Multiparametric characterization of healthy and diseased articular cartilage at 17.6T: Early results

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Introduction: Articular cartilage is a highly structured tissue and plays an important role in the pathogenesis of osteoarthritis (OA), which entails a global socio-economic burden. Therefore there is a need to early diagnose cartilage damage. The T2 relaxation time is a MRI parameter, which has been demonstrated being sensitive to the alterations of the collagen cartilage matrix. Other MRI parameters, such as apparent diffusion coefficient (ADC), fractional anisotropy (FA) and native T1 relaxation time, contain important information complementary to T2, which may help in the assessment of cartilage integrity. Diffusion tensor imaging (DTI) is sensitive to the microscopic movements of water, and thus it is influenced both by the architecture of the collagen matrix and the proteoglycan content [1,2]. Native T1 seems to correlate to cartilage mechanical properties [2]. The purpose of this work was to establish a protocol for multiparametric (T2, T1 and DTI) examination of articular cartilage at 17.6T for the study of healthy and diseased cartilage.

Methods: Healthy (n=1, 34y) and diseased (n=2, 55 and 60 y) excised human patellar cartilage samples harvested at time of autopsy ≤24 hours after death were examined on a 17.6-T MRI scanner with a maximum gradient strength of 1 T/m (Bruker Advance, Bruker Biospin GmbH, Rheinstetten, Germany). MRI protocols include a multiecho TSE sequence (TR/TE=900/5 ms, echo spacing=5 ms, 30 echoes, bandwidth (BW)=138.9 kHz, 16 averages (avg), acquisition time(TA)=30:47 min), a saturation-recovery FLASH sequence (TE=2.56 ms, flip angle=5°,TI=75, 125, 250, 500, 750 1000 and 2000 ms, BW=101.0 kHz, 2 avg, TA=22:49 min) and a diffusion-weighted spin-echo sequence (TR/TE=900/16.0 ms, b-values=0, 500 s/mm², 6 directions, BW=15.0 kHz, 16 avg, TA=3:35 h). All three sequences used the same FOV of 16×16 mm², image matrix of 256×128 (in plane resolution 62.5×125 µm²), and slice thickness of 1.5 mm. Sequence parameters were optimized in order to achieve a signal-to-noise ratio (SNR) greater than 10 at the bone-cartilage interface (BCI). T2 and T1 were calculated with a non-linear fit (Levenberg-Marquardt algorithm) to noise-corrected signal decays. Typical values of T2, T1, ADC and FA as well as SNR values (calculated with the two-region method) were calculated in each cartilage region (radial, transitional, and tangential zone) and in the physiological solution. The distribution of T2, T1, ADC and FA from the BCI to the articular surface (AS) was calculated in healthy and diseased cartilage.

Results: Typical values of T2, T1, ADC and FA as well as SNR are summarized in Table 1. Examples of the parameter maps are shown in Fig. 1. In healthy cartilage the radial zone is characterized by a low constant T2, a slowly increasing ADC and T1, and a rapid decaying large anisotropy (see plot A). In the transitional zone T2 reaches a maximum when most collagen fibers arrange with the orientation of the magic angle, whereas both ADC and T1 remain constant. The tangential zone with collagen fibers arranged parallel to the articular surface, and thus perpendicular to the magnet field, is clearly identifiable by a decrease in T2 and ADC and an increase on FA. Interestingly in healthy cartilage both T1 and ADC showed a very similar behavior.

