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 sclerosis1,2 and healthy subjects3 using T2* gradient echo images at 7 T MRI. Our data from healthy individuals showed that quantitative T2* mapping reveals patterns of cyto- and myeloarchitecture of the human cortex in vivo1. In this study, we probe the potential of mapping T2* in the cortex as a function of cortical depth to reveal the underlying laminar architecture. We also report the scan-rescan variability of T2* 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 T2*-weighted Fast Low Angle Shot (FLASH) spoiled gradient-echo images (resolution = 0.33 × 0.33 × 1 mm3), and once on a 3 T MR system to acquire T1-weighted data for cortical surface models reconstruction using FreeSurfer4. T2* signal at each voxel was corrected for background field gradients in the through-slice direction, associated with B0 inhomogeneities which were particularly present in the lower brain regions. The corrected T2* signal was fitted voxelwise using a Levenberg–Marquardt non-linear regression algorithm as described in Cohen-Adad et al (2012). The resulting T2* maps were registered to the cortical surface models from the corresponding 3 T data. T2* 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). T2* heterogeneity at these depths was assessed by comparing the average T2* 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. T2* is significantly heterogeneous across regions (p<0.001) as previously reported5, and there is no difference between hemispheres (p=0.27). Across the entire cortex (whole cortex mean± SD T2* = 33.77±1.47 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 T2* decreases with depth from the pial surface (Figure 1). On average T2* is 1.6ms greater at 50% relative to 75% depth and 2.9ms greater at 25% relative to 75% depth (p<0.001). Figure 2 illustrates the variation of T2* measures across a few cortical regions in the right hemisphere and consistent shortening of T2* with depth from the pial surface. As noted in Cohen-Adad et al (2012), average T2* was lower in sensorimotor, auditory and visual cortices, here represented using the ‘precentral’, ‘transversetemporal’ and ‘pericalcarine’ labels respectively. Similarly, the superior frontal and the cingulate regions show a substantially high T2* rate. Scan-rescan COVs across cortical 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 T2* detected in precentral, transversal and pericalcarine regions likely reflect higher iron and co-localized myelin content4. Across cortical areas, the layer-specific decrease in T2* 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 al4.

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.