social vs. nonsocial), and peripheral stimulus type (social vs. nonsocial) as within-subject effects; and group (ASD vs. TD) as a between-subjects effect, along with all one-to five-way interactions. See Table S2 in the Supplemental Material for a breakdown of SRTs by condition. There were significant main effects of trial type, $F(1, 82) = 198.10$, disengage slower than shift ($p < .0001$, $\eta_p^2 = .71$) and peripheral stimulus type, $F(1, 82) = 32.22$, shorter SRTs to social peripheral stimuli ($p < .0001$, $\eta_p^2 = .28$). The main effects of central stimulus type, $F(1, 82) = 2.15$, $p = .15$, $\eta_p^2 = .026$, central stimulus duration, $F(1, 82) = 3.56$, $p = .063$, $\eta_p^2 = .042$, and group, $F(1, 82) = 2.18$, $p = .14$, $\eta_p^2 = .026$, were not significant. One interaction, Disengagement Trial Type \times Peripheral Stimulus Type, was significant, $F(1, 82) = 5.47$, $p = .022$, $\eta_p^2 = .063$: Participants showed smaller disengagement costs when a social stimulus appeared in the periphery. Crucially, there was no interaction of group with any factor, Group \times Trial Type, $F(1, 82) = 0.74$, $p = .39$, $\eta_p^2 = .009$, Group \times Central Stimulus Duration, $F(1, 82) = 0.20$, $p = .65$, $\eta_p^2 = .002$, Group \times Central Stimulus Type, $F(1, 82) = 0.004$, $p = .95$, $\eta_p^2 < .001$, Group \times Peripheral Stimulus Type, $F(1, 82) = 0.067$, $p = .80$, $\eta_p^2 = .001$. All higher-order interaction effects were not significant ($p > .05$, $\eta_p^2 < .05$). Given the absence of significant group interactions, we proceeded with the planned comparisons to test for attentional disengagement and social orienting impairments in children with ASD.

Figure 2a shows SRTs and disengagement costs for trials in which both the central and peripheral stimuli were nonsocial images—within these trials we tested for a disengagement deficit in children with ASD independent of any influence of social stimulus content. Because the central stimulus duration did not interact with any other factor and was varied to make the peripheral stimulus timing unpredictable, we collapsed across the two durations. If children with ASD have impaired attentional disengagement, they should show a larger disengagement cost than TD children (Figure 2a). On the contrary, although each group independently showed significant disengagement costs (both $p < 0.001$; permutation tests; see the method section; $d = 1.06$ for ASD and $d = 1.10$ for TD), the disengagement cost did not differ across groups ($p = .52$, $d = 0.024$). Whether the nonsocial stimuli were trains or fruits/vegetables had no effect on SRTs in either

group (central stimulus—ASD: $p = .47$, $d = 0.025$; TD: $p = .12$, $d = 0.25$; peripheral stimulus—ASD: $p = .36$, $d = 0.11$; TD: $p = .15$, $d = 0.23$). Disengagement cost also did not differ across groups when trials with long SRTs were included in the analysis (no filtering by SRT; $p = .74$, $d = 0.01$ for the group difference), nor was there a group difference in disengagement cost when other stimulus types appeared in the central or peripheral locations ($p = .41$, $d = -0.009$ for social \rightarrow nonsocial; $p = .31$, $d = -0.13$ for non-social \rightarrow social; $p = .06$, $d = -0.30$ for social \rightarrow social).

To test for a social orienting deficit in ASD, we asked whether participants were faster to execute a saccade to a social stimulus than a nonsocial stimulus, an effect reported previously in typical adults (Crouzet, Kirchner, & Thorpe, 2010). Comparing SRTs for trials in which the peripheral stimulus was a social versus nonsocial image, we found that both groups were significantly faster to saccade to a social stimulus (Fig. 2b; both $p < .001$, $d = 0.54$ for ASD and $d = 0.73$ for TD). ASD and TD groups did not differ in the magnitude of faster orienting to social versus nonsocial peripheral stimuli (no Group \times Peripheral Stimulus Type interaction; $p = .41$, $d = -0.07$). This prioritized orienting to social stimuli was also present in both groups when trials with long (> 2 s) SRTs were included in the analysis ($p = .010$, $d = 0.23$ for ASD, $p = .016$, $d = 0.22$ for TD; $p = .63$, $d = 0.04$ for the Group \times Peripheral Stimulus Type interaction). Following their initial saccades to the peripheral stimuli, the ASD and TD groups did not differ in the total time spent looking at either social ($p = .29$, $d = 0.15$) or nonsocial ($p = .57$, $d = 0.024$) peripheral images. The social content of the central stimulus had no influence on SRTs in either group (ASD: $p = .066$, $d = 0.17$; TD: $p = .070$, $d = 0.16$).