Table 1. Typical values of healthy cartilage

	Multiecho		SR-FLASH		DTI		
	SNR (TE=5 ms)	T2 (ms)	SNR (TI=1 s)	T1 (ms)	SNR (b0)	ADC (10^{-3}mm²/s)	FA
Phys. sol.	56	45.1±0.7	123	1220±30	232	1.92±0.02	0.04±0.02
Radial z.	17	9.0±1.4	61.0	750±40	11.9	0.70±0.09	0.83±0.09
Transit. z.	56	35.0±1.8	132	920±20	218	1.35±0.03	0.12±0.05
Tangen. z.	40	15.1±1.7	94.4	890±40	270	1.08±0.04	0.26±0.05

Diseased cartilage alters all MR parameters. Both ADC and T2 demonstrate atypically high values in the region of the lesion, whereas the anisotropy markedly decreases. It is worth noting that the area of abnormal diffusion is much larger than the area of perturbed T2, which may indicate a higher sensitivity of DTI to the detection of pathological changes in the cartilage matrix. Plot B represents the distribution of T2, ADC and FA from the BCI to the AS through the lesion. Rapid decay of the FA, higher diffusion and a peak of increased T2 characterize the lesion. Plot C shows the distribution far away from the lesion. The smooth transition of T2 from the low values at the BCI to the values of water at the AS without trace of a tangential zone, and the reduced ADC in the transitional zone compared to the healthy sample may be additional landmarks of loss of cartilage integrity.

Conclusions: In spite of the reduced number of samples included, the early results presented here seem to provide evidence that a multiparametric study of the articular cartilage may contribute to gain insight into the mechanism of pathology in disease as well as to clarify the factors influencing the different MRI parameters. Results at high field strength can also be regarded as a first test bed for future clinical applications of MRI.

References: [1] Filidoro et al. Magn Reson Med. 2005;53:993-8; [2] Deng X et al. Magn Reson Imaging. 2007;25:168-71.

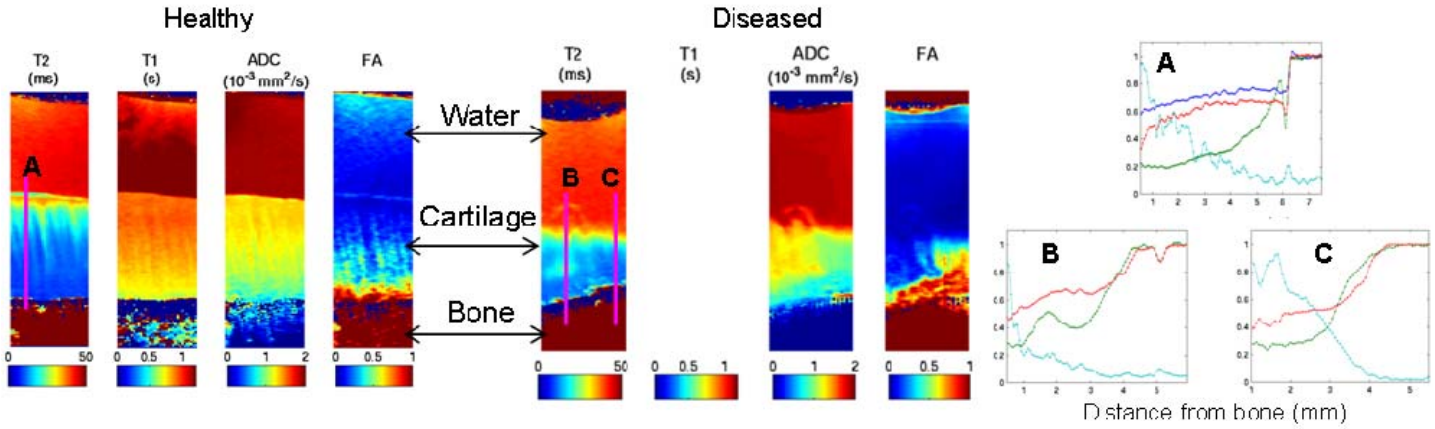

Figure 1: Example of T2, T1, ADC and FA maps on healthy (left) and diseased (right) cartilage. Plots A, B and C represent the distribution of T1 (blue), T2 (green), ADC(red) and FA(cyan) from the BCI to the AC along the magenta lines marked on T2 maps. T1, T2 and ADC are normalized to the values of water (Table 1).