**Fluorine-19 Magnetic Resonance at 21.1 Tesla to Detect Brain Inflammation**

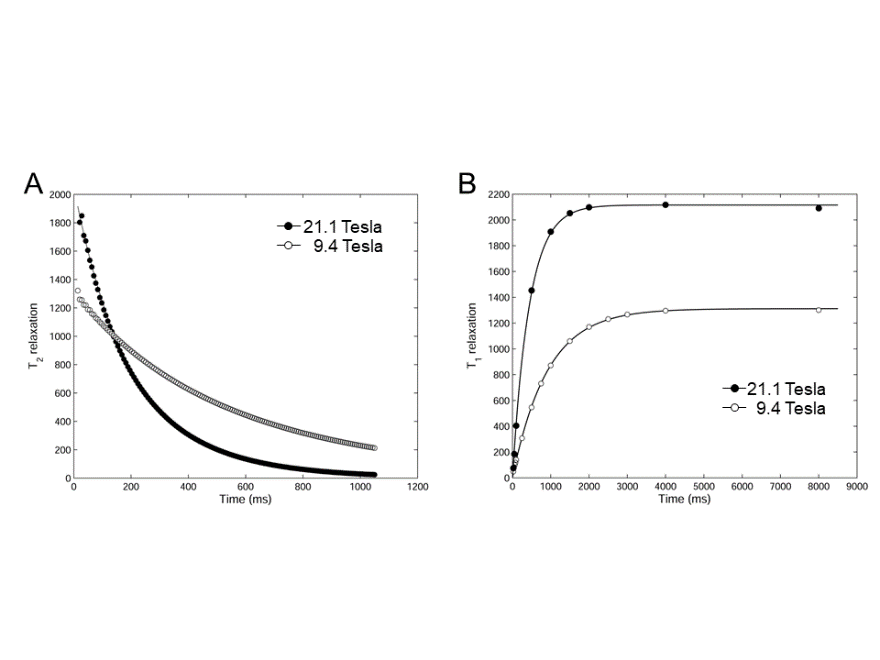
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**Introduction**

Neuroinflammation can be monitored with 19F MRI using 19F-nanoparticles (NPs) that label immune cells *in vivo*. The migration of these cells into the brain can then be studied in animal models of multiple sclerosis1,2. The low abundance of 19F nuclei *in vivo* poses a major challenge for MR detection in neuroinflammation. The theoretical SNR gain including increases in noise from sample and coil losses is about SNR B01.75 for solenoidal coils3. Recognizing these opportunities and challenges, we investigated the influence of 21.1 T on 19F relaxation times and SNR gain, compared to 9.4 T.

**Experimental**

Experiments were carried out on the 21.1 T at the NHMFL and a 9.4 T scanner at the Berlin Ultrahigh Field Facility (B.U.F.F.) using similar birdcage coils (1H/19Fat21.1 T=900/845 MHz and at 9.4 T=400/376 MHz) and parameters. For relaxation and SNR measurements, tubes of 19F-NPs (perfluoro-15-crown-5-ether)4 dilutions were submerged in saline. T1 and T2 mapping was performed on spin echo sequences using one 10-mm axial slices (FOV=30x30mm) with varying repetition times (TR) or echo times (TE). SNR was calculated on an axial 2D-RARE images (TR/TE=4000/9.1ms, slices=1-10mm). Animal experiments were carried out in accordance with local animal welfare protocols. EAE was induced in SJL/J mice and 19F NPs were administered daily for five days after which mouse tissue was prepared for *ex vivo* MRI. 3D 19F RARE sequence was acquired at low (matrix=90x60x60), medium (matrix=135x90x90) and high (matrix=135x90x90) resolution. A FLASH 1H image was acquired as an anatomical reference to the 19F image.

