Weak surround suppression of the attentional focus characterizes visual selection in the ventral stream in autism

Luca Ronconi, Simone Gori, Alessandra Federici, Maria Devita, Serena Carna, Maria E. Sali, Massimo Molteni, Luca Casartelli, Andrea Facoetti

1. Introduction

Autism spectrum disorder (ASD) is a pervasive neurodevelopmental condition that causes significant impairments in social-communicative and behavioural domains (American Psychiatric Association, 2013). In addition, a large amount of evidence demonstrates that individuals with ASD manifest abnormalities in the sensory domain (Dakin and Frith, 2005; Kellerman et al., 2005; Simmons et al., 2009), and sensory dysfunctions have been recently included as diagnostic criteria for ASD (American Psychiatric Association, 2013). Furthermore, low-level dysfunctions could be one of the main factors leading to impairments in the high-level social-cognitive domain, in accordance to the neuro-constructivist hypothesis (Johnson, 2011; Karmiloff-Smith, 1998; Ronconi et al., 2016b). For example, the face-processing deficit (Dawson et al., 2005) has been hypothesized to arise from deficits in low-level visual perception (McCleery et al., 2007; Vlamings et al., 2010). Moreover, the speech processing impairment has been linked to low-level multi-sensory integration (Facoetti et al., 2010; Stevenson et al., 2009).

Consistent evidence associated ASD with higher performance in detail-oriented tasks (Dakin and Frith, 2005; Simmons et al., 2009). Individuals with ASD detect targets faster in visual search tasks (Joseph et al., 2009; Plaisted et al., 1998) and in the Embedded Figure Test (Jolliffe and Baron-Cohen, 1997; Manjaly et al., 2007), and are also more resistant to visual crowding and visual illusions (Baldassi et al., 2009; Gori et al., 2016; Happé and Frith, 2006; Költa et al., 2010). However, individuals with ASD exhibit also an increased interference from irrelevant distractors (Adams and Jarrold, 2012; Burack, 1994; Remington et al., 2009; Ronconi et al., 2013a), and visual sensory overload has been well documented in autobiographical reports (Grandin, 2009), with caregiver-report questionnaires (Kern et al.,...
relevant target information without simultaneously boosting noise in a
Kleinschmidt, 2004; Slotnick et al., 2002; for a review see Carrasco, 2006; Leekam et al., 2007) as well as in electrophysiological studies (Belmonte, 2000; Pritchard et al., 1987). Many efforts have been made to clarify this intricate perceptual profile, with visual attention hypothesized to play a central role (for reviews see Ames and Fletcher-Watson, 2010; Keen et al., 2013).

Studies of the typical population have traditionally viewed spatial attention as a simple “spotlight”, enabled to move to a specific region in the visual space (Carrasco, 2011; Corbetta and Shulman, 2002; Petersen and Posner, 2012; Posner, 1980; Saalmann et al., 2007; Vidyasagar, 1999). Moreover, the focus of attention can be varied in its size as a “zoom-lens” (Castiello and Umiltà, 1990; Eriksen and St James, 1986; Facetti and Molteni, 2000; Müller et al., 2003; Ronconi et al., 2014a). Studies in the ASD population designed within these two theoretical frameworks seem to provide a consistent picture. Indeed, it has been consistently reported that ASD is associated with impairment in the disengagement of attention from a previously cued location (Courchesne et al., 1994; Landry and Bryson, 2004; Wainwright-Sharp and Bryson, 1993; Keen et al., 2017), a deficit that is associated with the emergence of autism in infants at-risk (Elsabbagh et al., 2013; Ronconi et al., 2014b). Moreover, individuals with ASD manifest an over-focused attention and an impairment in “zooming-out” the attentional focus (Mann and Walker, 2003; Robertson et al., 2013; Ronconi et al., 2012; Ronconi et al., 2013b).

Both the spotlight and the zoom-lens models, in their original conceptualization, predict that the resources fade progressively while the distance from the focus increases. However, more recent psychophysical and neurophysiological evidence in the typical population demonstrate that visual selection requiring spatial scrutiny elicits – in the immediate surround of the attentional focus – an area of attenuated excitability, forming a center-surround profile resembling a “Mexican hat” (Bahcall and Kowler, 1999; Boehler et al., 2009; Cave and Zimmerman, 1997; Hopf et al., 2006; Müller et al., 2005; Müller and Kleinschmidt, 2004; Slotnick et al., 2002; for a review see Carrasco, 2011). This inhibitory ring would be an optimal solution for amplifying relevant target information without simultaneously boosting noise in a visual scene.

