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Short reports

Short report presenting preliminary evidence of impaired corticomuscular coherence in an individual with Developmental Coordination Disorder

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ABSTRACT

Background: It has been suggested that developmental coordination disorder (DCD) could be caused by a 'dysconnection' in brain and skeletal muscle communication. To date no previous work has examined the integrity of this neuromuscular process in individuals with DCD.
Aims: To conduct a feasibility study for measuring functional connectivity of the brain and muscle in an individual with DCD using corticomuscular coherence (CMC).
Methods and Procedures: An individual with DCD and a typically developing (TD) participant completed a series of sustained 5-second voluntary isometric hand contractions (15 ± 5 % MVC) on a handheld dynamometer under both single and dual task (i.e., counting backwards) conditions. EEG, EMG and force data were collected.
Outcomes and Results: The participant with DCD displayed poorer force steadiness and higher mental demand compared to the TD participant and in dual task conditions. The TD participant displayed a commonly observed pattern of CMC that was highly localised over the contralateral hand area, the DCD participant displayed a less localised CMC across cortical regions.
Conclusions and Implications: These findings support the feasibility of measuring CMC in DCD populations and offer some, albeit preliminary, evidence of impaired communication between the brain and muscles in these individuals.

1. Introduction

Developmental coordination disorder (DCD) affects 5–6 % of children (American Psychiatric Association, 2013) and is associated with poor learning and performance of motor skills (Parr, Foster, Wood, Thomas, & Hollands, 2020). While the aetiology of this condition remains largely unknown, several studies have suggested that the condition is reflected by fundamental differences in skeletal muscle function (e.g., Yam & Fong, 2018). By contrast, other studies have suggested that differences in sensorimotor function can be explained by differences in the structure of focal brain regions (Scott et al., 2021). While both areas of research have provided much needed insight into the possible cause of DCD, isolating differences in muscle function or brain regions ignores the fact that

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motor coordination is a neuromuscular process that is dependent on the functional communication between the brain that initiates and controls movement and the muscles that translate the neural commands into the movement itself. Therefore, to fully understand movement coordination and, by extension, coordination disorders like DCD, researchers need to investigate how the brain and muscles interact to produce goal-directed, coordinated movements. Indeed, recent research has highlighted the need for an examination of brain-muscle connectivity as a cause of DCD (Tallet & Wilson, 2020).

A non-invasive method for establishing the integrity of corticospinal functional connectivity uses coherence analysis between electroencephalogram (EEG) signals from sensorimotor cortex and electromyography (EMG) signals from limb muscles. Cortico-muscular coherence (CMC) is proposed to reflect the cortical control of motor unit firing via the direct corticospinal pathway and is typically observed during isometric contraction, reaching its peak in the beta frequency band (approx. 15 – 35 Hz; Mima & Hallett, 1999). Beta CMC originates mainly from the primary motor cortex (M1) contralateral to the contracted limb muscle and is somato-topically organised. For example, during hand contractions in healthy individuals, CMC is localised to the hand area of the brain contralateral to the active hand. CMC is considered an established neurophysiological marker of motor impairment (see Liu, Sheng, & Liu, 2019 for review), which can result in CMC becoming reduced in strength (Roeder, Boonstra, & Kerr, 2020), or appear less topographically localised (Guo et al., 2020). Consequently, we propose that measures of beta CMC offer potential in investigating the aetiology of DCD.

Measures of CMC might also help to explain why individuals with DCD often struggle to perform motor tasks consistently under dual tasks conditions. For example, performing concurrent cognitive tasks (e.g., digit recall) has been shown to impair walking in children with DCD (Cherng, Liang, Chen, & Chen, 2009). Beta CMC decreases significantly when attention is divided between the motor task and another simultaneously performed task (Lattari et al., 2010). This suggests that beta-band CMC may reflect attention towards the motor task and supports the view that it plays an active role in motor control. To date, no studies have explored the integrity of brain-muscle communication under dual task conditions in individuals with DCD.

The aim of the present experiment was to conduct a feasibility study for measuring CMC between one individual with, and one without, DCD during a hand contraction task under single and dual-task conditions. We predicted that the individual with DCD would exhibit decreased force steadiness, report higher levels of mental demand, and display lower and less topographically localised CMC.