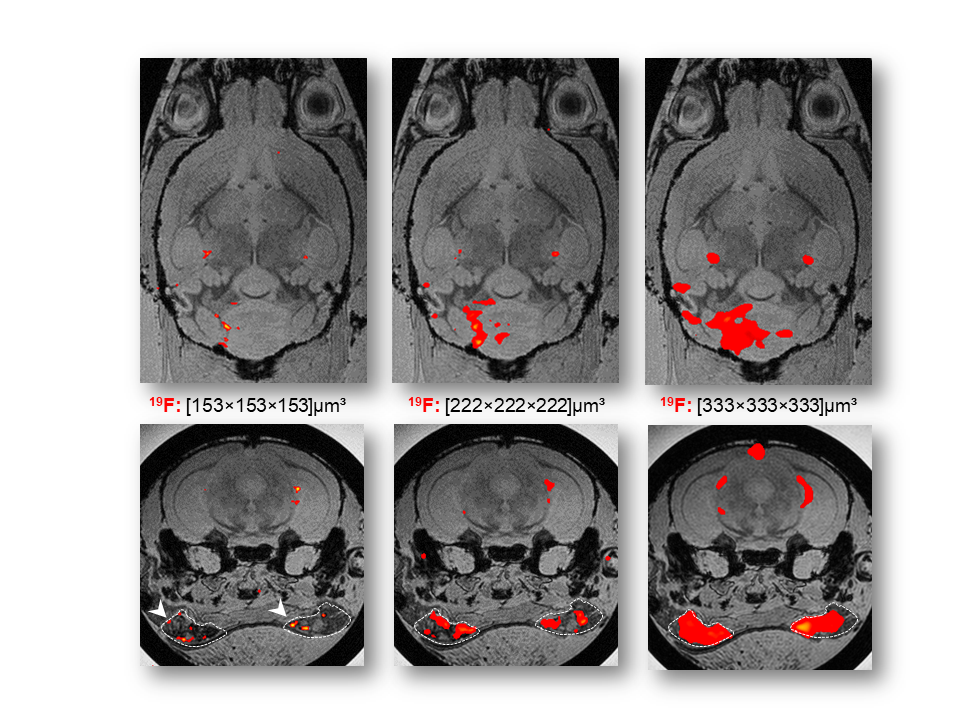
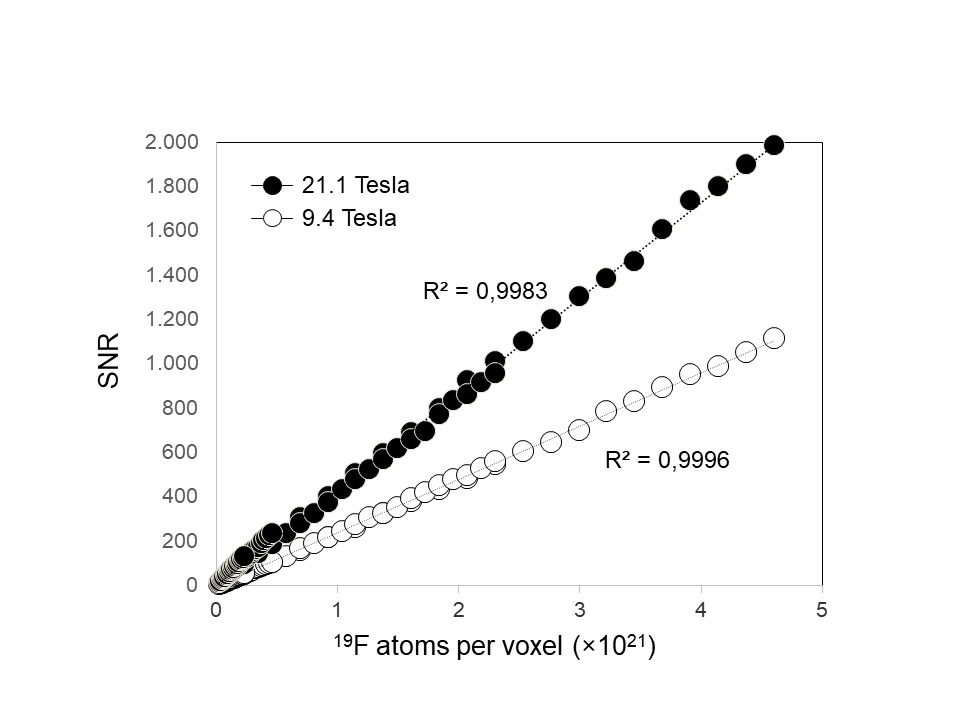


**21.1 T: T2 = 195 ms**

**9.4 T: T2 = 503 ms**

**21.1 T: T1 = 441 ms**

**9.4 T: T1 = 913 ms**



**C**

**D**

**Results and Discussion**

C:\Users\bickett\Pictures\301_19F_Figure1c-v2.tif Both T1 and T2 values for the 19F NPs were influenced by B0. The transverse relaxation was decreased at 21.1 T (Fig 1A). T1 of the 19F NPs decreased by nearly 50% at 21.1 T (Fig 1B), contrary to 1H T1 relaxation. For SNR measurements, slice thickness was varied and SNR was obtained as a function of the number of 19F atoms per voxel (Fig 1C). An SNR gain of 2.1 was achieved at 21.1 T versus 9.4 T using parameters optimized for 9.4 T. High resolved MRI of EAE mice at 21.1 T revealed a greater level of detail of the immune cell migration in the inflamed brain and draining lymph nodes (Fig 1D).

**Conclusions**

Our data demonstrate the feasibility of 19F MRI at 21.1 T for detecting inflammation in the brain and adjacent lymphatic system with higher SNR and as a result higher spatial definition. The shortened T1 is unexpected but consistent with previous studies5,6. The difference in the experimental SNR gain (2.1) and the maximum expected SNR gain (2.8) can be explained by coil and receive chain losses as well as preamplifier noise variations between both setups.

**Fig.1A**: Signal decay vs. TE yielding T2. **Fig 2B:** Signal increase vs. TR yielding T1. **Fig 2B**: Plots of SNR vs. 19F atoms per voxel at the two field strengths. **Fig 2C**: 19F MRI of an *ex vivo* EAE mouse brain acquired at 21.1 T and at different spatial resolutions with FLASH images as anatomical reference.

**Acknowledgements**

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**References**

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