

# Enhanced Access to Early Visual Processing of Perceptual Simultaneity in Autism Spectrum Disorders

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**Abstract** We compared judgements of the simultaneity or asynchrony of visual stimuli in individuals with autism spectrum disorders (ASD) and typically-developing controls using Magnetoencephalography (MEG). Two vertical bars were presented simultaneously or non-simultaneously with two different stimulus onset delays. Participants with ASD distinguished significantly better between real simultaneity (0 ms delay between two stimuli) and apparent simultaneity (17 ms delay between two stimuli) than controls. In line with the increased sensitivity, event-related MEG activity showed increased differential responses for simultaneity versus apparent simultaneity. The strongest evoked potentials, observed over occipital cortices at about 130 ms, were correlated with performance differences in the ASD group only. Superior access to early visual brain processes in ASD might underlie increased resolution of visual events in perception.

**Keywords** High-functioning autism · Asperger syndrome · Event timing · Visual simultaneity · Magnetoencephalography

## Introduction

There is increasing interest in the perceptual and sensory peculiarities associated with autism spectrum disorders (ASD), in part because although these symptoms are not currently included in the diagnostic criteria, they are common in affected individuals (Leekam et al. 2007).

Findings of superior performance on some perceptual tests have increased our knowledge about the autistic cognitive profile, particularly visuo-spatial cognition, including visual search (Plaisted et al. 1998), mental rotation (e.g., Falter et al. 2008; Soulières et al. 2011), and figure-disembedding (e.g., Edgin and Pennington 2005; Falter et al. 2008; Jarrold et al. 2005; Shah and Frith 1983). Additionally, individuals with ASD excel in a psychophysical test of temporal event structure (Falter et al. 2012). Temporal event structure coding is the processing of temporal relationships between events, such as the relative temporal order or the relative simultaneity or asynchrony of events. Short visual temporal intervals of about 57 ms have been reported to represent units of subjective time (Brecher 1932). These units are understood as temporal windows that determine whether events are perceived as simultaneous (if they fall within the same window) or asynchronous (if they fall into two different windows). Several higher-order functions that are impaired in ASD such as language processing (Tallal et al. 1993) and social interaction (Trevorthen and Daniel 2005) require temporal event structure coding and in particular correct sequencing of events.

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In previous studies of visual simultaneity perception (e.g., Elliott et al. 2007), participants were presented with two vertical bars to the left and right of the centre of the screen and had to judge whether the onset of the bars was simultaneous or asynchronous. Different stimulus-onset asynchronies (SOAs) were tested to determine the threshold of visual simultaneity for each individual. Using this paradigm with a group of individuals with high-functioning ASD, it was found that the average threshold of simultaneity was significantly lower in individuals with ASD compared to a typically-developing (TD) control group (Falter et al. 2012), indicating enhanced visual temporal resolution in ASD. The group difference was robustly reflected in individual data. One key question is what the psychophysical mechanism underlying superior resolution of visual events in time are. There are several cognitive accounts that incorporate findings of superior perceptual processing in ASD, such as Weak Central Coherence Theory (Happé and Frith 2006), Reduced Generalisation Theory (Plaisted 2001), and Enhanced Perceptual Functioning Theory (Mottron and Burack 2001). Although there is only inconsistent evidence of impaired global visuo-spatial processing in ASD (Falter et al. 2010; Mottron et al. 1999; Plaisted et al. 1999), evidence of superior performance in tasks requiring detail-focussed and piecemeal processing of visual information is more established (Dakin and Frith 2005). With regards to temporal event structure coding, this enhanced parsing of visual information could plausibly lead to increased discrimination of visual events in time and therefore to lower thresholds of perceptual simultaneity.

The focus of this study was on whether increased temporal resolution of visual events is reflected in brain activity in early visuo perceptual processing areas. In particular, we were interested in whether individuals with ASD would employ similar but superior functioning mechanisms to controls, or whether temporal event structure is coded using different mechanisms in ASD. The current study investigated brain activity associated with simultaneity perception. This was tested in perceived simultaneity in a condition in which simultaneity was indeed present and in a condition in which asynchrony was so small that stimulus onset appeared predominantly simultaneous.

We administered a modified version of the perceptual simultaneity task (Elliott et al. 2007) to facilitate robust between-condition comparisons of event-related brain activity measured using Magnetoencephalography (MEG). In particular, the task employed simple black-white transitions in order to enhance the temporal starting point of stimulus events compared to the previous study. We expected similar behavioural differences between individuals with ASD and controls as found in the extended task version (Falter et al. 2012).

