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**The temporal dynamics of coherent motion processing in autism spectrum disorder:  
evidence for a deficit in the dorsal pathway**

Greimel, E.<sup>1</sup>, Bartling, J.<sup>1</sup>, Dunkel, J.<sup>2</sup>, Brückl, M.<sup>1</sup>, Deimel, W.<sup>2</sup>, Remschmidt, H.<sup>2</sup>, Kamp-Becker, I.<sup>2</sup> & Schulte-Körne, G.<sup>1</sup>

<sup>1</sup>Department of Child and Adolescent Psychiatry and Psychotherapy, University Hospital Munich, Munich, Germany

<sup>2</sup>Department of Child and Adolescent Psychiatry, University Hospital Giessen and Marburg, Campus Marburg, Germany

**Address for reprints or correspondence:**

Ellen Greimel, Department of Child and Adolescent Psychiatry and Psychotherapy, University Hospital Munich, Pettenkoferstraße 8a, 80336 Munich, Germany

E-mail: [Ellen.Greimel@med.uni-muenchen.de](mailto:Ellen.Greimel@med.uni-muenchen.de), Phone: +49 89 51605914, Fax: +49 89 51605902



## **Abstract**

Individuals with autism spectrum disorder (ASD) show impairments in processing coherent motion which have been proposed to be linked to a general deficit in the dorsal visual pathway. However, few studies have investigated the neural mechanisms underlying coherent motion processing in ASD. Thus, the aim of this study was to further test the hypothesis of a dorsal pathway deficit in ASD using visual evoked potentials (VEPs).

16 children and adolescents with ASD and 12 typically developing controls were examined with VEPs elicited by a random dot kinematogram. After an initial experimental sequence, where subjects were presented randomly moving dots, a fraction of the dots moved coherently (dependent on the level of coherence, 20%, 40%, or 60% of the dots) to the left or right side. Subjects were asked to detect the direction of coherent motion via button press.

On the behavioural level, no significant group differences emerged. On the neural level, coherently moving dots elicited a N200 followed by a late positive potential (P400). ASD subjects exhibited a reduced N200 amplitude compared to controls. Moreover, in the ASD group, a trend for a negative relationship between N200 amplitude and a measure of autistic pathology was revealed.

The present study provides strong support of a dorsal stream deficiency in the disorder and renders alternative explanations for impaired coherent motion processing in ASD less likely. Together with findings from related research fields, our data indicates that deviances in the N200 during coherent motion perception might be fundamental to ASD.

**Keywords:** autism spectrum disorder; coherent motion; VEPs; psychophysiology; dorsal pathway

## 1. Introduction

Autism spectrum disorder (ASD) is a heterogeneous neurodevelopmental disorder characterised by impairments in social interaction, communication, as well as restrictive interests and behaviour [1]. Moreover, the disorder is associated with perceptual peculiarities [2]. In the visual domain, ASD individuals are less susceptible to visual illusions [3], show superior visual search [4] and outperform healthy controls in embedded figure tasks [5]. These advantages in processing local and featural information are contrasted by a *relative failure* to extract global information or meaning and to process information within its context. For example, ASD subjects have difficulties integrating visual elements to a coherent scene [6]. Moreover, using random dot kinematograms (RDK), several neuropsychological studies have shown that ASD subjects are less sensitive to coherent motion, i.e., the threshold for detecting coherently moving dots in an array of randomly moving dots is higher compared to healthy persons (e.g., [7,8], but see e.g., [9,10] for conflicting results). The influential Weak Central Coherence theory [11] assumes that such deficits in global processing in ASD are the result of a superiority in local processing characterized by a bias towards a detail-oriented cognitive style.

The characteristic perceptual profile in ASD, and particularly findings of deficient global motion processing as indexed by increased coherent motion thresholds, have prompted the idea that in ASD, there may be a general deficit in the magnocellular/dorsal pathway of the visual processing system [7,12], which can roughly be separated into two streams. *The dorsal pathway* projects via the magnocellular layer of the lateral geniculate nucleus (LGN) to the primary visual cortex (V1), forward to the middle temporal area (MT/V5) and then to the posterior parietal cortex. It is sensitive to stimuli of low contrast and is involved in processing movement and spatial relationships of objects. By contrast, *the ventral pathway*, which projects via the parvocellular layer of the LGN to V1 forward to V4 and then to the inferior

temporal lobe, is sensitive to stimuli of high contrast and plays an important role in object and colour discrimination [13,14]. The dorsal pathway has a higher processing speed compared to the ventral pathway, what has been referred to as the “magnocellular advantage” [15].

Based on this well-investigated theory of visual processing and findings of elevated motion thresholds [7,8], the idea that deficient coherent motion processing in ASD is the result of a general deficit in the dorsal stream [7,12] seems plausible. An alternative hypothesis that has been put forward to explain impaired global motion processing posits that ASD subjects have difficulties integrating complex perceptual information (including complex motion stimuli) regardless of the system involved in its processing [16]. In other words, this hypothesis claims that ASD is associated with a more general impairment of perceptual processing that not only affects the visual system, but rather can be found across different sensory modalities. Support for this assumption comes from studies in children with ASD showing that flicker contrast sensitivity, known to reflect lower-level dorsal stream functioning, is intact [17,18].

Moreover, Bertone et al. [16] found that in ASD subjects motion sensitivity is decreased for second-order (texture-defined) motion stimuli, but normal for first-order (luminance-defined) motion.

Neurobiological studies on coherent motion and related visual perceptual processes constitute a complementary and more direct approach to examine the underlying mechanisms of deficient coherent motion processing in ASD and to test the two alternative hypotheses that have been put forward. However, to date, only few studies have addressed the neural mechanisms of basic visual perception processes in ASD that give insight into dorsal pathway function (e.g., [19-21]) and knowledge on the neural processes underlying coherent motion perception in ASD is particularly sparse. One of the neurobiological studies that explored basic visual perception and its relation to autistic traits [22] included subjects from the normal population scoring high vs. low on a measure of autistic psychopathology (the Autism Spectrum Quotient, AQ) [23]. The authors recorded non-linear visual evoked potentials

(VEP) to stimulus sequences of low vs. high contrast and analyzed them by estimating Wiener Kernels to separate the inputs of the magnocellular and parvocellular pathway (for further details see also [24]). In the high AQ group, a weaker initial cortical response of magnocellular origin at low contrast was found compared to the low AQ group. Moreover, in high AQ subjects, a delay in magnocellular pathway processing was found for the high contrast condition. These findings imply that the temporal magnocellular advantage in these subjects is diminished which, in turn, might explain the abnormal visual perceptual profile in ASD subjects. However, as the study included low vs. high AQ scorers and no patient group, it is unclear to what extent the results are directly applicable to the disorder itself. To our knowledge, only one study addressed the neural processes underlying coherent motion perception in ASD. Using the RDK, which is particularly suited to assess dorsal pathway processing as it targets global motion processing [25], a recent functional Magnetic Resonance Imaging (fMRI) study [9] reported abnormal activation in V1, MT and superior parietal cortex in ASD subjects. These findings suggest that abnormal global motion processing in ASD might be associated with abnormalities at lower and higher processing stages of the dorsal stream.

