Relaxation rate enhancement from 1.5T to 3T in iron-loaded organs

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Target Audience Clinicians and engineers interested in imaging and quantifying the amount of iron in iron loaded tissues at 3.0 Tesla.

Purpose The use of MRI for tissue iron quantification is an essential component of clinical management of chronically transfused patients. This non-invasive technique was calibrated and developed at 1.5 Tesla over a decade ago and has become the gold standard for quantifying iron in the body. However, today 1/3 of all new MRI installations are 3T machines, and it is predicted that by 2016 the majority of new installations will be 3T magnets. Institutions that only have access to 3T machines will not be able to quantify and track iron overload in their patients, thus hindering clinical care. It is essential to extend the knowledge and calibration of assessing iron overload in a carefully controlled manner at 3 Tesla. We hypothesized that R2* at 3 Tesla in the liver, spleen, kidney, pancreas and heart would be approximately double the values observed at 1.5 Tesla, independent of the organ studied.

Methods 16 patients (9 male, 7 female, mean age 19.9 +/- 7.0) participated in the study. Patients had thalassemia syndromes (n=10), sickle cell disease (n = 5), or other rare anemias (n = 1). All patients underwent routine clinical evaluation of tissue iron at 1.5T (Philips Achieva, 8 element torso coil, system 3.2.1) per standard protocols and a subsequent MRI at 3.0T (Achieva, 16 element torso coil, system 3.2.1) for research purposes; exams were performed within 3 weeks of one another. Liver and spleen R2* (1.5T) were measured using a multiple echo gradient sequence with the following parameters: 3 slices, field of view 30 – 40 cm, 84 x 84 matrix, slice thickness 10 mm, repetition time 50 ms, bandwidth 3840 Hz/pixel, and 16 echoes equally spaced between 0.98 and 12 ms; at 3T, the TR was 80 ms, bandwidth 4419 Hz/pixel, and echoes were spaced from 0.76 – 8.8 ms. Pancreas and kidney R2* were measured using a similar protocol, but slice thickness was decreased to 6 mm, matrix increased to 96 x 96 pixels, and repetition time reduced to 30 ms to improve coverage. R2* was calculated using a 3-component model (exponential plus offset) and Levenburg-Marquadt fitting algorithm on a psuedopixelwise (adaptive binning) basis [1].

Results Figure 1 (left side) demonstrates a highly linear relationship between R2* at 3.0T and 1.5T for liver (r^2=0.98 p<0.0001). The slope is 1.98. The line of best fit (solid line) is nearly identical to previously published results from, Storey et al, 2007 (dotted line). Figure 1 (right side) compares the R2* in the spleen, kidney, pancreas and heart at the two field strengths. A logarithmic scale is used to better visual the differences at low iron concentrations in the tissues. Similar to the relationship shown in the liver, there is a linear correlation of R2* for 1.5T and 3T. There is an increase in scatter for R2* values below 50 Hz, as iron concentration decreases and has a lesser effect on the measured R2* value.

Discussion Previously, 3T-1.5T liver and heart cross validation of R2* was performed in a small thalassemia cohort [2] and later validated using Monte-Carlo modeling [3]. Our present cohort includes a larger, ethnically diverse patient population from multiple iron overload states. We confirmed the findings of Storey, et al, and found a strikingly similar relationship for spleen, kidney, pancreas and heart. In fact, we postulate that organ R2* values at 3T can be entirely predicted from values measured at 1.5T using a relaxivity enhancement factor of two and knowledge of organ R2* values at both field strengths collected from non-iron overloaded subjects. That is

$$R2^*_{3.0} = 2\left(R2^*_{1.5} - R2'_{1,5}\right) + R2'_{3.0}$$

where R2′ values indicate population means derived from non iron overloaded subjects. Note, it is necessary to use population mean values rather than intercepts from R2* - iron calibration curves (intrinsic R2* values) because true calibration curves have not been derived outside of the liver and the heart.

Conclusion These results suggest that R2* risk thresholds established at 1.5T can be systematically transferred to 3T for any organ. We are currently testing this hypothesis in a prospective manner.

References [1] Meloni et al, MRI 31(7):1074-80, 2013 [2] Storey, et al, JMRI 25(3):540-7, 2007 [3] [1]N. R. Ghugre, et. Al. MRM. Sep. 2014.

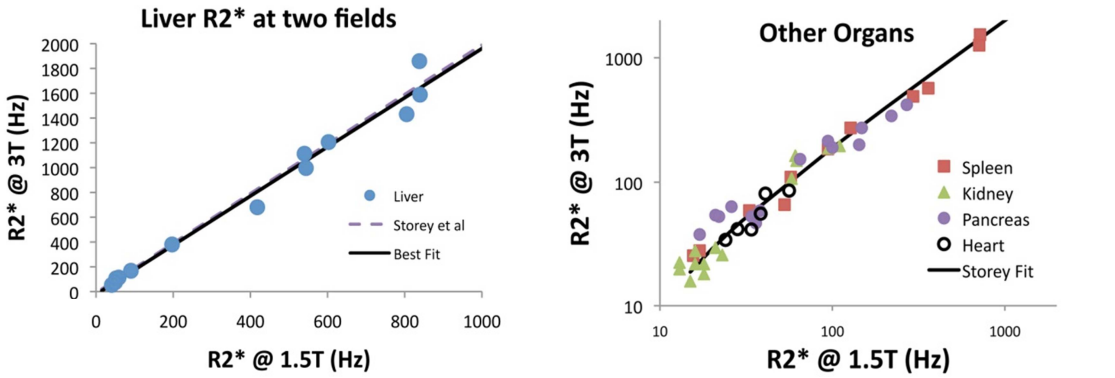

Figure 1 - Liver R2* at 1.5T and 3T (left) show a linear relationship with a slope of 2, matching predicitons in [2]. R2* relationships in other organs [right] follow the same linear relationship.