## Methods

### Participants

Eighteen adolescents and adults with a clinical diagnosis of ASD (16–38 years) were selected from amongst participants in previous ASD studies at the Department and through advertising. ASD diagnoses were confirmed using the Autism Diagnostic Interview—Revised, ADI-R (Lord et al. 1994) and the Autism Diagnostic Observation Schedule—Generic, ADOS-G (Lord et al. 2000). Verbal, performance, and full scale IQ scores were assessed using the Wechsler Abbreviated Scale of Intelligence, WASI (Wechsler 1999). Exclusion criteria for participation were a diagnosis of a comorbid psychiatric disorder, current medication, and a full scale IQ < 85. The ADI criteria for age of onset was not met by two of the participants with Asperger syndrome and two others scored one point below threshold on one ADI algorithm domain. Nevertheless, these four participants scored above the Autism cut-off on the other ADI domains and above the ASD cut-off on the ADOS-G algorithm and were included in the final analysis. Two clinical participants were not included (one because of falling below ADI algorithm cut-offs in multiple domains and one due to premature button presses during the task). Consequently data from 16 participants with a diagnosis of Asperger syndrome ( $N = 14$ ) or high-functioning autism ( $N = 2$ ) according to DSM-IV-TR (APA 2000), were included in the analysis (see Table 1). Eighteen TD participants (15–38 years) were recruited through their prior involvement in studies at the Department or through poster advertisements. Exclusion criteria for the TD group were any psychiatric diagnoses, any medication, and a full scale IQ < 85. One control participant was excluded from the analysis because IQ scores were not obtained. Consequently data from seventeen TD participants entered the final analysis.

The ASD and TD groups were well-matched on chronological age, verbal IQ, and performance IQ (largest  $t(31) = 1.14$ ,  $p = .261$ ). All participants had normal or corrected-to-normal vision. Written informed consent was obtained from all participants prior to any testing and ethics approval for the study was granted by the National Research Ethics Service UK. A large subgroup of individuals participating in this study also participated on the same day in a MEG study of auditory processing and Gestalt perception.

### Design and Procedure

The experiment was conducted in a dimly lit scanning room (mean screen surround luminance  $0.1 \text{ cd/m}^2$ ) to reduce the impact of onscreen persistence, with stimulus luminance maintained at  $0.4 \text{ cd/m}^2$  upon a background field of  $0.075 \text{ cd/m}^2$ . Stimuli were generated and presented using

**Table 1** Means, standard deviations, and ranges of age (years:-months), verbal IQ (VIQ), performance IQ (PIQ), and full IQ (FIQ) of participants with an autism spectrum disorder (ASD) and typically-developing (TD) participants

	ASD (N = 16; 1 female)			TD (N = 17; 4 females)		
	Mean	SD	Range	Mean	SD	Range
Age	24:1	7:0	16:9–38:3	26:3	6:6	15:9–38:6
VIQ	114	9	99–127	114	12	99–139
PIQ	112	15	75–136	117	9	104–136
FIQ	115	11	88–131	117	9	101–141
ADI-A	17	5	10–25			
ADI-B	15	4	9–21			
ADI-C	5	3	2–10			
ADOS-A	3	2	1–7			
ADOS-B	6	3	1–11			
ADOS-C	1	1	0–4			

ADI-R Social Interaction Domain (ADI-A), ADI-R Communication Domain (ADI-B), ADI-R Repetitive Behaviours Domain (ADI-C), ADOS-G Communication Domain (ADOS-A), ADOS-G Reciprocal Social Interaction Domain (ADOS-B), and ADOS-G Stereotyped Behaviours and Restricted Interests Domain (ADOS-C) of participants with ASD

**Table 2** List of abbreviations used in the description of results

SIM	0 ms Condition (the two bars appear simultaneously on screen)
SHORT	17 ms Condition (the second bar is delayed by 17 ms)
LONG	117 ms Condition (the second bar is delayed by 117 ms)
PERFDIFF	% Simultaneous responses in SIM—% simultaneous responses in SHORT
SIM_sim	Trials with simultaneous responses in SIM condition
SHORT_sim	Trials with simultaneous responses in SHORT condition
SHORT_asyn	Trials with asynchronous responses in the SHORT condition
LATDIFF	Peak latency difference between SIM_sim and SHORT_sim

Inquisit 3 (Millisecond Software 2003) and back-projected onto a translucent screen. Participants viewed two white bar stimuli (subtending 4.6° of visual angle vertically and 0.5° horizontally) which were positioned to the left and right of the centre of the screen (randomised side of first stimulus) with a distance of 7.2° visual angle between them. In contrast to a previous version of this task, in which stimuli faded in over several steps of luminance increase (Falter et al. 2012), the current experiment used simple onset stimuli in order to simplify the MEG analysis.

Three conditions were presented in pseudo-randomised order: the two vertical bars were presented (a) simultaneously

(SIM), (b) separated by a very short delay of 17 ms (SHORT), or (c) by a long delay of 117 ms (LONG), see Table 2 for an overview of used abbreviations. The main reason for inclusion of the long SOA condition was to maintain attention throughout the task by presenting perceptually different trials. Each trial started with a blank 500 ms ISI followed by stimulus presentation with simultaneous or asynchronous onset and simultaneous offset 17 ms after second stimulus onset. A response cue was presented 1,500 ms after stimulus offset. Participants were asked to respond if the two bars were simultaneous or asynchronous by pressing one of two buttons. Participants were instructed to avoid eye movements during stimulus presentations and to restrict eye blinks and button presses to when the response cue appeared. The response cue remained on screen until a response was given. There was no feedback on performance. Participants completed a practice block of 10 trials followed by four experimental blocks consisting of 60 trials each. Each SOA was presented 80 times (i.e. 240 trials overall).

