What does (multipole) Fourier tensor imaging tell us? – A simulation study

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TARGET AUDIENCE – Researchers interested in MR-based quantification of tissue microstructure through phase-based Fourier spectrum tensor imaging (FTI).

PURPOSE – In a recent paper Liu and Li[1] presented a novel MR phase-based technique to assess tissue anisotropy, in the following referred to as Fourier spectrum Tensor Imaging (FTI). The technique is entirely different from both diffusion tensor imaging (DTI) and the recently introduced susceptibility tensor imaging[2] (STI). FTI obtains information on microstructural anisotropy, such as axonal fibers, in a certain region-of-interest (ROI; e.g. a macroscopic voxel) from the fractional Fourier spectrum that corresponds to the complex-valued MR signal of the spin ensemble inside the ROI (*Fourier sub-spectrum*)[1]. The spatially resolved disentanglement of the ROI's Fourier sub-spectrum and the total *k*-space signal is achieved by applying physical dephasing field gradients prior to a gradient echo signal readout and then analyzing the phase of the signal averaged over the ROI as a function of the strength and direction of these gradients.[1] A fundamental assumption that makes FTI feasible is that the effect of the dephasing gradients can be mimicked numerically by simple *k*-space shifts (*shift assumption*), making the technique entirely post-processing based.[1] **In this contribution we show that the microstructural anisotropy measured by FTI, though being derived from the MR phase, does neither specifically reflect magnetic susceptibility anisotropy (as STI does; e.g. due to myelin) nor does it require an anisotropic distribution of magnetic susceptibility inclusions at all.** We show that FTI-derived anisotropy is a general measure of overall spatial signal anisotropy in the ROI. In other words, FTI-derived anisotropy can also arise from non-susceptibility related effects, such as chemical shift.

METHODS – *Numerical models:* To investigate the dependence of the FTI-derived anisotropy on the underlying tissue architecture we designed several different numerical tissue models. The models generally resembled a sample of the splenium of the corpus callosum: a cube with 1.3 mm side length was filled randomly with parallel axons (diameter=5μm; in *z*-direction) with intact myelin sheaths (thickness=5 μm) on a 5 μm numerical grid (**Figure 1** left). To the most complex model (**model ANISO**) the anisotropic magnetic susceptibility of myelin was assigned ($\chi_\perp$=-0.263 ppm, $\chi_\parallel$=-0.1ppm relative to surrounding water). In **model ISO** the susceptibility of myelin was assumed to be isotropic and equal to -0.1ppm. **Model PATH1** was constructed from model ISO and mimicked partial demyelination (not intact myelin sheaths). A *z*-*y*-slice of this model is shown in **Figure 1** (third from left). **Model PATH2** mimicked complete degradation of neurons (Figure 1 right-most). We calculated for all of these models the field perturbation at 9.4T by fast-forward field computation[3] using the apparent susceptibility[1] and a magnetic field tilt of 50° relative to the axon direction[1]. In **model CHEM** we assumed no susceptibility difference between myelin and water but a chemical exchange effect producing a field shift in the myelin that is invariant of the field direction. The field distributions of all models were converted to complex-valued signals at TE=20 ms assuming homogeneous magnitude. Transforming the models to the *k*-space and discarding the Fourier coefficients exceeding a certain nominal k_{MR} = 1/36 μm (i.e. a 7 times lower resolution than the nominal model resolution of 5 μm) mimicked MR acquisition with a voxel resolution of 36 μm. *FTI processing:* The MR signal matrices corresponding to the different models were zero-padded to increase the numerical spatial domain resolution to 5 μm. The FTI shift vectors (that mimic dephasing gradients G_j in the spatial domain[1]) were chosen as $\boldsymbol{p}=[q\ 0\ 0]^T$ (gradients in *x*-direction) and $\boldsymbol{p}=[0\ 0\ q]^T$ (gradients in *z*-direction) with $-0.5<q/k_{MR}<+0.5$ to visualize the dependence of the FTI-signal on the tissue type (model). For calculating the FTI anisotropy tensors of the models shift vectors (***p***) in 26 different directions were sampled[1]. Overall model size and MR voxel size were not chosen higher (i.e. more realistic) in this work due to numerical limitations. We showed in another contribution to ISMRM 2014 that FTI works independent of the exact settings of these parameters.

