Correlation between Cerebellar White Neuroanatomy and a Motor Coordination Task in Autism Spectrum Disorder

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Introduction

Structural neuroimaging studies suggest that Autism Spectrum Disorder (ASD) is accompanied by neuroanatomical differences in the cerebellum. For example, it has been shown that total cerebellar volume is significantly reduced in ASD (Hallahan et al., 2009). Also, a reduction in total cerebellar white matter volume in ASD has been observed (McAlonan et al., 2002). However, most prior studies were based on measures of cerebellar volume, which is a neuroanatomical highly unspecific feature as it is computed as a product of cortical thickness and surface area. Also, most studies investigated the cerebellum globally (e.g. total cerebellar volume) rather than locally (i.e. different cerebellar sub-regions). In addition, it is currently unknown how neuroanatomical cerebellar differences mediate specific autistic symptoms such as impaired motor control and coordination.

Objectives

Here we aim to establish neuroanatomical differences in the cerebellum between individuals with ASD and controls using a spatially unbiased, surface-based approach based on the cerebellar white-matter, and to correlate distinct morphometric features with autistic symptoms.

Method

Participants

75 male adults, 38 diagnosed with ASD and 37 healthy controls who did not differ significantly in mean age and full scale IQ (respectively 26 ± 7 years and 110 ± 14; and 28 ± 6 years and 114 ± 14).

Image processing

Figure 1: The FreeSurfer image analysis suite was used to derive 3D models of the cerebellar white matter surface in each T1-weighted image (A) and subsequently a single filled white matter volume (B) was generated for each hemisphere lobule of the cerebellum.

Purdue Pegboard Task

The main goal of this task is to test dexterity, via two types of activities: (1) gross movement of hands, fingers and arms, and (2) ‘fingertip’ dexterity in an assemble task (Tiffin et al., 1948).

Analysis

Initially, we examined between-group differences in total cerebellar white matter volume. At each cerebellar vertex (i.e. point on the surface), we also examined between-group differences in curvature (C), cortical folding (i.e. sulcal depth) (SD) and surface area (SA) using an exploratory vertex-level threshold of P < .05 (uncorrected). Furthermore, correlations between distinct neuroanatomical features and performance in the Purdue Pegboard test were examined at each cerebellar vertex using a general linear model (GLM) with a main effect of group, a main effect of task performance, and their interaction. A t-test for independent samples was used to compare task performance between groups.

Results

Total cerebellar and cerebral white matter volume

There was no significant difference between the ASD group and controls in volume (in cm3) of the cerebellar white matter (total volume: ASD=31.77, controls=33.44, p=0.076; right hemisphere: ASD=15.90, controls=16.81, p=0.052; left hemisphere: ASD=15.87, controls=16.63, p=0.116) and cerebral white matter (ASD=508.86, controls=529.81, p=0.071).

Between-group differences in cerebellar white matter morphometry

Figure 2: Blue: regions that increase in C/SD/SA in people with ASD. Red: regions that decrease in C/SD/SA in people with ASD. On the right side clusters are displayed that survived correction for multiple comparison.

Discussion

In summary, we found that individuals with ASD have significant differences in cerebellar white matter anatomy measured by sulcal depth and surface area at right hemisphere lobules IV, V, VI and VIIib. We also found significant differences in motor coordination and interactions between the task performance and having the condition that were largely overlapping with the regional cerebellar white matter differences between ASD and controls. These differences in geometric features may mediate alterations in motor coordination in ASD. Our study therefore provided an important first step into describing the neuroanatomy of cerebellar white matter, which may aid future investigations into the specific underlying neural mechanisms of ASD.