### Data Acquisition

Measurements were performed using a Neuromag-306 VectorView™ system, which consists of a helmet-shaped array of 102 pairs of orthogonal, first-order planar gradiometers. This system also has 102 magnetometers, whose output has not been analysed for this study. The outputs of each pair of gradiometers are most sensitive to tangential current flow in the region directly below the detectors. The local root-mean-square (rms) signal summed over the two readings is a measure of current strength.

The data were sampled at 1,000 Hz (0.03–330 Hz anti-alias filter; artifact correction using Maxfilter™; 40 Hz low-pass filter before subsequent analysis) and corrected for variation in individual head position before analysis using MaxMove™ (part of the scanner software package). Physiological artefacts were identified by recording the electrooculogram and electrocardiogram. MEG traces with coinciding eye-blinks were corrected using a PCA based approach (in-house software; algorithm based on spatial confound and sensor data correction routines provided by SPM8; see also Berg and Scherg 1994). A small number of epochs (<5 %) had to be excluded from the analysis due to other artefacts.

Evoked responses were averaged separately for each participant and for each of the three conditions. A baseline was established by averaging signal strength over the 100 ms preceding the onset of the first stimulus. A latency range of 0–800 ms after stimulus onset was chosen for analysis.

Statistical differences in event-related fields (ERFs) between the conditions of interest across participants were analysed employing a time-dependent measure  $P(t)$ , the details of which have been fully specified elsewhere (Braeutigam et al. 2004).

$$P(t) = \text{probability}(\chi^2) \quad (\chi^2) = -2 \sum_{i=1}^N \ln[f_i(t)]$$

$N$  denotes the number of channels. For each channel,  $f_i(t)$  denotes a non-parametric statistical test applied to evoked fields of interest. For each significant interval [ $P(t) < 0.01$ ], the spatial distribution of the significant difference was given by  $f_i$ . Correspondence between the spatial distribution of the significant difference and the spatial distribution of ERFs (using grand-average power maps = root-mean-square of the two gradiometers in a pair) was explored. Two different non-parametric tests ( $f$ ) were used in the formula above dependent on the nature of the comparison. Wilcoxon and Mann–Witney  $U$  tests were used for within and between group analyses, respectively.

## Results

### Behavioural Results

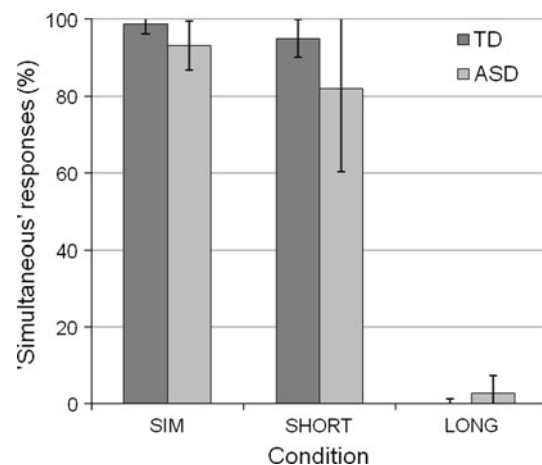
Mean percentages of ‘simultaneous’ responses and standard deviations for both groups are shown in Fig. 1. A mixed ANOVA with one between-participants factor of Group (ASD, TD) and one within-participants factor of Condition (SIM, SHORT, LONG) was performed. This analysis showed a significant main effect of Condition ( $F(2, 47) = 892.37$ ,  $p < .001$ , partial  $\eta^2 = .97$ , Huynh–Feldt corrected) and a trend towards a main effect for Group ( $F(1, 31) = 3.23$ ,  $p = .082$ , partial  $\eta^2 = .09$ ) indicating that the ASD group showed different percentages of ‘simultaneous’ responses across the three conditions compared with the TD group. The interaction between Group and Condition was significant ( $F(2, 62) = 8.15$ ,  $p = .001$ , partial  $\eta^2 = .21$ ). As revealed by post hoc analyses (Bonferroni–Holm corrected), the interaction was due to the ASD group giving significantly fewer ‘simultaneous’ responses in the SHORT condition ( $F(1, 31) = 6.40$ ,  $p = .017$ , partial  $\eta^2 = .17$ ;  $ASD = 76.53 \pm 21.84$ ,  $TD = 91.34 \pm 10$ ) and more ‘simultaneous’ responses in the LONG condition ( $F(1, 31) = 6.51$ ,  $p = .016$ , partial  $\eta^2 = .17$ ;  $ASD = 5.23 \pm 5.94$ ,  $TD = 1.37 \pm 1.88$ ) compared to the TD group. Performance in the LONG condition was at ceiling level preventing further analysis. The group difference in the LONG condition was in the opposite direction from the group difference in the SHORT condition; therefore, the effect in the SHORT condition is robust because any unspecific performance bias would have decreased rather than increased its magnitude.