The center-surround profile appears only under conditions requiring spatial scrutiny and, on the contrary, is not elicited when target identification can be achieved without precise spatial localization (Müller et al., 2005; Hopf et al., 2006; Hopf et al., 2010). The peculiarity of the center-surround profile is also suggested by the cortical activity underlying the surround suppression which is dissociated from the N2pc (Eimer, 1996; Luck et al., 1997; Luck and Hillyard, 1994) event-related potential (ERP). While the N2pc increases when adding distractors, the surround suppression is unaffected by it (Boehler et al., 2011). Although the precise neurophysiological mechanisms by which this center-surround profile arises is still unclear, a potential explanation has been proposed by the computational account called ‘selective tuning model’ (STM; (Tsotsos, 1990; Tsotsos et al., 1995; Tsotsos, 2005)). The STM claims that the center-surround profile is mediated by a recurrent top-down winner-take-all mechanism that starts from the winning units in higher-level visual areas most activated after the initial feed-forward flow and eliminates projections from lower-level units that do not contribute to the attended target location (Tsotsos et al., 2008).

The importance of this attentional mechanism in the typical population has been largely investigated. Nevertheless, there are no studies testing the attentional surround suppression in ASD. The aim of the present study was to evaluate the spatial profile of the attentional focus in individuals with ASD in order to disentangle whether contradictory aspects of perception in ASD could be linked to a particular spatial profile of the attentional focus. The central questions we attempted to answer are: How do individuals with ASD process visual information at different degrees of proximity from the attentional focus (see Fig. 1D)? How is this reflected in their cortical activity in the ventral visual stream? Two independent samples of children with ASD and age- and IQ- matched typically developing (TD) peers were tested in a psychophysical (Experiment 1) and in an EEG study (Experiment 2). In both experiments, visual attention was automatically captured onto a pop-out target (red C) among an array of non-target stimuli (blue C). In half of the trials (baseline condition), their task was to recognize the orientation of the red C that changed position from trial to trial. In the remaining half of the trials (probe condition), after the appearance of a red target C, a probe circle circumscribed a non-target C at various distances from the red target C. This latter condition allowed to measure the behavioural and neurophysiological manifestations of the spatial profile of the attentional focus.

2. Experiment 1 – psychophysical study

2.1. Methods

2.1.1. Participants

Forty-six children took part in the experiment. Both the ASD and TD groups were initially comprised of 23 children each. Four participants from the ASD group and 1 from the TD group were excluded from statistical analyses since they did not achieve 40% of overall accuracy in the probe condition (see Procedure below). Thus, the final samples comprised 19 children for the ASD group and 22 for the TD group. All participants with ASD were recruited at the Scientific Institute IRCCS “Medea” (Bosiso Parini, Italy) and at “Associazione La Nostra Famiglia” (Padua, Italy) according to the following criteria: (i) full-scale IQ > 70 as measured by the WISC (Wechsler, 1993); (ii) absence of gross behavioural problems; (iii) normal/corrected-to-normal vision, normal hearing; (iv) absence of drug therapy; and (v) absence of attention deficit hyperactivity disorder (American Psychiatric Association, 2013). Diagnosis of ASD was made by licensed clinicians in adherence to the DSM-IV criteria and to the Autism Diagnostic Observation Scale (Lord et al., 2002) (see Table 1). Children of the TD group were randomly sampled from local schools and did not have prior history of neurological and/or psychiatric disorders. Both groups were matched for chronological age and cognitive level (see Table 1) and the ASD group scored significantly higher both in the Current and in the Lifetime version of the Social Communication Questionnaire (SCQ; Rutter et al., 2003).

Informed consent was obtained from each child and their parents and the entire research protocol was conducted in accordance to the principles elucidated in the declaration of Helsinki. The ethical committee of both the University of Padua and Scientific Institute “E. Medea” approved the present study.

