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Research report

Zoom-out attentional impairment in children with autism spectrum disorder

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ABSTRACT

Autism spectrum disorder (ASD) has long been associated with an inability to experience wholes without full attention to the constituent parts. A zoom-out attentional dysfunction might be partially responsible for this perceptual integration deficit in ASD. In the present study, the efficiency of attentional focusing mechanisms was investigated in children affected by ASD. We measured response latencies to a visual target onset displayed at three eccentricities from the fixation. Attentional resources were focused (zoom-in) or distributed (zoom-out) in the visual field presenting a small (containing only the nearest target eccentricity) or large (containing also the farthest target eccentricity) cue, 100 or 800 msec, before the target onset. Typically developing children, at the short cue-target interval, showed a gradient effect (i.e., latencies are slower at the farthest eccentricity) in the small focusing cue, but not in the large focusing cue condition. These results indicate an efficient zoom-in and zoom-out attentional mechanism. In contrast, children with ASD showed a gradient effect also in the large focusing cue condition, suggesting a specific zoom-out attentional impairment. In addition, the ASD group showed an atypical gradient effect at the long cue-target interval only in the small cue condition, suggesting a prolonged zoom-in and sluggish zoom-out attentional mechanism. This abnormal attentional focusing—probably linked to a dysfunctional top-down feedback from fronto-parietal network to the early visual areas—could contribute to the atypical visual perception associated to individuals with ASD which, in turn, could have consequences in their social-communicative development.

1. Introduction

Autism Spectrum Disorder (ASD) is a complex neurodevelopmental disorder characterized by abnormalities in communication, social interaction and presence of markedly restricted interests and stereotyped behaviours (American Psychiatric Association, 1994).

Although the dysfunctions in social cognition and communication are typically considered the “core” deficits in individuals with ASD, there is growing evidence of abnormalities in
their visual perception and attention (e.g., Grandin, 2009; Vlamings et al., 2010; see Dakin and Frith, 2005; Happé, 1999; Mottron et al., 2006 for reviews). The idea that individuals with ASD pay attention to the world differently, and that the consequent atypical perception might contribute to abnormalities in both social and “non-social” (e.g., repetitive behaviours, insistence on sameness and preoccupation with parts of objects) domains, is perhaps one of the most intriguing aspects of the current ASD research (see Mazur, 2011 for a recent review). According to the neuro-constructivist approach (see Karmiloff-Smith, 1998; Johnson, 2011 for reviews) low-level attentional and perception abnormalities could, indeed, cause impairments in the higher level cognitive modules (e.g., Elsabbagh et al., 2011). It is well known that perception of relevant information is mediated by attention orienting (see Reynolds and Chelazzi, 2004 for a review). Attention orienting is often compared with a “spotlight” that moves to a specific region in the visual space, improving information processing in the attended area at the expense of other locations (see Posner and Petersen, 1990; Corbetta and Shulman, 2002 for reviews). However, the attention spotlight is not only oriented in a specific location, but has also to be adjusted in its size. This ability allows to process visual stimuli from a narrow (zoom-in) or a broad visual region (zoom-out). Eriksen and St. James (1986) suggested a “zoom-lens” model, in which the attentional spotlight size can be varied continuously (see also the attentional scaling by Luo et al., 2001). In particular, the zoom-lens model explicitly predicts an increase of processing efficiency within the focus when the attentional spotlight is decreased in size. This prediction has been supported by behavioural, neuro-imaging and neurophysiological data demonstrating a partial independence between the focusing and the orienting mechanisms (e.g., Castiello and Umiltà, 1990; Müller et al., 2003; Fu et al., 2005; Turatto et al., 2000).

