IMPACT OF T2* DECAY ON THE QUANTIFICATION OF HEPATIC STEATOSIS WITH MRI

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Introduction: Fat quantification using chemical shift based water fat separation techniques may reduce or ultimately eliminate the need for liver biopsy by quantifying hepatic steatosis, an important feature of chronic liver diseases such as non-alcoholic fatty liver disease (NAFLD). Most signal models for chemical shift separation of water and fat only consider a single, discrete resonant peak for each chemical species [1-5]. However, signal decay caused by T_2^* will cause line-width broadening that may corrupt attempts to quantify hepatic fat content [6][8-9]. Unfortunately, up to 40% of patients with NAFLD may have coexisting iron overload [7], which will cause additional T_2^* shortening. The purpose of this work is to explore the effects of line-width, that arises due to T_2^* decay, on the quantification of fat using 3-pt IDEAL water-fat separation technique, considering variable line-widths for water and fat.

Theory and Methods: The spectral equation of a specimen is the sum of individual spectral equations of all the different chemical species present in the specimen. If ρ_m is the area and f_m the central resonant frequency of the spectra of a chemical species m ($m=1,\ldots, M$), the spectral equation of the species is given by $\rho_m P_m(f - f_m)$. Here $P_m(f)$ is the point spread function (PSF) defining the line-width (caused by T_2^* decay) and shape of the chemical species. In the frequency domain the spectral signal is

$$S(f) = \sum_{m=0}^{M-1} \rho_m\left(\delta(f-(f_m+\psi))\otimes P_m(f)\right) \qquad (1)$$

and

$$s(t) = \exp(2\pi i\psi t)\sum_{m=0}^{M-1} \rho_m \exp(2\pi i f_m t) p_m(t) \qquad (2)$$

is the corresponding signal equation in the time domain, where ψ is the shift (Hz) of the entire spectrum caused by a local Bo field inhomogeneities If N echoes images are acquired at echo times of t_1 t_2 ….t_N, the resulting equations can be written in the matrix form as $\mathbf{S=D(\psi)A\Gamma}$, where $\mathbf{D(\psi)}$ is a $N\times N$ diagonal matrix dependent only on $\exp(2\pi i\psi t)$. $\mathbf{A}$ is $N\times M$ matrix dependent on the central resonant frequency and the PSF of the different species, while $\mathbf{\Gamma}=[\rho_1\ \rho_2 \ldots \rho_M]^T$ is the vector to be estimated, containing the signal for the M species. We define $\mathbf{A_b}$ as the matrix that arises if line-width is not considered, and thus the associated estimated quantities of the species $\mathbf{\Gamma_b = (A_b^H A_b)^{-1} A_b^H A\Gamma}$. The line-width arising from T_2^* decay corresponds to the full width at half maximum of a Lorentzian. Simulations are conducted to find the absolute percentage errors in quantification of fat, water and fat fraction that arise if line-width is not considered, at true fat fractions ranging from 0 to 0.4. The echoes times of 1.98, 3.57 and 5.15ms are chosen to produce optimal echo combination at 1.5T, corresponding to phase shifts between water and fat of $5\pi/6$, $3\pi/2$, $13\pi/6$ that maximizes SNR perfomance[5].

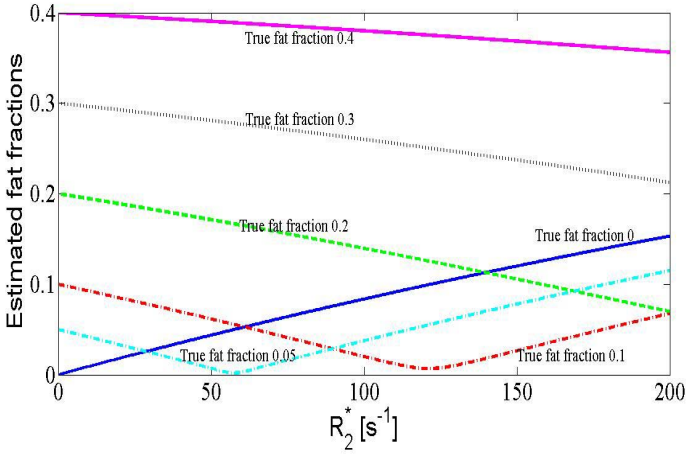

Figure 1: *Estimated fat fractions at different true fat fraction, 0, 0.05, 0.1, 0.2, 0.3 and 0.4 when R_2^* of water and fat are equal. It can be observed at R_2^* =0 (T_2^* very high) the estimated values of fat fraction are equal to the*

Results: The absolute percentage errors that arise from ignoring T_2^* decay when quantifying fat are computed with R_2^* (=$1/T_2^*$) ranging from 0 to 200 s^{-1} for both water and fat. The apparent fat-fraction for a range of R_2^* values 0-200s^{-1} was calculated for different true fat-fractions of 0-0.4 and plotted in figure 1. Over this range of R_2^* values, absolute errors in fat-fraction reached approximately 20%, a very large discrepancy from true fat-fraction. The error in fat fraction becomes less than 1%when $T_2^* \geq (R_2^* \leq 11\ s^{-1})$. To maintain an error of less than 5%, correction for T_2^* decay must be performed when R_2^* is less than 60s^{-1}, corresponding to a T_2^* >17ms, which is typically the case in normal livers (T_2^*=25-30ms). However, significant T_2^* shortening can occur in the presence of iron overload, which commonly occurs in NAFLD [6, 7].

Discussion: In this work, we have explored the impact of T_2^* decay and the resulting line-width broadening, and its impact on fat-fraction estimation. Several assumptions have been made, including the fact that the field inhomogeneity map, ψ, can be estimated accurately even in the presence of T_2^* decay. We have also assumed in these plots that T_2^* of water and fat are equal, although the equations described in this work can account for differences in T_2^* between these two species. To the first order, and to demonstrate the importance of T_2^* effects, these differences are minor.
Our results also indicate that an absolute error less than 5% should be easily achievable when iron overload is not present. However, an error of 5% may be unacceptably high for applications that attempt to detect and quantify steatosis early in disease. In such cases, correction for T_2^* decay must be made to reduce the error from signal loss to a minimum. Our simulations also indicate that the effects of T_2^* decay can only be ignored when T_2^* values of tissue are very long. For accurate quantification of fat in diseased states, particularly in the presence of iron overload, the effects of T_2^* must be considered and decoupled from the estimation of hepatic fat content.

References: [1]. Glover et al, MRM 1991;18:371-383. [2]. Xiang et al, JMRI 1997;7:1002-1015. [3]. Ma et al, MRM 2004;52:415-419. [4]. Reeder et al. MRM. 2004; 51:35-45. [5]. Reeder et al. MRM. 2005; 54(3):636-644. [6]. Yu et al. JMRI. 2007; 26(4): 1153-1161. [7]. George DK et al, Gastroenterology 1998; 114(2): 311-318. [8] Yokoo et al, ISMRM. 2007;1720. [9] Bydder et al, ISMRM 2006:2298. [10] Westwood et al, JMRI 2003;18(1):33-39.

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