As we were interested in the performance difference between the SIM and the SHORT conditions, which are perceptually similar but physically different, we calculated a difference measure between performance scores in the

two conditions. By subtracting the percentage of simultaneous responses in the SHORT condition from those in the SIM condition we took into account individual response biases (Falter et al. 2012). This performance difference index, PERFDIFF, was significantly different between groups ( $F(1, 31) = 7.02$ ,  $p = .013$ , partial  $\eta^2 = .19$ ) because participants with ASD distinguished better between the two conditions (simultaneous response SIM—simultaneous response SHORT:  $12.27 \pm 12.23$ ) than TD participants ( $3.97 \pm 4.03$ ). This group difference was not due to performance of only a few individuals with ASD but was shown in a majority of ASD individuals. Indeed, PERFDIFF of 10 ASD individuals (high-performing) was even larger than the upper 10<sup>th</sup> percentile of the TD group as compared to only two TD participants who showed such a high PERFDIFF. This difference in distribution was significant ( $\chi^2(1) = 9.17$ ,  $p = .002$ ; Yates corrected  $\chi^2(1) = 7.12$ , Yates corrected  $p = .008$ ).

### Event-Related Field Analysis

The event-related field analysis was performed on those trials of the SIM and SHORT conditions for which participants gave ‘simultaneous’ responses, henceforth referred to as SIM\_sim and SHORT\_sim. Comparing the ‘simultaneous’ trials in the two conditions allowed us to compare brain responses associated with real (physical) simultaneity and apparent simultaneity (i.e. asynchrony that is perceived as simultaneous). There were not enough trials with ‘asynchronous’ responses in both these conditions in the TD group. However, as the ASD group was more sensitive in the SHORT condition, their trials were divided for analysis into those trials in which they responded ‘simultaneous’ (SHORT\_sim) and those trials in



**Fig. 1** Means ( $\pm$ SD) of percentages of ‘simultaneous’ responses over conditions in the TD group (dark grey) and the ASD group (light grey)



which they responded ‘asynchronous’, henceforth termed SHORT\_asyn.

For both SIM\_sim and SHORT\_sim trials a complex sequence of neural responses was observed in both participant groups, where first appreciable evoked responses were detected over occipital cortices at about 90 ms after stimulus onset. Subsequently, widespread activity was found predominately over posterior parts of the brain including, to various extents, occipital, parietal and occipito-temporal regions at latencies between about 100 and 200 ms. During this latency span, a strong and prominent peak in evoked signal power was observed over occipital cortex in both groups at about 130 ms (Fig. 2); this signal was stronger over the left hemisphere in the TD group, whereas the ASD group showed a more balanced bilateral pattern. At longer latency, evoked activity became weaker and more widespread, including activity measured over frontal parts of the brain.

