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## Decreased interhemispheric time transfer of visual information in adults with Autistic spectrum disorder using the Poffenberger paradigm

Veema Lodhia<sup>a,\*</sup>, Curie. J. Suk<sup>b</sup>, Vanessa Lim<sup>a</sup>, Jeff. P. Hamm<sup>a</sup>, Ian. J. Kirk<sup>a</sup><sup>a</sup> Research Centre for Cognitive Neuroscience, School of Psychology, The University of Auckland, New Zealand<sup>b</sup> School of Psychology, The University of Sydney, Australia

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### ABSTRACT

**Background:** The Poffenberger task is a useful paradigm that measures the interhemispheric transfer time (IHTT) across the corpus callosum. Past research has demonstrated that the right to left transfer is faster in typically developing individuals compared to a left to right transfer. Numerous studies have found that the corpus callosum is structurally smaller and atypical in individuals with Autistic spectrum disorder (ASD) but whether this is associated with changes in interhemispheric time transfer in adult individuals with ASD is relatively unknown.

**Method:** The current study used a Poffenberger paradigm in combination with electroencephalography (EEG) to measure IHTT between individuals with ASD and typically developing controls. The IHTT in each direction was estimated by comparing the latencies of P100 and N170 components in hemispheres contralateral and ipsilateral to lateralised visual stimulation.

**Results:** Both groups demonstrated faster right to left transfer of information compared to the left to right transmission. Individuals with ASD exhibited faster IHTT latencies overall for the P100 and N170 components compared to matched typically developing controls. More importantly, these results demonstrate that the ASD group exhibited faster transfer in both directions compared to matched typically developing controls.

**Conclusion:** These findings suggest hyper-connectivity of local networks in parietal and occipital regions of the corpus callosum in ASD and may have implications in how information is integrated between hemispheres in ASD.

### 1. Introduction

Centre for disease control reports that 1 in every 68 individuals is affected by Autistic spectrum disorder (ASD) (Centres for Disease Control & Prevention, 2014). ASD is characterised with individuals having impaired social interaction abilities, difficulties in verbal and nonverbal communication, sensory deficits, and co-occurring restricted behaviours and interests (American Psychiatric Association, 1994; American Psychiatric Association, 2013). While the exact cause of ASD remains unknown, there is strong evidence that ASD has a neurobiological basis with a strong genetic origin (Constantino, & Todd, 2003; Hill & Frith, 2003; Minshe & Williams, 2007; Constantino, Gruber, Davis, Hayes, Passanante, & Przybeck, 2004). It is well established that the developmental trajectory of the brain is disrupted in ASD (Frith & Hill, 2003; Minshe & Williams, 2007; Landa, Stuart, Gross & Faherty, 2013) and that these disruptions occur early on in development (Courchesne et al., 2011). Abnormal growth patterns of white matter

\* Corresponding author at: School of Psychology (City), The University of Auckland, Private Bag 92019, Auckland, 1142, New Zealand.  
E-mail address: [v.lodhia@auckland.ac.nz](mailto:v.lodhia@auckland.ac.nz) (V. Lodhia).

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accelerate between the ages of 2–3 years in individuals with ASD, which is followed by atypically slowed growth in subsequent years of development. The rapid brain growth seen in children with ASD occurs during critical ages of development and is linked to disruptions in neural connectivity and structural abnormalities (Minshew & Williams, 2007) that manifest as core impairments seen in ASD (Kana, Libero, & Moore, 2011). However, it is unclear if the dysfunction seen in ASD is a result of structural abnormalities that arise from cellular morphological disruptions, neural connectivity, or a combination of both (Clawson, Clayson, South, Bigler & Larson, 2013; Kana et al., 2011).

The corpus callosum plays an important role in the communication and integration of information between the two hemispheres (Anderson et al., 2011; Van der Knaap and van der Ham, 2011; Vidal et al., 2006). Functional imaging and diffusion tensor imaging studies have provided great insight with respect to ASD brain connectivity and structure. Common findings among ASD studies is the reduction of the size of the corpus callosum (El-Baz et al., 2011; Frazier & Harden, 2009; Hardan, Minshew, & Keshavan, 2000; Manes et al., 1999; Egaas, Courchesne, & Saitoh, 1995) and the finding of altered connectivity (Travers et al., 2012; Travers et al., 2015; Vissers et al., 2012; Aoki, Abe, Nippashi, & Yamasue, 2013; Sparks et al., 2002). Post-mortem studies on individuals with ASD provide supporting evidence of altered structure, altered connectivity, and morphological disruptions of the corpus callosum (Courchesne et al., 2007; Wegiel et al., 2010; Wegiel et al., 2014).

