Title: Adults with motor impairment selectively process early visual, but not tactile information during action preparation. An electrophysiological study.

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RUNNING TITLE: Sensory processing during action in pDCD

ABSTRACT

Developmental coordination disorder (DCD) is a neurodevelopmental condition affecting motor coordination in children and adults. Here, EEG signals elicited by visual and tactile stimuli were recorded while adult participants with and without probable DCD (pDCD) performed a motor task. The task cued reaching movements towards a location in visible peripersonal space as well as an area of unseen personal space. Event-related potentials elicited by visual and tactile stimuli revealed that visual processing was strongly affected by movement preparation in the pDCD group, even more than in controls. However, in contrast to the controls, tactile processing in unseen space was unaffected by movement preparation in the pDCD group. The selective use of sensory information from vision and proprioception is fundamental for the adaptive control of movements, and these findings suggest that this is impaired in DCD. Additionally, the pDCD group showed attenuated motor rhythms (beta: 13-30Hz) over sensorimotor regions following cues to prepare movements towards unseen personal space. The results reveal that individuals with pDCD exhibit differences in the neural mechanisms of spatial selection and action preparation compared to controls, which may underpin the sustained difficulties they experience. These findings provide new insights into the neural mechanisms potentially disrupted in this highly prevalent disorder.

Keywords: DCD; Somatosensory; Motor; Beta; Body

1. Introduction

Developmental coordination disorder (DCD) is a neurodevelopmental disorder affecting the acquisition and execution of coordinated motor skills appropriate for an individual's age and opportunity for skill learning (American Psychiatric Association, 2013). Individuals with DCD experience slowness and inaccuracy of performance of motor skills, which significantly interferes with activities of daily living, as well as academic performance (Henderson & Hall, 2008; Zwicker, Missiuna, Harris, & Boyd, 2012). Recent estimates suggest that between 2 and 5% of school-aged children have DCD (Kirby & Sugden, 2007; Lingam, Hunt, Golding, Jongmans, & Emond, 2009) which ranks it among one of the most common neurodevelopmental disorders in school-aged children. Importantly, the motor impairment associated with DCD and its consequences are now widely believed to persist into adolescence (Cantell, Smyth, & Ahonen, 2003; Losse et al., 1991) and into adulthood (Cousins & Smyth, 2003; Visser, 2003) with nearly three quarters of children with DCD continuing to experience difficulties as adults (Kirby & Sugden, 2007; Losse et al., 1991).

Despite the high prevalence and continued difficulty across the lifespan, a detailed understanding of the aetiology of DCD is limited at best. However, studying adults with persistent motor impairment necessarily excludes those whose impairment in childhood may have resulted from a more benign developmental delay, thus reducing the impact of the known heterogeneity within the disorder (Kirby, Sugden, & Purcell, 2014). Described as a neurodevelopmental disorder, it is believed to result from abnormal development of neural pathways serving perceptual and visuo-motor processing (for reviews see Wilson, Ruddock, Smits-Engelsman, Polatajko, & Blank, 2013; Wilson et al., 2017; Wilson & McKenzie, 1998). Wilson and colleagues' recent review (2017) identified a range of neuropsychological domains with which there is strong evidence for a deficit in DCD. Key deficits include the anticipatory control of movement, basic processes of motor learning and cognitive control.

Whilst knowledge of the, likely multiple, causes of DCD is presently limited, research is beginning to uncover the brain mechanisms underlying the core motor and cognitive deficits in DCD (for detailed reviews see Brown-Lum & Zwicker, 2015; Peters, Maathuis, & Hadders-Algra, 2013; Wilson et al., 2017). A small but growing number of neuroimaging studies have identified differential patterns of cortical activation in DCD compared to typical controls during cognitive (Debrabant, Gheysen, Caeyenberghs, Van Waelvelde, & Vingerhoets, 2013;

Querne et al., 2008) and visuomotor tasks (Kashiwagi, Iwaki, Narumi, Tamai, & Suzuki, 2009; Licari et al., 2015; Zwicker, Missiuna, Harris, & Boyd, 2010, 2011). For example, Querne and colleagues (2008) showed that children with DCD exhibited differential patterns of connectivity within attentional and inhibitory networks compared to controls, and McLeod and colleagues (2014) found disrupted functional connectivity between primary motor cortex and areas serving sensorimotor processing, motor planning and executive functioning. Structural neuroimaging has also identified atypical white matter architecture in children with DCD, evidenced by decreased fractional anisotropy (FA) in the corpus callosum, which was associated with the children's poor motor and executive function (Langevin, Macmaster, Crawford, Lebel, & Dewey, 2014).

Importantly, a number of studies have reported deficits in covert orienting of visual-spatial attention (Wilson, Maruff, & McKenzie, 1997; Wilson, Maruff, Ives, & Currie, 2001; Wilson & Maruff, 1999; Zwicker et al., 2012), a mechanism that works to selectively amplify task relevant sensory information (e.g. Hillyard, Vogel, & Luck, 1998). These behavioural observations are supported by a small number of electrophysiological studies identifying a deficit in covert attentional orienting in children with DCD. For example, Tsai and colleagues (2009) found that children with DCD show longer N1 event-related potential (ERP) latencies in response to target stimuli compared to typically developing children during tasks of covert orienting. This pattern of findings appears to hold not only for severe instances of DCD, but also in more moderate cases (Chen, Wilson, & Wu, 2012).

The control mechanisms governing spatial attention have been shown to be inseparable from the control mechanisms governing goal-directed action. For example evidence using functional MRI has identified overlapping activation of brain regions during tasks of covert orienting and movement preparation (Astafiev et al., 2003; Corbetta et al., 1998; Perry & Zeki, 2000). Therefore, further investigation of these mechanisms may shed light on the efficiency of this type of processing in adults with motor impairments. In healthy individuals, target detection is facilitated not only at locations of space that are covertly attended to, but at locations that are the target of upcoming eye movements (Deubel & Schneider, 1996; Irwin & Gordon, 1998). Recent electrophysiological work has shown enhanced ERPs evoked by visual stimuli presented at the goal location of planned manual movements (Eimer, Forster, Van Velzen, & Prabhu, 2005; Eimer, Van Velzen, Gherri, & Press, 2006), as well as during reaching movements (Gherri, Van Velzen, & Eimer, 2009; Job, de Fockert, & van Velzen, 2016) and sequences of reaching movements (Baldauf, Cui, & Andersen, 2008; Baldauf &

Deubel, 2009). Together these effects demonstrate an adaptive prioritisation of sensory processing at action-relevant spatial locations (for a review see Baldauf & Deubel, 2010). Furthermore, recent electrophysiological evidence has shown enhanced visual processing at locations along the reach trajectory taken around obstacles (Baldauf, 2018). Given these known links between mechanisms of spatial attention and motor preparation, as well as the growing evidence for deficits of spatial attention in DCD, a detailed investigation of these processes in DCD is required.

The ERP method is well suited to investigating early sensory perception due to its fine temporal resolution in the millisecond range. ERPs provide a useful index of attentional processing, given that stimuli falling within an attended area of space receive increased processing resources and therefore typically elicit larger ERP component amplitudes, regardless of the stimuli's task relevance (Heinze, et al., 1994). Enhanced ERP components can therefore be obtained in response to task-irrelevant visual 'probe' stimuli presented in an attended area relative to an unattended area of space (Hillyard et al., 1998; Hillyard & Anllo-Vento, 1998).

Early studies investigating sensory perception at movement relevant locations have focused entirely on movements towards peripersonal space, the visible areas of space within arms' reach, which is functionally relevant for object exploration and manipulation. However, many movements have goal locations in personal space, on the body surface, and many of these are located in unseen space (e.g. movements towards the face when eating, toward the upper chest, or back). Such movements rely more on tactile and proprioceptive information rather than vision and they differ functionally, in that they likely serve to touch the body (e.g. touch the mouth when eating or to scratch an itch). Previous findings have shown that early processing of tactile information on the hands is influenced by movement preparation: tactile ERP components (e.g. N140) are enhanced during the preparation of eye-movements (Gherri & Eimer, 2008), manual movements (Eimer et al., 2005) as well as reaching movements (Forster & Eimer, 2007) toward the stimulated area. Recent work from our lab found that tactile processing on an unseen area of the body surface was similarly enhanced when a reaching movement to this area was prepared (Job *et al.*, 2016). These findings, together with the marked similarities in preparatory activity across attention and movement preparation, suggest that similar mechanisms modulate visual and tactile sensory perception during planned movements.