Although several studies investigated the attentional orienting in ASD (e.g., Townsend et al., 1996a, 1996b), only a few of them are related to the ability to adjust the size of the attentional spotlight (hereafter, attentional focusing). In a recent review Ames and Fletcher-Watson (2010) reported that only two studies attempted to explore the attentional focusing mechanisms in ASD (Burack, 1994; Mann and Walker, 2003). In the Burack’s study (1994) participants (four mental-age matched groups composed by subjects: with autism, with organic mental retardation, with familial mental retardation, and with no handicap) performed a forced-choice reaction time (RT) task to assess the filtering component of selective attention. The independent variables were the presence/absence of a window which narrowed the attentional spotlight (zoom-in), the number (zero, two, or four) and the location of distractors. The RTs of the subjects with autism improved relative to the other groups in the presence of the window without distractors, but this effect was negated when distractors were also presented. Performance of the autism group was, indeed, the most impaired in the presence of distractors. These findings represent a behavioural evidence of an inefficient broad attentional lens among persons with autism. In the second study, Mann and Walker (2003) employed a paradigm requiring participants to make a judgement about which one of the two pairs of cross-hairs was the longer. ASD participants were less able than comparison group in making this judgement when the previous pair of cross-hairs was smaller than the one to be judged. The authors argued that individuals with ASD have a difficulty in the zoom-out of the attentional spotlight, even if they speculated that this deficit could arise from a general difficult in orienting attention to a target in the periphery.

We hypothesise that the “inability to experience wholes without full attention to the constituent parts” (Kanner, 1943, p. 246) in ASD could be related to an abnormal attentional focusing mechanism. Precisely, we suppose that children with ASD present a poorer ability to enlarge the size of their attentional spotlight: i.e., a specific zoom-out attentional impairment. This deficit in the zoom-out of the attentional spotlight, although it could lead to superior performances in several perceptual tasks (see Dakin and Frith, 2005; Mottron and Burack, 2001 for reviews), it could also result in poor performance in other visual paradigms. For example, in coherent dots motion detection paradigm (Newsome and Pare, 1988), observers with ASD require about 10% more of coherent motion to correctly report direction (e.g., Milne et al., 2002; Pellicano and Gibson, 2008; Ronconi et al., under review; Spencer et al., 2000; but see De Jonge et al., 2007; see Grinter et al., 2010 for a recent review). A narrow attentional spotlight could contribute to worsen the coherent motion performance because it would filter the information outside the attentional focus, leading individuals with ASD to base their judgement on a restricted portion of moving dots. Moreover, Navon Task (Navon, 1977) performance in ASD indicates a preference for the local level of hierarchical stimulus analysis – maybe due to a deficit in the zoom-out of the attentional spotlight (e.g., Milne et al., 2002; Rinehart et al., 2000). These findings suggest that a detail-oriented visual perception could be a possible mechanism for the “weak central coherence” (Frith and Happé, 1994; Happé and Frith, 2006; see Happé, 1999 for a review).

In the present study, we investigated the attentional focusing mechanisms (i.e., zoom-in and zoom-out) in children with and without ASD, to verify the hypothesis for which children with ASD present a specific deficit in zooming-out their attentional spotlight. We employed a simple RTs task to measure the target detection – presented at three eccentricities from the fixation point – when a non-informative small or large focusing cue guided participants to scale the attentional processing in a restricted or enlarged visual field area, respectively. The “attentional gradient” is defined as the specific RTs pattern evoked in presence of a small cue-size that focuses the attentional spotlight (i.e., zoom-in mechanism): it predicts that the RTs to the target are slower at the farthest in comparison with the nearest eccentricity. In contrast, when a large cue-size enlarges the attention spotlight this gradient should be reduced or nullified because the target is presented inside the focus regardless target eccentricity (i.e., zoom-out mechanism; e.g., LaBerge, 1983; see LaBerge and Brown, 1989 for a review).

We predict that typically developing (TD) participants will be able to zoom-in their attention, generating a gradient effect, only when a small cue anticipates the target onset. On the other hand, with a large cue, they should be able to zoom-out their attention, nulling the gradient effect of the target
eccentricity. This prediction should be valid only at the shorter stimulus-onset-asynchrony (SOA, i.e., 100 msec), because when a longer SOA is employed (i.e., 800 msec) the time between the cue and the target will be too long to sustain the zoom-in of the attentional focus (Turatto et al., 2000). Thus, our prediction is that if the zoom-out attentional mechanism is specifically impaired in children with ASD, these children will show an abnormal gradient effect in the large focusing cue only at short cue-target SOA.