2. Methods

2.1. Participants

Two right-handed male adults participated in the study, one formally diagnosed with DCD (age = 34 years) and one typically developed individual (age = 29 years). Participants gave written informed consent and ethical approval of the experimental protocol was approved by the institutional ethics committee.

2.2. Recordings

A handheld dynamometer attached to a PowerLab 4/25 T (AD Instruments, Bella Vista, Australia) was used to record hand contraction force (in kilograms) via Labchart 8 software (ADinstruments) at a sampling rate of 1000 Hz. The EEG signals were recorded with a conventional gel-based cap (eego sports, Ant Neuro, Hengelo, Netherlands) with 64 AgCl electrodes in a layout based on the extended 10–20 international system (Jurcak, Tsuzuki, & Dan, 2007). Electrodes CPz and AFz were used as reference and ground, respectively. The EEG cap was connected to an EEG mobile amplifier (eego sports, ANT Neuro, Hengelo, Netherlands) with additional auxiliary channels to allow for synchronous recording of bipolar surface EMG. A pair of bipolar EMG electrodes were placed on the surface of the flexor digitorum superficialis (FDS) of the right forearm with an interelectrode distance of 2 cm. Both the EEG and EMG signals were recorded at a sample rate of 1000 Hz. Time synchronisation of the force, EEG and EMG signals was achieved using a LabJack U3 device (LabJack Corporation, Lakewood, United States) that was controlled by a custom Psychopy experiment. As such, each contraction "onset" auditory stimulus produced a square wave trigger timestamp that would be treated as timepoint zero in subsequent analyses.

2.3. Procedure

Upon arrival, participants were prepared for EEG and EMG data collection. Participants were sat with their right forearm strapped into a cushioned rig fixed onto the desk in front of them. Participants held the dynamometer in their right hand with their left arm freely resting on their lap. A computer screen was positioned approximately 60 cm from participants. Participants were asked to perform two 5-second maximal voluntary contractions (MVCs) upon the handheld dynamometer. A minimum of 1-minute of rest was given between each MVC. Each participant's MVC was subsequently determined as the max force value (in kilograms) obtained across the two attempts (TD = 45.38 kg, SD = 1.18 vs. DCD = 29.32 kg, SD = 3.19).

The experimental task consisted of eight blocks of ten isometric hand contractions. The task was initially performed under single-task conditions (8 × 10 contractions), before being performed under dual-task conditions (8 × 10 contractions; total of 160 experimental contractions). In the single-task condition, participants were asked to contract for 5-seconds at 15 ± 5% of their MVC, with both the force produced and the goal error boundary (i.e., ± 5%) visually presented on a computer screen using Labchart 8.0 software (ADinstruments). Each 5-second contraction was followed by 5-seconds of rest, and each block of ten contractions were separated by an additional 1-minute of rest. The onset and offset of each 5-second contraction were indicated via auditory beeps (10 ms duration)

that were controlled using Psychopy software (Pierce et al., 2019). In the dual-task condition, participants were additionally required to count backwards in threes from a random number for the entirety of an experimental block (i.e., 10 contractions).

2.4. Data analysis

EEG and EMG signals were down-sampled (250 Hz) and band-pass filtered (1 – 45 Hz; finite impulse response) prior to being cut into epochs ranging from − 2 to + 6 s relative to the onset auditory stimulus. Epochs were visually inspected, and those showing large EEG contamination from muscular artifacts were discarded (from both EEG and EMG analyses). No bad EEG channels were identified. Independent component analysis (ICA) weights were obtained through the RunICA infomax algorithm (Jung, Makeig, Bell, & Sejnowski, 1998) running on EEG signals. ICA weights that presented obvious nonneural activity upon visual inspection were manually rejected. Finally, the EEG signals were average referenced. These processing steps were performed using EEGLAB functions (Delorme & Makeig, 2004) for MATLAB.

2.5. Measures

2.5.1. Force steadiness

The force exerted by each participant was analysed to determine the steadiness of contraction during the "steady phase" of the task, defined in the present study as occurring between 2 and 5 s following the auditory 'go' stimulus (i.e., 3 s of steady contraction). Steadiness was defined as the coefficient of variance (CoV), calculated as the ratio between force standard deviation and the mean force value during the steady phase (Fig. 1).