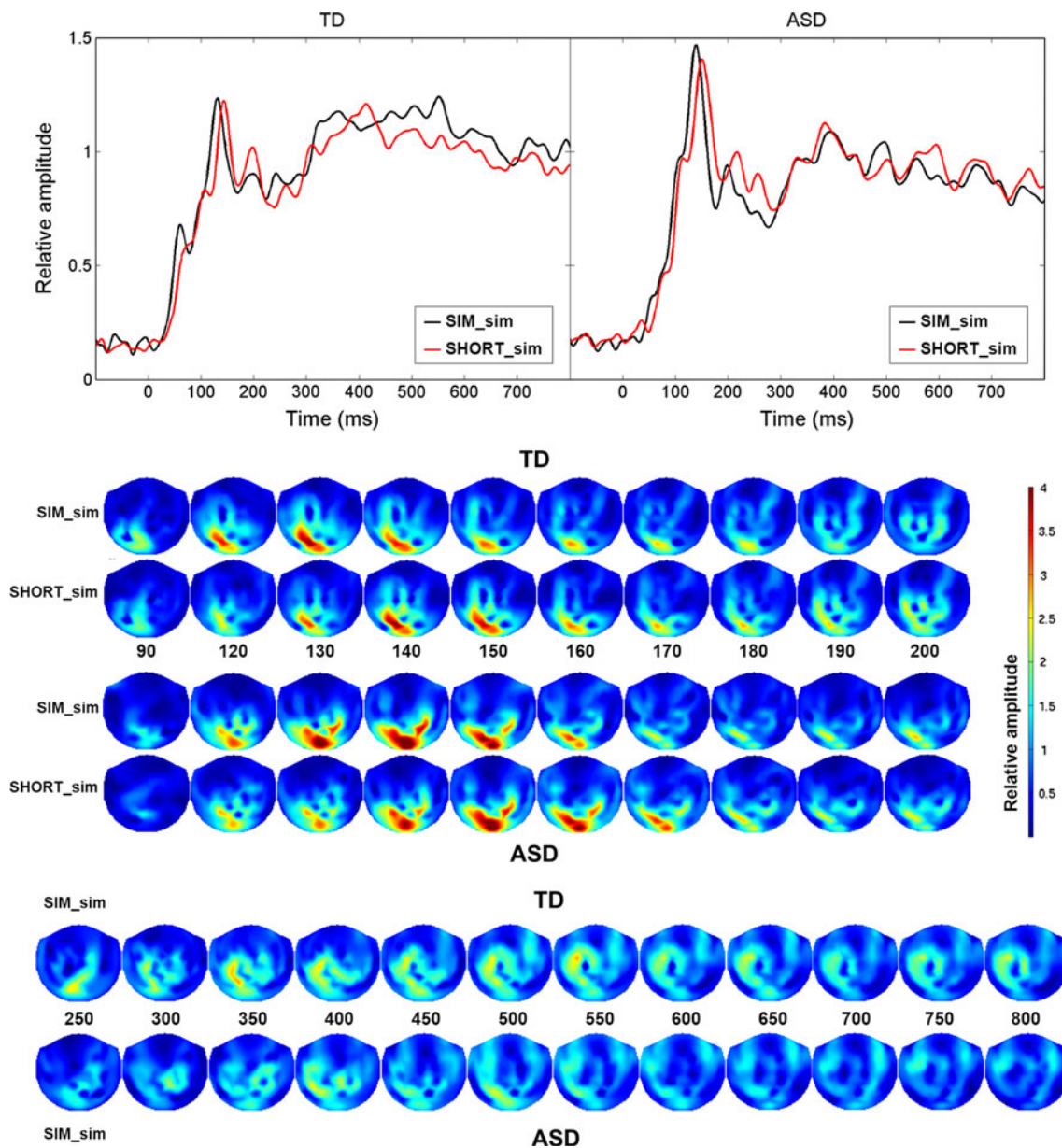
In the remainder of this section, the results obtained from two different but interrelated analyses are presented. First, an analysis of latencies at peak power corresponding to the strongest average brain response observed at about 130 ms is presented. Overall, evoked activity following SIM\_sim stimuli (average latency TD:  $118 \pm 17$  ms, ASD:  $125 \pm 21$  ms) peaked before SHORT\_sim (TD:  $127 \pm 19$  ms, ASD:  $135 \pm 22$  ms) and SHORT\_asyn (ASD:  $143 \pm 28$  ms). This difference was quantified by considering a simple measure LATDIFF calculated as the differences between individual SIM\_sim and SHORT\_sim peak latencies of the 130 ms component. This measure was correlated with the measure PERFDIFF in both groups. The correlation was significant in the ASD group (Spearman  $r(16) = -.595$ ,  $p = .015$ ) but not in the TD group ( $r(17) = .263$ ,  $p = .308$ ; see Fig. 3). A selective analysis based on the 10 high-performing individuals with ASD and the 10 TD participants with highest PERFDIFF showed that this effect was robust, i.e. a negative and significant correlation between the two measures was only present in the ASD group ( $r(10) = -.712$ ,  $p = .021$ ) but not the TD group ( $r(10) = .286$ ,  $p = .423$ ). In addition, the effect in the ASD group was not only due to increased attention or motivation in comparison with the TD group, as the performance benefit for the ASD group was restricted to the SHORT condition rather than generalised across conditions. Indeed, comparing the performance of the 10 individuals with ASD and the 10 TD participants with highest PERFDIFF reveals that it is only the SHORT condition in which individuals with ASD excel ( $F(1, 20) = 13.79$ ,  $p = .002$ , partial  $\eta^2 = .43$ ; ASD =  $63.75 \pm 18.01$ , TD =  $88.75 \pm 11.35$ ). In the SIM condition, the TD group performed better than the ASD group ( $F(1, 20) = 4.52$ ,  $p = .048$ , partial  $\eta^2 = .20$ ; ASD =  $82.88 \pm 15.47$ , TD =  $95.25 \pm 9.96$ ) and in the LONG condition there was no group difference ( $F(1, 20) = 2.75$ ,  $p = .114$ , partial  $\eta^2 = .13$ ; ASD =  $4.38 \pm 5.08$ ,

TD =  $1.5 \pm 2.11$ ). Thus, there were clear differences in the performance pattern between the ASD and the TD group.

Note that the group of high-performing individuals with ASD identified above showed a trend ( $z = -1.926$ ,  $p = .054$ ) for increased symptom severity in the non-verbal communication sub-scale of the ADI-R (median =  $5 \pm .63$ ) in comparison to the remaining six individuals with ASD (median =  $3 \pm .38$ ) and a trend of earlier symptom onset ( $z = -1.876$ ,  $p = .061$ ). Note also that, according to the power curves, there were no latency or amplitude differences between the groups in the SIM\_sim and SHORT\_sim conditions or LATDIFF (largest  $F(1, 31) = 1.311$ ,  $p = .261$ ) for the 130 ms response.

The second analysis was a within-group analysis of the evoked responses to SIM\_sim and SHORT\_sim stimuli utilising time dependent measure P in conjunction with a Wilcoxon test (see Fig. 4a). At 30 ms after stimulus onset, an interval of significant differences was observed only in individuals with ASD (Fig. 4a, yellow), where SIM\_sim stimuli evoked higher activity compared to SHORT\_sim stimuli over bilateral prefrontal cortices. Centered at about 150 ms, a sequence of significant intervals was observed consistent with the latency shift in peak power reported above. In both participant groups, neural activity elicited over posterior regions was stronger for SIM\_sim compared to SHORT\_sim stimuli, most notably during the early parts of this interval, whereas activity was stronger for SHORT\_sim compared to SIM\_sim stimuli during the later parts. There were, however, three important differences between the patterns of significance in the two groups. (A) In TD participants, significance was confined to a shorter interval 120–200 ms compared to 90–220 ms in individuals with ASD (Fig. 4a blue). (B) Strong and modulated (by condition) activity was more left lateralised in TD compared to ASD (Fig. 4b top; this lateralisation effect was confirmed by a between-group analysis of the SIM\_sim and SHORT\_sim responses using a Mann–Whitney  $U$  test;  $p < .01$ ). (C) Significance extended anteriorly in ASD compared to TD. In particular, significantly modulated signal amplitudes were found over bilateral anterior temporal cortices in ASD, although these components were relatively weak compared to the strong posterior signals (Fig. 4b bottom).