The comparison between the target RTs at the two SOAs (across the cue-sizes and target eccentricity) will be a good control to test whether children with ASD were able to process the cues or they simply ignored them. The presence of a SOA effect (i.e., faster target RTs at the long SOA compared to the short SOA) should, indeed, suggest that the observers processed the cues.

2. Methods

2.1. Participants

Twenty-three children took part of the experiment. The ASD group comprised 11 children. All the participants with ASD were included according to the following criteria: (i) full scale IQ > 70 as measured by the Italian version of Wechsler Intelligence Scale for Children- Revised (WISC-R, Wechsler, 1993); (ii) absence of gross behavioural problems; (iii) normal or corrected-to-normal vision and hearing; (iv) absence of drug therapy; and (v) absence of attention deficit hyperactivity disorder on the basis of DSM-IV criteria (American Psychiatric Association, 1994). Children with ASD were recruited at the Developmental Neuropsychology Units of Scientific Institute “E. Medea”. Diagnosis of ASD was made by licensed clinicians experienced in the assessment of ASD in respect to DSM-IV diagnostic criteria and to the Autism Diagnostic Observation Scale (ADOS; Lord et al., 2002). The control group comprised 12 TD children randomly sampled in Padua public schools. According to the parents’ report, TD children did not have prior history of any psychiatric disorders. Both groups were matched for chronological age [t(22) = −1.9, p > .05] and gender [χ² = 1.54, p > .05]. Cognitive level in TD children was estimated with two Verbal (Vocabulary and Similarities) and two Performance (Block Design and Pictures Completion) subtests of the WISC-R (Wechsler, 1993). ASD and TD group differed only in the Vocabulary subtest [mean ASD: 9.09, mean TD: 12.27; t(21) = 2.7, p < .05], while they did not differed in the other three subtests (Similarities: mean ASD = 11.27, mean TD = 12.43; Block Design: mean ASD = 11.18, mean TD = 11.9; Pictures Completion: mean ASD = 11.45, mean TD = 10.83; all ps > .05). Informed consent was obtained from each child and their parents. For details about participants’ characterization see Table 1.

2.2. Material and stimuli

The experiment was conducted in a dimly lit and quiet room. Participants were seated 40 cm far from an LCD screen (20 inch, 75 Hz). A chinrest was used to stabilize the head. Stimulus presentation and data acquisition were performed with E-Prime 1.1 (Schneider et al., 2002). All stimuli were middle grey displayed on a black background. The fixation point consisted of a cross (5 deg) presented in the centre of the screen. In the Small cue condition (see Fig. 1 panel A) a circle with a ray of 4 deg was presented concentrically to the fixation point. In the Large cue condition (see Fig. 1 panel B) a circle with a ray of 12.5 deg was presented concentrically to the fixation point. The target stimulus was a dot of .5 deg which could appear at one of the three possible distances from the fixation point on the horizontal axis (i.e., 2, 6 and 12 deg, named: Eccentricity 1, Eccentricity 2, Eccentricity 3, respectively). In the Small cue condition, the target was displayed inside the focusing cue at the Eccentricity 1, whereas at Eccentricity 2 and at Eccentricity 3 it fell outside. In the Large cue condition the target was always displayed inside the focusing cue. The target was randomly presented in the left and in the right visual hemi-field.

