Differing patterns of white matter connectivity in Autism Spectrum Disorders versus Sensory Processing Disorders

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Purpose

Over 90% of children with Autism Spectrum Disorders (ASD) demonstrate atypical sensory behaviors¹. In fact, hyper- or hyporeactivity to sensory input or unusual interest in sensory aspects of the environment is included in the DSM-5 diagnostic criteria for ASD². There are, however, many children with isolated sensory processing disorders (SPD) who do not show primary language or social deficits, but do show atypical sensory reactivity and/or sensory interests to the same, or greater, extent as children who meet an ASD diagnosis³. The comparison of children with SPD and ASD will help to define the neural underpinnings of each of these conditions.

We have previously demonstrated that these children with Sensory Processing Disorders (SPD) have impaired white matter microstructure, primarily in posterior cerebral tracts involved in unimodal sensory processing as well as multisensory integration⁴. We further found that this white matter microstructural pathology correlates with atypical sensory behavior. In this study, we use a diffusion tensor imaging (DTI) fiber tractography approach to contrast the structural connectivity of specific white matter tracts in children with ASD and SPD, with comparison to age-matched typically developing controls.

Methods

DTI was acquired in 16 boys with SPD, 15 boys with ASD, and 23 neurotypical boys, all ages 8-11 years. Probabilistic diffusion fiber tractography was used to define white matter tracts (examples in Fig. 1), and strength of tract connectivity was assessed using mean fractional anisotropy (FA). Group differences in connectivity were assessed with two-tailed nonparametric permutation tests, using a two-sample t-statistic, at a significance level of p<0.05.

Results

Both the SPD and ASD cohorts demonstrate decreased connectivity relative to controls in parieto-occipital tracts and the inferior longitudinal fasciculus (ILF) involved in sensory perception and multisensory integration. However, the ASD group alone shows impaired connectivity in the lingual-orbitofrontal tracts and the fusiform-amygdala and fusiform-hippocampus tracts thought to be relevant to emotional processing of face information as well as other forms of sensory processing.

Discussion

Our results demonstrate both overlapping and divergent white matter pathology affecting SPD and ASD, with both groups demonstrating extensive white matter microstructural pathology in parieto-occipital and temporoparietal-sensory processing tracts, but with temporal and occipito-orbitofrontal tracts traditionally associated with ASD being relatively unaffected in SPD. The preservation of connectivity in these tracts for the SPD cohort indicates a more direct association between decreased connectivity within these tracts and impaired social and emotional processing.

References