A recent study by Wilkinson, Wang, van der Kouwe & Takashi (2016) used high angular resolution diffusion imaging (HARDI) and diffusion tensor imaging (DTI) to investigate white and grey matter in pre-adolescent ASD children and matched control children. By using HARDI in conjunction with DTI these authors were able to cross the fibre data with voxel data in juvenile brains (2–3-year-old) and verified the accuracy of the combined imaging data by post-mortem examination. Wilkinson et al. (2016) found that the corpus callosum was thinner in the ASD group compared to the control group, supporting numerous studies indicating reduced functional anisotropy (FA) of the corpus callosum in ASD (Travers et al., 2015). In addition, they found that grey matter areas were smaller overall, and that fine grey matter tracts were abnormal in the ASD group compared to controls. However, given the small sample size of the study, it is hard to conclude whether grey matter is altered across all individuals with ASD.

Shortened tract length, atypical pathways, and reduced volume of the corpus callosum are thought to contribute to the heterogeneity of symptoms seen among individuals diagnosed with ASD (Demopoulos et al., 2015; Wilkinson et al., 2016; Clawson et al., 2013; Courchesne et al., 2007; Kana et al., 2011). A recent study by Travers et al. (2015) tracked the developmental trajectory of the corpus callosum of a 100 ASD male participants and 56 matched male controls from early childhood to full development (3–41 years). Using DIT measures to track the maturation of the corpus callosum (genu, body, and splenium), they found that the ASD group demonstrated an altered developmental trajectory for white matter microstructure compared to the controls, especially prior to 10 years of age. The ASD group showed decreased FA across the entire corpus callosum, but prominently in the genu and body, consistent with the wider DTI literature in ASD. Moreover, the results of the study suggest that these developmental differences are sustained through adolescence and into adulthood supporting atypical brain connectivity theories of ASD.

While post mortem and imaging studies provide us with an in-depth understanding of the corpus callosum, very few studies incorporate behavioural tasks to functionally measure the transfer of information in the corpus callosum. Understanding how information is transferred between hemispheres may provide vital clues about how information is integrated across verbal, auditory, and motor domains, and how these are affected in ASD. The Poffenberger paradigm (1912) is often utilized to measure interhemispheric time transfer (IHTT) of the corpus callosum. A typical behavioural experiment involves participants responding to lateralized visual stimuli on screen and the estimated IHTT can be measured by subtracting the reaction time of the uncrossed hemi-field condition (i.e. visual stimuli and manual response both on the same side) from the reaction time of the crossed hemi-field condition (i.e. visual stimuli and manual response are contralateral). The response times in the uncrossed condition is faster than the crossed condition presumably because the visual and motor regions are in the same hemisphere, whereas the crossed condition involves the additional callosal transfer of information (Marzi, 2010; Poffenberger, 1912).

By combining electroencephalography (EEG) techniques with the Poffenberger paradigm, researchers can assess IHTT by measuring event-related potential (ERP) latency differences (Brown, Larson, & Jeeves, 1994). With this method, IHTT is obtained by subtracting the latencies of evoked potentials such as the P1 and N1 components that recorded over the hemisphere contralateral to the visual stimulation from that recorded at the hemisphere ipsilateral to the stimulation (Brown et al., 1994; Iwabuchi & Kirk, 2009; Moes et al., 2007). Past studies using ERPs to assess IHTT have found that typically developing controls had shorter peak latency of the ERP components when the signal was recorded at the hemisphere directly connected to the hemi-field where the stimulus was presented (i.e., direct pathway) compared to the hemisphere connected through the callosal pathway (i.e., indirect pathway) (Marzi, 2010).

There are a few studies where the visual interhemispheric time transfer in ASD individuals has been investigated. Nyden, Carlsson, Carlsson, & Gillberg (2004) investigated interhemispheric time transfer in children with ASD and matched control children using Poffenberger paradigm across visual, auditory and motor modalities. They hypothesised that the ASD group would perform worse compared to the control group and this would be indicative of interhemispheric transfer deficits. For the visual Poffenberger paradigm, they presented visual stimuli for 3 ms either in the centre, left or right side of the computer monitor. The stimulus presentation that is flashed in the centre of the screen is presumed to be projected to both hemispheres, while the stimulus presentation to the right visual field is projected to the left hemisphere and presentations in the left visual field to the right hemisphere. They tested two conditions, in condition one participants, were asked to name the stimulus presented on screen (i.e. naming task) and in the second condition participants pointed to the object presentation (i.e. object pointing task). Reaction time was used as a measure. They found that the ASD children produced significantly slower reaction times compared to the non-ASD children for the left visual field but not for the right or centre visual fields. In contrast, for the pointing task the ASD group performed significantly slower to visual stimuli presented across all visual fields, however as this task included motor function, it is unclear whether the

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