2.3. Procedure

Participants were instructed to keep their eyes on the fixation point throughout the duration of the trial. Each trial started with the onset of the fixation point. After 500 msec, a non-informative (i.e., the probability of the target location was equal in the two focusing cue condition) small or large focusing cue was presented. The target was displayed for 200 msec. In order to measure the time-course of attentional focusing the cue-target SOA was manipulated (i.e., 100 or 800 msec). Participants were instructed to press the space bar on the keyboard as fast as possible at the target onset, and the computer recorded RTs and accuracy. Response was recorded within 2 sec from the stimulus onset. If any response was given, participants were advised with a 800 Hz sound played for 500 msec. Catch trials, in which the stimulus was not presented and the participant did not have to respond, were intermixed with response trials. The experimental session consisted of 132 randomised trials. Precisely, 120 response trials (2 focusing cue-sizes × 2 SOAs × 3 Eccentricities × 10 times) and 12 catch trials. At the end of each trial a blank screen was presented until the experimenter pressed the mouse button to start the next trial.
3. **Results**

The two groups (TD and ASD children) did not differ significantly in the overall response accuracy as revealed by the error analysis. Precisely, we counted as errors the omissions in the response trials and the false alarms in the catch trials. Mean accuracy for the response trials was 98.3% (±2.3) for the children with ASD and 98.9% (±1.2) for the TD children [t(21) = 1.0, p > .05], while for the catch trials mean accuracy was 87.2% (±27.8) for children with ASD and 95.9% (±6.5) for the TD children [t(23) = -1.06, p > .05]. RTs faster than 150 msec and slower than 1500 msec were filtered-out from the statistical analysis. The two groups did not significantly differ for the number of trials excluded by this procedure [ASD group mean = 6.7 ± 2.6, TD group mean = 6.1 ± 0.8, t(21) = 1.16, p > .05].

RTs mean of corrected trials was computed by a mixed analysis of variance (ANOVA) with 2 x 3 x 2 design in which within-subject factors were the focusing cue-size (Small and Large) and Eccentricity (2, 6 and 12 deg), while the between-subject factor was the Group (ASD and TD). ANOVA was performed separately for the two SOAs (100 and 800 msec).

ANOVA on the first SOA (100 msec) presented a significant main effect for Eccentricity [F(2,42) = 8.37, p < .05, η² = .29], showing that RTs were modulated by target eccentricity (mean RTs was 393 ± 62 msec at Eccentricity 1, 402 ± 76 msec at Eccentricity 2, and 423 ± 66 msec at Eccentricity 3). Importantly, focusing cue-size by Eccentricity by Group interaction (see Fig. 2, panels A and B) was significant [F(2,42) = 4.96, p < .05, η² = .19], suggesting that the two groups presented a different eccentricity effect as a function of the focusing cue-size. In the Small cue condition both ASD [mean RTs at Eccentricity 1 was 400 ± 44 msec while at Eccentricity 3 it was 432 ± 68 msec; F(1,10) = 5.34, p < .05, η² = .35] and TD children [mean RTs at Eccentricity 1 was 382 ± 66 msec while at Eccentricity 3 it was 423 ± 69 msec; F(1,10) = 16.57, p < .05, η² = .60] showed the typical attentional gradient effect (i.e., faster RTs for stimuli displayed at Eccentricity 1 and slower RTs for stimuli displayed at Eccentricity 3, i.e., inside and outside the small focus, respectively). In contrast, in the Large cue condition, the gradient effect disappeared in the TD group, as predicted by the attentional zoom-lens model: i.e., no significant differences in the target RTs at two different eccentricities (F < 1) were observed. In contrast, children with ASD still presented an abnormal gradient effect, showing a significant difference [F(3,42) = 5.09, p < .05, η² = .34] between target RTs at Eccentricity 1 (mean RTs was 404 ± 61 msec) and Eccentricity 3 (mean RTs was 452 ± 86 msec). No other main effects and interactions were significant (all ps > .05).

ANOVA at the second SOA (800 msec, see Fig. 2, panels C and D) showed a significant main effect for Eccentricity [F(2,42) = 4.05, p < .05, η² = .16; mean RTs was 375 ± 15 msec at Eccentricity 1, 375 ± 15 msec at Eccentricity 2, and 398 ± 17 msec at Eccentricity 3]. No other main effects and interactions were significant (all ps > .05).