To summarise, despite growing evidence for shared mechanisms of attention and movement preparation and the demonstrated attentional difficulties in DCD, little is known about how individuals with DCD prioritise sensory processing at spatial locations relevant for upcoming action. Here, movement preparation and its consequences for sensory processing are investigated in adults with probable DCD (pDCD) and compared to that of matched controls using a delayed movement task (Job et al., 2016). In this task, an auditory cue instructs participants to prepare a movement towards a goal location either in peripersonal space in front of them or to an unseen area of space on the body surface, however the movement is to be withheld until a 'GO' signal. During the delay, visual or tactile task-irrelevant probe stimuli are delivered at the peripersonal or personal movement goal locations, respectively. The analyses will focus on early neural responses to these probes, as indexed by visual and tactile ERPs, which should be influenced by movement preparation such that preparing to move to a location selectively biases early sensory processing at that location. Difficulties with adaptively prioritising sensory processing in individuals with pDCD would be reflected by reduced modulations of early sensory processing during movement preparation compared to controls. Further, given that individuals with DCD display particular difficulties with movements based on proprioception (Summers, Larkin, & Dewey, 2008), it is expected that sensory processing at the goal of upcoming movements towards an unseen location on the body will be particularly affected.

Additionally, cued motor preparation is also typically accompanied by a prominent decrease in the power of beta oscillations (13-30Hz) relative to the pre-cue baseline over central electrode sites (Cheyne, 2013; Kilavik, Zaepffel, Brovelli, MacKay, & Riehle, 2013; Pfurtscheller, Stancák, & Neuper, 1996). These power decreases are often referred to as an event-related desynchronisation (ERD) (Pfurtscheller & Lopes da Silva, 1999) with the most common source being the contralateral pre-Rolandic 'sensorimotor' region (Pfurtscheller & Berghold, 1989). While there are controversies regarding the exact functional role of ERD in cued movement tasks (Kilavik et al., 2013), there is a general consensus that beta ERD provides a reliable indicator of the onset of movement preparation, execution and motor imagery (Kuhn et al., 2006; Pfurtscheller & Lopes da Silva, 1999; Pfurtscheller & Neuper, 1997) and may reflect an active process promoting existing motor or cognitive states (Engel & Fries, 2010). Whether or not individuals with pDCD exhibit the same pattern of beta ERD during delayed movement tasks is unknown, as no studies to date have investigated oscillatory dynamics during cued movements in DCD. An exploratory approach to

investigating beta ERD following movement cues in pDCD is therefore taken, with the aim of identifying any stable differences in beta ERD between the groups.

2. Materials and methods

2.1. Participants

A total of 24 adults (See Table 1, upper panel, for mean ages) took part in return for travel expenses and £20. The pDCD group consisted of 12 adults (10 females) who reported a diagnosis of developmental coordination disorder (DCD) given either in adulthood (10) or in childhood (2). As well as reporting a diagnosis of DCD all participants in the pDCD group had self-reported motor difficulty in the form of scores within probable range on the Adult DCD Checklist (ADC, Kirby, Edwards, Sugden, & Rosenblum, 2010). The ADC is a 40-item questionnaire designed to screen for motor-related deficits in adulthood, with higher scores indicating greater levels of impairment. The control group consisted of 12 adults (10 females) who reported no diagnosis of DCD and scored outside of the range considered at risk of DCD on the ADC. All participants were right handed (see Table 1 upper panel) and reported normal or corrected to normal vision. All participants provided written informed consent and were debriefed at the end of the experiment as appropriate. The Local Ethics Committee at Goldsmiths, University of London, approved all experimental protocols and the experiment adhered to the ethical guidelines presented in the 1964 declaration of Helsinki.

2.2. Motor assessments

All participants completed the Adult Developmental Coordination Disorder Checklist (Kirby et al., 2010), a screening tool for identifying DCD in adulthood. In order to ensure individuals in the pDCD group experienced continued coordination difficulty in comparison to the control group, the highest age band (16 years, 11 months) tasks of the Movement Assessment Battery for Children (MABC-2, Hendersen, Sugden, & Barnett, 2007) were administered. Although it is not appropriate to formulate a diagnosis using these tasks, as standardised normative scores are available only up to the age of 16 years and 11 months, comparisons to the control group validate the placement of individuals into the respective pDCD and control groups (see table 1 middle panel for a summary of the MABC-2 results). The use of scores standardised to the upper age band of the MABC has been used to validate group inclusion in previous studies investigating adult DCD sample (Wilmut & Byrne, 2014; Wilmut, Byrne, &

Barnett, 2013). The MABC-2 was administered as instructed in the manual and consists of tasks assessing manual dexterity, ball skills as well as static and dynamic balance.

2.3. Intelligence assessments

In accordance with the DSM-5 diagnostic criterion D - The motor skills deficits are not better explained by intellectual disability (intellectual developmental disorder) - an assessment of intelligence using subtests from the Wechsler Adult Intelligence Scale third edition (WAIS-III, Wechsler, 1997) was used. Although no IQ cut-off or discrepancy is specified by the diagnostic manual, participants were only included in the study if their scores were not below two standard deviations from mean on a number of subtests of the WAIS-III, as recommended by Sugden (2006). Verbal measures included vocabulary and similarities and performance measures included picture completion, block design and matrix reasoning. All tests were administered according to the manual instructions.

2.4. Stimuli and task

A delayed movement task (Job et al., 2016) was implemented using E-Prime software (Schneider, Eschman, & Zuccolotto, 2002). See Fig. 1 for a schematic illustration of the task trial procedure. The auditory cue and the response required were counterbalanced across participants; half of participants in each group prepared a movement towards the peripersonal goal after the presentation of a high tone (1 kHz) and a movement towards a personal goal following a low tone (0.4 kHz) and the reverse was true for the remaining participants. The visual probe consisted of an LED (2 cm in diameter, subtending 1.91° in visual angle at 60 cm from participant) presented for 200ms. The tactile probe was delivered by a solenoid, driving a blunt ended metal rod that contacted the skin when a current was passed through them. The tactile device was attached with an adhesive sticker to the upper sternum. White noise (62 dB SPL) was played throughout the experiment in order to mask any sounds made by the tactile device. Responses were collected using a custom-made response panel consisting of two centrally located infrared response devices embedded at 30 cm and 60 cm from the panel's edge. The infrared device situated closest to the body (30 cm) acted as the starting point for movements and as the fixation point. The device registered the moment the hand was lifted from the central starting point of the movement. Responses made towards peripersonal space were recorded using the further infrared response device (60 cm from the participant), which registered a response when the hand landed on the device. A touch

sensitive device attached to the chest recorded the time at which the hand arrived at the personal goal.