Attentional disengagement and social orienting effects were not independent, as revealed by a significant interaction of these two factors in the omnibus ANOVA ($p = .022$, $\eta_p^2 = .063$), reflecting reduced disengagement costs when a social rather than a nonsocial stimulus appeared in the periphery. Of importance, the magnitude of this effect did not differ across groups (no Group \times Trial Type \times Peripheral Stimulus Type interaction; $p = .32$, $\eta_p^2 = .012$). There was no interaction between central stimulus content and disengagement (no Trial Type \times Central Stimulus Type interaction; $p = .29$, $\eta_p^2 = .014$).

These findings collectively show a remarkable degree of similarity between children with ASD and TD children in both attentional disengagement and social orienting. Both groups had significant disengagement costs in all conditions, and both showed prioritized orienting to social stimuli, demonstrating that our study had sufficient power to detect even small differences in SRTs, yet the magnitude of the effects was nearly identical across groups in every case. Further analyses showed that ASD and TD children did not differ in their fixation patterns

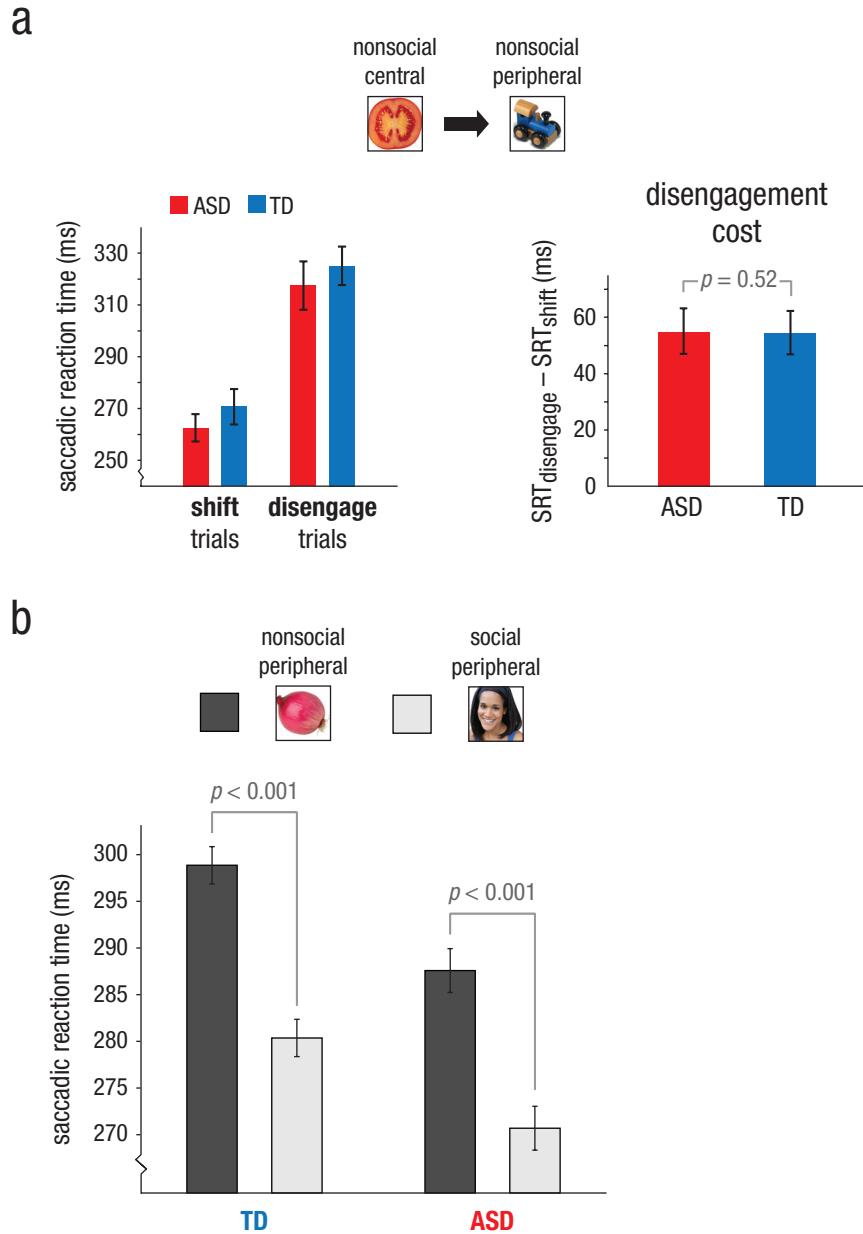


Fig. 2. Tests of attentional disengagement and social orienting. (a) Disengagement costs. SRTs (when both central and peripheral stimuli were nonsocial) were computed as the time for the eyes to arrive at the peripheral stimulus after its onset. Red bars are SRTs for ASD children and blue bars are SRTs for TD children. Error bars are $\pm 1 SD$ of the bootstrapped distribution (see the method section). Both groups showed a significant disengagement cost ($SRT_{disengage} - SRT_{shift}$, shown at right; both $p < .001$, both $d > 1$), but there was no difference in disengagement cost between groups ($p = .52$, $d = 0.024$). (b) Social orienting. We pooled trials according to whether the peripheral stimulus was a social image (dark gray bars) or a nonsocial image (light gray bars). Both groups arrived significantly faster on social stimuli (both $p < .001$, $d = 0.54$ for ASD and $d = 0.73$ for TD), and there was no Group \times Stimulus Type interaction ($p = .41$, $d = -0.07$).

on either social or nonsocial stimuli (see Figure S3 in the Supplemental Material) and that our results cannot be explained by group differences in language abilities (see Figure S2).

Discussion

The present study tested two key signatures of attentional function that have been widely implicated in autism:

attentional disengagement and social orienting. Each of these putative impairments has been proposed to lie at the root of other core aspects of the cognitive phenotype of autism (e.g., restricted interests and deficits in social cognition). However, we find no evidence that high-functioning children with ASD suffer from impairments in either attentional disengagement or social orienting (as indexed by saccadic eye movements). Instead, we find both attentional signatures are present in children with ASD and both are of very similar magnitude to those found in age- and IQ-matched typical children. Children with ASD disengaged attention as quickly as TD children and were significantly influenced by social stimulus content, showing the same increased speed to orient to social versus nonsocial stimuli that TD children did. Our study used a relatively large number of participants, affording good statistical power to detect a difference between ASD and TD children if it existed. The effects we report are not simply null findings when comparing across groups; they are positive findings that are independently significant in each group individually and that are virtually identical in magnitude across groups. Although deficits in attentional disengagement or social orienting may exist under different circumstances from those tested here, our data indicate that high-functioning children with ASD do not suffer from across-the-board impairments in either attentional disengagement or social orienting.