2.1.2. Apparatus and stimuli

The experiment was conducted in a dimly lit and quiet room. Participants were seated 50 cm from a Philips 19S” LCD screen (19 in., 75 Hz). A chinrest was used to avoid head movement. Stimulus presentation and data acquisition were performed with E-Prime2. The choice of stimulus parameters was based on pilot testing. All stimuli were presented on a middle grey background (42.4 cd/m²). The fixation point consisted of a black cross subtending 0.5°, presented in the screen center. The search array was comprised of nine blue non-target C-shapes (10.2 cd/m²), while the target C-shaped stimulus was coloured in red (18.3 cd/m²). Both target and non-target C stimuli subtended a visual angle of 1.2°, and were presented at an iso-eccentric distance of 8.25° from the fixation point (the angular extent from the fixation point ranged from 7 to 8.5°). All C were obtained by removing from a ring-shaped stimulus portion subtending 45°’ angle. The missing portion of each C varied randomly in position (up, down, left and right; chance level = 0.25). One C was presented aligned with the horizontal axis, and the other C were presented four in the upper and four in the lower quadrant of the randomly chosen left or right hemisphere, separated by an angle of 0.6° edge-to-edge. The stimulus used as a probe was a white circle (83 cd/m²) with a diameter of 2.12°. Mask stimuli were
obtained from the complete ring-shaped stimuli used to create the C stimuli.

2.1.3. Procedure

Children were instructed to keep their eyes on the fixation point for the entire duration of the trial. Each trial started with the onset of the fixation cross (1000 ms). The array of nine randomly oriented (non-target) blue C then appeared unpredictably to the left or right side of the fixation point. After 53 ms (4 refresh cycles), a target C was coloured in red for 107 ms (8 refresh cycles). The target (red C) appeared in each trial randomly in one of the nine possible stimulus locations among the other eight non-targets (blue C). Thus, children were required to focus their attention in different positions from trial to trial. On 50% of the trials (baseline condition, Fig. 1A), after the presentation of the red target C the trial ended with all C replaced by mask stimuli (mask duration was 13 ms, 1 refresh cycle). In the other 50% of the trials (probe condition, Fig. 1B), the appearance of the target (red C) was followed by the probe circle appearing around the central blue C for 53 ms (4 refresh cycles). In this case, the red C persisted for the entire duration of the probe circle and subsequently, as for the baseline condition.

Table 1

Descriptive statistics for the two groups of participants in the Experiment 1 (psychophysical experiment) and Experimental 2 (EEG experiment) (ASD = autism spectrum disorder; TD = typically developing; TIQ = Total Intelligence Quotient).
condition, all C were replaced by the mask stimuli. As the probe position was kept constant and the target position (i.e. the position of the red C) varied, there were five target-to-probe distances, called probe distance (PD), ranging from probe distance 0 (PD0; probe at the target location) to probe-distance 4 (PD4; probe at the farthest distance; see Fig. 1C). Trials for the baseline and the probe condition were randomly selected during the experiment. After a blank screen (1000 ms), the response screen with the four possible orientations of the C was presented for an unlimited time. Participants then indicated their assumed correct response, i.e. the orientation of the red C in the baseline condition and the orientation of the blue C highlighted by the probe in the probe condition. The experimenter entered the selected choice. It was specified to the children that only accuracy would be assessed. The entire experiment consisted of 144 trials (preceded by 12 practice trials), 72 for the baseline and 72 for the probe condition.

This experimental design was optimal to test the center-surround profile of the attentional focus since attention was first pre-allocated to one of the two hemifields by the appearance of the stimulus array for ~50 ms. Then the focus of attention was captured by the pop-out target for ~100 ms, which was followed by the probe for another ~50 ms. This sequence of stimuli was finalised before participants could make an eye-movement toward the peripheral array of stimuli, and nevertheless it was compatible with the timing used in previous psychophysical studies to measure the surround suppression of the attentional focus (Cave and Zimmerman, 1997; Bahcall and Kowler, 1999; Müller et al., 2005) and, in general, with the time-course of automatic attentional capture (Carrasco, 2011).

2.2. Results

Response accuracies were analysed by two 5 × 2 mixed design ANOVAs, separately for the baseline and the probe condition. The ANOVAs were carried out with the within-subjects factor PD (5 levels: PD0, PD1, PD2, PD3 and PD4), and with the between-subjects Group (ASD vs. TD).

