Alterations of the inferior longitudinal fasciculus in congenital and late blindness

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Introduction

We present preliminary results from a study of the structural changes that occur in congenital (CB) and late blind (LB) humans compared to normal sighted controls (NS). Blind subjects offer a unique opportunity to study brain plasticity and to understand neuro-adaptive processes in response to the absence of a sensory input. There is now ample evidence that the occipital cortex in congenital blind subjects remains metabolically active and is recruited by a wide variety of auditory, tactile, olfactory and cognitive tasks [1, 2]. There is a general consensus [3-6] that congenital blindness leads to atrophic changes in the fibre tracts projecting from the retina, through the optic chiasm, lateral geniculate nuclei, and optic radiation, to the visual cortex. However it is currently unknown to what extent other pathways of the visual system react to visual deprivation and how congenitally and late blind subjects differ in this respect. The fibre tracts of the dorsal and ventral visual pathways include the superior longitudinal fasciculus (SLF) and the inferior longitudinal fasciculus (ILF) as well as the fronto-occipital fasciculus (FOF). These tracts connect the occipital cortex with the parietal and temporal lobes, respectively, thereby integrating multiple sensory inputs (e.g. somatosensory, auditory, and visual), which play an important role in normal mammalian brain functioning. In this exploratory study, we investigated the effects of congenital and late-onset blindness on the integrity of the ILF.

Methods

Subjects: Six CB (mean age 43 ± 12), 6 LB (mean age 47 ± 14), and 6 NS (mean age 46 ± 13.2), matched for gender, age, education and handedness, were included in the analysis. Inclusion criteria of blind subjects were constrained to blindness caused by pathology of peripheral origin. Mean onset of blindness in the LB group was 16.6 ±8.9 years.

Image acquisition: Data acquisition was performed on a 3.0 Tesla Siemens Verio scanner with a 32-channel head coil. Whole brain diffusion weighted images (DWI) were acquired in 61 non-collinear directions with a b-value of b = 1500 s/mm^2 and 10 non-diffusion weighted images (b = 0 s/mm^2) using the twice refocused spin echo sequence (TE = 89 ms, TR = 11440 ms, 61 axial slices, isotropic 2.3 mm^3 voxels and Grappa =2) [7]. Also, a field map using a double gradient echo sequence (TR = 479ms, TE1 = 4.92ms, TE2 = 7.38ms, isotropic 3mm^3 voxels) was acquired.

Data processing: Geometric distortions due to susceptibility artefacts were minimized with the application of a voxel displacement map estimated from the acquired field map and using the field map toolbox of SPM8 [8]. The field map was resliced to DWI resolution. Affine transformation between the DWI volumes within each subject using normalised mutual information was applied to reduce motion and eddy current induced distortions. Finally, the orientation of the 61 non-collinear directions was reoriented similar to the orientation introduced by the applied displacement map and affine transformation. The diffusion tensor model was fitted to the corrected DWI and DTI parameters were calculated using Camino [9, 10]. Deterministic tractography was performed in ExploreDTI [11] using constrained spherical deconvolution as multi-fibre reconstruction [12]. We delineated the ILF individually in each hemisphere by using a two ROI approach. In a coronal view the first "AND" gate was placed in the anterior temporal lobe, right after the splitting between the ILF and the FOF, making sure not to include the fibres of the FOF. The second "AND" gate covered a large area of the white matter in the occipital lobe. For analysis only the mid-part of the ILF was included, where the projection of streamlines was most dense and uniform as shown in figure 1. This reduced the effect of fanning fibres being most pronounced at tract termination sites, lowering anisotropy. A region-of-interest (ROI) of the mid-part ILF was generated by including only voxel with FA > 0.3 and streamline counts >10.

Statistics: We calculated for each subject fractional anisotropy (FA), mean diffusivity (MD), axial diffusivity (primary eigenvalue) and radial diffusivity (RD) (average of second and third eigenvalue) from the ILF ROI in each hemisphere. To test for group differences in mean parameters, we calculated an average value for each diffusion parameter within each ROI. Statistical tests were applied for left and right ILF respectively. The non-parametric Kruskal-Wallis ANOVA test is applicable for small sample sizes, hence used to compare samples of the three groups.

Results

It was possible to extract the ILF consistently in all three groups.

For both CB and LB the left and right ILF ROIs showed a decrease in mean FA compared to NS (Figure 2). Increased RD mainly controlled changes in FA. No changes were found in axial diffusivity.

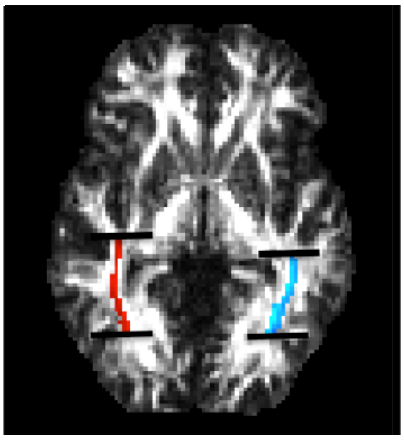

Figure 1: Illustration of the right and left ILF ROI, overlaid on an axial FA-map. Black lines illustrate tract split limits.

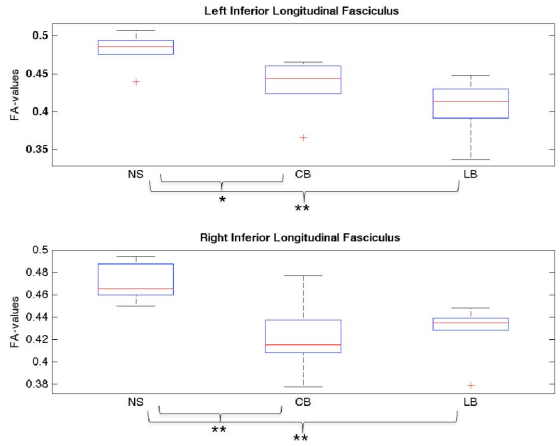

Figure 2: Boxplot of the extracted FA-values for each group for the left and right ILF. (* p<0.025 and ** p<0.005).

Discussion

We found a significant difference of structural changes in both CB and LB compared to NS when assessing microstructural markers from diffusion weighted MRI.

Both groups of blind showed a decrease in FA, suggesting that plastic structural changes also occur in late onset blindness. Based on our findings we speculate that loss of vision at birth or later in life leads to microstructural changes of the ILF. The ILF is an important pathway within the ventral stream that is involved in object recognition.

Although vision plays an important role, tactile and auditory information also contributes to object recognition. In the absence of vision, object recognition relies solely on the latter as visual information is no longer available. Consequently, the ILF will lose structural connectivity, suggested by the lower FA values in CB and LB. Our results are in agreement with earlier findings of volumetric white matter changes of the ILF [6]. Our findings need to be confirmed in larger study cohorts.

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References [1] Klinge et al., 2010 J. Neurosci.; [2] Kupers et al., 2011 Frontiers in Psychology; [3] Noppeney et al., 2005 Curr. Biol.; [4] Shimony et al., 2006 Cereb Cortex; [5] Shu et al., 2009 HBM, [6] Ptito et al., 2008 Exp Brain Res; [7] Reese et al., 2003 MRM [8] Jezzard et al., 1995 MRM; [9] Cook et al., 2006 ISMRM; [10] Basser et al., 1994 Biophys; [11] Leemans et al., 2009 Intl Soc Mag Reson Med.; [12] Tournier et al., 2007 NIMG.