Figure 1. Trial Procedure

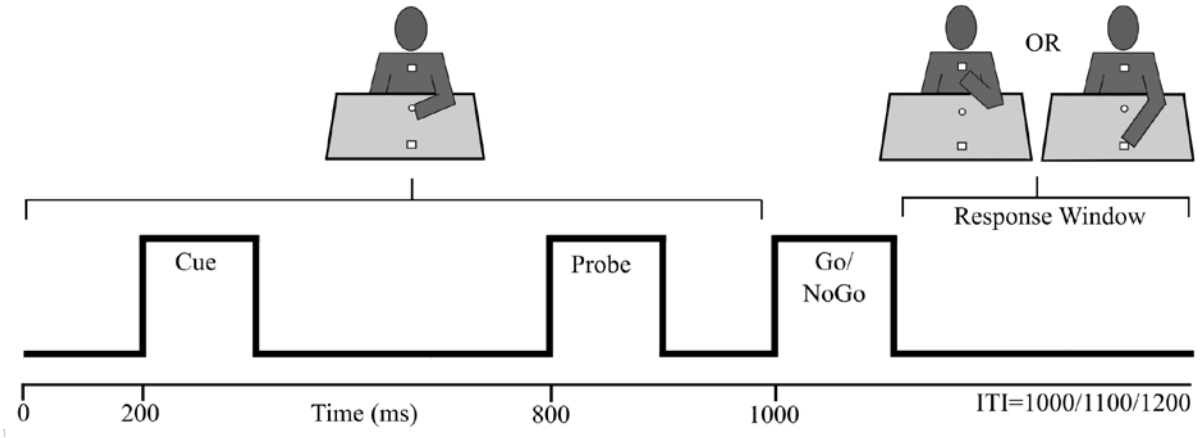

Figure 1. Schematic illustration of the delayed movement task trial procedure. Auditory cues are first presented that signal the preparation of the reaching movement either to a goal location in visible peripersonal space in front of participants, or to a goal location in personal space on the body surface. Before the execution of the movement is signalled by an auditory GO/NoGO stimulus, a task irrelevant visual or tactile probe is delivered to the peripersonal or personal goal location, respectively. Participants are told to ignore the probes and to execute the movements as fast and accurately as possible. The inter-trial-interval (ITI) is randomly varied to be 1000, 1100 or 1200 milliseconds.

2.5. Procedure

Participants were seated in a sound-attenuated, dimly lit and electrically shielded chamber. They first completed a practice block of 40 randomised trials, followed by a block of 480 trials with a pause every 60 trials after which participants verbally informed the experimenter to continue with the next 60 trials. With each block of 60 trials participants were instructed to change the arm with which they completed the movement, with the order counterbalanced between participants. There were therefore 120 trials per condition (peripersonal/visual probe, peripersonal/tactile probe, personal/visual probe, and personal/tactile probe). Within each condition, there were 96 Go trials and 24 No-go trials. The total duration of the experiment was approximately 45 min.

2.6. EEG recording and processing

EEG was recorded from 64 Ag–AgCl electrodes at a digitisation rate of 2048Hz and down sampled offline to 512Hz. Electrodes were referenced to the average of electrodes placed on the left and right earlobes. Activity from horizontal eye movements was recorded from pairs of electrodes placed on the outer canthi of the eyes. Vertical eye movement activity was recorded from electrodes placed above and below the left eye. Offline pre-processing of the EEG data was conducted using EEGlab toolbox (Delorme & Makeig, 2004). Further offline analysis was conducted using a combination of Fieldtrip toolbox (Oostenveld, Fries, Maris, & Schoffelen, 2011) and custom MATLAB scripts.

Continuous EEG data were high-pass filtered at 1Hz and divided into 600ms epochs locked to the onset of the visual or tactile probe including a 200ms pre-stimulus baseline. Probes presented before a Go and No-go signal were included in the analyses. Cue-locked data were divided into 1600ms epochs locked to the onset of the movement cues including a 400ms pre-stimulus baseline. Epochs including voltages exceeding + and/or - 100 μV were automatically rejected prior to analysis. Eye-blink artefacts were corrected for using Independent Component Analysis (ICA).

For probe-locked epochs, the peak amplitudes of ERP components within pre-defined time windows were extracted for analysis, in line with previous investigations (Eimer, 1993; Hillyard et al., 1998; Hillyard & Anllo-Vento, 1998; Mangun, Hillyard, & Luck, 1993). For the visual probe the maximal positive amplitude between 50 and 150ms post stimulus onset was extracted and analysed as the P1 peak amplitude. Maximal negative amplitudes between 100-200ms post-stimulus were extracted and analysed as the N1 peak amplitude. The tactile probe-evoked P60 peak amplitude was detected as the largest positive amplitude between 20 and 120ms post stimulus onset and the tactile N140 component peak amplitude was detected between 100 and 200ms post stimulus onset.

For cue-locked epochs, the time-varying spectral content of the signal was estimated using Morlet Wavelet based time-frequency representation (TFR). The signal of individual epochs was convolved in the single-trial using Complex Morlet wavelets and then averaged for each participant. The number of cycles per wavelet was linearly scaled with the spectral frequency from 3 cycles at 5Hz to 10 cycles at 50Hz. Changes in the spectral power following the cue were calculated by dividing it by its baseline value at each frequency, averaged from -400ms to the onset of the cue. The power at frequencies within the beta range (13-30Hz) was averaged. Epochs were subsequently averaged such that right hemisphere electrodes for

epochs where movements were executed with the left hand were averaged with left hemisphere electrodes in right hand epochs (contralateral electrodes). Similarly, left hemisphere electrodes in left hand epochs were averaged with right hemisphere electrodes in right hand epochs (ipsilateral electrodes). This averaging procedure was used in order to merge cue-locked data from left and right hand blocks while maintaining information about hemispheric lateralisation.

2.7. Statistical analysis

For ERP analysis the peak amplitude values were extracted and analysed. Central electrode sites over left and right hemispheres (C1/2, C3/4, C5/6, CP3/4, CP5/6) as well as posterior-occipital sites over left and right hemispheres (O1/2, PO3/4, PO7/8) were pooled for analyses. Electrode sites were chosen based on the latency and scalp distribution of grand average components averaged across conditions. A mixed ANOVA was used for visual and tactile evoked responses separately, with one between-subjects factor of group (pDCD vs. Control) and three within-subjects factors of movement preparation (personal vs. peripersonal), electrode hemisphere (left vs. right) and electrode region (central vs. posterior). Corrections for multiple comparisons were made using Bonferroni adjustment.

For cue-locked epochs, the lack of previous literature regarding beta oscillations in individuals with DCD meant that an a priori hypothesis regarding when or where the spectral content of the EEG signal might differ between the groups was not made. An exploratory analysis approach, non-parametric cluster permutation (Maris & Oostenveld, 2007), of the cue-locked data was therefore adopted. This approach to the analysis of multidimensional neuroimaging data extracts spatiotemporal regions showing significant differences between conditions or groups without any a priori on spatial regions or time windows. It therefore identifies effects that are robust within a cluster, rather than highly significant on one dimension (i.e. a single electrode and/or time point). The method is as robust against Type I error as Bonferroni's correction, as Type I error is intrinsically controlled for by evaluating only the maximum cluster-level statistics under the null hypothesis. The method has been successfully applied in a number of EEG studies (Lindsen, Jones, Shimojo, & Bhattacharya, 2010; Luft & Bhattacharya, 2015; Park, Correia, Ducorps, & Tallon-Baudry, 2014; Sandkühler, Bhattacharya, Schoffelen, Maris, & Oostenveld, 2008).

The following steps were taken to identify significant clusters: 1) Independent samples t-statistics comparing pDCD and control data were gathered for each of the samples in the

multidimensional data structure; 2) t-statistics above a p-value threshold ($p<.05$) were then gathered; 3) Neighbouring data points exceeding the threshold were found; 4) The t-statistics were summed to calculate the cluster level statistic; 5) The maximum cluster statistic under its permutation distribution (shuffled data), derived from the test statistics obtained from the independent samples t-tests based on 1000 random permutations, was evaluated. The cluster-level significance threshold was set at the two-tailed level of 0.025. Electrodes with distances of less than 5cm were considered neighbouring, yielding an average of 6.6 neighbours per electrode. Following identification of significant clusters, an ANOVA with a between-subjects factor of group (pDCD vs. Control) and a within-subjects factor of movement preparation (personal vs. peripersonal) was used on the beta power values relative to baseline, averaged across significant cluster electrodes/time points.