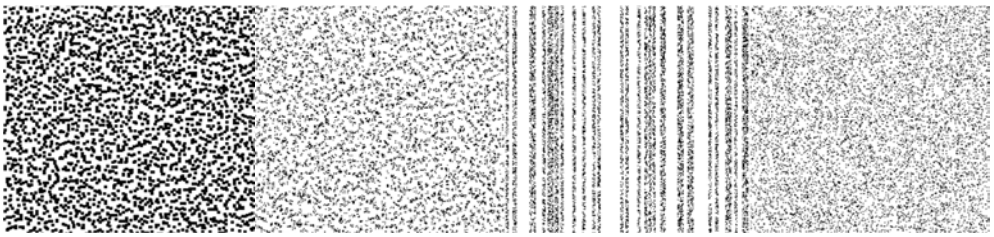

FIGURE 1. Schematic illustration of the corpus callosum tissue models used in this study. From left to right: ANISO/ISO/CHEM (*xy*-slice), PATH1 (*xy*-slice), PATH1 (*zy*-slice), and PATH2 (*zy*-slice).

RESULTS – Figure 2 shows the frequency of the FTI signal in all models for gradients in *x*-direction. All curves showed the typical shape of a quadrupolar FTI signal (cf. Ref. 1, Fig. 2c) with a slightly different scaling depending on the model. **Figure 3** shows the frequency of the FTI signal for gradients in *z*-direction (i.e. in the direction of anisotropy). In this case, FTI signal was zero for all models (as expected, cf. Ref 1, Fig. 2c) except for models PATH1 and PATH2, which mimicked reduced anisotropy in *z*-direction (see above). **Table 1** lists the angle of the tensor main axis relative to the z-direction and the fractional anisotropy (FA) calculated from the Eigenvalues. Though mimicking different biophysical properties models ANISO, ISO and CHEM yielded relatively high FA values (>0.7) and the tensor orientation was aligned with the axons (angle to *z*-axis in the order of 1°). When using the models mimicking pathologies, PATH1 and PATH2, the calculated directions were inaccurate (>39°) and the FA of PATH2 was lower than that of PATH1, reflecting the decreased anisotropy in model PATH2.

DISCUSSION – For gradients perpendicular to the axon direction (*x*-gradients) similar FTI signal characteristics were observed for all models (Fig. 2 left; even for model CHEM,which had no susceptibility variation), demonstrating that FTI is not specific to susceptibility-related anisotropy but sensitive to tissue architecture in general. The observed signal for *z*-gradients with models PATH1 and PATH2 indicate the potential of FTI to detect subtle pathologic tissue changes such as demyelination.

CONCLUSION – The FTI signal reflects the geometric anisotropy of the complex-valued MR signal rather than magnetic susceptibility-related anisotropy. The technique shows potential to identify different degrees of pathologic breakdown of anisotropy such as demyelination. Further work remains to be done in what concerns more complex structures, such as, e.g., crossed fibers.

REFERENCES – [1] Liu C and Li W, 2013. *Neuroimage*. 67:193-202. [2] Liu C, 2010. *Magn Reson Med*. 63(6):1471–7. [3] Marques JP and Bowtell RW, 2005. *Concepts Magn Reson B Magn Reson Eng*, 25B(1):65–78.

model	angle relative to the *z*-axis	FA
ANISO	0.1°	0.71
ISO	0.1°	0.71
CHEM	0.07°	0.73
PATH1	58°	0.42
PATH2	40°	0.41

TABLE 1. Tensor measures derived from the different models. FA is the fractional anisotropy.

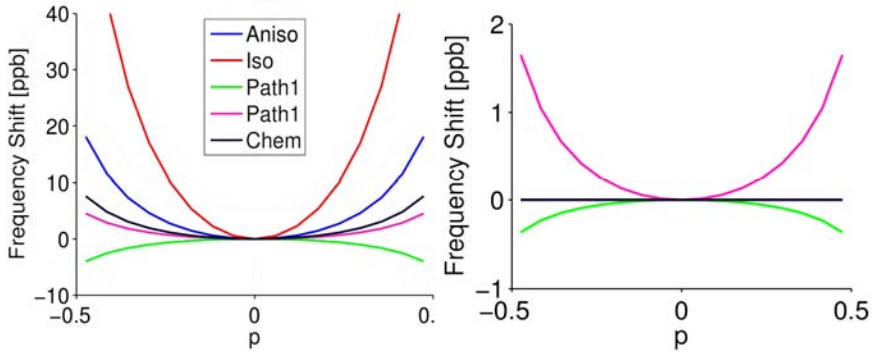

FIGURE 2. FTI frequency shift (in ppb) for gradients in *x*-direction (left) and *z*-direction (right). Note that the ordinates have different scaling.