For the baseline condition, in which the probe stimulus was absent, the variable PD was used to identify the position of the target in the array (PD0 represents a target aligned with the horizontal axis, while PD1 to PD4 represent positions progressively farther from it).

ANOVA performed in the baseline condition revealed a main effect of the PD (F(4, 156) = 12.96, p < .001), revealing that overall accuracy varied as a function of the position of the C in the array (mean ± SEM were: PD0 = 89.2% ± 2.5, PD1 = 84.4% ± 2.3, PD2 = 82.1% ± 2.1, PD3 = 74.3% ± 2.2, PD4 = 83.3% ± 2.6). The main effect of Group and the PD by Group interaction were not significant (p = .33 and p = .59, respectively; see Fig. 2A). These results confirm that both groups were equally efficient in orienting and focusing attention onto the peripheral pop-out target.

ANOVA performed in the probe condition revealed a main effect of PD (F(4, 156) = 17.40, p < .001; mean ± SEM were: PD0 = 80.4% ± 3.1, PD1 = 58.4% ± 2.7, PD2 = 64.1% ± 2.7, PD3 = 68.7% ± 2.3, PD4 = 73.3% ± 2.5). Importantly, a significant PD by Group interaction emerged (F(4, 172) = 4.38, p = .002). Planned comparisons showed that ASD exhibited a higher accuracy as compared to the TD group both at PD1 (t(39) = 2.17, p = .036) and PD2 (t(39) = 2.15, p = .038; Fig. 2B). Comparisons at the other PDs were not found significant (all ps > .15).

These results indicate that the ASD group showed a significant weaker suppression at PD1 and PD2 where the effect of surround suppression should be the strongest.

3. Experiment 2 – EEG study

3.1. Methods

3.1.1. Participants

Initially, 18 children with ASD were recruited from the Scientific Institute IRCCS “E. Medea” (Bosisio Parini, Italy) and 18 TD children were recruited from local schools in the same area. This sample was fully independent relative to that of Experiment 1.

Participants were selected following the same criteria of Experiment 1, and the two groups were again matched for chronological age and cognitive level (the only difference was observed in the Vocabulary; see Table 1).

Three children of the ASD group and three children of the TD group were excluded from the analysis because their EEG data were excessively contaminated by artefacts.

3.1.2. Apparatus, stimuli and procedure

Stimuli and procedure were identical to Experiment 1, except for the following details: i) the mask was absent, in order to prevent overlap between target/probe and mask ERPs; ii) the duration of the blank screen displayed after each trial was 1500 ms (note that variation in response time resulted in the total inter-trial interval being jittered with respect to the time of stimulus-locked signals); iii) the total number of trials was 552 divided as follows: 276 for the baseline condition and 276 for the probe condition. Each condition comprised 120 trials for PD0, 120 trials for PD1, and 36 trials for PD2, PD3, PD4 (12 each).

In this second experiment the conditions PD0 and PD1 were privileged since they allow the measurement of the fundamental aspects of the center-surround profile, and thus to focus on the most important features of this attentional mechanism in children with ASD. Indeed, it is important to consider that in this EEG experiment it was not
affordable to test the entire profile (PD0 to PD4) in a sample of children with ASD. This would require an excessive amount of trials for an EEG study in this specific population. Thus we decided to focus only on the most important conditions for our aim (PD0 and PD1). Nonetheless, in order to prevent children developing a strong attentional bias for PD0 and PD1, also some trials for the condition PD2–PD4 were included. For obvious reasons, only PD0 and PD1 conditions had a sufficient number of trials to analyse the relative ERP data, and the EEG data from PD2–PD4 were not taken into account.

3.1.3. EEG recording and pre-processing

EEG was recorded using the Electrical Geodesics system and a 64-channel Hydrocel Geodesic Sensor Net (Electrical Geodesics, Inc.). Input data were analog-filtered between 0.01 and 100 Hz and the sampling rate was 500 Hz. Offline, data were down-sampled at 250 Hz, notch-filtered at 50 Hz (non-causal Parks-McClellan Notch, order = 180), band-pass filtered between 0.1 and 30 Hz (non-causal infinite impulse response filter, order = 2) and re-computed to an average reference. Data analysis was performed using EEGLAB (Delorme and Makeig, 2004) and Brainstorm (Tadel et al., 2011), running under MATLAB.