Based on these previous findings, it would be important to further shed light on the neural bases of coherent motion processing in ASD. Given different assumptions on the processing stages of motion perception that are assumed to be impaired in ASD subjects, it would be of particular importance to elucidate the temporal dynamics of global motion processing in the disorder. In healthy controls, VEP have been a preferred experimental approach to study the neurocognitive bases of motion perception including coherent motion processing due to the high temporal resolution of the method. VEP evoked by motion are dominated by transient negativity at a latency around 150-200ms, (N200) which is generated over the extrastriate occipito-temporo and parietal cortex (for a review see [25]). In the majority of studies, the N200 was evoked by the onset of coherent motion [26]. Among other factors that influence

this component, the N200 is modulated by motion coherence levels with higher amplitudes and shorter latencies at higher levels of coherence [27].

Besides the N200, two VEP studies in children and adults also reported a late positivity to coherent motion [28,29], which emerges between 300 to 800 ms after stimulus onset at posterior sites (P400 [29]). The P400 likely reflects the integration of perceptual information and has been supposed to be modality-independent [30]. One of the VEP studies on coherent motion processing could show that this late positive potential was modulated by coherence, with larger deflections at higher levels of motion coherence [28]. However, based on ERP findings from other research fields, the reported modulation effect might rather be the result of differential task difficulty associated with different coherence levels [30-32] than an effect of coherent motion per se.

To our knowledge, the temporal course of coherent motion processing in ASD has not been investigated in subjects with ASD using VEPs. Thus, the present study was set up to fill this gap and to further investigate the neural bases of global motion perception in ASD. We included children and adolescents with ASD as well as healthy controls and applied a RDK paradigm, which has been repeatedly proven to predominantly target dorsal pathway processing and to reliably elicit the N200 [25].

If the hypothesis of a general dorsal stream deficit in ASD were true, we would expect an attenuated N200 in subjects with ASD. If, on the other hand, the hypothesis of an abnormal perceptual integration of complex information (regardless of the perceptual system involved) were true, we would assume that ASD subjects exhibit attenuated later ERP components that are involved in the integration of visual and other perceptual information, e.g., in the P400 [29,30].

## **2. Methods**

### ***2.1 Participants***

17 children and adolescents with ASD, and 17 control subjects took part in this study. 16 ASD subjects, diagnosed with Asperger syndrome ( $n=10$ ) or high-functioning autism ( $n=6$ ) and 12 controls were included in the final statistical analysis (see “Data analysis” for further details on exclusion criteria). Only subjects with an  $IQ \geq 85$  (based on the WISC-III [33]) were included. The groups of the final study sample were comparable with regard to age, IQ, and gender (see Table 1).

-----Insert Table 1 about here-----

ASD subjects were recruited from the Department of Child and Adolescent Psychiatry in Marburg. They had been diagnosed by experienced clinicians according to ICD-10 [34] and DSM-IV [1]. Diagnoses were confirmed by the Autism Diagnostic Observation Schedule- Generic (ADOS-G) [35,36], a standardized observational instrument for assessing behaviour relevant to autism, and a semi-structured interview for caregivers of children with ASD (Autism Diagnostic Interview-Revised) [37,38], which were conducted by a certified examiner (I.K.-B.).

With regard to psychiatric comorbidities, one subject had been diagnosed with comorbid specific phobic disorder and one with enuresis. A comorbid diagnosis of attention deficit hyperactivity disorder was an exclusion criterion for the present study.

Control subjects were screened to exclude psychiatric disorders using the Child Behaviour Checklist [39,40]. Moreover, ASD symptoms in controls were screened based on a German screening instrument for Asperger syndrome or high-functioning autism [41].

None of the ASD subjects and control subjects received any medication or suffered from any relevant neurological or somatic disorders. In all subjects, visual acuity was assessed based on a Landolt C-chart. Only subjects were included whose normal or corrected visual acuity (near vision) in both eyes and during binocular viewing was 0.5 or better.



The study was approved by the institutional review board of the Department of Child and Adolescent Psychiatry in Marburg and was performed in accordance with the latest version of the Declaration of Helsinki and in compliance with national legislation. All participants were informed in detail about the experimental procedures and the aims of the study, and provided written informed assent. Written informed consent was obtained by at least one parent/legal custodian, after the parent(s)/legal custodian(s) had been informed about all aspects of the study.

## ***2.2 Experimental procedure***

### ***2.2.1 Stimuli***

The stimuli presented during the coherent motion task comprised a rectangular patch containing 300 randomly arranged white dots on a black background. At 60 cm viewing distance the patch of dots subtended  $8 \times 12^\circ$ . The luminance of the dots was 86 cd/m<sup>2</sup> and background luminance was 1.2 cd/m<sup>2</sup> resulting in a Michelson contrast of 97%. The angular size of each pixel was 0.038 and the speed of moving dots was 5°/s. To make it difficult for subjects to track single dots and to force them to process motion globally, 10% of the dots disappeared after each frame change (60 Hz) and reappeared at a random location within the stimulus patch.

### ***2.2.2 Coherent motion task***

Each experimental trial comprised three sequences (for a similar approach see [27]). In all sequences, moving dots surrounded by a box were presented. In the initial sequence, each dot moved independently of the others in a random direction for 1080 ms. This phase was followed by a coherent motion sequence, where a fraction of the dots moved coherently (depending on the level of coherence, 20%, 40%, or 60% of the dots) to the left or right side

horizontally for 420 ms. Following the coherent motion sequence, dots moved again randomly for 1080 ms, until the box with the dots disappeared from the screen. The inter-trial interval was set to 2000 ms. In each trial, participants indicated the direction of coherent motion by pressing the respective mouse button (left button for leftwards and right button for rightwards movement). The response window started with the onset of coherent motion and lasted 3500 ms (onset of the next trial). The direction of motion and the level of coherent motion were presented randomly, with 30 trials for each direction and each level of coherent motion, respectively. Additionally, 20 control trials with 90% coherence level (10 leftwards, 10 rightwards shifts) were randomly interspersed with the experimental trials. To familiarize participants with task requirements, subjects first practiced the task before ERP data were acquired.

