

# Prolongation of T2 Stratification after Microfracture Does Not Indicate Normal Cartilage

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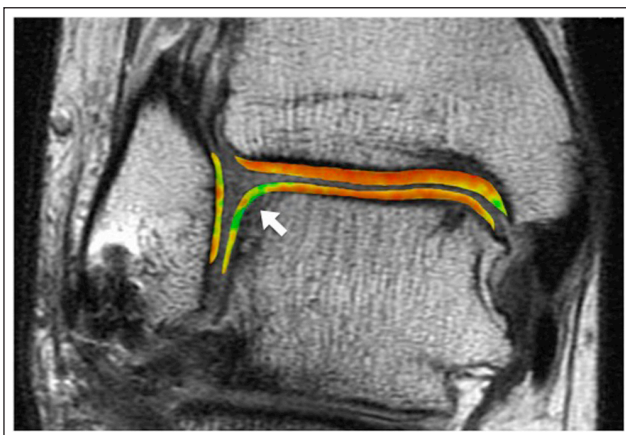
Dear Editor,

We read with interest the recent article by Domayer *et al.* titled "Microfracture in the Ankle: Clinical Results and MRI with T2-Mapping at 3.0 T after 1 to 8 Years" (Cartilage Online First, October 18, 2010). The authors concluded that microfracture surgery as a means of treatment for osteochondral lesions of the talus can provide a repair tissue that is similar to that of "adjacent" cartilage when assessed with a relaxation time analysis via quantitative T2-mapping MRI.

The inferior biological and mechanical properties of fibrocartilage are well recognized throughout the literature.<sup>1-5</sup> Normal articular cartilage is composed primarily of type II collagen, whereas the differentiation of fibrocartilage results in an increased type I collagen composition. We were therefore surprised to learn that the fibrocartilage infill reported in the analysis by Domayer *et al.* resembled normal cartilage.

In our experience, the T2 values of fibrocartilage infill following microfracture never approach the stratification of normal cartilage. In fact, the T2 values are typically diffuse and prolonged, thereby reflecting poor collagen fiber architecture within the repair cartilage (**Fig. 1**).

We must emphasize that a single T2 measurement within the respective region of interest should not be considered adequate in accurately quantifying the cartilage repair tissue.



**Figure 1.** Coronal quantitative T2-map performed 24 months following microfracture to the lateral aspect of the talar dome demonstrates prolongation of T2 values without color stratification at the site of cartilage repair.

Standardized regions of interest for quantitative imaging techniques such as T2-mapping should include multiple standardized measurements of the repair tissue (i.e., the central aspect of the defect, the peripheral defect, the interface between the repair and host tissues, the area adjacent to the defect, and a control value that is remote from the defect). These measurements should also be taken in both the deep and superficial halves of repair cartilage. Interestingly, a significant area of prolongation within the repair tissue over the defect is evident in Figure 3A, but it is impossible to know whether or not this was taken into account in the authors' analysis without a standardized protocol with multiple measurements.

We stress that the quantification of repair cartilage following microfracture surgery for osteochondral lesions of the talus must be evaluated further before definitive conclusions can be drawn.

Sincerely,

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and John G. Kennedy

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