3. Results

3.1. Movement assessment results

All individuals in the pDCD group scored above the higher cut off of 65 on the Adult DCD Checklist placing them within the 'probable DCD' category. All of the individuals in the control group scored below the lower cut off of 56 for 'at risk' of DCD. Results of the MABC-2 can be seen in the middle panel of Table 1. Group performance on each measure total as well as the grand total was compared using one-way ANOVAs with group (pDCD vs. Control) as the between-subjects factor and measure total as the dependant variable. As can be seen in Table 1, the controls outperformed the pDCD group for tasks of manual dexterity as well as balance. The groups did not differ on their performance on the ball skill tasks. Overall, however, the control group significantly outperformed the pDCD group on the grand total for the MABC-2. This corroborates previous demonstrations of not only the continued motor difficulty experienced by adults with DCD compared to controls (Cousins & Smyth, 2003), but the utility of the MABC-2 in validating group inclusion criteria when comparing adult DCD and control groups (Wilmut & Byrne, 2014; Wilmut et al., 2013).

3.2. Intelligence assessment results

Table 1 shows performance on each subtest of the WAIS-III. In line with DSM5 Criterion D, all participants performed within the normal range (i.e. not below two standard deviations from the mean, as recommended by Sugden (2006) on each subtest). Furthermore, the groups did not differ on any of the intelligence measures administered; therefore, the groups are sufficiently matched on intelligence.

3.3. Delayed movement task

Table 1 (bottom panel) shows the mean reaction times and results of one-way ANOVAs comparing the groups for movements towards personal and peripersonal space. Although the pDCD group's reaction times were overall slower than controls, this difference did not reach statistical significance either for movements towards personal or peripersonal movement targets.

Table 1. Means (M) and standard deviations (SD) for pDCD and control groups, as well as F-ratios and p-values for the effect of group

	pDCD (n=12)		**Control (n=12)**			
	M	**SD**	**M**	**SD**	**F-Ratio (1, 22)**	
Age (years)	26.33	(4.20)	27.92	(2.96)	F=1.13	p=.298
Edinburgh Handedness Inventory	89.32	(12.55)	88.67	(13.74)	F=.012	p=.913
Adult DCD Checklist	89.67	(12.78)	23.00	(14.12)	F=133.72	p<.001
Movement Assessment Battery (MABC-2)						
Manual Dexterity Total	31.83	(7.61)	39.17	(9.06)	F=4.61	p=.043
Ball Skills Total	20.83	(10.87)	24.17	(7.83)	F=0.74	p=.398
Balance Total	29.42	(10.88)	39.75	(6.20)	F=8.18	p=.009
Grand Total	82.08	(21.22)	103.08	(15.70)	F=7.60	p=.012
Wechsler Adult Intelligence Scale (WAIS-III)						
Picture Completion	11.58	(1.78)	12.08	(2.43)	F=.33	p=.298
Vocabulary	13.42	(3.45)	13.75	(1.54)	F=.09	p=.571
Similarities	12.00	(3.22)	14.00	(1.54)	F=3.77	p=.065
Block Design	11.92	(2.94)	13.17	(1.95)	F=1.51	p=.232
Matrix Reasoning	13.83	(2.08)	14.33	(1.77)	F=.40	p=.533
Delayed movement task (ms)						
Personal movement RT	645	(156)	596	(123)	F=.717	p=.406
Peripersonal movement RT	655	(148)	631	(142)	F=.167	P=.686

3.4. Visual probe-evoked potentials

Fig. 2 and 3 show the grand averaged ERPs elicited by the visual probe during movement preparation both towards personal and peripersonal space. As shown, the visual ERP components are most pronounced over occipital electrode sites.

Statistical analysis confirmed the distribution of the P1 was largest at posterior electrode sites with a main effect of electrode region ($F(1,22)=14.58$, p=.001, η_p^2=.399). A significant interaction between movement preparation, (personal vs. peripersonal), group (pDCD vs. controls) and electrode region (central vs. posterior) was present, $F(1,22)=4.68$, p=.042, η_p^2 =.175. An ANOVA with a between-subjects factor of group (pDCD vs. controls) and within-subjects factors of movement preparation (personal vs. peripersonal) was therefore used on posterior sites only. This revealed a significant interaction between movement preparation and group ($F(1,22)=10.10$, p=.004, η_p^2=.315) and indicates that the pDCD group modulated the visual probe evoked P1 component as a function of movement preparation to a greater extent than the control group. Post-hoc t-tests confirmed that P1 amplitudes were significantly larger during movement preparation towards the probed location in peripersonal space (M=4.37, SD=2.23) relative to the opposite location in personal space (M=3.12, SD=2.18) in the pDCD group, $t(11)=-2.56$, p=.026. This difference was not significant in the control group, $t(11)=1.22$, p=.248 (peripersonal space M=2.61, SD=1.59, personal space M=3.13, SD=1.59). See Fig. 6 for bar graphs summarising the mean peak amplitudes of early ERP components.

For the visual probe-evoked N1 component, no main effect of group (pDCD vs. control) or interactions involving group were present (all F-values < 2.5, all p-values > .1). An interaction between movement preparation (personal vs. peripersonal) and the electrode region (occipital vs. central) was present, $F(1,22)=5.88$, p=.024, η_p^2=.211. Post-hoc t-tests revealed that, for all participants, at occipital sites, the N1 was enhanced during movement preparation towards the probed location in peripersonal space (M=-3.96, SD=3.03), compared to during movements towards the opposite location in personal space (M=-3.24, SD=3.39), $t(23)=2.34$, p=.028. This difference was not significant at central electrode sites, $t(23)=.68$, p=.503.

Figure 2. Control Group Visual ERPs

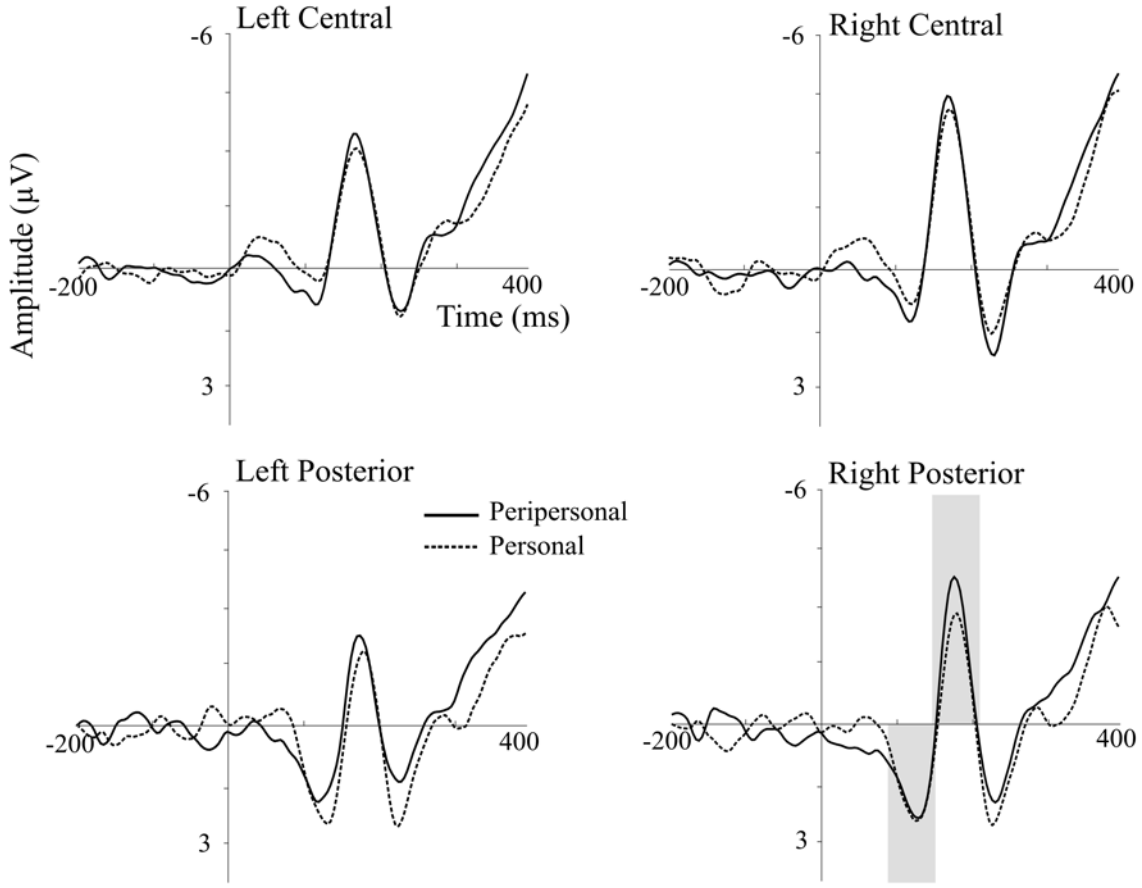

Figure 2. Control group grand averaged ERPs locked to the visual probe stimuli (onset time point 0ms) as a function of movement preparation (Peripersonal vs. Personal) at electrodes pooled into four regions: left/right central sites (C1/2, C3/4, C5/6, CP3/4, CP5/6) and left/right posterior sites (O1/2, PO3/4, PO7/8). Highlighted are the visual P1 and N1 components, which were most pronounced at right posterior electrode sites.