3.1.4. Data analysis: event-related potential (ERPs) and source reconstruction

EEG epochs extracted for the analysis ranged between ~200 and 700 ms relative to the target onset (baseline-corrected from ~200 to 0 ms). Interpolation was carried out on individual bad channels if required. Epochs containing eye movements before the target onset, as well as massive muscular artefacts (i.e. high-frequency activity affecting the majority of channels and time points) were discarded. The remaining activity evoked by eye-movements and eye-blinks was removed using the Independent Component Analysis (ICA). Epochs containing voltage deviation exceeding ± 100 μV were also removed. Following this procedure, the mean percentage and SD of interpolated channels was 4.6 ± 1.54 for the ASD and 5.3 ± 1.07 for the TD group and 90% of trials for the ASD and 89% for the TD were retained after artifact rejection.

The visual inspection of the ERP waveforms revealed that the ASD and TD groups differentiated their evoked activity in two ERP components: i) the P1, with a positive peak around 210 ms relative to the target onset (i.e., 130 ms relative to the probe), and ii) the N2, with a negative peak around 440 ms relative to the target onset (i.e., 360 ms relative to the probe) (Fig. 3). These two components differentiated between the two groups at their maximum in occipito-temporal channels (left hemisphere: channels 29, 30, 32; right hemisphere: channels 43, 44, 47). The mean amplitude evoked by contralateral stimuli in these two clusters of channels were analysed in the two different time windows of interest (P1: 180–240 ms relative to the target onset, 100–160 ms relative to the probe onset; N2: 400–475 ms relative to the target onset, 320–395 ms relative to the probe onset) with a Group (ASD vs. TD) by Hemisphere (Left vs. Right) by PD (PD0 vs. PD1) ANOVA, performed separately for the baseline and the probe condition.

For estimating cortical sources of ERP results, individual epoched data were used to estimate neural activity in the same time windows used in the ERP analyses. A depth-weighted minimum-norm estimation inverse solution (Baillet et al., 2001) with constrained dipole orientation (i.e. one dipole at each vertex, which orientation is the normal to the cortex surface at that point) was applied. A cortical mesh template surface, composed by 15,000 vertices and derived from the default anatomy MNI/Colin27, was used as a brain model to estimate the current source distribution. To compute the forward model, a symmetric boundary element method with the OpenMEEG software (Kybic et al., 2005; Gramfort et al., 2011) was employed. The noise covariance matrix was calculated for each of the subjects taking data from the baseline period (~200–0 ms) of all single-trial epochs.

3.2. Results

3.2.1. ERP results and relative cortical sources

3.2.1.1. Baseline condition (ERPs)

The ANOVA on the P1 mean amplitude revealed no significant main effects or interactions (all ps > .17). The ANOVA on the N2 mean amplitude revealed a significant effect of PD (F1, 28) = 11.64, p = .002) and a PD by Hemisphere interaction (F1, 28) = 7.69, p = .01), demonstrating that, across participants, the N2 evoked by a target appearing in PD0 was significantly smaller than in PD1 only in the left (mean amplitude ± SEM at PD0 = 2.28 ± 0.52 μV and at PD1 = 3.53 ± 0.59 μV; t28 = −4.71, p < .001, but not in the right hemisphere (t28) = −0.36, p = .72). This result is in agreement with previous evidence of the left hemisphere specialization for the processing of letter-like stimuli (e.g. Ronconi et al., 2016a). All the other main effects and interactions were not significant (all ps > .17).