At longer latency, an interval of significant differences between the SIM\_sim and SHORT\_sim evoked signals was found at about 280 ms in both participant groups (Fig. 4a green), where significance was distributed over orbito-frontal and temporal (ASD only) cortices. It was not possible, however, to relate this effect to a condition-specific increase or decrease in evoked power. There were three subsequent intervals of significance found only in individuals with ASD at about 340, 400, and 460 ms after stimulus onset (Fig. 4a purple). Here,



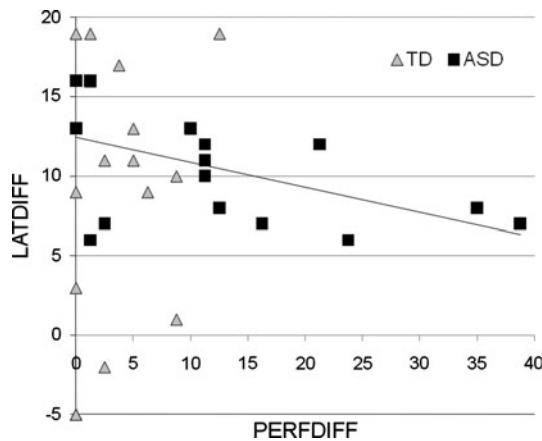
**Fig. 2** *Top*: grand root-mean-square (rms) signals in response to SIM\_sim (SIM condition, ‘simultaneous’ response) and SHORT\_sim (SHORT condition, ‘simultaneous’ response) conditions in the TD and ASD group. First stimulus onset is at 0 ms. The global rms-curves have been obtained by summation over all channels and all participants within a participant group. *Bottom*: spatial distribution

of local rms signals to SIM\_sim and SHORT\_sim between 90 and 200 ms and to SIM\_sim between 250 and 800 ms. The maps have been obtained by summation over all participants within each group. For presentation of the spatial distribution of data the detectors have been projected into two dimensions (right ear on the *right*, front at the *top*)

significant differences were widespread over central and right hemispheric areas implying increased evoked activity following SHORT\_sim compared to SIM\_sim stimuli.

In addition to the above analyses, brain activity was compared with respect to *perceived* event differences (but same physical event) in those ten individuals with ASD who had performed better than 90 % of the TD group; i.e.

evoked fields associated with SHORT\_sim and SHORT\_asyn. In the TD group the number of SHORT\_asyn trials (in which ‘asynchronous’ responses were given) was insufficient for a similar evoked analysis. This analysis revealed a significant increase in evoked activity following SHORT\_asyn stimuli over left anterior and left frontal cortices at about 100 ms compared to SHORT\_sim stimuli. Further intervals of significance were found at 290 and



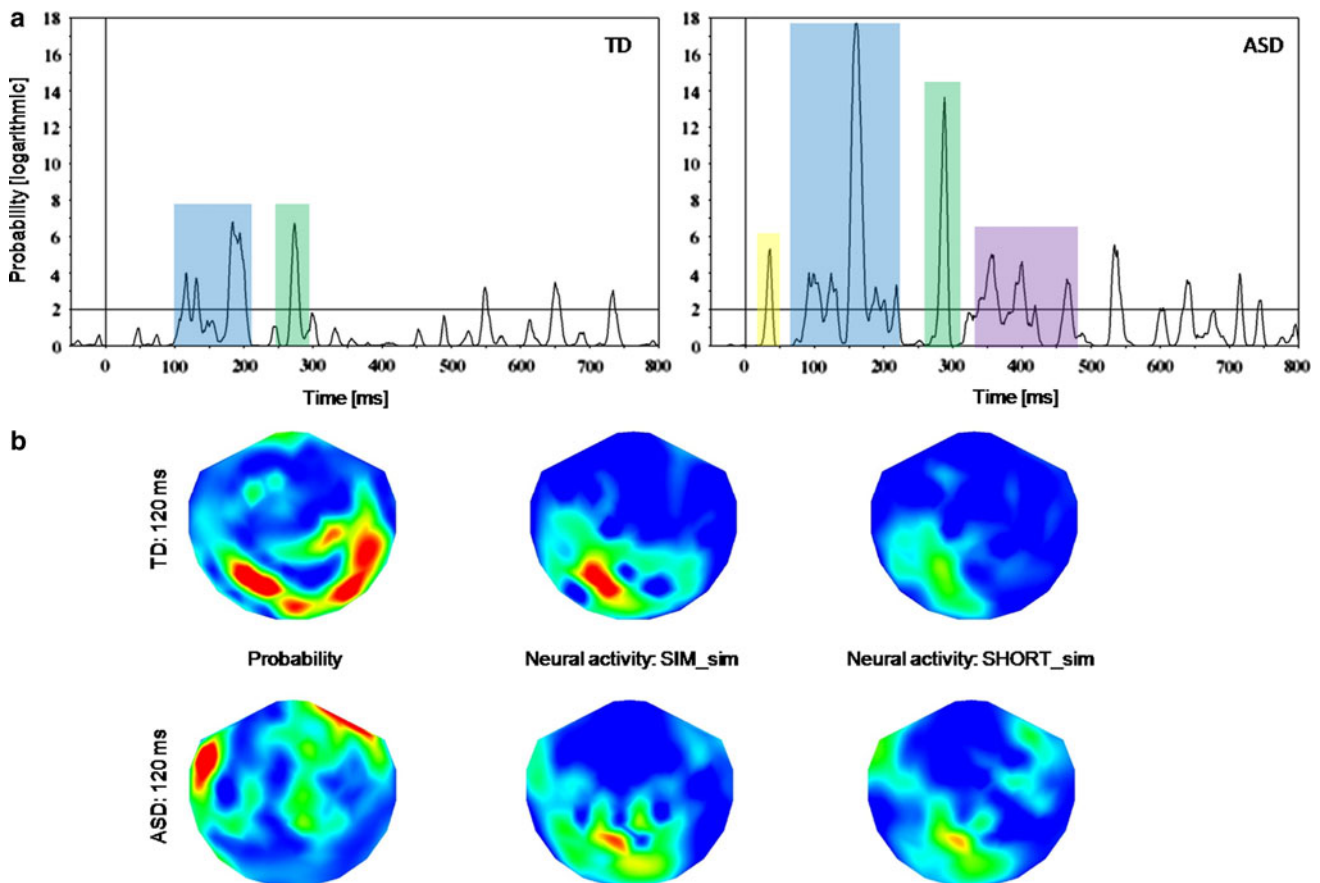
**Fig. 3** Correlation of the performance difference (PERFDIFF) with the latency difference (LATDIFF) between the SIM\_sim and SHORT\_sim conditions in the TD group (*grey triangles*) and the ASD group (*black squares, trend line*). Note that only 14 TD and 15 ASD symbols are visible due to overlapping of latency/performance values at the chosen numerical resolution