Our findings are consistent with some prior studies of disengagement in ASD, especially those that required a saccade to the peripheral stimulus (Kelly et al., 2013). However, they are inconsistent with the majority of the studies that examined disengagement in a free-viewing paradigm. How can the discrepancies between our results and prior free-viewing findings be reconciled? For attentional disengagement, the best-known prior report of a deficit in ASD (Landry & Bryson, 2004) found an attentional disengagement cost of about 2 s. This is more than an order of magnitude larger than the disengagement cost reported in other studies (Kawakubo et al., 2007) and the disengagement costs we found here in children with ASD. One possibility is that the disengagement cost in Landry and Bryson's (2004) study was overestimated because of the handful of trials where SRTs were 7 to 8 s—many times what would be expected for even very slow attentional disengagement. Later studies showed that very slow SRTs are associated with global developmental delay rather than with autism (Chawarska et al., 2010).

Elsabbagh et al. (2009, 2013) reported slower disengagement in infant siblings of children with ASD, especially those who went on to develop autism themselves. Substantial design differences between their study and ours may account for the conflicting results. Elsabbagh

et al. presented the same peripheral stimulus on every trial, which might have interacted with the speed of orienting, for example, through repetition priming (Maljkovic & Nakayama, 1994). They also used animated cartoons as the central stimuli that switched to static images at the time of the peripheral stimulus onset; this change in motion was likely a salient attentional cue itself, and may well have interacted with disengagement. Finally, global developmental delay (as reflected by lower Mullen scores in the infants who later developed autism) may contribute to disengagement deficits. In another study of adults performing a gap-overlap task, Kawakubo et al. (2007) found that observers with ASD were slower to disengage attention than TD controls in the overlap condition. However, their participants with ASD had very low IQ (~40). In fact, an IQ-matched mental retardation group showed similar disengagement delay as the autism group. Thus, although our results run counter to the prevailing belief that attentional disengagement is impaired in autism, this was the first rigorous test of this hypothesis in a large sample of IQ-matched children using a free-viewing paradigm.

With regard to social orienting, Chawarska et al. (2010) reported that toddlers with ASD had shorter SRTs when shifting attention away from social stimuli than did TD toddlers, but not when shifting attention away from non-social stimuli. Though their study did not measure disengagement costs directly, their results are suggestive of smaller social disengagement costs for toddlers with ASD. However, Chawarska et al. presented only 10 nonsocial trials, all blocked together at the beginning of the study before the 64 social trials, confounding the social versus nonsocial manipulation with participants' time spent performing the task. Any trend in reaction times over the course of the experiment would have differentially affected social and nonsocial trials in their study, so it is not clear whether the larger social disengagement costs Chawarska et al. report are truly the result of social orienting.