### **2.3 EEG recording**

The EEG was recorded from Ag/AgCl electrodes, mounted on an elastic electrode cap configured according to the equidistant 61-channel-arrangement (model M10; EASYCAP GmbH, Herrsching, Germany; Fig. 1). The inter-electrode distance is approximately 3.7 cm (given a head circumference of 58 cm). The arrangement is made up of triangles, which are measured on the threedimensional head surface and are placed around Cz. The horizontal electrooculogram was recorded from two Ag/AgCL electrodes positioned at the outer canthus of each eye. The vertical electrooculogram was recorded via two electrodes placed below and above the left eye, respectively. Electrodes were referenced to the right mastoid.

-----insert Figure 1 about here-----

Electrode impedances were kept below 5 k $\Omega$ . The EEG was amplified with a BrainAmp system (Brain Products GmbH, Gilching, Germany), with a low frequency cut-off at 0.1 Hz and an upper frequency cut-off at 70 Hz (sampling rate: 500 Hz).

## ***2.4 Data analysis***

### ***2.4.1 ERP data***

EEGs were analysed using Brainvision Analyzer 1.05 (Brain Products GmbH, Gilching, Germany). After filtering (lowpass 40 Hz, highpass 0.53 Hz), manual removal of EOG artefacts based on Independent Component Analysis and exclusion of other artefacts based on an automatic mode (gradient: max 50  $\mu$ V; max-min: 150  $\mu$ V for 200 ms windows; amplitude: max 100  $\mu$ V min -100  $\mu$ V; low activity 100  $\mu$ V for 100 ms windows; no individual channel mode), the signal was re-referenced to the linked mastoid. The data was segmented into epochs (-100 to 1000 ms), baseline-corrected and averaged separately for each participant and coherence condition (20%, 40%, or 60% coherence). For inclusion into the final analysis described in the following sections, ASD and control subjects had to meet two criteria: (1) a minimum of 20 artefact-free trials in each coherence condition, and (2)  $\geq 65\%$  correct responses in control trials with 90% coherence level. One subject with ASD and five control subjects did not meet these statistical criteria (the ASD subject and 2 control subjects not meet either of these criteria; 3 control subjects did not meet the second criterion), resulting in a final sample of 28 participants (16 ASD subjects and 12 control subjects). Individual ERPs were averaged for each condition. Finally, grand averages were computed separately for the control and the ASD group.

Based on previous findings on coherent motion processing [27], we defined a ROI for the N200 over the occipital cortex and a second ROI over the and parietal-occipital cortex.

Moreover, in line with the literature [29], two ROIs were defined for the P400, one including parietal electrodes and a second ROI including occipital electrodes. The occipital ROI for the N200 and P400 included electrodes 41 42 43, 44, 45, 54, 55, 56, 57, 58 and the parietal-

occipital ROI for the N200 electrodes 26, 27, 28, 29. The ROI over the parietal region for the P400 included electrodes 4, 5, 6, 12, 13, 14, 15, 16 (see Fig. 1). “

Upon visual inspection, the ERP response across all subjects comprised a negative potential at around 220-280 ms (N200) over occipital and parietal-occipital electrodes and a large positive deflection peaking at around 450-500 ms (P400) over parietal and occipital electrodes.

Based on visual inspection of single electrodes, the time window used to determine individual peak amplitudes and latencies for the N200 was set to 130-300 ms for the parietal-occipital ROI and to 150-360 ms for the occipital ROI. The time window used to determine individual peak amplitudes and latencies for the P400 was set to 300-800 ms for the parietal ROI and to 380-950 ms for the occipital ROI. Moreover, we assessed the area under the curve for the P400 using the identical time windows for the two ROIs (300-800 ms for the parietal site and 380-950 ms for the occipital site). VEPs from single electrodes within the above defined ROIs were averaged for statistical analysis.

Since exploratory analyses did not reveal lateralisation effects, the data of all electrodes were averaged for each condition. Group differences in latency and amplitudes of the N200 and P400 were investigated using repeated measures ANOVAs with group as between-subject factor, and coherence level (20%, 40%, 60%) and electrode site (parietal/parietal-occipital for the N200 and parietal/occipital for the P400) as within-subject factors. Moreover, group differences in the area under the curve for the P400 were investigated based on a repeated measures ANOVA with group as between-subject factor, and coherence level (20%, 40%, 60%) and electrode site (parietal/occipital) as within-subject factors.

#### *2.4.2 Behavioural data*

For reaction times (RTs) and the percent of correct responses, repeated measures ANOVAs with coherence (20%, 40%, 60%) as within subject factor and group as between subject factor were computed.

Statistical analyses of the ERP data and behavioural data were conducted with IBM SPSS Statistics 20. For all analyses, the significance level was set to  $p=.05$  (two-tailed). In all ANOVAs, Huynh-Feldt correction was applied to correct for violations of the sphericity assumption (Mauchly's test).

### **3. Results**

#### ***3.1 Behavioural results***

##### ***3.1.1 Percent correct responses***

The percentage of correct responses was comparable across groups ( $F(1,26)=2.0$ ,  $p=.184$ ,  $\eta_p^2=0.067$ ) and coherence levels ( $F(1.4,26)=1.3$ ,  $p=.283$ ,  $\eta_p^2=0.046$ ). The interaction between group and coherence level was found to be non-significant ( $F(1.4,26)=0.1$ ,  $p=.829$ ,  $\eta_p^2=0.004$ ). Separate group results are summarized in Table 2.

-----Insert Table 2 about here -----

##### ***3.1.2 Reaction times***

RTs of correct responses were comparable across groups ( $F(1,26)=0.3$ ,  $p=.579$ ,  $\eta_p^2=0.012$ ). RTs became faster with increasing coherence level ( $F(2,26)=38.4$ ,  $p<.001$ ,  $\eta_p^2=0.606$ ). The interaction between group and coherence level failed to be significant ( $F(2,26)=2.5$ ,  $p=.091$ ,  $\eta_p^2=0.091$ ). RTs for the ASD and control group are summarized in Table 2.

