



Clinical Study

Brain morphometry in blind and sighted subjects



Jerome J. Maller^{a,*}, Richard H. Thomson^a, Amanda Ng^b, Collette Mann^c, Michael Eager^d, Helen Ackland^e, Paul B. Fitzgerald^a, Gary Egan^{f,g}, Jeffrey V. Rosenfeld^{h,i,j,k}

^a Monash Alfred Psychiatry Research Centre, The Alfred & Monash University Central Clinical School, 607 St Kilda Rd, Melbourne, VIC 3181, Australia

^b Howard Florey Institute, University of Melbourne, VIC, Australia

^c Monash Vision Group, Department of Electrical and Computer Systems Engineering, Monash University, Melbourne, VIC, Australia

^d Monash Biomedical Imaging and Monash e-Research, Monash University, Melbourne, VIC, Australia

^e National Trauma Research Institute, The Alfred hospital and Department of Epidemiology and Preventive Medicine, Monash University, Melbourne, VIC, Australia

^f Monash Biomedical Imaging, Monash University, Clayton, VIC, Australia

^g School of Psychology & Psychiatry, Monash University, Melbourne, VIC, Australia

^h Division of Clinical Sciences and Department of Surgery, Central Clinical School, Monash University, VIC, Australia

ⁱ Department of Neurosurgery, Alfred Hospital, Melbourne, VIC, Australia

^j Monash Institute of Medical Engineering, Melbourne, VIC, Australia

^k F. Edward Hebert School of Medicine, Uniformed Services University of the Health Sciences, Bethesda, MD, USA

ARTICLE INFO

Article history:

Received 19 October 2015

Accepted 17 January 2016

Keywords:

Blindness

Magnetic resonance imaging

Morphometry

Visual cortex

ABSTRACT

Previous neuroimaging studies have demonstrated structural brain alterations in blind subjects, but most have focused on primary open angle glaucoma or retinopathy of prematurity, used low-field scanners, a limited number of receive channels, or have presented uncorrected results. We recruited 10 blind and 10 age and sex-matched controls to undergo high-resolution MRI using a 3T scanner and a 32-channel receive coil. We evaluated whole-brain morphological differences between the groups as well as manual segmentation of regional hippocampal volumes. There were no hippocampal volume differences between the groups. Whole-brain morphometry showed white matter volume differences between blind and sighted groups including localised larger regions in the visual cortex (occipital gyral volume and thickness) among those with blindness early in life compared to those with blindness later in life. Hence, in our patients, blindness resulted in brain volumetric differences that depend upon duration of blindness.

© 2016 Elsevier Ltd. All rights reserved.

1. Introduction

Whilst brain structure has been extensively examined in normal subjects, relatively few studies have investigated patients with blindness (Table 1). Many of these studies have reported regions of increased size or thickness in blind subjects, whilst others have found the opposite. For example, using a whole brain voxel-based morphometry approach, Chen and colleagues [1] reported that a bilateral advanced glaucoma study group (N = 15) compared to a control group (N = 15) showed significantly decreased gray matter volume in widespread regions such as the lingual gyrus, calcarine gyrus, postcentral gyrus, and an array of frontal regions; gray matter volume was significantly larger in the study group than in the control group in occipital and parietal gyri, and left occipital gyri. Zikou and colleagues [2] found reduction in the left

visual cortex volume, left LGN, and chiasm in those with POAG (N = 18). Similarly, Yu and colleagues [3] found POAG patients (N = 36) to show significant bilateral cortical thinning in the anterior half of the visual cortex around the calcarine sulci (left BA17 and BA18, right BA17) and in some smaller regions located in the left middle temporal gyrus (BA37) and fusiform gyrus (BA19) when compared to 40 matched controls. In a smaller sample, Bridge and colleagues [4] reported the banks of the calcarine sulci to be significantly thicker in six anophthalmic subjects compared with controls. The authors hypothesized that a lack of visual input in these patients from an early age may have prevented axonal and synaptic pruning in this region hence resulting in thicker cortex. Other groups have also found increased cortical thickness in these regions in early/congenital blind subjects [5–8].