To rule-out that children with ASD were simply ignoring uninformative spatial cue, mean RTs of corrected trials was computed by a mixed ANOVA 2 (the within-subject factor was the SOA) x 2 (the between-subject factor was the Group) design. In particular, if children with ASD process the spatial cue, faster target RTs should be shown at long SOA in comparison with the short one. On the other hand, if children with ASD ignored the spatial cue, no target RTs difference between long and short SOA should be present. The ANOVA showed a significant main effect of the SOA [F(1,21) = 8.07, p < .05, η² = .28], demonstrating that target RTs were affected by the SOA (RTs mean at 100 msec SOA was 407 ± 65 msec, while RTs mean at 800 msec SOA was 383 ± 72 msec). The SOA by Group interaction was not significant (F < 1), highlighting that the SOA effect did not differ in the two groups. Planned comparisons reveal that the SOA effect was significant for both ASD [F(1,10) = 3.28, p < .05, η² = .25; RTs mean at 100 msec SOA was 421 ± 61 msec, while RT mean at 800 msec SOA was 401 ± 84 msec] and TD group [F(1,11) = 4.95, p < .05, η² = .31; RTs mean at 100 msec SOA was 393 ± 68 msec, while RTs mean at 800 msec SOA was 364 ± 57 msec], ruling-out that children...
with ASD were simply ignoring the cue during the attentional focusing task.

This last result, however, is not informative regarding the spatial effect of the large and small cue-size in children with ASD. Thus, a 2 (focusing cue-size) × 2 (SOA) ANOVA in the ASD group, was performed to investigate whether children with ASD processed the large and small cue. Focusing cue-size × SOA interaction was significant \[F(1,10) = 5.18, p < .05, \eta_p^2 = .34\], showing that the large versus small focusing cue were differently processed at the two SOAs. In particular, target RTs (across the three eccentricities) were faster in presence of the small cue-size than with the large one at the short SOA (415 ± 19 msec and 427 ± 20 msec, respectively), whereas they were slower in presence of the small cue-size than with the large one at the long SOA (409 ± 26 msec and 394 ± 26 msec, respectively).

4. Discussion

In the present study, we investigated the visual spatial attentional focusing mechanism (see Reynolds and Heeger, 2009 for a recent neuro-computational review) in a group of children affected by ASD using a simple target-detection task. Our aim was to verify a possible deficit in adjusting the size of the attentional focus in ASD. We manipulated the allocation of attentional resources in the visual field presenting a small or large spatial cue (e.g., Ericksen and St. James, 1986; Castiello and Umiltà, 1990; Turatto et al., 2000). In addition, in order to measure the spatial distribution of the “attentional gradient”, participants were asked to respond as fast as possible to a visual target, which could appear at three eccentricities from the fixation point along the horizontal axis (e.g., Facoetti and Molteni, 2001; see LaBerge and Brown, 1989 for a review). When a small focusing cue (a circle that included only the first target eccentricity) is presented, it is expected to induce in participants a specific attentional gradient (i.e., zoom-in mechanism). RTs should increase with target eccentricity (gradient effect). On the contrary, when a large cue is presented, it is not expected a gradient effect because attentional processing resources spread on the entire cue-delimited visual space (i.e., zoom-out mechanism; e.g., LaBerge, 1983). These results are expected only in presence of a short cue-target SOA (i.e., 100 msec), because of the...
specific time-course of attentional focusing (Turatto et al., 2000).

RTs pattern of TD children was consistent with these predictions, showing an attentional gradient when a small focusing cue preceded by 100 msec the target onset, whereas no attentional gradient emerged when a large focusing cue was displayed. Thus, the presence of a gradient effect in the small focusing cue condition only at 100 msec SOA, indicates that our experimental manipulation was appropriated.

Children with ASD, in contrast, showed narrowed focused attention not only in the small but also in the large attentional cue condition at 100 msec SOA. Thus, we find a deficit in the distribution of the attentional resources in ASD. In particular, children with ASD show an impairment in zooming-out the spotlight of visual attention. The cue-target SOA effect was similar in both groups, showing that the participants with ASD did not ignore the temporal feature of the spatial cue. Moreover, the different effect of the cue-size at the two cue-target SOAs confirmed that the children with ASD processed the size of the attentional cue as well. Interestingly, the attentional gradient abnormally present in the ASD group with a large cue at the short cue-target SOA disappeared at the long SOA (Fig. 2, Panel D), suggesting a specific sluggish zoom-out attentional mechanism. Thus, the zoom-out attentional deficit seems to be the more appropriate explanation for our findings. Finally, the ASD group, unlike the TD children, showed, in the Small cue condition, an atypical gradient effect also at the long cue-target SOA (Fig. 2, Panel C), suggesting a prolonged zoom-in attentional mechanism.

