

# Long-term Endurance Running is Associated with Elevated T2 of Superficial Femoral Cartilage

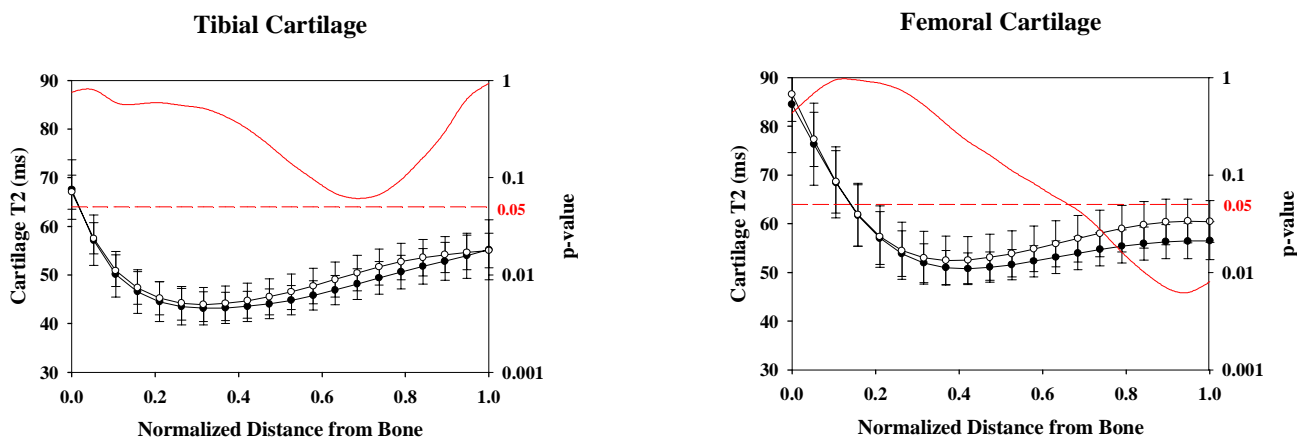
T. J. Mosher<sup>1</sup>, Y. Liu<sup>1</sup>, M. B. Smith<sup>1</sup>

<sup>1</sup>Radiology, Penn State University College of Medicine, Hershey, PA, United States

**Introduction:** Although epidemiology studies suggest high impact loading exercise such as running increases risk for developing osteoarthritis (1), there have been very few animal studies and no published human studies correlating exercise and altered cartilage structure. In addressing this question, MRI can be a useful research tool. Correlation studies have shown that the T2 of cartilage is sensitive to content and anisotropic organization of the type II collagen matrix in cartilage, and thus may be an important image marker for evaluation of early degenerative changes. The purpose of this study is to apply quantitative T2 mapping to test the hypothesis that long-term endurance running is associated with elevated cartilage T2 indicative of damage.

**Methods:** A cross sectional study design was used to compare 19 trained marathon runners (Mean Age: 40.0 years (range: 19-64), Mean Body Mass Index (BMI): 23.1 kg/m<sup>2</sup> (range: 18.7 – 28.8)), and 16 sedentary controls (Mean Age: 40.9 years (range: 21-60), Mean BMI: 25.1 kg/m<sup>2</sup> (range: 19.6 – 29.3)). Quantitative T2 maps were obtained using a Bruker 3T MR spectrometer, a 24 cm gradient insert, and 15 cm linear Litz coil (Doty Scientific). Sagittal T2 maps of the femoral tibial joint were calculated from a 6 section, 12 echo sequence with TR/TE = 1500/9-106 ms, 4 mm section thickness, 384 x 384 matrix and a 12.75 cm field of view (FOV). Cartilage T2 maps and profiles of weight-bearing femoral/tibial cartilage were generated using automated subroutines in CCHIPs/IDL software. Pooled T2 profiles (mean T2 as a function of normalized distance from bone) from each cohort were compared using 1-way ANOVA to determine statistical significance (p < .05).

**Results:** As demonstrated in Figure 1, spatial distribution of T2 in femoral/tibial cartilage T2 was similar for running and sedentary cohorts. Although there was no difference in tibial cartilage T2, runners demonstrated significantly higher T2 in the superficial 30% of weight-bearing femoral cartilage compared to sedentary controls (p < .05).



**Figure 1:** Pooled Cartilage T2 profiles: Mean cartilage T2 ( $\pm$  s.d.) profiles of tibial and weight bearing femoral cartilage for runners ( $\circ$ ) and sedentary controls ( $\bullet$ ). On the right axis, P-value plotted as function of normalized distance ( $\text{---}$ ) refers to 1-way ANOVA comparison of mean cartilage T2 between groups. Threshold level of significance ( $p=0.05$ ) is indicated by ( $\text{---}$ ). There is no statistically significant difference in tibial cartilage T2 between groups. Compared to sedentary controls, runners have longer T2 values ( $p < .05$ ) in the superficial 30% of femoral cartilage.

**Discussion:** These results demonstrate long-term endurance running is associated with increased T2 values of superficial femoral cartilage similar to that previously reported with aging (2). Histological studies in dogs suggest long term, high endurance running damages the superficial collagen matrix and produces loss of superficial collagen anisotropy on polarized light microscopy (3). Given the strong inverse correlation between cartilage T2 and collagen anisotropy (4), the elevated femoral cartilage T2 in marathoners is likely due to lower fiber anisotropy and suggests running is associated with reorganization/disorganization of the superficial collagen matrix. The ability to monitor these changes *in vivo* provides the unique opportunity to study important co-variables, such as age, body habitus, and form of exercise as they modulate the effect of exercise on cartilage structure.

## References:

1. Felson DT, Zhang Y, Hannan MT, et al. Arthritis Rheum 1997; 40:728-733.
2. Mosher TJ, Liu Y, Yang QX, et al. Arthritis Rheum 2004; 50:2820.
3. Arokoski JP, Hyttinen MM, Lapvetelainen T, et al. Ann Rheum Dis 1996; 55:253-264.
4. Nieminen MT, Rieppo J, Toyras J, et al. Magn Reson Med 2001; 46:487-493.

**Acknowledgements:** Research support provided through grants from the Arthritis Foundation, NIH/NIAMS, and NIH/NCRR