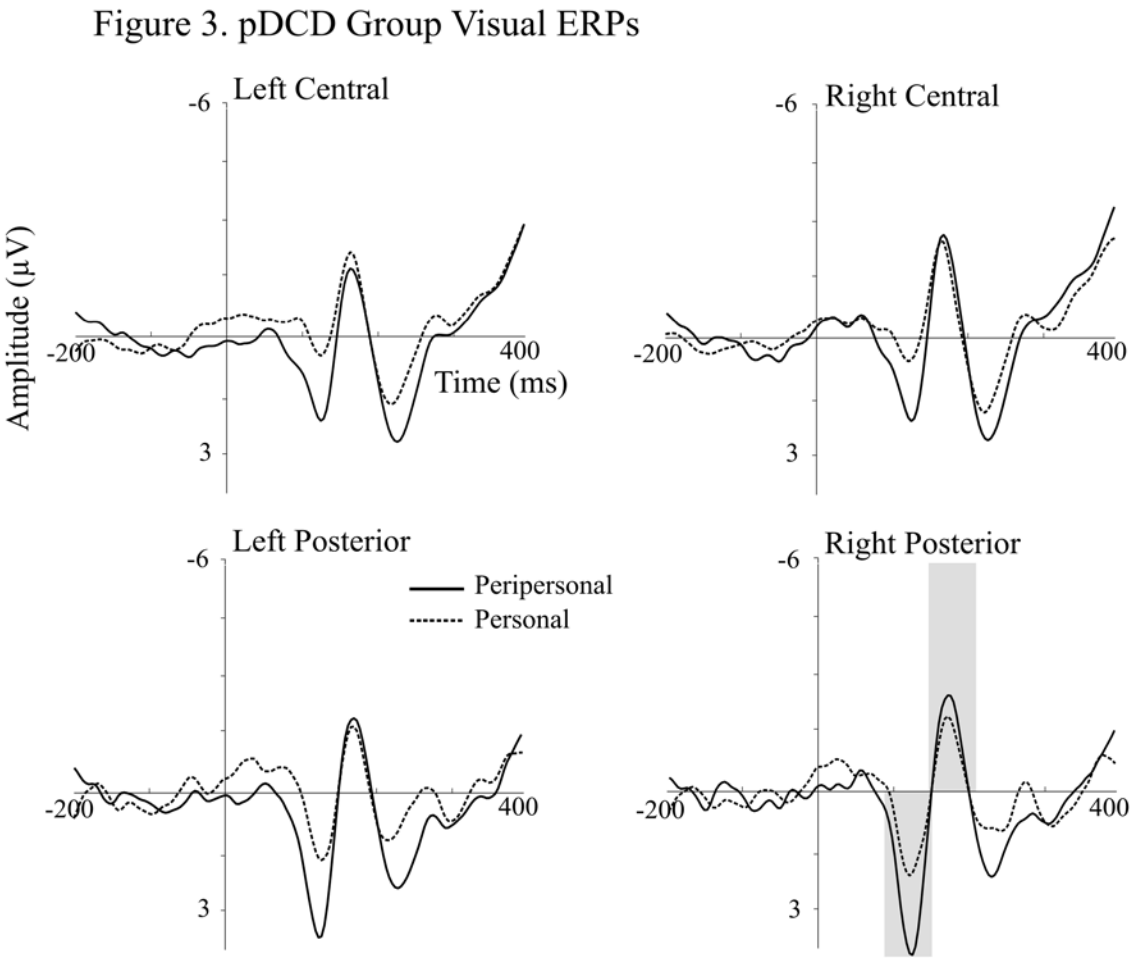

Figure 3. pDCD group grand averaged ERPs locked to the visual probe stimuli (onset time point 0ms) as a function of movement preparation (Peripersonal vs. Personal) at electrodes pooled into four regions: left/right central sites (C1/2, C3/4, C5/6, CP3/4, CP5/6) and left/right posterior sites (O1/2, PO3/4, PO7/8). Highlighted are the visual P1 and N1 components, which were most pronounced at right posterior electrode sites.

3.5. Tactile probe-evoked potentials

Fig. 4 and 5 shows the grand averaged ERPs elicited from the tactile probe during movement preparation both towards personal and peripersonal space. As shown, the tactile ERP components are most pronounced over central electrode sites.

Statistical analysis confirmed that the distribution of the P60 was largest at central electrode sites ($F(1,22)=6.11$, $p=.022$, $\eta_p^2=.217$). A main effect of movement preparation

($F(1,22)=5.03$, p=.035, η_P^2=.186) was also present. An interaction between group (pDCD vs. control) and movement preparation (personal vs. peripersonal) was marginally significant ($F(1,22)=6.11$, p=.059, η_P^2=.159). This indicates that the control group modulated the tactile probe-evoked P60 component as a function of movement preparation to a greater extent than the pDCD group. Post-hoc t-tests confirmed that for the control group the P60 component was significantly larger during movement preparation towards the probed location in personal space (M=3.68, SD=2.54) relative to the opposite location in peripersonal space (M=2.89, SD=2.56), $t(11)=3.15$, p=.009. This difference was not significant for the pDCD group, $t(11)=.620$, p=.548 (personal space M=3.63 SD=1.59, peripersonal space M=3.58, SD=1.93).

The tactile probe-evoked N140 component was largest at central electrode sites in the right hemisphere, as evidenced by main effects of both electrode region and hemisphere, respectively ($F(1,22)=22.83$, p<.001, η_P^2=.509, $F(1,22)=7.55$, p=.012, η_P^2=.256) as well as an interaction between region and hemisphere $F(1,22)=14.86$, p=.001, η_P^2=.403. No significant main effects or interactions with the factors of group, or movement preparation were present for the N140 (all p-values > .09).

