R2* estimation in the presence of fat and macroscopic B0 field variations

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Introduction: Quantitative, non-invasive estimation of R_2* is an important biomarker of hepatic iron overload. Unfortunately, estimates of R_2* may be confounded by the presence of fat, as well as the signal dephasing that occurs from macroscopic magnetic field gradients caused by external susceptibility. This can lead to systematic error in the R_2* maps [1-3], potentially complicating the detection and quantification of iron overload. In this work, we extend complex chemical shift based fat-water imaging methods that provide simultaneous, unconfounded estimates of water, fat, R_2* and Bo field maps. Using the Bo field maps, estimates of R_2* that are corrected for macroscopic susceptibility can be achieved.

Theory: The complex-valued signal in a multi-echo SPGR acquisition can be modeled as follows, including R_2* decay [4,5], multi-peak fat signal [4], and macroscopic field effects [1-3]:

$$s_{model}(TE;W,F,R_2^*,f_B) = (W + F\, c_F(TE))\exp[-R_2^*\, TE]\, \exp[i2\pi f_B TE]\, h(TE), \qquad (1)$$

$$c_F(TE) = \sum_m \alpha_m \exp(j2\pi f_m TE), \qquad (2)$$

where W and F are the water and fat signal amplitudes, respectively, $c_F(TE)$ is the multi-peak fat signal model (fat peaks with frequencies f_m and relative amplitudes α_m, respectively) [4], $R_2^* = 1/T_2^*$, f_B is the local frequency offset due to B_0 field inhomogeneity, and $h(TE)$ accounts for the additional signal decay caused by macroscopic B_0 variation within each voxel. The additional decay $h(TE)$ is generally non-exponential, and is a confounding factor for R_2* estimation (typically leading to overestimation of R_2* if not accounted for). Assuming constant signal amplitude and linear B_0 variation (gradient $\vec{g}_B$) over the voxel (with spatial response function $SRF(\vec{r})$), $h(TE)$ can be expressed as:

$$h(TE) = \int SRF(\vec{r}) \exp[i2\pi \vec{g}_B \cdot \vec{r}]\, d\vec{r} \qquad (3)$$

where $SRF(\vec{r})$ can be approximated as a *rect* function in the slice direction for 2D experiments (in which case the typically dominant through-slice decay is a *sinc* function), and a *sinc*-like function in 3D experiments (in which case the decay can be approximated numerically by integrating over the main lobe of the *sinc*). An initial B_0 map estimate obtained (from the same data) using the standard fat-water signal model (without $h(TE)$) is used to calculate the gradient $\vec{g}_B$ needed for the B_0-corrected model.

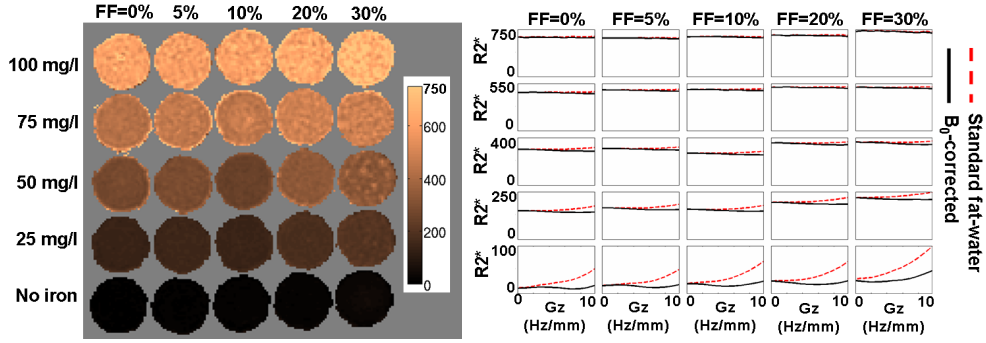

Figure 1: (Left) Phantom R_2* map obtained with the proposed method, for G_z=0Hz/mm. The R_2* values vary from ~20 s^{-1} in the no-iron vials up to 700 s^{-1} in the 100 mg/l iron vials. (Right) Mean R_2* in each of the vials for varying G_z, using the standard model and the B_0-corrected method. The presence of a B_0 gradient can lead to severe overestimation of R_2* (bias of nearly 60 s^{-1}), particularly in the low iron vials. Correction for B_0 gradients results in stable estimates over the measured range of field gradients.