Thus, prior findings are not inconsistent with our conclusion that children with autism do not have across-the-board impairments in attentional disengagement or social orienting. On the other hand, these deficits may exist in circumscribed situations or participant groups not tested here. For example, it is possible (if unparsimonious) that attentional deficits exist in very young children but quickly disappear. Indeed, the severity of some symptoms in autism tends to decrease with age (Elsabbagh & Johnson, 2007). Yet in separate analyses of our data on the younger half of our participants (ages 5–9 years), we still find no evidence for impairments in either attentional disengagement or social orienting, and neither disengagement nor social orienting was correlated with age in either group in our participant pool (see Figure S1 in the Supplemental

Material available online). Nonetheless, it will be important in the future to test whether ASD children younger than those tested here have an attentional impairment (while unconfounding autism from global developmental delay, ideally by testing children with normal cognitive abilities). Even so, autism is a lifelong disorder, and the defining characteristics of ASD should persist to some degree across development and interventions. That we find no disengagement or social orienting deficits in school-aged children with ASD substantially weakens the purported link between these abilities and autism.

Second, deficits in attentional disengagement and/or social orienting may emerge under real-world conditions, even if they are not apparent in more constrained laboratory tests. The findings of Dawson et al. (2004), for example, point toward this possibility. In their experiment, conducted during face-to-face interaction with children with ASD, social and nonsocial auditory stimuli (e.g., humming and snapping fingers vs. a phone ringing or blowing a whistle) were produced by one experimenter while another experimenter was interacting with the child. Children with ASD were less likely than TD children to orient toward social sounds, an effect that was significantly reduced for nonsocial sounds. Similarly, studies using movies of naturalistic social situations as stimuli, perhaps a halfway point toward real-life interactions, have found atypical eye gaze patterns in ASD, although these findings are not without challenges (Chawarska, Macari, & Shic, in press; Jones et al., 2008; Klin, Jones, Schultz, Volkmar, & Cohen, 2002; Norbury et al., 2009; Rice, Moriuchi, Jones, & Klin, 2012; Speer, Cook, McMahon, & Clark, 2007). For all these reasons, it will be worthwhile to study attentional function in ASD in more naturalistic, real-world contexts.

Our results show that the core attentional functions of disengagement and social orienting are intact in ASD. Together with the results of other recent studies (Grubb et al., 2013; Jiang, Capistrano, Esler, & Swallow, 2013; Koldewyn, Jiang, Weigelt, & Kanwisher, 2013; Koldewyn, Weigelt, Kanwisher, & Jiang, 2013), these findings challenge the once-popular idea that deficits in attentional function may be the root cause of autism. Instead, these data are more consistent with the idea that domain-specific deficits in social cognition are the crux of the matter in autism. In its strongest form, this hypothesis holds that social deficits appear first in development in autism and play a causal role in the etiology of the rest of the autism phenotype. Further research in two key veins will be necessary to examine this possibility. First, because attention includes a variety of functions, it is important to test whether other core aspects of attention, particularly those involved in planning and other central executive functions, are also intact in ASD during early

development. Such tests should control for comorbid conditions such as intellectual delay, attention-deficit/hyperactivity disorder, and reduced inner speech. Second, future research should examine how a primarily domain-specific disorder could produce broad deficits in behavior, including some that may resemble attention deficits in everyday function. Atypical social functioning may modulate attentional priority in the real world even in people whose core attentional functions (e.g., disengagement, endogenous cueing) are intact. Furthermore, early social deficits may change the way the brain learns and develops, producing an array of later deficits in nonsocial domains. Indeed, understanding the causal trajectory that leads to autism within the first 3 years of life remains the most fundamental question in autism research, and the most important if we are ever to intervene effectively to alter that developmental trajectory.

Author Contributions

J. Fischer and K. Koldewyn contributed equally to this work. The study was designed by Y. V. Jiang, K. Koldewyn, and N. Kanwisher. Testing and data collection were performed by K. Koldewyn and J. Fischer. Data analysis and interpretation were performed by J. Fischer and K. Koldewyn under the supervision of Y. V. Jiang and N. Kanwisher. The article was written by J. Fischer, K. Koldewyn, Y. V. Jiang, and N. Kanwisher.

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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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Supplemental Material

Additional supporting information may be found at <http://cpx.sagepub.com/content/by/supplemental-data>

Note

1. One study (Kawakubo et al., 2007) did report larger disengagement costs in autism spectrum disorder versus typically developing participants, but a similar deficit was found in IQ-matched mental retardation controls. The deficit likely derived from low IQ rather than autism.

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