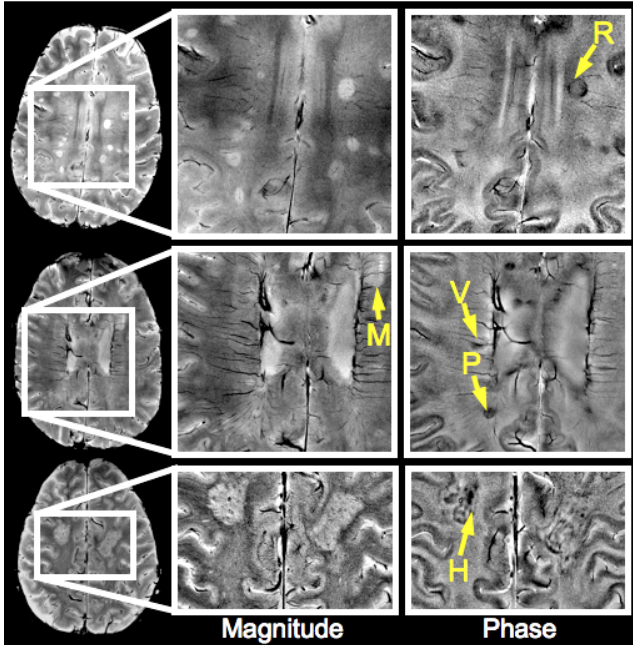


## Phase Imaging of Multiple Sclerosis at 7T

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**Introduction:** Studies of patients with neurodegenerative diseases would benefit from the availability of imaging methods that provide alternative mechanisms for generating contrast. The phase of the MR signal is sensitive to the presence of magnetic-susceptibility-shifted compounds such as iron. We have developed a technique for creating phase images that quantified the B<sub>0</sub> field shifts in MS lesions and the basal ganglia using standard gradient echo images acquired at 7T with a multichannel coil and phase unwrapping.



**Figure 1:** MS white matter lesions had (R)inged contrast, penetrating (V)essels or (H)eterogeneous contrast. Some lesions were visible in only either (M)agnitude or (P)hase.

**Methods:** Thirty-two subjects including seventeen RRMS patients (age 37.3±12.6 years, disease duration 11.3±8.2 years, EDSS 1.9±1.3) and fifteen controls (age 36.5±9.9 years) were scanned on a whole-body 7T MRI (GE Healthcare) equipped with an 8-channel receive phased array coil (in-house or Nova Medical) and a head transmitter coil with active detuning. Gradient echo images (TE/TR 15/250ms, 20° flip, 2mm slices, 3 NEX, 9 or 6.5min) were acquired at a spatial resolution of 195 by 260 microns or 180 by 350 microns. The gradient echo data, which is traditionally used to produce anatomical magnitude images, was post-processed to produce phase images of the B<sub>0</sub> field [1].

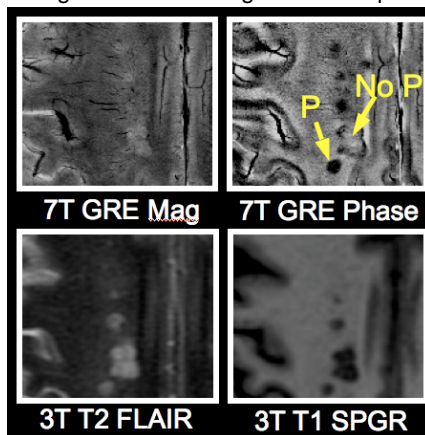
**Results:** The phase images demonstrated hypointense plaques with penetrating vessels in the region of the deep medullary veins (Fig.1, V). Some white matter plaques only had hypointense phase contrast at the periphery (Fig. 1, R). Fourteen of the MS patients had hyperintense phase shifts in the choroid plexus. The mean B<sub>0</sub> field shift in patients relative to controls was significantly higher (Wilcoxon rank sum test) in the deep gray matter (Table 1; Fig. 3).

Anatomical Region	MS Patients	Controls	P
Globus pallidus	4.8±0.8 Hz	2.6±1.3 Hz	<0.005
Caudate	4.1±0.6 Hz	2.5±0.8 Hz	<0.005
Thalamus	0.9±0.3 Hz	0.4±0.3 Hz	<0.05

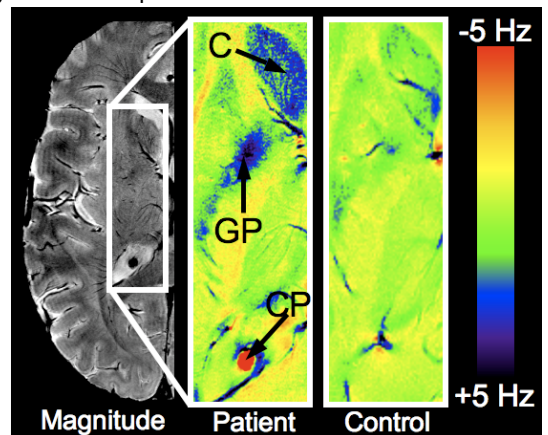
**Table 1:** MS subjects had significantly higher phase contrast in the basal ganglia than control subjects.

**Discussion & Conclusions:** 7T MR phase showed novel contrast to magnitude in high-resolution images of MS plaques and enabled quantitative assessment of iron in the deep gray nuclei. The frequent observation of plaques containing penetrating vessels (Fig. 1, V) supported the contention that MS progresses along the vasculature [2]. The B<sub>0</sub> field shift observed at the periphery of lesions (Fig. 1, R) supported existing histological data identifying iron-rich macrophages in acute plaques and globular structures of non-heme iron in chronic plaques [3]. The ability to use phase images to detect magnetic susceptibility-shifted compounds such as iron in MS lesions is especially important because in magnitude images the increased water content could lengthen T<sub>2</sub> and mask the signal dropout from iron (e.g. Figs. 1 and 2, P). As a result, only phase *definitively* shows the presence of susceptibility-shifted compounds.

Paramagnetic compounds such as iron increase the local B<sub>0</sub> field, while diamagnetic compounds such as calcifications decrease the B<sub>0</sub> field. The significantly increased field in the deep gray nuclei suggests increased iron deposition [4], while the decreased field in the choroid plexus likely results from calcification (Table 1; Fig. 3). Phase imaging may be valuable for monitoring disease severity and furthering understanding of MS biology.



**Figure 2:** Only a subset of T<sub>2</sub>-hyperintense T<sub>1</sub>-hypointense lesions showed phase contrast (P).



**Figure 3:** Paramagnetism in the (C)audate, and globus pallidus (GP) and diamagnetism in the choroids plexus (CP) was more prominent in MS patients (patient: age 41, control: age 49).

**References:** [1] Hammond et al., (2007) *NeuroImage In Press* [2] Kesseling et al., (1997) *SMW* 127:500-5 [3] Connor et al., (2001): *Pediatr. Neurology* 25:118-29 [4] Ge et al., (2007) *AJNR* 28:1639-44. **Acknowledgments:** This research was supported by UC Discovery grants LSIT-01-10107 and ITL-BIO04-10148 (in conjunction with GE Healthcare) and NSF NDSEG.