Figure 4. Control Group Somatosensory ERPs

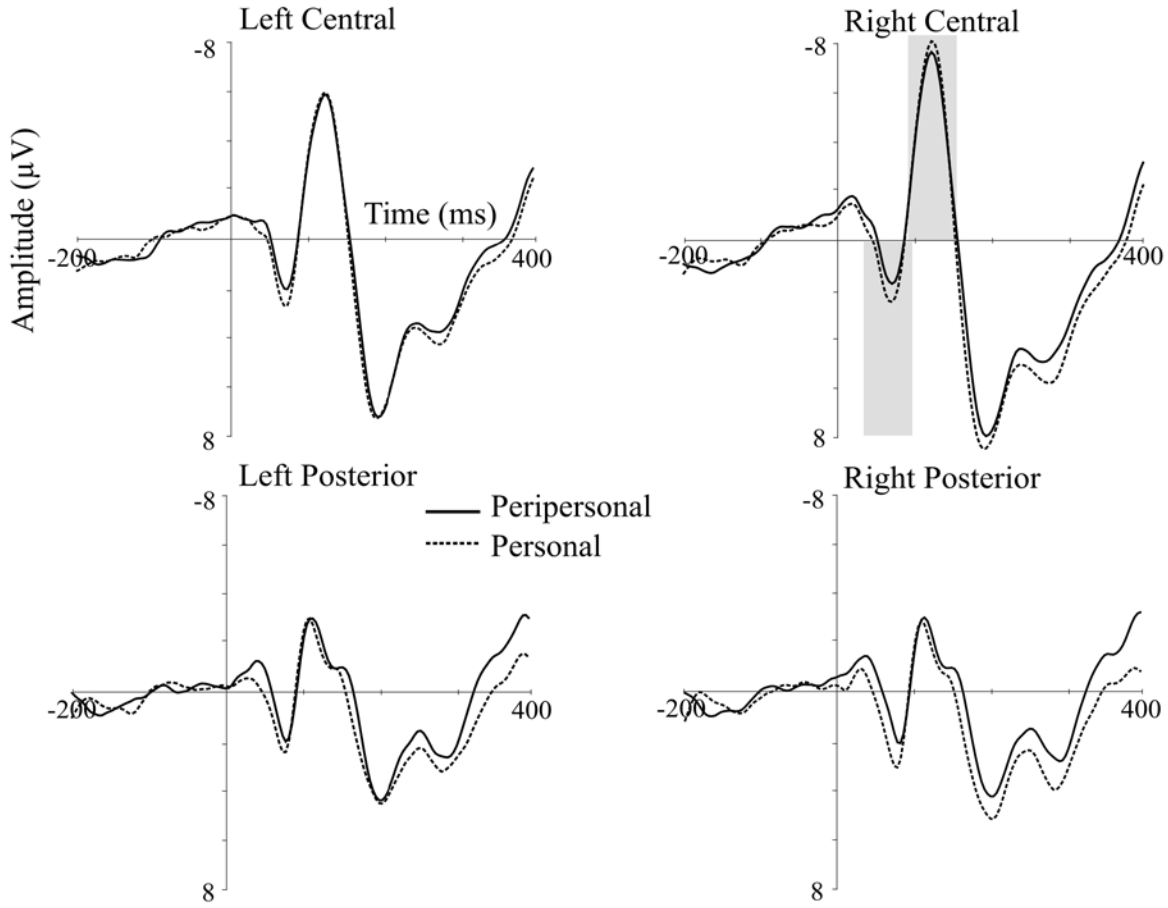

Figure 4. Control group grand averaged ERPs locked to the tactile probe stimuli (onset time point 0ms) as a function of movement preparation (Peripersonal vs. Personal) at electrodes pooled into four regions: left/right central sites (C1/2, C3/4, C5/6, CP3/4, CP5/6) and left/right posterior sites (O1/2, PO3/4, PO7/8). Highlighted are the somatosensory P60 and N140 components, which were most pronounced at right hemisphere central electrode sites.

Figure 5. pDCD Group Somatosensory ERPs

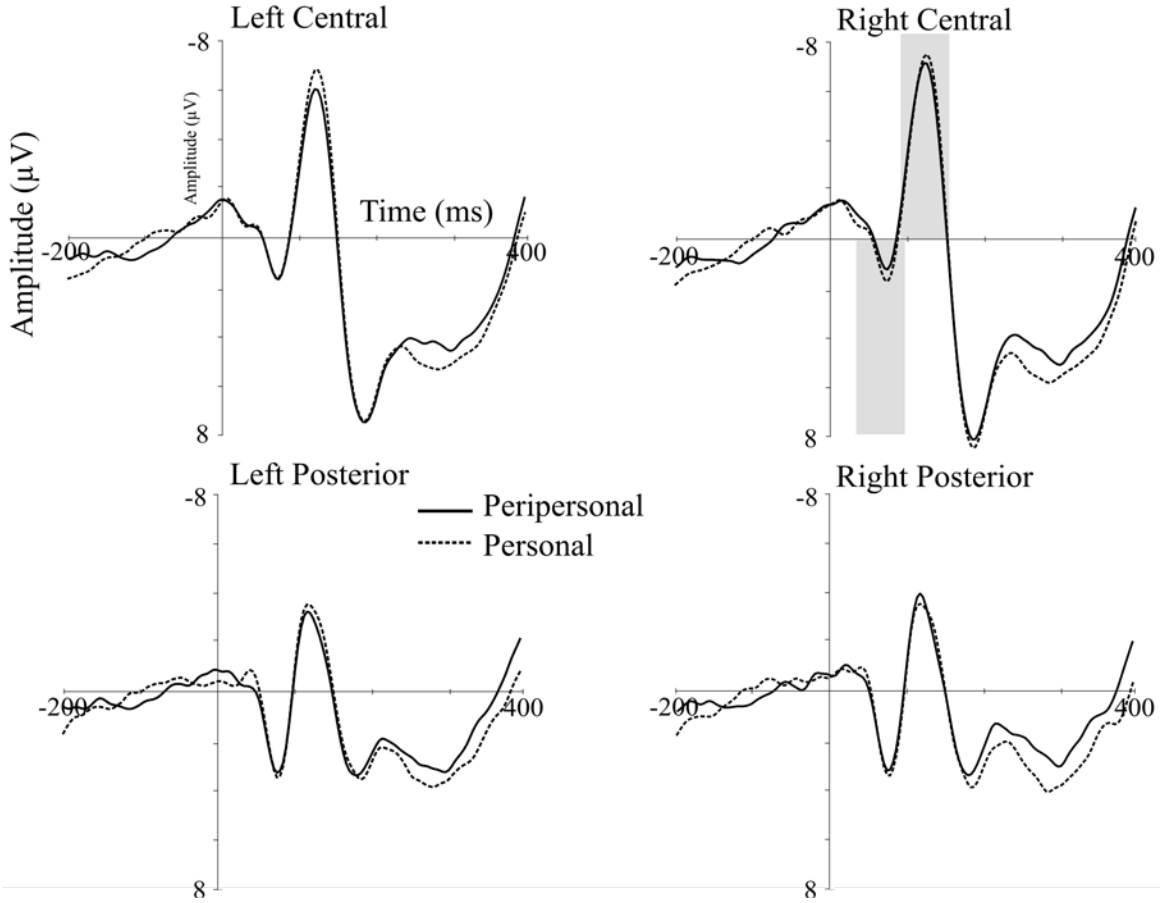

Figure 5. pDCD group grand averaged ERPs locked to the tactile probe stimuli (onset time point 0ms) as a function of movement preparation (Peripersonal vs. Personal) at electrodes pooled into four regions: left/right central sites (C1/2, C3/4, C5/6, CP3/4, CP5/6) and left/right posterior sites (O1/2, PO3/4, PO7/8). Highlighted are the somatosensory P60 and N140 components, which were most pronounced at right hemisphere central electrode sites.