As Table 1 indicates, field strength, number of receive channels, and voxel dimensions have been inconsistent in morphometric MRI studies of blindness, as have the analysis methods employed. Cause of blindness has also been heterogeneous. All of these factors prevent strong conclusions from being formed.

* Corresponding author. Tel.: +61 3 9076 2404; fax: +61 3 9076 8545.

E-mail address: jerome.maller@monash.edu (J.J. Maller).

Table 1
Previous structural brain MRI studies in blind patients

Study	Clinical N (M:F), age (years)	Control N (M:F), age (years)	Cause of blindness	Scanner and T1-weighted parameters	Analysis technique	Findings	Comments
Anurova (2015)	12 (5:7), 34–58, X = 49 ± 9.2	12 (5:7), X = 45 ± 10.9	EB	3T, 12 channel head coil, 1 mm isotropic	BrainVoyager QX Cortical thickness; preselcted ROIs	↓ Occipital, frontal STG, superior parietal, anterior cingulate	12 channel head coil
Bridge (2009)	6 (4:2), 18–31, X = 24.2 ± 5.2	6 (3:3), X = 27.0 ± 6.0 and 20 (10:10), 20–35, X = 28.0 ± 4.6	Anophthalmia	3T, 12 channel head coil, 1 mm isotropic	Whole brain; VBM FSL and FS PD (for LGN): 2 × 0.75 × 0.75 mm	VBM: Nothing when corrected; (uncorrected: ↓ Primary visual cortex) FS: ↑ Thickness and GM volume (corrected) calcarine sulcus, MTG, putamen, WM; thalamus , internal capsule, occipital . Smaller LGNs.	12 channel head coil; very small sample
Chebat (2007)	10 (5:5), 23–48, X = 38.6	10, 24–49, X = 34.0	EB	1.5T, 1 mm × 0.94 × 0.94 mm	Display (only hippocampus)	↓ GM lingual, calcarine, postcentral, SFG, IFC, Rolandic operculum, inferior occipital , paracentral, supramarginal, cuneus	1.5T, low intra-class reliabilities (0.73–0.98)
Chen (2013)	15 (9:6), 40–50, 43.3 ± 4.1	15 (9:6), X = 43.9 ± 3.8	POAG	3T, 8 channel head coil, 1 mm isotropic	SPM	↓ MTG, IFC, angular, parietal, precuneus, middle occipital	8 channel head coil; first reduced the number of voxels entering the statistical computation
Dai (2011)	26 (21:5), 25–58, X = 35.4	26 (21:5), 21–57, X = 35.4	POAG	3T, 8 channel head coil, 1 mm isotropic, PD: 1.8 × 0.64 × 0.64 (for LGN measurements)	Advantage workstation (only LGN)	↓ LGN height and volume	8 channel head coil
Fortin (2008)	12 EB (9:3), 19–55, X = 33.8 7 LB (4:3), 22–57, X = 33.9	19 (13:6), 19–56, X = 36.0	EB and LB	1.5T, 1 mm isotropic	Display (only hippocampus)	↓ hippocampal head (in both EB and LB compared with controls)	1.5T
Gupta (2009)	10 (3:7), 52–76, X = 63.1 ± 7.7	8 (3:5), 46–71, X58.6 ± 10.0	POAG	1.5T, 8 channel head coil, 4 mm gap 10 mm, PD: 2 mm and Av = 5 (for LGN measurements)	Magicweb (only LGN)	↓ LGN height	1.5T, 8 channel head coil
Jiang (2009)	17 EB (9:8), 15–31, X = 22.6 ± 4.0 19 LB (12:7), 18–35, X = 24.2 ± 5.0 22 (14:8), X = 36.9 ± 11.0	29 (15:14), X = 22.6 ± 3.1	EB and LB	3T, 1 mm isotropic	FreeSurfer; whole brain	↓ L temporal lobe ↓ EB primary visual cortex (compared with controls or LB) ↓ EB L fusiform (compared to LB) ↓ R hippocampal head ↓ hippocampal tail	Analyses between EB and LB not corrected for multiple comparisons