#### ***3.2 VEP results***

Group means of amplitudes and latencies of the N200 and the P400 are summarized in Table 3.

-----Insert Table 3 about here -----

### 3.2.1 N200 amplitude

A significant main effect of group was revealed for the N200 amplitude ( $F(1,26)=4.3$ ,  $p=.048$ ,  $\eta_p^2=0.142$ ), with ASD subjects showing a smaller N200 amplitude compared to controls (see Fig. 2a and 2b.)

-----insert Fig. 2a and 2b about here -----

Moreover, the N200 exhibited a smaller amplitude over occipital ( $M=-2.4\pm 1.3$ ) compared to parietal-occipital electrodes ( $M=-3.6\pm 2.2$ ;  $F(1,26)=22.0$ ,  $p<.001$ ,  $\eta_p^2=0.458$ ). No differences emerged between the different coherence levels ( $F(2,52)=0.4$ ,  $p=.669$ ,  $\eta_p^2=0.015$ ). However, on a mere descriptive level, the N200 amplitude over both sites increased at higher levels of coherence across all subjects. None of the interactions were found to be significant (all  $p\geq .177$ ).

### 3.2.2 N200 latency

N200 latency did not significantly differ between groups ( $F(1,26)=0.1$ ,  $p=.732$ ,  $\eta_p^2=0.005$ ) or coherence levels ( $F(1.8,46.0)=0.4$ ,  $p=.647$ ,  $\eta_p^2=0.015$ ). Again, descriptive data collapsed across groups indicated shorter N200 latencies over both occipital and parietal-occipital electrodes with increasing motion coherence. The N200 occurred earlier over parietal-occipital ( $M=228.7\pm 30.1$ ) compared to occipital sites ( $M=279.8\pm 30.4$ ;  $F(1,26)=50.4$ ,  $p<.001$ ,  $\eta_p^2=0.695$ ). Again, none of the interactions proved to be significant (all  $p\geq .108$ ).

### 3.2.3 P400 amplitude

P400 amplitude over both sites (occipital, parietal ROI) was comparable across groups ( $F(1,26)=0.5$ ,  $p=.822$ ,  $\eta_p=0.002$ ). A significant main effect for coherence level was revealed ( $F(2,52.0)=39.7$ ,  $p<.001$ ,  $\eta_p=0.327$ ), which was due to a higher amplitude at higher levels of coherence (see Fig. 3a and 3b).

-----insert Fig. 3a and 3b about here -----

A significant main effect was also found for electrode site ( $F(1,26)=97.8$ ,  $p<.001$ ,  $\eta_p=0.790$ ), with a smaller amplitude over occipital compared to parietal electrodes. The interaction between electrode site and coherence level was found to be significant ( $F(2,52)=7.6$ ,  $p<.01$ ,  $\eta_p=0.225$ ). Further investigation of the interaction revealed that the P400 amplitude increased at higher coherence levels over occipital electrodes but not over parietal electrodes across both groups. No further significant interactions were revealed (all  $ps>.629$ ).

#### *3.2.4 P400 latency*

No significant main effect of group was revealed for P400 latency ( $F(1,26)=1.4$ ,  $p=.245$ ,  $\eta_p=0.052$ ). By contrast, a significant main effect of coherence was found ( $F(2,52.0)=20.8$ ,  $p<.001$ ,  $\eta_p=0.444$ ; earlier peaks at higher coherence levels). Moreover, the P400 peaked earlier over parietal compared to occipital electrodes ( $F(1,26)=68.1$ ,  $p<.001$ ,  $\eta_p=0.724$ ). None of the interactions proved to be significant (all  $ps>.282$ ).

#### *3.2.5 P400 area*

Groups did not differ with regard to P400 area ( $F(1,26)=0.3$ ,  $p=.617$ ,  $\eta_p=0.010$ ). Again, significant main effects were found for coherence ( $F(2,51.2)=3.6$ ,  $p=.035$ ,  $\eta_p=0.122$ ; larger area at higher levels of coherence) and electrode site ( $F(1,26)=91.5$ ,  $p<.001$ ,  $\eta_p=0.779$ ; larger area over parietal electrodes). Moreover, a significant interaction between coherence level and

electrode site was revealed ( $F(2,26)=9.0$ ,  $p<.001$ ,  $\eta_p^2=0.309$ ) resulting from an increased P400 area at higher levels of coherence over occipital, but not parietal electrodes.

### *3.2.6 Additional analyses: Correlations between N200 amplitude and autism severity.*

To examine the relationship between autism severity and the N200 amplitude in the ASD group, Pearson's  $r$  correlations were computed between the ADOS-G score and the three ADI-R subscale scores (see Table 1), and the N200 amplitude averaged across coherence levels and the a priori-defined ROIs for this component (parietal-occipital and occipital ROI, see "Data analysis"). These analyses revealed a marginal significant negative correlation between N200 amplitude and the ADI-R communication scale ( $r=-.44$ ;  $p<.097$ ). The remaining correlations were all non-significant (all  $ps>.223$ ).

## **4. Discussion**

The present VEP study sought to examine the neural correlates of coherent motion processing in ASD. On the neural level, ASD subjects showed a deviant N200 across coherence levels. On the behavioural level, no significant group differences were observed. It may seem striking that although at the behavioural level, no differential effects could be observed, distinct neural mechanisms were detected between groups. However, our findings are in line with a recent meta-analysis on visual functioning in ASD [2], which reported comparable behavioural performance in ASD and controls along with robust group differences in neural processes. With this regard, it is worth stressing that neurobiological measures can be more sensitive than behaviour [42]. Moreover, the absence of group difference on the performance level warrants that neurophysiological differences between ASD and control subjects cannot be dismissed as artifacts related to a differential capacity to perform the task.



#### ***4.1 Behavioural data***

In accordance with other studies using the RDK [27,43], RTs decreased with increasing coherence. This expected finding supports the validity of the experimental procedure and presumably reflects shorter visual detection time at higher levels of motion coherence [27].

Our finding of comparable behavioural performance in the RDK paradigm in ASD and control subjects might in part be explained by a ceiling effect, as the percentage of correct identification of coherent motion exceeded 90% in both groups for all levels of coherence (see [27] for similar findings).

Our results are in line with a previous fMRI study [9], which applied a similar experimental design as in the present study by using fixed coherence levels above coherent motion detection thresholds. Such an approach might not be as sensitive to group differences as studies of coherent motion detection thresholds that repeatedly reported impaired performance (i.e., increased thresholds) in ASD subjects (e.g., [7,8]; but see, e.g., [10]). Intact performance of the ASD group in our study might also pertain to the fact that the majority of ASD subjects were diagnosed with Asperger syndrome, as previous studies focusing on this diagnostic subgroup have failed to find evidence of impaired coherent motion processing [9,44].

