Laminar-specific variations of T2* relaxation decay in the cortex at 7 Tesla MRI

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Purpose. We recently demonstrated the ability of a surface-based technique to study the spatial distribution of cortical myelination in patients with multiple sclerosis^{1,2} and healthy subjects³ using T_2^* gradient echo images at 7 T MRI. Our data from healthy individuals showed that quantitative T_2^* mapping reveals patterns of cyto- and myeloarchitecture of the human cortex in vivo³. In this study, we probe the potential of mapping T_2^* in the cortex as a function of cortical depth to reveal the underlying laminar architecture. We also report the scan-rescan variability of T_2^* measurements at various depths from the pial surface.

Methods. We scanned 8 healthy subjects (mean±SD age = 38.5 ± 8.7 years) twice, a week apart, on a 7 T Siemens system to acquire multi-echo T_2^* -weighted Fast Low Angle Shot (FLASH) spoiled gradient-echo images (resolution = $0.33 \times 0.33 \times 1 \text{ mm}^3$), and once on a 3 T MR system to acquire T1-weighted data for cortical surface models reconstruction using FreeSurfer⁴. T_2^* signal at each voxel was corrected for background field gradients in the through-slice direction, associated with B_0 inhomogeneities which were particularly present in the lower brain regions. The corrected T_2^* signal was fitted voxelwise using a Levenberg–Marquardt non-linear regression algorithm as described in Cohen Adad et al (2012). The resulting T_2^* maps were registered to the cortical surface models from the corresponding 3 T data. T_2^* rates were sampled along the cortex at 14 different depths (5-10% intervals ranging between 10-95% depths) from pial surface (0% depth) towards white matter (WM, 100% depth). T_2^* heterogeneity at these depths was assessed by comparing the average T_2^* rates within FreeSurfer labeled cortical regions using Kruskal-Wallis test. Scan-rescan reproducibility was measured using coefficients of variations (COV=SD/mean) of each couple (scan-rescan) of measurements, at cortical depths 25%, 50% and 75%.









Results. T_2^* is significantly heterogeneous across regions (p<0.001) as previously reported³, and there is no difference between hemispheres (p=0.27). Across the entire cortex (whole cortex mean± SD $T_2^* = 33.77\pm1.47$ ms, 32.18 ± 1.42 ms, and 30.29 ± 1.54 ms at 25%, 50% and 75% depths respectively) and in most individual cortical regions T_2^* is 1.6ms greater at 50% relative to 75% depth and 2.9ms greater at 25% relative to 75% depth and 2.9ms greater at 25% relative to 75% depth (p<0.001). Figure 2 illustrates the variation of T_2^* measures across a few cortical regions in the right hemisphere and consistent shortening of T_2^* with depth from the pial surface. As noted in Cohen-Adad et al (2012), average T_2^* was lower in sensorimotor, auditory and visual cortices, here represented using the 'precentral', 'transverse temporal' and 'pericalcarine' labels respectively. Similarly, the superior frontal and the cingulate regions range from 0.5% to 6.5% (average COV across regions, across depths = 1.64%) as can be seen in Figure 3, and COVs for whole cortex T2* at 25%, 50% and 75% depths were 0.83%, 1.78% and 0.87%.

Discussion. Patterns of lower T_2^* detected in precentral, transversal and pericalcarine regions likely reflect higher iron and co-localized myelin content³. Across cortical areas, the layer-specific decrease in T_2^* from pial surface to WM is consistent with the greater degree of myelination in deeper cortical layers. We are aware that greater partial volume effect is likely to affect the estimated $T2^*$, particularly close to the WM and pial boundaries, however the same trends are observed after weighting voxels affected by partial volume effects, as proposed in Polimeni et al⁵.

Conclusion. Surface-based methods used to map T2* as a function of depth are reproducible and can prove useful to study the layered structure of the cortex *in vivo*. T2* mapping can be used to understand and potentially quantify the pathophysiology and progression of diseases associated with changes in iron and/or myelin concentration.

References. [1] Cohen-Adad, *NeuroImage* 2011. [2] Mainero, *Neurology* 2009. [3] Cohen-Adad, *NeuroImage* 2012. [4] Dale, *NeuroImage* 1999. [5] Polimeni, *NeuroImage* 2010. This study was supported by a grant of the National MS Society (NMSS) RG-4281-A-1.