2.5.2. Mental demand

The mental demand Likert scale derived from the NASA-TLX (Hart & Staveland, 1988) was self-reported after each block of contractions. Responses ranged from "very low" (1) to "very high" (21). The mean levels of mental demand were then calculated for each participant across each experimental condition.

2.5.3. Corticomuscular coherence

Time-frequency decomposition was performed through short-time fast Fourier transform (FFT) conducted on 76 overlapping windows, each of 500 ms, with central points ranging from − 1.75–5.75 s relative to the onset of the auditory go stimulus. Prior to FFT, data points in each window were Hanning tapered and zero padded to reach 1 s. This procedure generated complex-valued coefficients in the time-frequency plane with a precision of 100 ms and 1 Hz. The correlations between EEG and EMG signals were calculated by magnitude-squared coherence using the following equation:

$$C_{xy}(f) = \frac{|P_{xy}(f)|^2}{P_{xx}(f) \bullet P_{yy}(f)}$$

where P_{xx} and P_{yy} are the averaged power spectral densities of the EEG and EMG signals throughout the segments for a given frequency f, respectively, and $P_{xy}(f)$ is the averaged cross-spectral density between the two parameters throughout the segments. The coherence function provides a normative measure of linear correlation on a scale from 0 to 1, where 1 indicates a perfect linear correlation.

To quantitatively evaluate the contraction related differences in beta CMC, we set the frequency range for our subsequent analyses

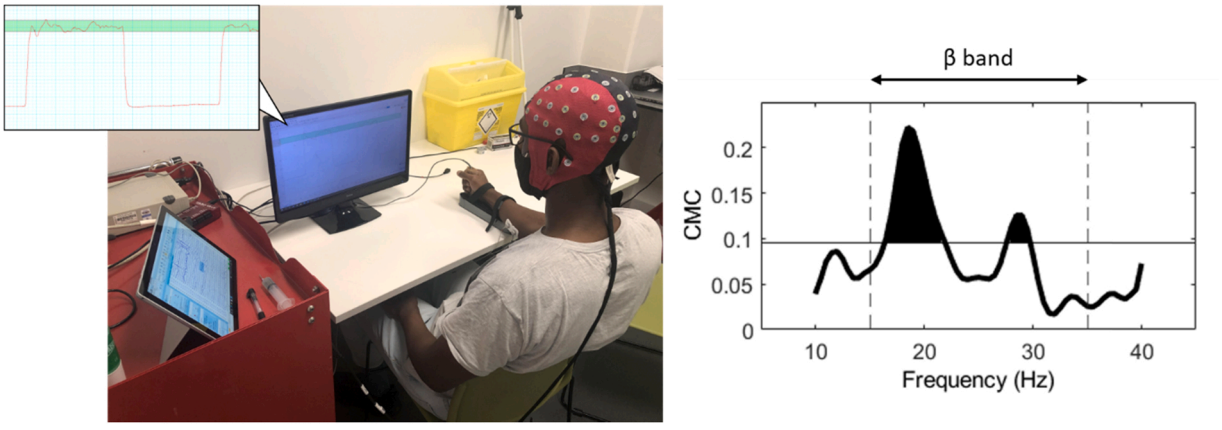

Fig. 1. Visual representation of the experimental task (left), with the displayed force trace enlarged. Example CMC spectrum (right) between the C3 electrode of the EEG and the EMG electrode positioned over the FDS during the steady state period (i.e., between 2 and 5 s following auditory stimulus) of the contraction at 15% MVC. The area under the coherence curve and above the SL (area shaded in black) were determined for the beta band (beta Coh_{area}). The estimated SL of $P < 0.05$ is shown as the horizontal line at 0.096 on the y-axis.