400 ms, but these did not show modulation of activity beyond brain areas already identified above.

In summary, as an overall pattern, the ASD group showed earlier and longer relevant activity differences between conditions compared to the TD group. There was evidence for more power measured over the left than the right hemisphere in the TD group compared to a more balanced pattern in the ASD group. Activity in this task was generally strongest over occipital regions in both groups, but there was more modulation of signals over other areas in the ASD group than the TD group.

**Discussion**

Many cognitive functions such as communication (Tallal et al. 1993), social interaction (Trevarthen and Daniel 2005), and the planning and execution of actions (e.g., Hommel et al. 2001) rely on coding of the relative temporal



**Fig. 4 a** Significant difference between SIM\_sim and SHORT\_sim stimuli in TD individuals (*left*) and individuals with ASD (*right*). The curves are based on measure  $P(t)$  reflecting cumulative levels of significance over all sensors. For subsequent analysis, intervals with  $2 \leq P(t)$  (corresponding to  $p \leq 0.01$ ) were considered significant (see text for colour coding). **b** An illustration of the differential effects in TD and

ASD individuals at about 120 ms after stimulus onset. In TD participants SIM\_sim stimuli evoked more neural activity over left posterior cortices compared to SHORT\_sim stimuli. Note that not all significant areas (probability maps; red corresponds to 0.01 and green to 0.1) relate to easily identifiable changes in signal power (neural activity). The maps have been normalised to TD data in condition SHORT\_sim (Color figure online)

structure between events and integration of events in terms of correct sequencing. In a previous psychophysical study it was shown that individuals with ASD show abnormal event structure processing in that they showed increased temporal resolution in a perceptual simultaneity task as compared to TD controls (Falter et al. 2012). The aim of the current study was to measure event-related brain activity associated with real and apparent simultaneity in a perceptual task. In line with the psychophysical study we found an enhanced temporal resolution in the ASD group similar to the lower simultaneity threshold in behavioural performance previously found (Falter et al. 2012). Indeed, the ASD group in the current study showed even higher temporal resolution than in the previous study, which is most likely due to a difference in task design. While the onset of stimuli in the earlier, psychophysical study were luminance-defined, with a fading in sequence of several steps of increasing luminance, the current version of the task included simple stimulus onsets, for reasons of simplicity of MEG analysis. The sudden stimulus onsets made the temporal starting point of the stimulus events more salient in the current study and might have increased the resolution of perceptual simultaneity.

The overall topography and timing of neural activity measured with MEG was similar in both groups. Nevertheless, the current study provided some evidence for a left hemisphere dominance of MEG activity associated with perceptual simultaneity in the TD group. This was in contrast to the more bilateral activity in the ASD group, which is in line with previous imaging studies (e.g., Chiron et al. 1995; Gage et al. 2009) as well as behavioural findings (Efron 1963; Falter et al. 2012). The finding of additional engagement of the right hemisphere in the ASD group suggests different underlying mechanisms. Indeed, more detailed analysis of MEG data showed that there were an increased number of significant event-related power differences between the simultaneous and the apparently simultaneous condition in the ASD group compared to the TD group. This might reflect an increase in neural processes aimed at perceptual differentiation between conditions in the ASD group even if behavioural responses are the same. The differential response in the ASD group might be indicative of the ability to discriminate SOA differences which were not discriminated by the TD group.