According to the “weak central coherence” hypothesis (Happé and Frith, 2006; Frith and Happé, 1994), our results suggest that in children with ASD attentional resources appear to be rigidly allocated in a narrow region of the visual field. These results could provide a better understanding of the detail-oriented perception exhibited by individuals with ASD. There is a large experimental literature describing this detail-oriented perception in visual domain in ASD (Happé and Frith, 2006; Mottron et al., 2006), which results, sometimes, in superior performances compared to typical developing children (see Dakin and Frith, 2005; Mottron and Burack, 2001 for reviews). Superior detail-oriented visual perception in ASD was found, for example, in serial search tasks, in which a target stimulus has to be discriminated from distractors (e.g., Joseph et al., 2009; O’Riordan et al., 2001). Superior search performances were confirmed also in typical individuals with high autistic traits (Almeida et al., 2010), and in the embedded figure test, in which a shape has to be found within a larger design (e.g., Jolliffe and Baron-Cohen, 1997; Manjaly et al., 2007). However, the findings by Joseph et al. (2009) and O’Riordan et al. (2001) can not be fully explained by “weak central coherence” because they showed that the superiorities were most prominent in a conjunctive search condition, where the targets shared features with both of the distractors, and which thus requires some integration process. Recently, Baldassi et al. (2009) showed that children with ASD present reduced interference effect in visual crowding. Remington et al. (2009) reported similar results for the visual perceptual load, confirming detail-oriented visual perception in ASD. This large body of literature could be plausibly related to this prolonged zoom-in attentional mechanism. In particular, it is demonstrated that orienting of attention improves performance in several visual tasks, such as serial visual search and crowding, by diminishing the noise effect outside the spotlight while intensifying the signal processing inside the attentional focus (e.g., Montagna et al., 2009; Yeshurun and Rashal, 2010; see Reynolds and Heeger, 2009 for a recent review).

Thus, our results suggest that the sluggish attentional zoom-out combined with prolonged zoom-in of the attentional spotlight in ASD could be linked to the superior performances in several visual tasks requiring efficient perceptual noise-exclusion mechanisms. Joseph et al. (2009) reported, indeed, that the ASD advantage in a visual search task derived from an enhanced ability to discriminate between targets and distractors at the locus of attention. The attentional focusing impairments, suggested by our findings, could allow individuals with ASD to better inhibit distractors in a visual search display (Baldassi et al., 2009; Remington et al., 2009).

On the other hand, these attentional focusing deficits, reducing the global spatio-temporal integration processing, could be related to the characteristic social domain impairments associated to ASD. For example, the efficient faces and emotions processing development, as well as the biological motion detection (Klin et al., 2009; Simion et al., 2008), could be impaired when the size of attentional focus is not spread to integrate local elements of the global visual scene. Accordingly, previous studies have found a relation between basic abnormal visual perceptual skills and autism symptomatology. In particular, visual search performance (Joseph et al., 2009), biological motion processing (Koldewyn et al., 2010), and visual fixation pattern (Klin et al., 2002) predicted communication and social interaction impairments in individuals with ASD.