Experiments: An oil-water-iron phantom was built as in Ref. [6], with fat-fractions (FFs) of 0, 5, 10, 20 and 30%, and SPIO (Ferridex, Bayer Inc., Wayne, NJ) concentrations of 0, 25, 50, 75 and 100 mg/l. Phantom data were acquired at 1.5T using an investigational version of a 3D multi-echo SPGR "IDEAL" sequence, with FA=10°, slice thickness 4mm and 15 echoes (TE_{min}=1.3ms and ΔTE=0.7ms, obtained in three interleaved "shots"). The phantom vials were positioned parallel to the B_0 field, and 11 axial datasets were obtained by intentionally varying the shim gradient along the "z" direction over a range of 0-10 Hz/mm, in order to generate controlled macroscopic magnetic field gradients. Additionally, liver data were acquired in patients with fatty liver disease, in accordance with our Institutional Review Board, using a 3D SPGR IDEAL sequence with FA=5°, slice thickness 10mm and 6 echoes (TE_{min}=1.20ms, ΔTE=2.00ms).

Results and Discussion: Figures 1 and 2 show R_2* estimation results from a water-oil-iron phantom and liver acquisition, respectively. As the B_0 gradient increases, the standard fat-water model (including multi-peak fat and R_2*, but no background B_0 variation) results in severe overestimation of R_2* (up to ~60 s^{-1} in the phantom and 20 s^{-1} in the liver). The B_0-corrected method is able to largely remove this overestimation.

A limitation of the proposed method is that it uses a locally linear model for B_0 variations. In regions of very severe susceptibility-induced field variation (with significant higher order terms in the B_0 field variation), it is still advantageous to acquire thinner slices, which result in reduced susceptibility effects and allow better approximation by a locally linear B_0.

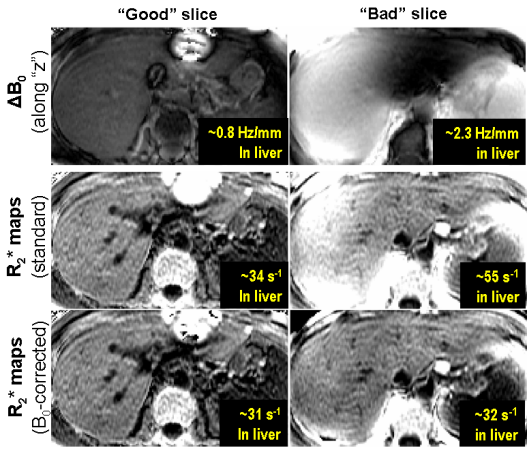

Figure 2: *In vivo* liver results on two slices (one with rapid B_0 variation). The standard fat-water signal model results in severe bias in regions of rapid B_0 variation, which is largely removed by the B_0-corrected model. Fat-fraction maps (not shown) remained largely unaffected, showing ~4% fat fraction in the liver for both slices and both signal models.

Conclusion: Improved mapping of R_2* in the liver can be achieved by correcting for confounding factors, including macroscopic B_0 variations and the presence of fat. The proposed method uses the complex signals to estimate and correct for macroscopic B_0 variations.

References: [1] Fernandez-Seara et al, MRM 2000;44:358-366. [2] Wild et al, MRM 2002: 48:867-876. [3] Dahnke et al, MRM 2005;53:1202-1206. [4] Yu et al, MRM 2008;60:1122–1134. [5] Bydder et al, 2008, MRI 2008;26:347-359. [6] Hines CDG et al, JMRI 2009;30:1215-1222.

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