#### ***4.2 Psychophysiological responses***

We found a deviant N200 in ASD subjects during coherent motion processing, as reflected in a reduced amplitude of this component compared to controls. The N200 can be regarded as the main motion specific component of motion-onset VEPs and has been predominantly linked to dorsal pathway processing [25]. Thus, the present study provides strong support of a dorsal stream deficiency in ASD [7,12] and extends previous findings from neurobiological studies in ASD (e.g., [9,22]).

In the ASD group, we found a trend towards a negative relationship between N200 amplitude and the ADI-R communication scale, indicating that somewhat smaller amplitudes were

evoked in subjects with elevated scores on this measure. This correlational trend is ~~intriguing~~ rather unexpected as a number of previous studies using behavioural measures have failed to find evidence of a relationship (or a trend thereof) between performance in coherent motion perception and autistic psychopathology (e.g., [18,46]). Communication problems in ASD, as assessed by the respective ADI-R subscale, include problems in processing more global aspects of language, such as language generalization, and context-sensitive language use or comprehension. Indeed, previous research has demonstrated a reduced ability to infer global meaning from sentences [47,48] and stories [49] in ASD. The trend for a negative relationship between the communication subscale and N200 amplitude in ASD subjects might suggest that disturbed neural mechanisms of global perceptual processing perhaps contribute to impairments of autistic subjects to process language in a global fashion (see also [11,49]). However, given the facts that (1) the correlation between the two measures did not reach significance and that (2) a replication of our result is needed, this suggestion remains speculative and needs to be followed up in future studies.

It should be mentioned that deficient coherent motion processing is not unique to ASD, but has been reported for a number of neurodevelopmental disorders, including dyslexia and Williams syndrome (e.g., [50,51]), prompting the idea that a “dorsal stream vulnerability” might be present in these disorders [7,12]. Of note, previous VEP studies on motion processing in dyslexia [28,52] have identified abnormalities in dyslexic children in components other than the N200, including the P100 and the P200. Thus, taken together with the present findings, this suggests that deficits in dorsal stream processing are qualitatively different in autism and dyslexia. As similarly suggested before [18], dorsal stream processing might be disrupted at different development stages in autism and dyslexia, resulting in different impairments in coherent motion processing. In future studies, it would be worthwhile to shed light on the developmental trajectories of neural processes underlying

coherent motion processes in autism, dyslexia and other neurodevelopmental conditions to draw a clearer picture of “dorsal stream vulnerability” in these patient groups.

It needs to be discussed that we did not find a significant effect of coherence level on N200 amplitude and latency. This finding might perhaps pertain to insufficient statistical power to reveal a modulation effect. Indeed, our descriptive data are in line with findings from Patzwahl et al. [27], who reported shorter latencies along with larger amplitudes of the N200 component at higher levels of motion coherence. The absence of a coherence effect in the present study can also be explained by the fact that our experimental procedure only comprised three different experimental conditions (20%, 40%, 60% coherence) that spanned only a limited range of motion coherence levels (20-60%). By contrast, previous studies that reported significant effects of coherence level on the N200 applied more different coherence levels (5-10) comprising a wider range of motion coherence levels (up to 0-100%) [26,27]. The ladder approach is likely to be more sensitive to reveal significant coherence effects. Moreover, it should be mentioned that N200 latency in the present study was shorter than latencies typically reported in previous studies on motion processing in adults [25,54]. This difference can be presumably drawn back to a prolonged development of the magnocellular system beyond adolescence, which is (amongst other characteristics) reflected in a shortening up of N200 latency until the age of 18 [55].

The P400 component was observable both in subjects with ASD and in healthy controls. Coherent motion perception represents a complex task that requires integrating visual information across space and time [45]. In a previous study, the P400 component has been shown to be evoked by different classes of complex visual stimuli, including ~~coloured~~ stationary coloured dots and moving stimuli [29]. In line with our findings, this study reported maximal amplitudes of the P400 over the parietal electrode sides. The P400 has been suggested to reflect integration of perceptual information independent of the nature of the perceptual stimuli [29]. Our finding of shorter P400 latencies along with greater amplitudes at

higher levels of motion coherence can be brought in line with this suggestion; stronger motion coherence represents a stronger visual signal what might result in a stronger and faster neural response related to integration processes (see also [27]). In accordance with our results, previous ERP studies reported enhanced P400 responses with decreasing difficulty of detection or discrimination of different perceptual stimuli [30-32]. Thus, taken together, the modulation effect observed for the P400 might not be driven by motion coherence per se but is likely to be an epiphenomenon of the differential difficulty associated with the three coherence levels.

The absence of group difference in the P400 component renders the hypothesis that deficits in motion processing in ASD can be explained by abnormal perceptual integration of complex information less likely [16]. However, future VEP studies in ASD should present visual stimuli that differ both in complexity and visual class (e.g., motion vs. colour stimuli) to further examine the complexity hypothesis.

#### ***4.3 Limitations and conclusion***

A limitation of the present study is the relatively small sample size in combination with a relatively large age- and IQ-range of the control and the ASD group. This might perhaps explain the absence of group differences on the behavioural level and the non-significant effect of coherence on the N200 amplitude due to restricted statistical power. Therefore, our results need to be replicated in a larger sample of ASD and control subjects.

Moreover, our clinical sample comprised only high-functioning ASD subjects. Based on the findings from the present investigation, future studies should investigate whether the results can be generalized across the whole spectrum of autistic disorders. Despite these caveats, this study is the first to explore neurophysiological mechanisms underlying coherent motion processing in ASD using VEPs. Our finding of a reduced N200 amplitude during the RDK paradigm substantially adds to previous findings on abnormalities in the dorsal visual

pathway in ASD [9,22]. Abnormalities in processing visual information in ASD are present early in development and have been shown to contribute to social problems in affected individuals, including impairments in face processing [19]. Thus, in the long run, a deeper understanding of the neurobiological bases of abnormal visual perception in ASD is crucial to target key issues related to the disorder.

Based on the suggestion of a prolonged development of the motion processing system in healthy subjects [25], it would be an important future research aim to explore the developmental time course of dorsal visual processing in ASD. Such an approach would help to understand at which age exactly the deficits emerge, are present or become more pronounced, and thus provide important information on when interventions targeting deficits in dorsal visual processing (e.g., guided instruction to pay attention to global information [56]) might be most beneficial for subject suffering from ASD.