Figure 6. ERP component amplitudes

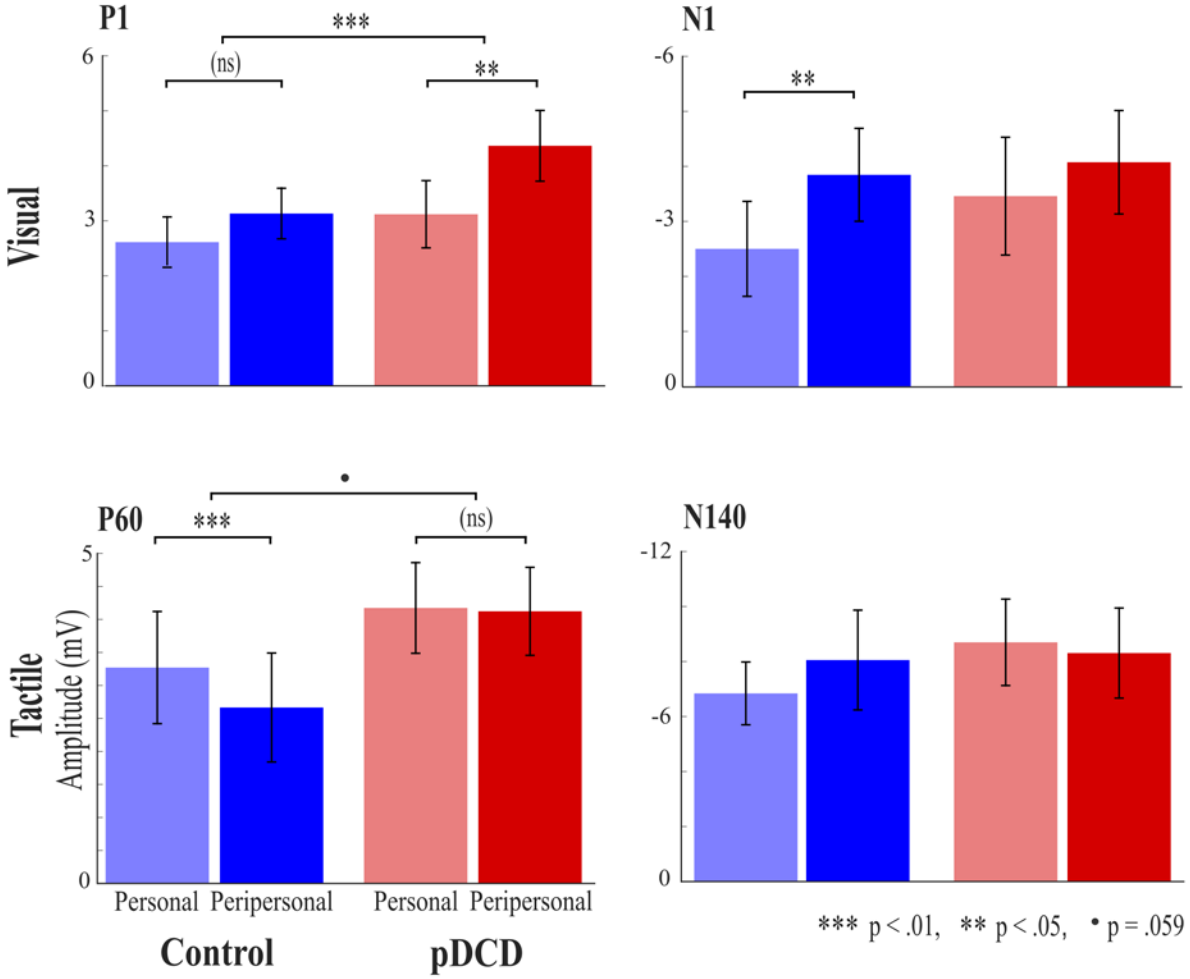

Figure 6. Bar graphs show the mean peak amplitudes of the visual P1 and N1 components at posterior electrode sites (O1/2, PO3/4, PO7/8) as well as the tactile P60 and N140 component amplitudes at central electrode sites (C1/2, C3/4, C5/6, CP3/4, CP5/6) for each group and movement condition (control peripersonal, control personal, pDCD peripersonal and pDCD personal). Error bars show 1+/- SD.

3.6. Cue-locked beta oscillations

For the cue-locked activity, time-frequency representations of the data were subjected to non-parametric cluster permutation (Maris & Oostenveld, 2007) in order to explore differences in preparatory activity during delayed movements between the groups.

The control group showed a greater event-related decrease in beta power following cues to prepare a movement towards personal space on the body surface compared to controls (see Fig. 7a), while no differences were found for movements away from the body. Fig. 7b shows the difference (controls vs. pDCD) in beta (13-30) power following cues to prepare a movement towards the goal location in personal space. The difference resulted in a negative cluster, which started around .5 seconds following the movement cue at sensorimotor electrodes (significant cluster electrodes are highlighted, $p<.025$). The cluster remained significant until the onset of the GO signal (1 second). The cluster began in the hemisphere contralateral to the movement hand but became quickly bilateral and finally progressed anteriorly across time. An ANOVA with a between-subjects factor of group (pDCD vs. Controls) and a within-subjects factor of movement preparation (personal vs. peripersonal space) was run on beta power values averaged across significant cluster electrodes from .5 to 1 second post-cue. A main effect of group was found (see Fig. 7c) with control participants exhibiting a significantly larger reduction from baseline beta power, which was standardised to 1.0 (M=.90, SD=.017) compared to the pDCD group (M=.98, SD=.017), $F(1,22)=9.59$, $p=.005$ $\eta_P^2=.303$. A significant group by movement preparation interaction was also found, $F(1,22)=6.99$, $p=.015$, $\eta_P^2=.241$. Post-hoc t-tests revealed that this interaction was driven by movements towards personal space in which the control group showed a larger reduction from baseline beta power (M=.89, SD=.07) compared to the pDCD group (M=.99, SD=.06), $t(22)=-3.91$, $p=.001$. This difference was not significant for movements towards peripersonal space, $t(22)=-1.73$, $p=.098$ (controls: M=.91, SD=.07, pDCD: M=.95, SD=.06).

Figure 7. Post-cue beta power

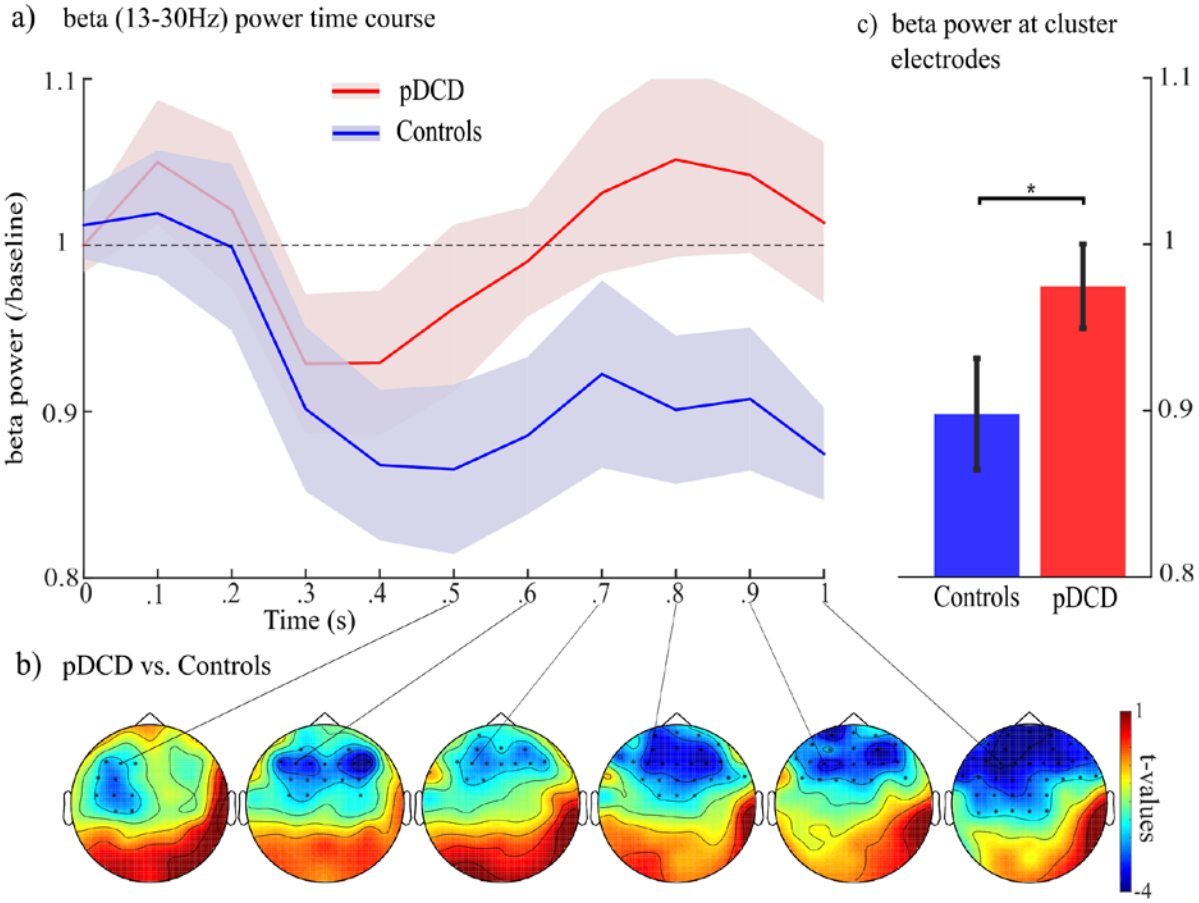

Figure 7. Power in the beta band (13-30Hz) following movement cues. a) Beta power across time at a representative cluster electrode (F3) following cues to prepare a movement towards an unseen area of personal space on the body surface. Values below 1 (dotted line) indicate a decrease from baseline. Shaded areas represent +/- SD. b) Topographies of the differences in beta power between the pDCD and control groups at six time points following the movement cue towards personal space on the body surface. Significant cluster electrodes are marked. c) Beta power relative to baseline for each group, averaged over both movement cues (personal vs. peripersonal) from .5 – 1 second. Error bars represent +/- SD.