However, measures of symptom severity were, despite of trends for earlier symptom onset and a trend of a non-verbal communication difference, not significantly different between the high-performing group of individuals with ASD and remaining individuals with ASD. This finding may suggest that better temporal resolution does not necessarily improve autistic symptoms; instead, enhanced temporal resolution tends to be associated with increased communication difficulties and earlier symptom onsets,

which corroborates previous findings that problems with non-verbal communication are associated with increased visual temporal resolution (Falter et al. 2012).

Of particular interest is the finding that relative latency of early brain responses associated with the SIM\_sim and SHORT\_sim conditions predicted performance in the ASD group, but not in the TD group. This finding could be interpreted in terms of judgement or decision making processes that rely on the input/pre-processing provided by the primary and secondary visual system in the ASD group. This putative input level was characterised by earlier and longer lasting significant brain activity in the ASD group, with more areas involved in processing of the short latency compared with the TD group. These characteristics of early processing might have made a ‘not simultaneous’ response more likely. In other words, the physical difference between stimuli might be received but is not sufficiently processed in TD individuals to make them depart from the assumption of simultaneity. The early effects (at about 30 ms; SIM\_sim versus SHORT\_sim) observed over anterior regions in the ASD (but not TD) group might reflect atypical involvement of higher-order control in primary and secondary visual processing. Although it cannot be ruled out that enhanced anterior activity in the ASD group could be an epiphenomenon (related to, say, atypical but task-irrelevant attention processes), it might well be indicative of alternative strategies in ASD developed, in part, as compensatory mechanisms necessitated by abnormal executive or other higher-order processes. Note that differential effects over anterior regions were not observed before 100 ms when comparing SHORT\_sim and SHORT\_asyn stimuli. This suggests that physical stimulus differences but not perceived stimulus differences elicited a very early brain activity difference; we may, therefore, argue that early visual processing in ASD is characterised by increased temporal resolution of physical event structure, but not necessarily perceived event structure at this stage of processing.

Thus, the current findings suggest that processing of temporal event-structure in ASD is characterised by two components: (a) elevated resolution at a basic physiological level, and (b) atypical involvement of higher order function in primary/secondary visual processing.

Over and above the differences in neural processing between TD and ASD, the question arises as to why participants responded ‘simultaneous’ in many SHORT trials given that the asynchrony was clearly associated with differential neural activity (compared to SIM) at both early and long latency. Although the current study did not measure experience of simultaneity, and therefore one cannot judge whether real and apparent simultaneity were truly perceived the same, we emphasise the discrepancy in simultaneity judgements contrasting with the apparent



neuronal processing of asynchrony. Such a discrepancy between performance and brain activity is not unusual and has been reported in previous studies testing typical participants. For instance, there is substantial evidence of perceptual phenomena other than stimulus timing, showing a discrepancy between neuronal coding of stimulus features and phenomenal experience of these stimulus features (e.g. Corbin 1942; Gur and Snodderly 1997; Haynes et al. 2003; Haynes and Rees 2005; Rock and Broscole 1964; Rock and Ebenholtz 1962). In their study on chromatic flicker perception, Gur and Snodderly (1997) describe dissociation between brain activity and perception. Their participants perceived colour fusion for two isoluminant colours alternating with a rate of >10 Hz. This perceived colour fusion happened despite colour opponent cells in primary visual cortex (V1) resolving chromatic flicker up to 60 Hz, i.e. a rate strongly exceeding the colour fusion threshold. The visual system therefore showed processing of physical stimulus features that were not perceived. Similarly, in a recent fMRI study Haynes and Rees (2005) reported that although information on the orientation of invisible stimuli could be found in V1, participants were not able to use this information and performed at chance level. In general, these previous findings show that participants sometimes cannot consciously access the neuronal processes that pick up stimulus differences. This explanation could relate to observations in the current study; TD participants might generally be unable to access fine temporal differences between stimuli, which nevertheless are neurally processed. In contrast, individuals with ASD appear to have more access to those neuronal processes coding differences in temporal event structure. The enhanced access to early visual processes and thereby to the factual (rather than interpreted) stimulus characteristics could underlie increased parsing of visual information in space and time by individuals with ASD (Dakin and Frith 2005; Falter et al. 2012).

We are aware that this study is limited in scope making it difficult to generalise the results beyond what was tested explicitly. In particular, the range of tested SOAs was limited to three in order to balance the duration of testing time for patients. As a consequence, the TD group showed little variation in the SHORT condition—responding almost exclusively ‘simultaneous’—which prevented the analysis of brain activity sorted by responses that was performed in the ASD group. Thus, future studies might consider a wider range of SOAs and include a confidence rating rather than binary decisions. This approach could provide more detailed insight into the relationship between perceived timing and event-related brain activity.

In conclusion, we found increased temporal resolution in a perceptual simultaneity task and enhanced access to early visual processing stages in ASD. Increased parsing of

temporal event structure, similar to the spatial local processing bias observed in ASD (Dakin and Frith 2005), might be the result of the direct and increased access to sensory information from early visual processing stages.

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