Lepore (2009)	28 (17:11), X = 34.1 ± 10.6	28 (17:11), X = 34.1 ± 10.6	NS	1.5T, 0.98 mm (note: this is NS, but matrix = 256 × 256 and FOV = 250 mm)	Display (only hippocampus)	↓ EB and LB, but greater in EB) V1/V2, cingulate, I SMA, PMA, L entorhinal	1.5T
Lepore (2010)	16 EB (10:6), 19–55, X = 36.2 ± 9.8 16 LB (10:6), 22–56, X = 38.2 ± 10.2	32 (20:12), divided into: 16 (10:6) 22–44, 35.3 ± 9.5 (matched for EB) 16 (10:6), 22–57, X = 38.2 ± 10.3 (matched for LB)	EB and LB	1.5T, 32d, 1 mm isotropic (reconstructed to 1 × 0.66 × 0.66 mm)	ANIMAL, Multitracer (for CC), BrainSuite (whole brain)	↓ (EB and LB, but greater in EB) V1/V2, cingulate, I SMA, PMA, L entorhinal	1.5T
Manara (2014)	11 (6:5), 6–45, X = 23	19 (6:5), X = 23 (6–43)	Alstrom Syndrome	1.5T, 32d, 1 mm isotropic (reconstructed to 1 × 0.66 × 0.66 mm)	SPM; whole brain	calcarine cortex and fusiform gyri	1.5T
Pan (2007)	14 (7:7), 38–58, X = 47.1 ± 5.8	16 (8:8), 42–55, 49.8 ± 4.1	EB	1.5T, 1.2 × 0.94 × 0.94 mm, Av = 2.	SPM; whole brain	↓ GM V1 and cuneus/lingual gyrus	1.5T
Park (2009)	21 EB (16:5), 17–36, X = 27.1 ± 5.4 12 LB (7:5), 18–38, X = 28.8 ± 6.6	35 (21:14), 22–37, 26.7 ± 4.1	CB and LB	3T, 1.2 × 0.98 × 0.98 mm	FreeSurfer; whole brain	↓ WM bilateral optic radiation and R anterior temporal lobe CB: ↓ pericalcarine, lateral occipital , lingual	1.5T
Pitto (2008)	11 (6:5), 22–68, X = 33.0	21 (11:10), 20–54, X = 35.6	CB	1.5T, 1 mm × 0.94 × 0.94 mm	SPM; whole brain	↓ L somatosensory R auditory cortex (compared to LB and controls) ↓ whole brain GM and WM ↓ GM: LGN and R pulvinars, bilateral BA17/18/19, MTG, caudate, posterior hippocampus, R SFG, R IFC, R lateral orbital, R insular	1.5T
Shimony (2006)	5 (3:2), 27–54, X = 40.4	7 (4:3), 20–56, X = 34.7	EB	1.5T, 48d, SI = 1 × 1 × 1.25 mm reformatted to 2.5 mm isotropic	In-house 3D Slicer; and Analyze; LGN, V1/V2 WM and GM	↓ WM: R OF, SLF, genu CC ↓ WM V1/V2 (but not GM)	Small sample size; 1.5T; T1-weighted data reformatted to low resolution
Trampel (2011)	13 (6:7), 21–70, X = 46	15 (8:7), 20–31, X = 25	CB	7T, 24 channel head coil, 0.5 mm isotropic	Signal intensity of Stria of Gennari	Stria of Gennari is detectable in CB subjects	7T
Voss (2011)	14 EB (10:4), 20–59, X = 38.2 ± 13.8 13 LB (5:8), 29–60, X = 46.6 ± 8.5	19 (8:11), 37.6 ± 12.0	EB and LB	3T, 1 mm isotropic	GIVET	EB ↓ L lingual and R lateral occipital (compared with controls) LB ↓ lingual, cuneus , L inferior/middle occipital (compared with controls)	Small sample size; 1.5T; T1-weighted data reformatted to low resolution
						lateral occipital (compared with EB)	

Download English Version:

<https://daneshyari.com/en/article/5630013>

Download Persian Version:

<https://daneshyari.com/article/5630013>

[Daneshyari.com](https://daneshyari.com)