

7 Tesla Sodium MRI of patients with Multiple Sclerosis: A preliminary study

G. Madelin¹, N. Oesingmann², G. Johnson¹, S. Nielles-Vallespin³, B. Stoeckel², E. Barberi⁴, R. Pinkerton⁴, D. Sodickson¹, R. I. Grossman¹, and M. Ingles^{1,5}

¹Radiology, New York University, New York, NY, United States, ²Siemens Medical Solutions, New York, NY, United States, ³Siemens AG, Erlangen, Germany, ⁴XLR imaging Inc, London, Ontario, Canada, ⁵Neurology, New York University, New York, NY, United States

Introduction: Multiple Sclerosis (MS) is a multi-focal, inflammatory, demyelinating disease with pathological involvement of neurons and axons¹. Pathological and imaging studies have shown that neuro-axonal loss occurs progressively from the onset of the disease, and that it correlates with disability. These findings have prompted the search for mechanisms of neurodegeneration and for surrogate markers to monitor *in vivo* degeneration and neuroprotection. There is increasing evidence that the intra-axonal accumulation of sodium ions may contribute to degeneration^{2,3}. In addition, partial blockade of sodium channels protects axons from degeneration in experimental models of MS, and it is currently under investigation in clinical trials⁴. Sodium MR Imaging (²³Na MRI) provides an indicator of cellular and metabolic integrity and ion homeostasis and has been applied to the study of patients with brain tumors and stroke⁵. The low sensitivity and spatial resolution of ²³Na MRI make higher field strengths desirable to improve this imaging modality. The aim of this study was to optimize ²³Na MRI at 7T and investigate its feasibility in the study of patients with MS.

Material and methods: Three patients with MS (mean age 40±8.5 years) and three healthy controls (mean age 36±6.5 years) underwent ²³Na MRI on a 7T whole body imager equipped with multi-nuclei options (Siemens Medical Solutions, Erlangen, Germany). Approval for this study was obtained from the Institutional Board of Research Associates of New York University Medical Center and informed consent was obtained from all subjects. The MR experiments were performed using a prototype single tuned ²³Na unshielded TX-RX head coil (XLR Imaging Inc.) and a 3D GRE radial sequence. The pulse sequence's parameters were: TR: 120 ms, TE: 0.05 ms, 1440 radial views, Flip Angle: 90°, 5 averages, BW: 130 Hz/pixel, FOV: 240 × 240 mm², matrix size: 60×60, nominal resolution: 4×4×4 mm³, Acquisition time: 14:26 min. Two calibration tubes with two different known concentrations of sodium (100 mM and 50 mM) in Agar gel 4% were placed in the FOV as references and allowed quantification of the sodium signal as a voxel-wise concentration.

Results and discussion: Good quality brain ²³Na images were obtained in patients and controls. The specific absorption rate (SAR) was monitored in real time and was within FDA guidelines (<60% FDA limit). None of the subjects experienced side effects. Selected 3D radial images and the corresponding total sodium concentration (TSC) maps from a 33 year old patient with MS and a 35 year old healthy control are shown in Figure 1. For the patients, sodium hyper-intensity was observed in most of the lesions that were hypo-intense on T1-weighted images in line with the notion that these lesions are characterized by more severe tissue destruction. Serial studies and further patients recruitment is needed to investigate the dynamic of sodium changes in relation to disease activity and progression.

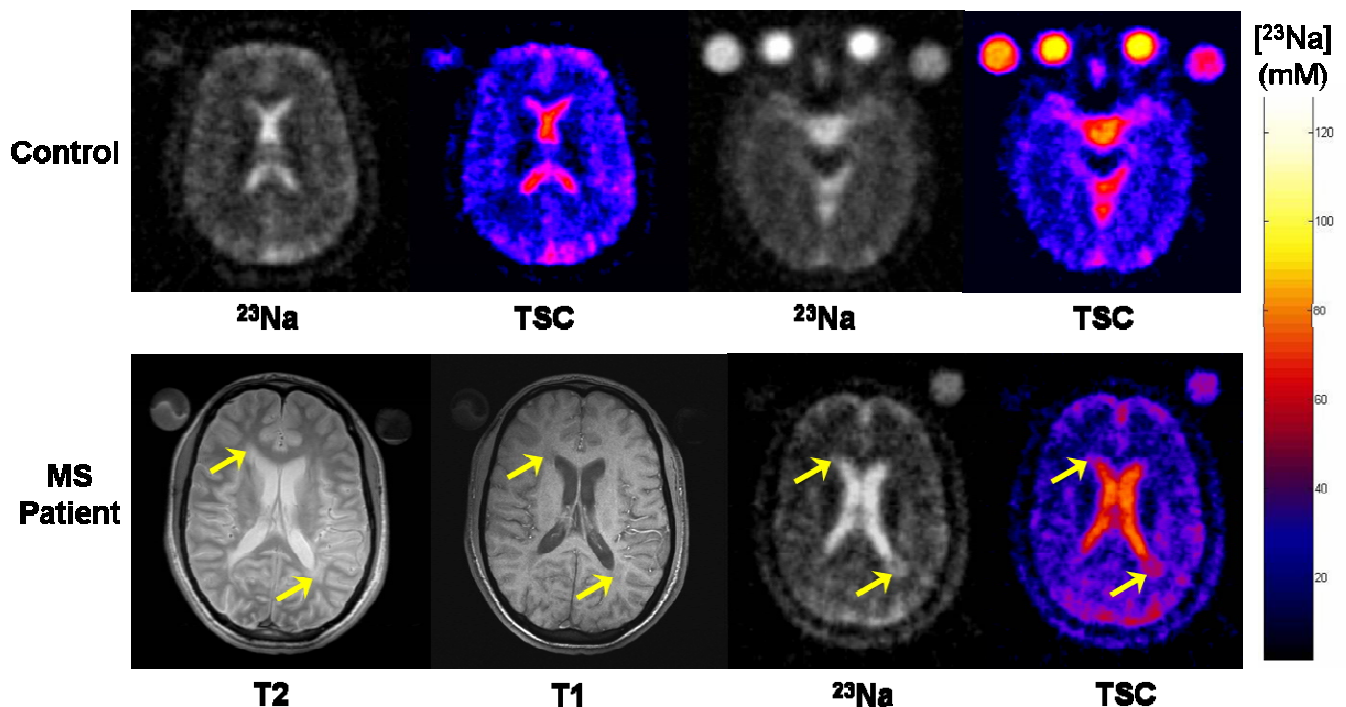


Figure 1. Selected images from a 3D ²³Na dataset and corresponding TSC maps for a 35 year old healthy control and for a 33 year old patient with MS. Note the higher TSC value in MS plaques (arrows) which appear hypo-intense on the corresponding T1 weighted images obtained at 3T.

Conclusion: This study demonstrates the feasibility of ²³Na MRI in MS at 7T. Future studies will focus on the validation of sodium measurements in lesions and normal-appearing brain tissue in MS patients with different disease course and on the development of methods to measure intra- and extra-cellular sodium changes.

References: 1. Trapp BD *et al.*, N Engl J Med 1998. 2. Waxman SG, Brain 2005. 3. Bechtold DA *et al.* J Neurol Sci 2005. 4. Kapoor R, Current Opinion in Neurology 2006. 5. Thulborn KR *et al.*, Neuroimaging Clin N Am 2005.

Acknowledgments: This study was supported in part by NIH RO1 NS051623.