

Detection of the Grey Matter Lesions in MTR and MPRAGE in 7T

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Introduction The majority of MRI studies of multiple sclerosis (MS) examine changes in white matter (WM), although the correlation of the WM lesion load with the clinical disability is not good. Histopathological findings suggest that cortical lesions are very common in MS and might provide additional information regarding its pathogenesis. GM lesions are difficult to detect on MR due to their small size, however recently some success has been reported using T2* weighted images at 7T and DIR at 3T. We have previously shown that 7T MPRAGE can detect a number of cortical lesions [1]. In this work we compared detection of GM lesions on high resolution 7T Magnetization Transfer Ratio (MTR) maps and MPRAGE images.

Methods 8 MS (7 RRMS, 1 SPMS) patients (age mean=48±9), and 4 healthy controls (HC) (age mean=33±8) were scanned on 7T Philips Achieva scanner using (i) high resolution MPRAGE images (0.5x0.5x0.5mm, TI=1070ms, FA=8°, TE/TR=7/15ms, 280 slices, FOV=205x215x140mm, tailored inversion pulse to reduce effects of B1 inhomogeneities [2]); (ii) MT-TFE acquisition giving two images (MT_{sat} and MT_{noSat}) used to produce MTR maps: the MT_{sat} was acquired by applying

20 off-resonance pulses (sinc pulses, bandwidth=300Hz, off-resonance=1.0kHz (3.4ppm), 21ms between each pulse); these pulses were omitted for the MT_{noSat} reference image. The sequence used a Turbo-Field echo readout (TR/TE=12/6.4ms, flip angle=8°, 0.5x0.5x1mm³, FOV 205x175x80mm, centre-out sampling, shot to shot interval (SSI) of 10s, acquisition time=8min 50s). Images were then coregistered and high resolution MTR maps were calculated from (MT_{noSat}-MT_{sat})/MT_{noSat} on a pixel by pixel basis. T2*-images were also acquired with a 3D FFE sequence (TR/TE=50/17ms, flip angle=16°, acquisition time=9min). Segmentation of the cortical grey matter ribbon (CGMR) was obtained via Freesurfer run on the MPRAGE data. The mask of the CGMR was dilated, and then manually corrected for any missegmentation due to WM lesions. Finally the CGMR mask was visually checked to ensure the cortical ribbon would be blinded to the reviewers. The mask was applied to the MPRAGE, MTR and T2*w data sets which were previously coregistered to the MPRAGE volume via FSL. Lesions were manually segmented by a trained researcher separately in MTR and MPRAGE images of randomized subjects and reviewed post-hoc by another observer to separate purely cortical and mixed lesions (which lie in both GM and WM). Numbers of lesions identified for each subject were counted automatically. Masks of the non lesional GM (NLGM) surrounding each lesion were created by subtracting two masks created by dilating the lesions masks dilated by 2 and then 5 voxels. Contrast between the NLGM and lesion was calculated for each lesion on MTR and MPRAGE (normalised by dividing by the mean of NLGM).

Results Fig. 1 shows example of both lesion types in both modalities. Fig. 2 and Table 1 summarize the detected lesions, and show that GM abnormalities were significantly more common in MS patients than HC. MTR detected almost twice as many cortical lesions as MPRAGE, but was less efficient in case of mixed ones. Relatively few lesions were detected on both sequences. MTR and MPRAGE contrast between lesion and NLGM were plotted in Fig. 3 for both cortical and mixed lesions. In 4 randomly chosen cases (2 controls and 2 MS) T2* scans were examined and showed only 8 lesions in total between them.

Discussion We have shown an increased rate of detection of intracortical lesions for MTR compared to MPRAGE at 7T, but MPRAGE was more sensitive in detecting mixed leukocortical lesions. Since only very few lesions were detected on HC, it seems unlikely that artefacts were misinterpreted as lesions. Both sequences were better at detecting cortical lesions than T2*w scans, possibly due to the sensitivity of T2*w scans to shimming variations and motion and in contrast to previous work comparing T2* and T1 weighted scans [3]. The spatial resolution made it possible to localize the position of the cortical lesions precisely, for instance as purely intracortical, which was not possible in previous studies using DIR results [4]. Since the cortical levels of myelin are low, demyelination is difficult to detect in the GM. MTR is more specific for demyelination than other sequences which may explain why more lesions were detected with MTR than with MPRAGE, despite the fact that the MTR maps are noisier than the MPRAGE images, and require intrinsic registration. The longitudinal relaxation time (to which MPRAGE is sensitive) is sensitivity to different tissue characteristics compared to MT (eg inflammation), which may explain its sensitivity to the mixed lesions. Perilesional contrast (fig. 3) may allow automatic classification of lesions in future which can be used in longitudinal studies of patients.

References : [1] Tallantyre EC, et al. JMRI: 2010; 32(4):971-7. [2] Hurley A, et al. MRM: 2010; 63(1): 51-8. [3] Mainiero C, et al. Neurology: 2009; 73:941-948. [4] Geurts JJG, et al. Neurology: 2011; 76:418-424. Funded by MRC.

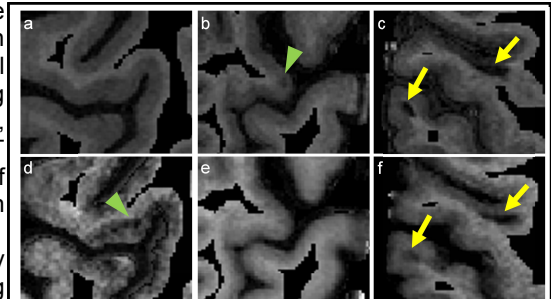


Fig. 1: Example lesions in MPRAGE (a,b,c) and MTR (d,e,f). Cortical lesions (green arrow heads) detected only in MTR (a,d) and only in MPRAGE (b,e) and mixed lesions (yellow arrows) detected in both images (c,f).

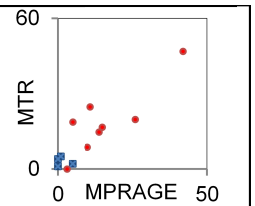


Fig. 2: Number of lesions found in HC ■ and MS ● in MTR versus MPRAGE.

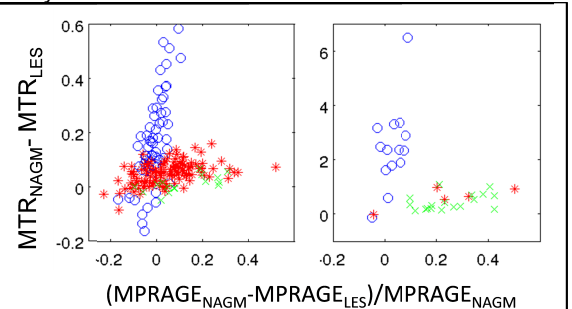


Fig. 3: Scatterplots of contrast between lesions and NLGM in MTR and MPRAGE for lesions detected in MPRAGE ○, MTR * and both ×. Left: cortical and right: mixed lesion.

	MPR		MTR		MPRAGE & MTR	
	cortical	mixed	cortical	mixed	cortical	mixed
MS (N=8)	79	37	129	22	16	18
HC (N=4)	5	0	12	0	0	0

Tab.1: Summary of the lesion detection in 12 subjects in MTR and MPRAGE.