3.2.1.2. Probe condition (ERPs and related cortical sources)

The ANOVA on the P1 mean amplitude revealed a main effect of PD (F1, 28) = 5.98, p = .021) and, importantly, a PD by Group interaction (F1, 28) = 5.57, p = .026). This interaction was further explored with post-hoc comparisons. The TD group showed no significant difference between the P1 mean amplitude for trials where the probe appeared in PD0 relative to PD1 (t14) = 0.68, p = .95). Contrarily, this difference was significant in the ASD group, which showed a higher mean amplitude of the P1 when the probe appeared in PD0 relative to when it appeared at PD1 (t14) = 3.1, p = .008; PD0 = 4.87 ± 0.48 μV and PD1 = 3.89 ± 0.62 μV). All the other main effects and interactions were not significant (all ps > .13). The ANOVA on the N2 revealed a main effect of PD (F1, 28) = 14.33, p = .001) and, importantly, a PD by Group interaction (F1, 28) = 5.57, p = .026). This interaction was further explored with post-hoc comparisons. They revealed opposite findings relative to what was previously found for the P1. In the ASD group there was no significant difference between the N2 mean amplitude for trials where the probe appeared in PD0 relative to PD1 (t14) = 1.21, p = .246). However, this difference was significant in the TD group, which showed relatively more negative mean amplitude of the N2 when the probe appeared in PD0 in comparison to when it appeared at PD1 (t14) = −4.00, p = .001; PD0 = 1.56 ± 0.63 μV and PD1 = 2.41 ± 0.62 μV). All the other main effects and interactions were not significant (all ps > .23).

Cortical sources of the P1 and the N2 (minimum size = 10 vertices, minimum amplitude = 30% of the maximum value) evoked by a left hemifield stimuli array (for which the modulation of both P1 and N2 were more evident) are displayed in Fig. 4 as the difference between the probe and the baseline condition. As visible, the strongest cortical sources of this probe-baseline difference in the N2 time windows were localized for both groups in the contralateral occipito-temporal areas (see Fig. 4). In the TD group a clear modulation of the activation in these regions was visible, with stronger activations when the probe stimulus appeared in PD0 relative to PD1. Such a modulation was not evident in the ASD group. The ASD group showed, indeed, similar activation in right occipito-temporal areas for both PD0 and PD1 probe stimuli. (if any difference can be highlighted, the activation appears stronger in the condition PD1).

The strongest cortical sources of the P1 difference were localized in different regions in the two groups. In the TD group, the strongest sources were localized in the rostral part of the right middle frontal gyrus, with stronger activation in PD0 relative to PD1. In the ASD group, on the contrary, the strongest activations were visible in the left inferior parietal lobule and were stronger for PD1 relative to PD0.

In addition, Supplementary Fig. 1 contains the cortical sources evoked in the probe condition only (i.e. without subtraction of baseline trials). Although the effect of surround suppression in occipito-temporal areas is less evident in this case, it is clear that the strongest cortical sources are present in occipito-parietal and occipito-temporal areas.
This pattern of activity suggests that the ERPs modulation in occipito-temporal channels (Fig. 3) are mainly reflecting visual processing and attentional selection of the probe stimulus, and not frontal ERPs volume-conducted to the occipito-temporal electrodes reflecting a later processing stage of the preceding target stimulus (this could be expected, for example, by a target-evoked P2a/P3f connected to the evaluation of task-relevance; see Potts, 2004).

3.2.2. Weak surround suppression correlated with autistic symptomatology

We considered the possible relationship between the individual measure of surround suppression and the ASD severity measured by the SCQ in the two independent samples of participants from Experiment 1 and 2. Partial correlations were performed in order to control for the effect of chronological age.

From the psychophysical data (Experiment 1), an individual Surround Suppression Index (SSI) was calculated as the mean of accuracy rate in PD1 and PD2 subtracted from the accuracy rate at PD0 (SSI = PD0 - Mean [PD1, PD2]). A lower SSI corresponds to a weaker suppression outside the focus of attention, and vice versa. Results showed that individual SSI was negatively correlated with SCQ scores (Current version; \( r(16) = -0.418, p = .042 \); see Fig. 5A). From the ERP data (Experiment 2), we calculated the individual difference in the mean amplitude of the N2 between the two probe conditions (PD0–PD1; subtraction of signed values). Results showed that the
Fig. 4. Cortical sources of the weak surround suppression in ASD. Cortical sources in the P1 and in the N2 time windows estimated for the right hemisphere (a left hemifield probe stimulus), presented at the center (PD0) or in the surround (PD1) of the attentional focus. These images were obtained by subtracting from the probe trial the activity of the related baseline trials (i.e. where only the red target appeared in the same spatial position). Contrarily to TD children, children with ASD did not show modulation of activity in the ventral visual stream in the N2 time window.