According to the neuro-constructivist approach (see Karmiloff-Smith, 1998 for the original review), development itself is the key to understand developmental disorders. ASD characteristic features include impaired social-communication skills and atypical visual attention. A recent debate focused on whether the later emergence of atypical communicative skills is a consequence of attention problems during early life (see Johnson, 2011 for a recent review). For example, 9 months infants at familial-risk for a later diagnosis of ASD, differed from controls, both in measures of social perception and attentional disengagement. Preliminary data from an ongoing longitudinal research program, suggest an association between attentional measures and autism-related characteristics in children of 3 years old. The emergent nature of ASD could be the result of a complex developmental interactions among attentional- and social-brain networks (Elsabbagh et al., 2011). Thus, low-level attentional and perception abnormalities could cause impairments in the high-level communication and language modules (e.g., Mundy et al., 1987; Lum et al., 2007). In particular, a specific zoom-out attentional dysfunction in ASD – hampering the integration of distributed communication cues – might be partially responsible for the typical social “core” deficit.

However, as found by Ronconi et al. (under review) by using central and peripheral coherent dots motion tasks, the inability to enlarge the focus could be overcome. When the
information relevant to the task is, indeed, completely absent inside the attentional focus children with ASD seem to be able to zoom-out the focus of attention in the same way the TD group do. In contrast, when a small amount of relevant information falls into the attentional focus, the ASD group is unable to enlarge the focus even if it would be beneficial for the task performance.

4.1. What could be the neural correlate of these attentional focusing deficits in children with ASD?

Neuroimaging studies in non-clinical population could suggest a plausible physiological explanation of zoom-out attentional deficit found in children with ASD. It has been demonstrated that the neural activity preceding the object presentation was finely modulated by the size of attended region in the early visual areas (Müller et al., 2003). The increasing size of the attended region causes a reduction in the blood oxygen level-dependent response in early visual areas (Müller et al., 2003; see also Brefczynski and DeYoe, 1999). In addition, the temporal dynamics of the attentional focusing has been examined by recording event-related potentials. In particular, the attentional zoom-out was associated to a decreased in N1 amplitude (Luo et al., 2001; Fu et al., 2005). Recent studies shed some lights on the influence of top-down fronto-parietal attention network (e.g., Saaßmann et al., 2007; see Corbetta and Shulman, 2002 for a review) on the early visual processing. Combining transcranial magnetic stimulation (TMS) and functional magnetic resonance imaging (fMRI), Ruff et al. (2006, 2008) found that the TMS application on the right prefrontal cortex (frontal eye fields – FEF) and on the right parietal cortex (intra-parietal sulcus – IPS), increased fMRI activity for representations of the peripheral visual field, and reduced activity for the central field in all retinotopic visual areas.

One possible explanation for the children with ASD inability to zoom-out the attentional spotlight size in response to a large visual cue could arise from a dysfunction in the connectivity between top-down fronto-parietal attention network and early visual areas, where the “zoom-lens” of the spatial attention is modulated (Müller et al., 2003; see also Brefczynski and DeYoe, 1999). A large number of recent studies suggest a possible under-connectivity between frontal and occipital areas in ASD (e.g., Bartfeld et al., 2010; Courchesne and Pierce, 2005; see Belmonte et al., 2004 for a review). Accordingly, two fMRI studies have shown, in individuals with ASD, a dysfunction of the dorso-lateral prefrontal and the intra-parietal cortex during visual attention task (Manjaly et al., 2007; Ring et al., 1999). A recent study showed that atypical prefrontal activations seem to be present also in unaffected sibs (Belmonte et al., 2010). Thus, both the impaired zoom-out as well as the prolonged zoom-in attentional focusing here shown in children with ASD could be probably linked to a sluggish top-down feedback from fronto-parietal network to the early visual areas.

We conclude that if this attentional focusing deficit will be confirmed also in infants at familial-risk for ASD, the zoom-out attentional impairment could be considered an important neuropsychological marker for the early identification of ASD.

Acknowledgements

This work was supported by a grant from University of Padua ("Progetto di Ateneo 2009 and 2011" to A.F. and "Assegni di Ricerca 2009 and 2011" to S.G.). The contributions of staff members of "E. Medea" Scientific Institute as well as of children and their families are gratefully acknowledged. We thank Laura Zamponi and Barbara Urbani for their help in recruitment and clinical characterization of participants. Finally, we thank the Editor Mike Anderson and the two anonymous Reviewers for their valuable comments.

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