### **Conflicts of interests**

None

### **Acknowledgments**

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## Tables

**Table 1** Demographic characteristics of the study sample

	<b>ASD group (n=16)</b>	<b>control group (n=12)</b>
Age (M, SD)	12.7 (2.2)	12.0 (2.2)
Age range (Min-Max)	9 - 16	8 - 15
IQ (M, SD)	110.1 (16.2)	114.0 (10.2)
IQ range (Min-Max)	(89 – 141)	(93 – 125)
sex (male/female)	15/1	11/1
Handedness <sup>a</sup> (right/left)	15/1	11/1
ADOS-G Total (M, SD)	12.4 (3.6)	n.a.
ADI-R Social Interaction (M, SD)	17.5 (6.3)	n.a.
Communication (M, SD)	13.7 (4.7)	n.a.
Stereotyped Behaviour (M, SD)	5.9 (3.0)	n.a.

Abbreviations: ASD = autism spectrum disorder

<sup>a</sup>Edinburgh handedness inventory (Oldfield, 1971)

**Table 2** Behavioural performance in the coherent motion task

	<b>ASD group (n=16)</b>	<b>control group (n=12)</b>
Correct responses (%)		
20% coherence (M, SD)	92.0 (11.3)	96.0 (4.5)
40% coherence (M, SD)	94.5 (7.2)	97.4 (3.1)
60% coherence (M, SD)	93.9 (8.2)	96.9 (5.0)
Reaction time		
20% coherence (M, SD)	871.5 (130.5)	825.6 (94.9)
40% coherence (M, SD)	775.4 (102.6)	775.8 (116.4)
60% coherence (M, SD)	778.4 (107.0)	752.4 (118.2)

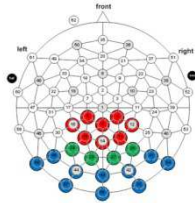
Abbreviations: ASD = autism spectrum disorder

**Table 3** Amplitudes and latencies of the N200 and the P400

	ASD group (n=16)	control group (n=12)		ASD group (n=16)	control group (n=12)
N200 amplitude <i>occipital ROI</i>			P400 amplitude <i>occipital ROI</i>		
20% coherence (M, SD)	-1.9 (1.4)	-2.8 (1.5)	20% coherence (M, SD)	4.5 (2.0)	4.1 (1.7)
40% coherence (M, SD)	-2.3 (1.2)	-2.7 (2.0)	40% coherence (M, SD)	4.3 (2.5)	4.9 (1.5)
60% coherence (M, SD)	-1.9 (1.7)	-3.3 (1.8)	60% coherence (M, SD)	5.0 (2.4)	5.1 (1.5)
N200 amplitude <i>occipital-parietal ROI</i>			P400 amplitude <i>parietal ROI</i>		
20% coherence (M, SD)	-2.5 (2.2)	-4.5 (2.9)	20% coherence (M, SD)	9.1 (3.4)	9.3 (3.5)
40% coherence (M, SD)	-3.2 (2.4)	-4.5 (2.7)	40% coherence (M, SD)	10.5 (3.9)	11.1 (3.5)
60% coherence (M, SD)	-3.2 (2.5)	-4.6 (2.2)	60% coherence (M, SD)	11.6 (4.4)	11.9 (4.4)
N200 latency <i>occipital ROI</i>			P400 latency <i>occipital ROI</i>		
20% coherence (M, SD)	287.0 (42.0)	271.1 (51.8)	20% coherence (M, SD)	699.1 (78.8)	653.9 (90.3)
40% coherence (M, SD)	283.0 (38.5)	278.2 (36.6)	40% coherence (M, SD)	642.0 (85.9)	616.9 (7.5)
60% coherence (M, SD)	276.6 (28.0)	280.7 (36.2)	60% coherence (M, SD)	627.8 (75.9)	592.1 (95.2)
N200 latency <i>occipital-parietal ROI</i>			P400 latency <i>parietal ROI</i>		
20% coherence (M, SD)	237.1 (43.4)	233.4 (45.9)	20% coherence (M, SD)	540.6 (97.2)	520.4 (84.3)
40% coherence (M, SD)	228.0 (46.2)	226.7 (27.3)	40% coherence (M, SD)	481.4 (59.5)	457.3 (60.2)
60% coherence (M, SD)	222.4 (42.6)	223.8 (35.6)	60% coherence (M, SD)	470.6 (41.9)	480.2 (102.6)

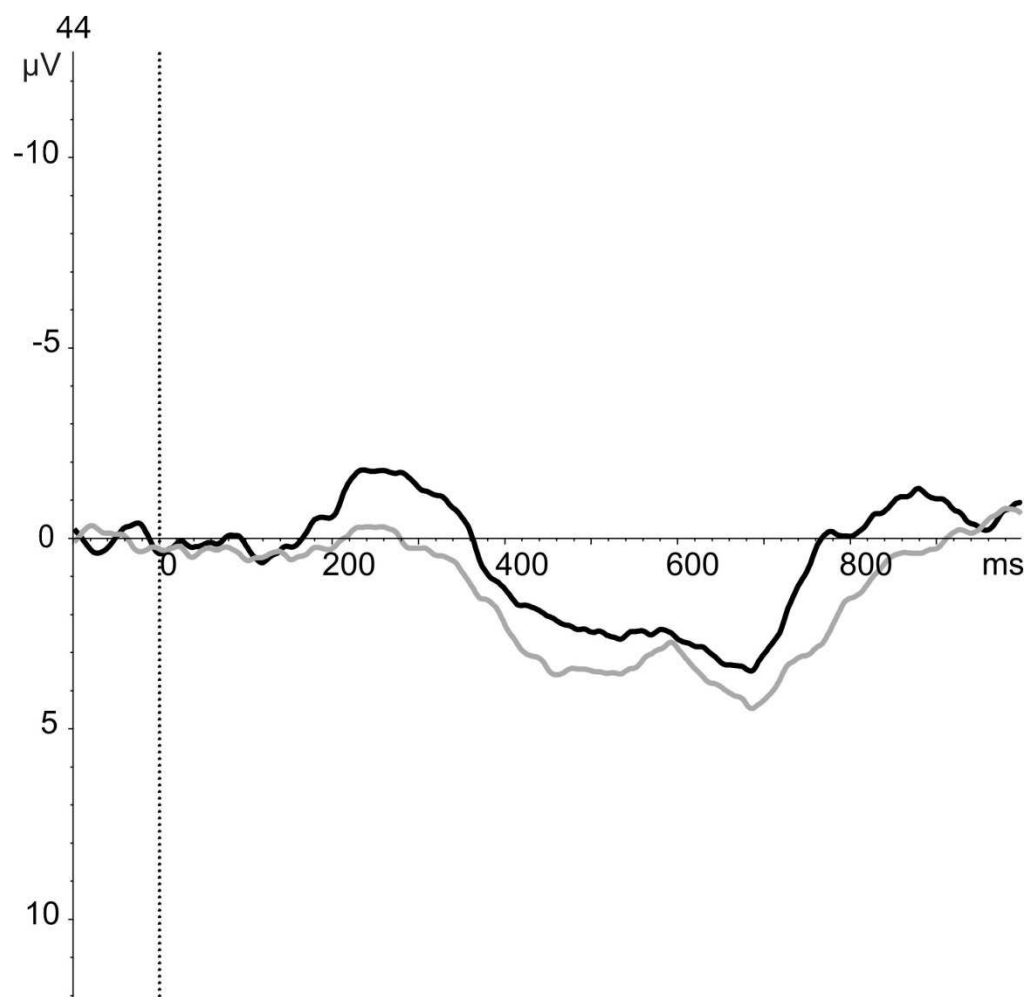
Abbreviations: ASD = autism spectrum disorder; ROI = Region of Interest