4. Discussion

This study investigated the neural underpinnings of motor preparation in adults with and without probable developmental coordination disorder (pDCD) by recording EEG signals during a delayed movement task. When planning the movements, healthy controls selectively processed visual and tactile information at the movement goal, as previously reported.

However, adults with pDCD did not selectively process tactile information at the movement goal to the same extent as controls. Instead individuals with pDCD selectively processed visual information to a greater extent than matched controls. While the overall group differences were marginal, they may provide important information about how sensory information is processed during movement preparation in adults with motor impairment. Additionally, the groups showed marked differences in the power of sensorimotor beta rhythms (13-30 Hz) during movement preparation.

Spatial selection during motor preparation was measured with visual and tactile ERP responses elicited by task-irrelevant probe stimuli delivered following a cue to prepare a reaching movement either towards or away from the body. Individuals with pDCD showed a greater enhancement of the P1 component elicited by the visual probes presented at the goal of the planned movement. However, when preparing a reaching movement towards an area of unseen personal space on the body surface the reverse effects, although marginal ($p=.059$), were observed for tactile ERPs. Individuals with pDCD showed little or no enhancement of the tactile probe-evoked P60 component compared to controls, who showed larger P60 amplitudes when preparing to move to the probed location.

Previous studies have shown that movement preparation by healthy subjects modulates visual and tactile information in a similar manner, such that visual and tactile perception at movement goals is enhanced prior to executing the movement (Forster & Eimer, 2007; Gherri & Eimer, 2008). Our more recent work (Job et al., 2016) has also shown that the mechanisms involved in preparing movements towards unseen body locations are similar to those underlying movements into visible peripersonal space. The finding that the pDCD group is selectively impaired in modulating (somato)sensory processing during movement preparation towards the body, could suggest a problem with the integrity of the available sensory input, which would critically affect these mechanisms. The question remains as to the exact nature of such compromised sensory input in DCD. Whether it is the quality of afferent tactile/proprioceptive information that is impaired in DCD, the ability of parietal regions to represent such information appropriately, or the effective use of this information during motor preparation is so far unknown. A combination of these factors across development may be likely; indeed, it is conceivable that impaired afferent proprioceptive information in early childhood could result in later under-reliance of this sensory information for movement planning downstream in development.

The pattern of ERP results observed here is consistent with a suggested relative over-reliance on visual as opposed to tactile information in DCD, as this group modulated visual information in peripersonal space to a greater extent as well as earlier than controls, but showed little or no modulation of tactile processing in an area of unseen space. There is some support for an over-reliance on visual information in DCD from behavioural studies of postural control (Bair, Barela, Whitall, Jeka, & Clark, 2011; Hill, 1998), walking (Deconinck et al., 2008) as well as reaching (Zoia, Castiello, Blason, & Scabar, 2005). Zwicker and colleagues (2010) also found greater activation during a trail tracing task in regions of the frontal, parietal and temporal lobes of children with DCD, areas associated with visual-spatial processing, whereas controls activated primarily the precuneus to support their motor performance. This may suggest that the DCD group relied more on visual and spatial processing to complete the task compared to the controls. Other fMRI findings (Debrabant et al., 2013; Querne et al., 2008) have identified relative hypoactivation of attentional brain network areas such as the dorsolateral prefrontal cortex (DLPFC), suggesting a greater requirement of processing resources for motor performance in DCD. The apparent over-reliance on visual information in DCD could be the result of either compensatory mechanisms for poor sensory feedback from proprioceptive systems, poor use of such afferent information for motor preparation, from atypical development of internal models for movement, or a combination of these factors. The challenge for future research investigating the brain mechanisms involved in DCD should seek to elucidate this further.

Alternatively, the finding that the pDCD group is selectively impaired at modulating somatosensory information during movements towards the body could suggest that the mechanisms with which movement preparation modulates visual and somatosensory information may in fact differ, contrary to previous conclusions (Forster & Eimer, 2007; Gherri & Eimer, 2008; Job et al., 2016). Rather than one uniform mechanism controlling modulations of visual and somatosensory processing during movement preparation, distinct mechanisms may be at work for movements towards different functional spaces (i.e. personal/peripersonal) that depend on differing sensory information that is typically available for such movements.

Notwithstanding the wider implications of the findings, the relatively inefficient selection mechanisms for tactile perception on the body surface observed here may help to explain some of the more precise output problems reported in descriptive studies of DCD, for example difficulty with activities such as eating, grooming and dressing (Summers et al.,

2008). Such activities often involve movements towards personal space on the body surface, many of which are located in regions not typically accessible to vision (e.g. the face and upper torso). It further suggests that the sensory modulations that are observed at (unseen) locations relevant for motor behaviour reflect the influence of a mechanism that is a necessary requirement for adaptive and smooth motor behaviour. It should be noted that while sensory perception during movement preparation differed between the groups, as indexed by early ERP components, no differences were observed in the behavioural responses of the delayed movement task. It could be that the relatively crude measure of motor behaviour in this task (reaction times to initiate a reaching movement) was not sensitive enough to detect more fine-grained differences in the kinematics of the movements between the groups. Nevertheless, our findings show that the task was performed differently by the two groups, with the pDCD group demonstrating an over-reliance on visual information.

In addition to the probe-evoked potential findings, differences between the groups in the oscillatory dynamics induced by movement cues were also identified, for the first time in the known literature. Following cues to prepare a movement towards the unseen goal in personal space, differences between the groups emerged in the beta band (13-30Hz). The pDCD group showed significantly less event-related desynchronisation (ERD) of beta oscillations from approximately 500ms after the cue onset. This difference in beta ERD was initially distributed over sensorimotor electrodes contralateral to the movement hand and became bilateral and finally progressed anteriorly over time. There is a general consensus that sensorimotor beta ERD in delayed movement tasks reflects an active process promoting existing motor or cognitive states (Engel & Fries, 2010) and provides an index of motor preparation. This therefore suggests that the DCD group's attenuation of beta ERD, relative to controls, reflects less efficient recruitment of sensorimotor regions responsible for movement programming towards unseen personal space. It is interesting that this difference manifested only following cues to prepare movements towards the unseen goal location in personal space and not towards the visible peripersonal goal location. This may reflect a relative difficulty in representing areas of space for movement preparation that cannot be directly accessed by vision. Further studies are required in order to track the contribution of beta desynchrony to motor performance in DCD and its potential implications for how individuals with DCD plan and represent different actions before executing them.

5. Conclusion

A detailed understanding of the brain mechanisms responsible for the deficits experienced by individuals with DCD is profoundly lacking in the literature. This is particularly evident for the literature concerning adults with DCD. From the few studies to date, key findings suggest a deficit in visual-spatial attentional processing in DCD. Mechanisms of covert attentional orienting, and their consequences for perceptual processing are increasingly being linked to the mechanisms of effective movement preparation. Compared to a group of matched controls, adults with pDCD demonstrated a distinct pattern of sensory prioritisation during movement preparation. The pDCD group appeared to selectively enhance visual processing at the goal location of movements towards visible peripersonal space in front of them to a greater extent than controls, however the pDCD group did not show prioritisation of tactile processing in an area of unseen personal space. The findings suggest that individuals with DCD may be selectively impaired at using somatosensory signals during movement preparation, which could underlie their suggested over-reliance on vision. Taken together the results of this study contribute to our understanding of the continued difficulties associated with pDCD in adulthood and suggest reduced efficiency in motor preparation towards the body and a difficulty with adaptively modulating sensory processing in the context of movement preparation.

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