at 15 – 35 Hz (Fig. 1). For multiple corrections across the 21 frequency bins (i.e., between 15 and 35), we applied a Bonferroni correction to the following equation defining the significance level of coherence (SL; Rosenberg, 1989):

$$SL(\alpha) = 1 - \left[\frac{1}{N} \bullet (1 - \frac{\alpha}{100})\right]^{1/(L-1)}$$

where α is the confidence limit (%), N is the number of frequency bins, and L is the number of epochs. As $N = 21$, $L = 61$, and $\alpha = 95$ were used in the present study, SL was determined to be 0.096. This revision eliminates the potential risk that the coherence value is judged to be significant owing to statistical error. We measured the individual area under the coherence curve and above the significance level in beta frequency range (beta Coh_{area}) during the steady phase (as per the force analyses) of the force contraction (e.g., Omlor, Patino, HeReymond, & Kristeva, 2007; Parr, Gallicchio, Canales-Johnson, Uiga, & Wood, 2022; Fig. 1). The primary focus of this work was to assess beta CMC between the EEG in the left motor cortex (C3) and EMG in the right forearm. However, for exploratory purposes, topographical scalp maps are presented to evidence the global organisation of beta CMC between participants.

3. Results

3.1. Force steadiness

For the single-task condition, the TD participant displayed steadier force output than the participant with DCD, with an average CoV of 1.43 (± 0.60) compared to an average of 1.72 (± 0.94), respectively. The dual-task condition disrupted task performance for both participants, with the TD participant displaying a mean CoV of 1.80 (± 0.72) and the DCD participant displaying a mean CoV of 2.67 (± 1.85; Fig. 2).