## Figures



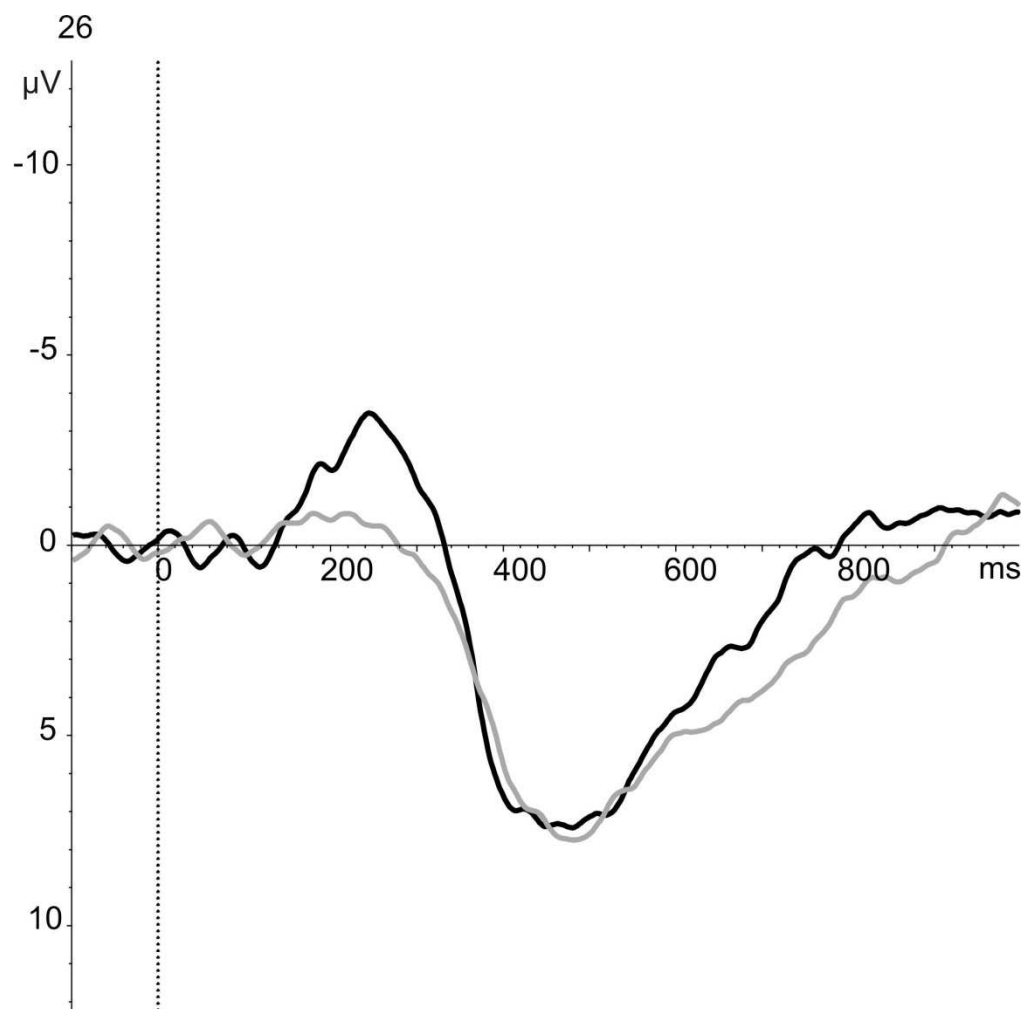
**Figure 1**

Illustration of the 61-channel-arrangement and electrode position taken from Easycap GmbH, Herrsching, Germany. Electrodes included in the parietal region of interest (ROI) are depicted in red, electrodes included in the parietal-occipital ROI in green, and electrodes included in the occipital ROI in blue.