Fig. 5. Partial correlation plots showing the relationship between individual weak surround inhibition measures (calculated both using the psychophysical and the neurophysiological data) and autistic symptomatology in the two independent samples of participants with ASD. (A) Scatter plot showing the relationship between individuals Surround Inhibition Indexes derived from the Experiment 1 (see Results section) and the Social Communication Questionnaire (SCQ) score (Current version). (B) Scatter plot showing the relationship between individuals N2 mean amplitude difference (PD0–PD1) derived from the Experiment 2 and the SCQ score (Lifetime version). For both correlations, the effect of chronological age has been controlled for.
individual N2 differences were significantly correlated with the SCQ scores (Lifetime version; \( r(15) = 0.626, p = .017 \). No significant correlation was found for the PD0–PD1 differences in the P1.

Overall, these findings showed that a weaker suppression in the surround of the attentional focus corresponded to a higher ASD symptom severity.

4. Discussion

The present study systematically evaluates the spatial profile of the attentional focus in individuals with ASD, in order to provide an insight on why many contradictory aspects of perception in ASD have been reported. Psychophysical results of Experiment 1 showed that the ASD group exhibits a weaker suppression in the surround of the attentional focus relative to the TD group. In Experiment 2, neurophysiological evidence confirmed these findings. In this second study an independent sample of participants with ASD were tested with a simplified version of the psychophysical task used in Experiment 1 with concurrent dense-array EEG recording. The ERPs results showed that in the TD group, the posterior N2, which is part of a family of components that reflect the attentional selection of relevant objects in space (Bocquillon et al., 2014) and time (Ronconi et al., 2016c; Sergent et al., 2005), was suppressed when the target appeared in the surround of the attentional focus. The strongest cortical sources of this N2 (after subtraction of the activity in baseline trials) were located in lateral occipital and ventral temporal areas, which are part of the ventral visual pathway for object discrimination (Kobatake and Tanaka, 1994; Kravitz et al., 2013; Milner and Goodale, 2012). Stronger activations in these regions for the TD group were visible for probe stimuli appearing at the center of the attentional focus (P0) as compared to probe stimuli appearing in the surrounding zone (P1), a result which is in agreement with previous findings in neurotypical adults. The timing of the N2 modulation evident in TD participants is consistent with a vast amount of evidence on the time course of attentional feedback to early visual areas (for a review see Wyatte et al., 2014). Signals reflecting attentional feedback occur within the range of 100–300 ms after stimulus onset both in macaque (Vidyasagar, 1998; Roelfsema et al., 1998; Mehta et al., 2000) and human (Martínez et al., 2001; Noesselt et al., 2002). Here, in TD participants we found a (attentional) surround-modulated N2 effect arising around 300 ms after the probe, and considering that in the present study adolescents were tested – who might have longer latencies due to an incomplete maturation of the fronto-parietal network (Giedd et al., 1999) – the timing seems to be in line with what has been reported in previous adult studies.

On the contrary, for the ASD group, the same suppression was not visible in the ventral visual pathway for target appearing in the surround of the attentional focus. Moreover, individual measures of surround suppression, calculated using both behavioural and neurophysiological data, predicted the autistic symptomatology as measured by the SCQ score. Specifically, the degree of inefficiency in inhibiting visual information outside the focus of attention was associated with higher ASD symptom severity.

Although no difference in the N2 temporal window was found in the ASD group, they showed an earlier modulation of neural activity as a function of the position of the probe stimulus in the P1 component of the ERP, with stronger cortical sources estimated in the inferior parietal lobe. These findings appear in line with previous results of an abnormally strong “spotlight” of visual spatial attention in autism as measured by P1 ERP modulation (Townsend and Courchesne, 1994). This stronger activation of the inferior parietal cortex in the ASD group may reflect a stronger recruitment of areas that are important for automatic attentional processes (Cabeza et al., 2012). Specifically, we speculate that this may reflect the effect of a ‘hyperfocused’ attention exhibited by the ASD group, in agreement with previous psychophysical reports (Robertson et al., 2013; Ronconi et al., 2012; Ronconi et al., 2013b). This stronger focalization of visual attention has been associated in the neurotypical population with an increased in the P1 amplitude (Fu et al., 2005), as well as with higher activations in the left inferior parietal lobule (Chen et al., 2009), compatibly with our ERPs results and their source reconstruction. This interpretation seems to be in agreement with other findings regarding the relationships between attentional ERP modulation, visual search performance, and the broader ASD spectrum (e.g. Milne et al., 2013), which seems to suggest a greater demand on downstream suppressive mechanisms in ASD (for a review see Belmonte, 2017).

