How water T2 shortening affects ^{1}H Magnetic Resonance Spectroscopy quantification

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Introduction

Iron accumulation has been reported to affect the MR signal [1, 2]. In this study, an *in vitro* correspondence is established between the iron accumulation and the shortening of water T2 relaxation times. Later, *in vivo* relationship between water T2 decay and metabolite quantification was assessed in a cohort of 10 Parkinson's patients.

Methods

Seven spherical phantoms (phantom1 to phantom7) with a 40 mm in diameter were filled with an identical homogeneous aqueous solution mimicking the metabolic properties of a healthy human brain. Phantoms 2 to 7 were doped with an increasing concentration of iron metal nanoparticles solution as shown in Table 1. Additionally, *in vivo* correlation between water T2 times and metabolite concentrations was estimated for a cohort of 10 diagnosed Parkinson's patients (5 males/5 females) aged between 58 and 75 years. All MR images and spectroscopic data were acquired on a clinical 3.0T GE Signa HDx scanner using a single channel quadrature head coil. The spectroscopy protocol consisted of a PRESS acquisition (TR/TE=1500/35 ms). In the *in vitro* experiments a single voxel (SV) with a nominal size of 12x12x12 mm was prescribed at the centre of the phantom, while for the *in vivo* acquisition a SV with a nominal size of 11x25x15 mm was placed in the corpus striatum. All spectroscopy data was subsequently processed off-line. Metabolite quantification was performed with the LCModel software [3].The water T2 imaging protocol consisted of a contiguous coronal T2-weighted Spin Echo pulse sequence using TR=1500 ms and eleven echo times (TE=20, 35, 40, 60, 70, 80, 105, 140, 144, 216 and 288 ms). Signal intensity was recorded from the same spatial localization prescribed during the SV acquisition. T2 maps were then computed after a mono exponential fitting of the Signal Intensity as a function of TE using in-house developed software [4].

	Conc. (mM/l)
phantom1	0
phantom2	2.16
phantom3	6.47
phantom4	10.8
phantom5	15.1
phantom6	21.6
phantom7	25.9

Table 1. Iron concentrations

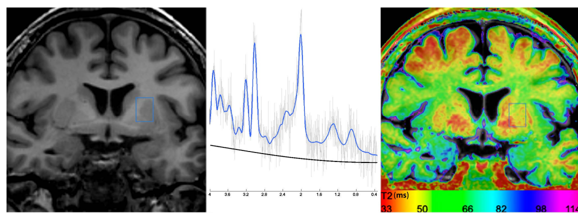

Figure 1.(a) T1, (b) LCModel spectrum, (c) water T2 map for one patient. SV prescription in blue (a) and (c), at striatum

Results

Figure 1 shows the typical single-voxel spectroscopy prescription (rectangular region of interest in blue) in the corpus striatum region from one of the Parkinson patients, registered onto a high-resolution T1-weighted image (1a) and onto a colour-coded water T2 map (1c). The resulting LCModel spectrum is shown in (1b). In the *in vitro* approach, the presence of iron nanoparticles is causing a signal intensity attenuation in the computed water T2-maps (Figure 2, top row), which is also accompanied by noticeable changes in the reconstructed spectra (Figure 2, bottom row). All spectra shared the same amplitude scale. Figure 3 displays plots of water T2 values for each phantom against absolute metabolite concentrations for *in vitro* (3a) and *in vivo* (3b) studies. Correlation coefficients were on average [r]=0.97 and [r]=0.35, for the *in vitro* and *in vivo* cases respectively. Similarly, in Figure 4 water T2 are plotted against metabolite ratios for *in vitro* (4a) and *in vivo* (4b) cases. Their corresponding correlation coefficients were on average [r]=0.85 and [r]=0.27 for the *in vitro* and *in vivo* respectively.

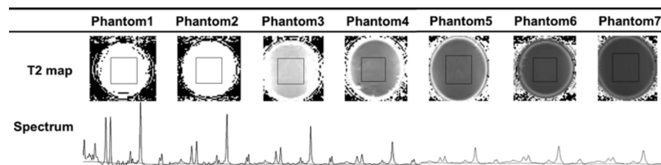

Figure 2. Water T2-maps and corresponding short echo time spectra

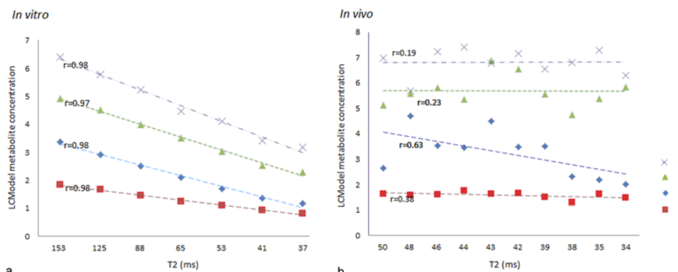

Figure 3. Water T2 values vs. LCModel absolute metabolite concentrations

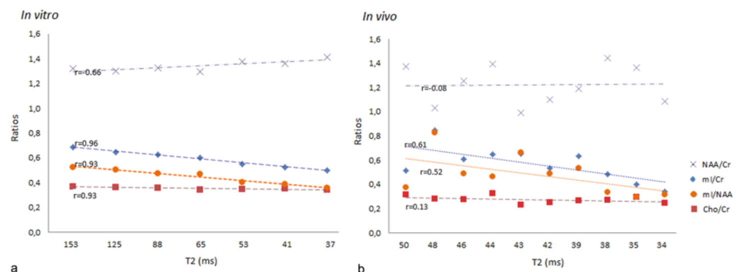

Figure 4. Water T2 values vs. LCModel absolute metabolite ratios

Conclusions

The signal attenuation shown in the computed water T2-maps (Figure 2) is causing an underestimation in the absolute metabolite quantification. This effect is also demonstrated in the observed *in vitro* correlation between water T2 relaxation times and both metabolite concentrations and ratios (Figure 3a and 4a respectively). Results in Figure 3b and 4b indicate a comparable behaviour for the cohort of Parkinson's patients. Although there is a less degree of correlation between water T2 and metabolite concentrations both, *in vitro* and *in vivo*, share similar linear trend. *In vivo* presence of iron deposits or other factors such as microcalcifications and microbleeds affecting the water T2 decay could explain part of the inter-subject metabolite quantification variability reported in the literature. This work suggest the need to introduce the measurement of water T2 relaxation times as a new statistical cofounding variable in those studies involving MRS and elder patients or pathologies with iron deposits, mirocalcifications or microbleeds.

References:

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