**Figure 2a**

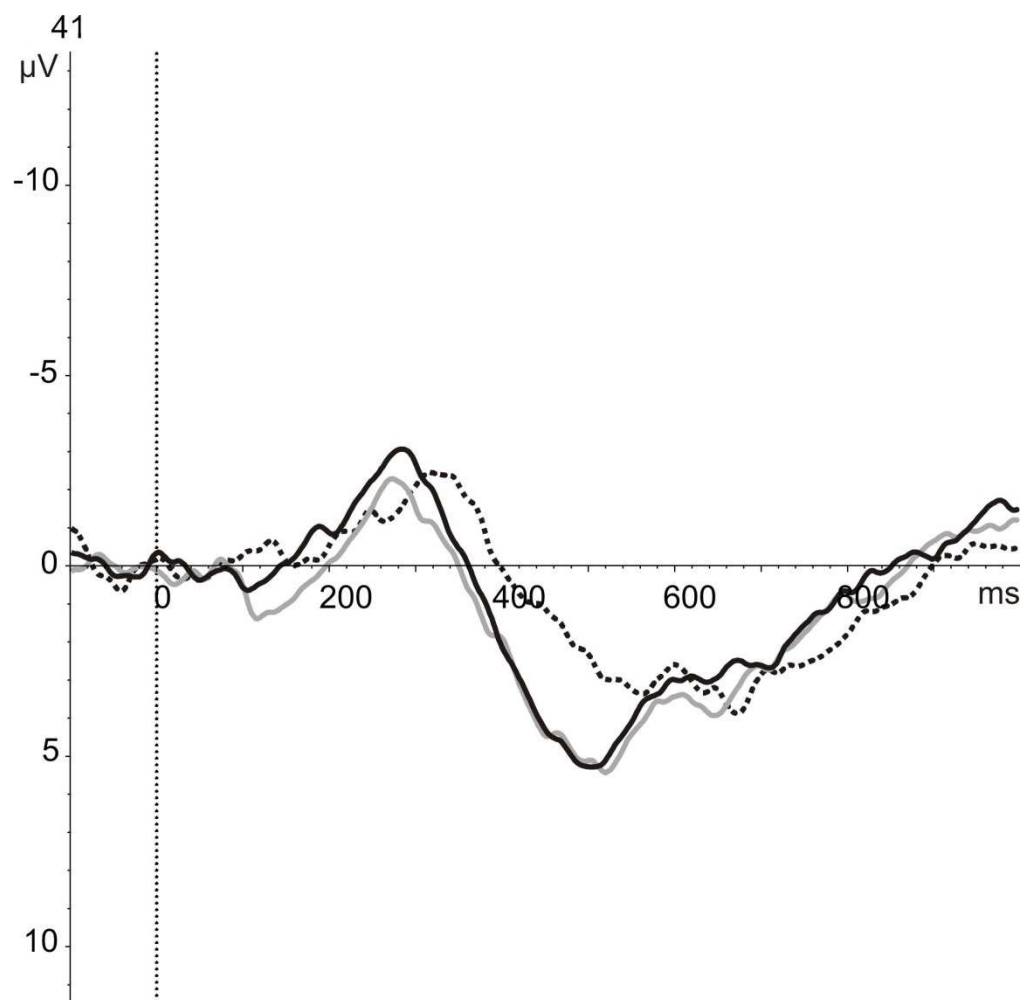
Grand average ERP response in the control group (black line) and in the ASD group (grey line) at sample electrode 44 (included in the occipital region of interest) in response to coherent motion. For illustrative purpose, the ERP response is shown for the 60% coherence level.



**Figure 2b**

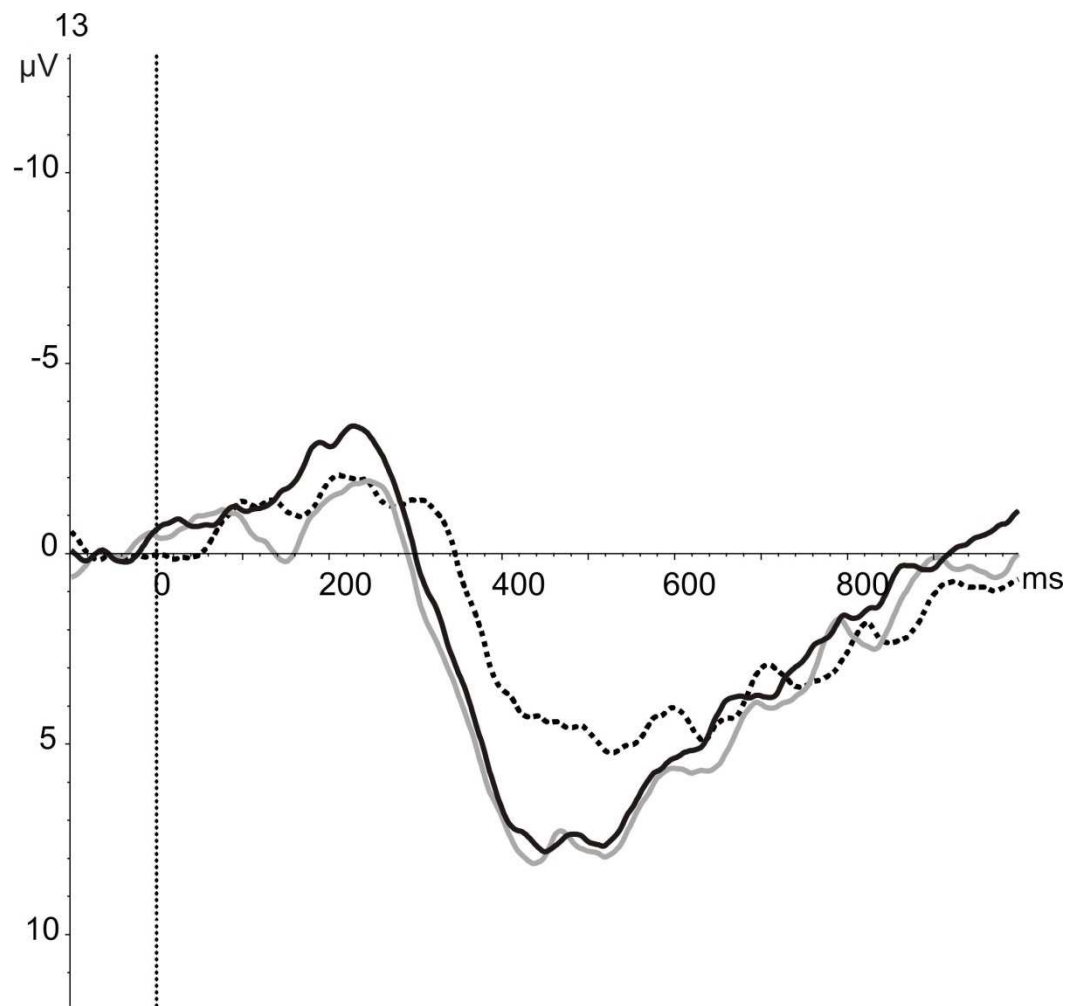
Grand average ERP response in the control group (black line) and in the ASD group (grey line) at sample electrode 26 (included in the parietal-occipital region of interest) in response

to coherent motion. For illustrative purpose, the ERP response is shown for the 60% coherence level.



**Figure 3a**

Grand average ERP response across all subjects at sample electrode 41 (included in the occipital region of interest) in response to 20% (dotted line), 40% (grey line) and 60% (black line) coherent motion.



**Figure 3b**

Grand average ERP response across all subjects at sample electrode 13 (included in the parietal region of interest) in response to 20% (dotted line), 40% (grey line) and 60% (black line) coherent motion.