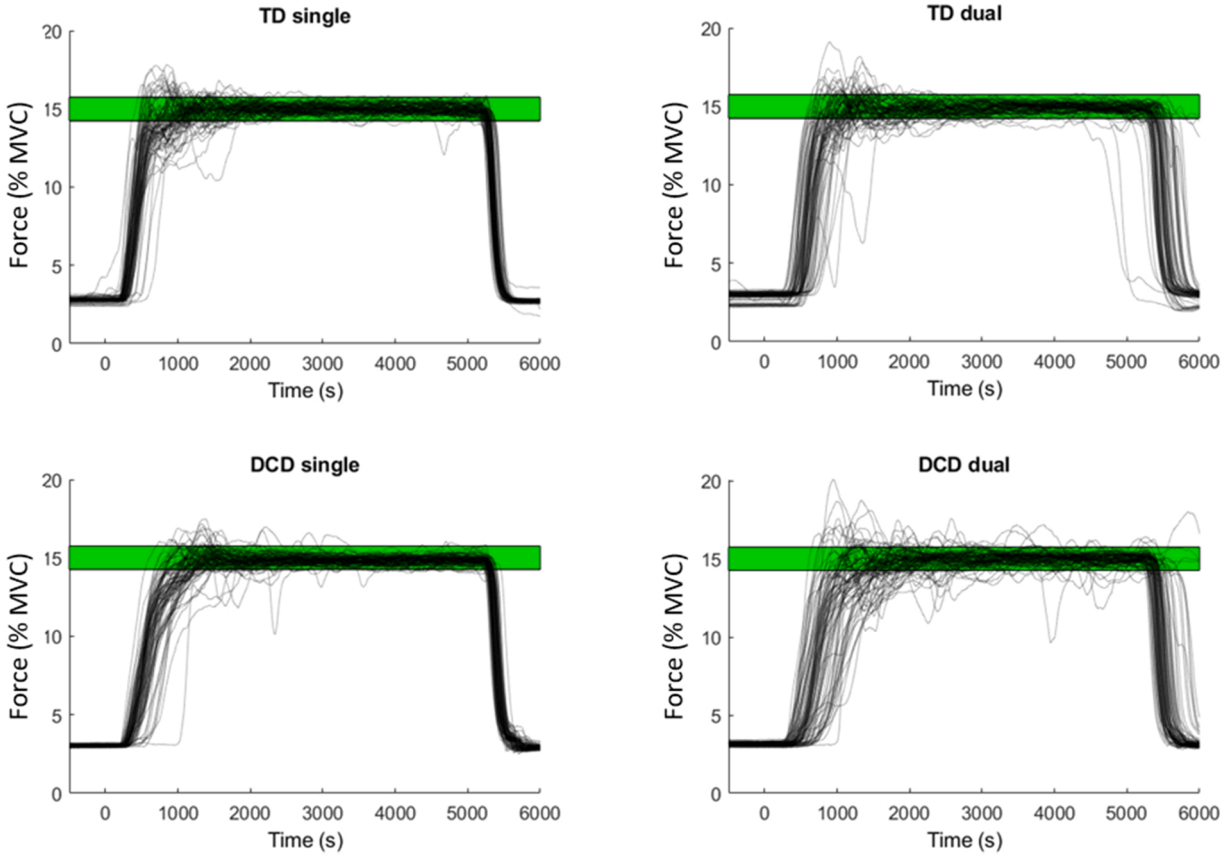

Fig. 2. Line plots representing all experimental trials overlayed for each participant across each condition. The black lines represent the exerted force (as % of MVC) across time (x-axis), with time-point "0" representing the onset of the auditory "go" stimulus. The green area represents the goal-boundary within which participants were required to keep their force production within, set at 15% (± 5%) MVC.

3.2. Mental demand

For the single-task condition, the TD participant reported the lowest possible levels of mental demand (1 ± 0.00), whilst the DCD participant reported moderate levels of mental demand (12.63 ± 1.41). For the dual-task condition, the TD participant still reported low levels of mental demand (3.75 ± 0.46) whilst the DCD participant reported high levels of mental demand (18.38 ± 1.51).

3.3. Corticomuscular coherence

3.3.1. C3 beta Coh_{area}

For the single task condition, the TD participant (1.18 ± 1.05) displayed higher levels of C3 beta Coh_{area} compared to the DCD participant (0.93 ± 0.86). The dual-task condition appeared to disrupt C3 beta Coh_{area} in the TD participant (0.94 ± 0.83, $\Delta = -0.24$), but have little effect on the DCD participant (0.99 ± 0.87, $\Delta = 0.06$; Fig. 3).

3.3.2. Topographical inspection

For the single task condition, the TD participant's beta Coh_{area} appeared highly localised to electrode C3 (1.176 ± 0.618) and thus the right-hand area of the contralateral motor cortex. For the dual-task condition, the TD participant displayed a less topographically localised beta CMC, appearing more posteriorly (e.g., P1 = 1.171 ± 0.590, P3 = 1.118 ± 0.514) and across the ipsilateral motor cortex (e.g., CP2 = 1.131 ± 0.679, C4 = 1.103 ± 0.638). For the single task condition, the DCD participant's CMC appeared maximal over the ipsilateral motor cortex, peaking at electrode C6 (1.112 ± 0.636). For the dual-task condition, the DCD participant displayed no clear distribution of CMC across motor-related cortices (Fig. 3).

4. Discussion

We hypothesised that the individual with DCD would have poorer force steadiness and higher levels of mental demand across all conditions compared to the TD control participant. As performance in this task is reliant of the ability to maintain a constant force, it is unsurprising that the individual with DCD exhibited poorer performance as deficits in the ability to produce force steadiness are well documented in the DCD literature (Smits-Engelsman, Westenberg, & Duysens, 2008). The DCD participant also reported markedly higher levels of mental demand whilst performing all conditions - a difference that was exacerbated during the conditions that required secondary task performance. This again concurs with previous research that has demonstrated that individuals with DCD often attempt to maintain performance by recruiting compensatory attentional resources representative of more effortful performance (Koch, Miguel, & Smiley-Oyen, 2018). Therefore, the person with DCD exhibited performance characteristics that are strongly associated with the condition.

The individual with DCD displayed lower CMC compared to the TD individual (Fig. 3). This finding is consistent with previous research in movement impaired individuals and adds to the evidence suggesting that CMC is a neurophysiological marker of motor impairment (Liu et al., 2019). While the dual task condition markedly decreased CMC in the TD individual, it had little effect on the CMC of the individual with DCD. In agreement with findings here, previous work has shown that dual-task conditions cause a reduction in CMC in TD individuals under dual-task conditions (Lattari et al., 2018), and we expected similar reduction in CMC for the individual with DCD. While this was surprising, it could reflect a potential floor effect in the CMC in the individual with DCD. Alternatively, the TD individual could have flexibly adopted alternative cognitive or motor strategies in the face of the increased demands of the task – as suggested by the shift towards posteriorly localised CMC. Perhaps no such reductions in CMC were evident in the individual with DCD