A weaker suppression surrounding the focus of attention in the ASD group suggests an unbalanced relationship between neural mechanisms of enhancement and suppression at the locus of visual attention, and is likely to dramatically affect the way in which persons with ASD engage with the visual environment. Weak surround suppression may also explain different aspects of visual perception in ASD. Arguably the ASD group did indeed manifest a better representation of visual information (e.g., enhancing local contrast sensitivity) in the vicinity of the attentional focus, which could be related to better performance in task such as visual search (Joseph et al., 2009; Plaisted et al., 1998) and visual crowding (Baldassi et al., 2009; Kösta et al., 2010). Nevertheless they concurrently showed less inhibition of the visual information outside the focus of attention. This peculiar visual profile could lead to tremendous problems when irrelevant information is concurrently presented with relevant information. An anomalous interference from irrelevant lateral information has been found for example using an Eriksen flanker task (Eriksen and Eriksen, 1974) in a study by Adams and Jarrold (Adams and Jarrold, 2012) where the nature of impaired distractor inhibition found was not ascribable to a higher-order inhibitory problem. Accordingly, in a previous work of our group, it was demonstrated that people with ASD experienced a deeper and prolonged backward interference (i.e., attentional masking) relative to controls only when a laterally displayed irrelevant object, but not a central one, was presented after a central target that they had to discriminate (Ronconi et al., 2013a).

At a neurophysiological level, it may be speculated that a weak suppression surrounding the focus of attention can arise from diminished top-down modulation from the fronto-parietal attentional network coupled with an augmented neural representation of visual objects in visual areas. There is a consistent amount of evidence from both human (Brefczynski and DeYoe, 1999) and macaque (e.g., Vidyasagar, 1998; Nasi et al., 2013) studies, showing surround effects as early as V1 due to top-down attention. In addition, increasing evidence supports the idea that ASD is characterized by reduced functional connectivity between distant neural areas (Belmonte et al., 2004; Di Martino et al., 2013; Just et al., 2004; Khan et al., 2013; Minshew and Williams, 2007; Rubenstein and Merzenich, 2003; Vissers et al., 2012), with a conspicuous reduction in fronto-occipital connection (Bartfeld et al., 2011; Courchesne and Pierce, 2005; Jou et al., 2011). There are also recent reports lending support to the hypothesis of diffuse focal local over-connectivity in occipitotemporal region, where the visual objects are processed (Di Martino et al., 2013; Keown et al., 2013). Thus, the inefficient surround suppression that leads to a non-modulated activation of the ventral visual stream in ASD is likely to result from impaired feedback projections from the attentional network (i.e. fronto-parietal areas) coupled with an augmented visual representation of irrelevant objects in visual associative areas (i.e., occipito-temporal areas).

To conclude, the present findings show that individuals with ASD manifest a spatial profile of the attentional focus characterized by a weak suppression surrounding the attended area. This altered inhibitory ring can be one of the main factors underlying the profile of strengths and weaknesses in the visual domain typically associated with ASD. Remarkably recent findings show a direct relationship between superior attentional abilities and ASD diagnosis in infants at 9 and 15 months of age (Gliga, Bedford, Charman, Johnson, and BASSIS, 2015; Cheung et al., 2016). Moreover, as attention is known to be a supramodal function that operates on multiple sensory modalities (Banerjee...
et al., 2011; Farah et al., 1989; Green et al., 2011), this altered center-surround profile can be postulated also for the altered sensory processing present in the tactile and auditory domains (Kern et al., 2006; Leekam et al., 2007). Finally, recent findings demonstrated that similarly the representation of information in working memory is maintained with the same center-surround profile that acts in visuo-spatial selection (Kiyonaga and Egner, 2016). This suggests interesting hypotheses – which could be tested in future studies – about how this altered general mechanism for selection of information can influence higher cognitive functions in people with ASD.

Supplementary data to this article can be found online at https://doi.org/10.1016/j.nicl.2018.02.014.

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