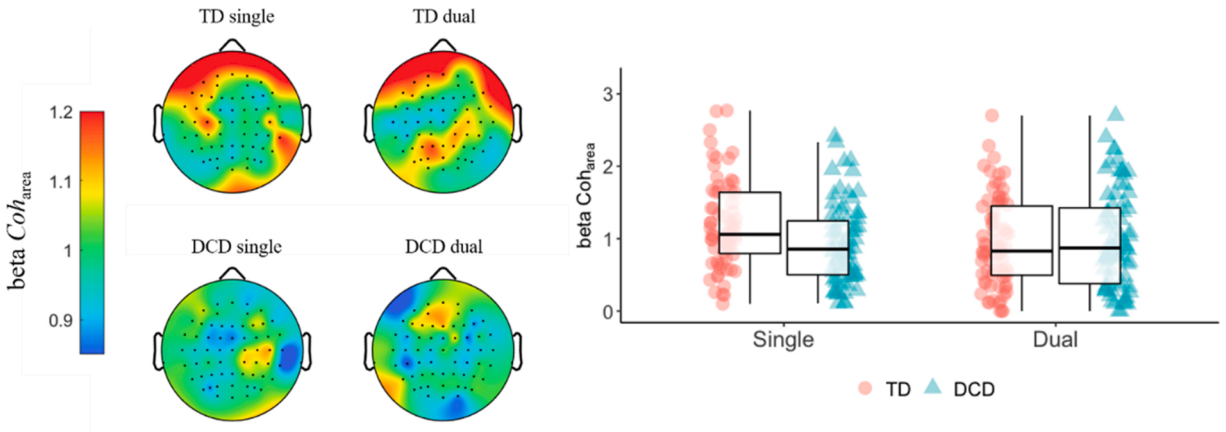

Fig. 3. . (Left) Topographical scalp maps displaying the mean beta Coh_{area} for both the TD and DCD participant during both the single and dual task conditions. (Right) Box plots with jitter points displaying the distribution of trial-level C3 beta Coh_{area} for both the TD (red) and DCD (blue) participants across both the single and dual task conditions.

as such individuals are known to persist with ineffective strategies in the face of increased task demands (Biotteau, Chaix, & Albaret, 2016).

Finally, the TD participant displayed a commonly observed pattern of CMC that was highly localised over the contralateral hand area (Fig. 3), reflective of neurally efficient corticospinal organization that optimises upper-limb function. The absence of this topographical pattern in the DCD participant supports previous work that has highlighted neurally inefficient processes in these individuals (Brown-Lum & Zwicker, 2015) and extends these findings by suggesting that such inefficiency may extend beyond cortical regions to impact brain-muscle communication.

Taken together, these findings support the feasibility of measuring CMC in DCD populations and offer some, albeit very preliminary, evidence of impaired communication between the brain and muscles in these individuals. While acknowledging the limitations of a case-study design and the generalisability of these findings, these initial insights into the potential impairment in CMC in individuals with DCD demonstrates that further investigation of these processes is warranted.

What this paper adds?

It has been recently suggested that the movement impairment associated with Developmental Coordination Disorder (DCD) may be related to impaired corticomuscular coherence (CMC) using coherence analyses. In this short report we present preliminary findings of a feasibility study where we measured, for the first time, the integrity of brain-muscle communication in an individual with DCD. Using corticomuscular coherence analysis between EEG and EMG signals during muscular contractions, we show that the impairments in force production and steadiness exhibited by the individual with DCD were mirrored in the impaired communication between the brain and muscle. Specifically, we show that the individual with DCD displayed lower CMC and displayed less localised CMC across cortical regions. These results provide initial evidence of differences in this type of neuromuscular function in an individual with DCD and provide some support to the suggestion that DCD is a 'dysconnection' syndrome. These initial insights demonstrate the feasibility of applying CMC based analysis in individuals with DCD, and indicate that further investigation is warranted in the wider DCD population. Continued investigation across this area may allow CMC to become a recognised neurophysiological marker of DCD and a tool to assess the effectiveness of rehabilitation strategies in the future.

Data Availability

Data will be made available on request.

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