Measuring T2 in the liver. A comparison between 1H spectroscopy and SE-EPI

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Introduction
There has been much recent interest in using MRI and MRS to replace liver biopsy to determine liver fibrosis [1], liver iron content [2] and liver fat fraction [3]. An important parameter is the transverse relaxation time, T2; it can be combined with T1* to estimate iron content and is used in correcting 1H spectra for T2 decay when calculating fat fractions [4,5]. It has also been suggested as a parameter for staging liver fibrosis [6,7]. Currently 1H liver spectra are corrected using literature T2 values taken from MRI studies of bulk tissue, averaged over many individuals [8]. However, if fibrosis and iron content alter the tissue T2 then it will be necessary to correct clinical spectra on an individual basis. The aim of this study was to compare liver tissue water T2 measured using 1H spectroscopy with that measured using Spin-Echo (SE) EPI T2 mapping to determine whether a small number of echo times can be used for the T2 decay correction in MRS, and to investigate the variation of T2 in patients with chronic liver disease.

Methods
The study was approved by the local NHS Ethics Committee and all patients gave written, informed consent. Patients (n=18, 13 male) with chronic liver disease confirmed by liver biopsy were scanned on a single occasion using a 1.5 T Philips Achieva scanner with body transmit and 5-element SENSE cardiac coil. MRS 1H spectra were acquired from a 30x30x30 mm3 voxel (positioned to minimise contributions from large blood vessels) using a varying TPE PRESS sequence (90°-TE1=10 ms-TE2=180°-TE3=10 ms). MRI T2 maps were generated from SE-EPI data (9 slices, 3x3x8 mm3 voxels, 4mm slice gap, 96x96 image matrix, SENSE 2, SPIR fat saturation). Data were respiratory triggered and acquired during the expiration phase of the breathing cycle (minimum TR 3 s). 3 volumes were acquired at each of 6 TEs (27, 35, 42, 50, 60, 70 ms). Phantom data was also acquired from a 4 quadrant Gd-DTPA doped agar gel phantom which had T2 values of 35-90 ms, and T1 values of 100-700 ms, across the quadrants. Data were acquired using the same protocol as for the liver patients using simulated respiratory triggering to give a TR of 6 s.

Data Analysis
Spectroscopy: All spectra were individually phase- and frequency-offset corrected before averaging spectra from the same TE. Areas under the water peak (and CH2 fat peak if in-vivo data) were calculated in Matlab (Mathworks Inc, Natick, Mass.). T2 was then calculated from the linear least squares fit of ln(area under peak) vs. TE. Imaging: If respiratory triggering was poor, some through-plane misalignment between slices occurred and these volumes were discarded from the analysis. A mask was drawn around the liver region in a single TE volume (9 slices). All voxels from the SE-EPI data set within this mask were then fitted for T2 using a weighted least squares fit, using 1/TE as the weighting factor to generate a 9 slice T2 liver map. A histogram of the T2 maps was then generated and the peak histogram value (which excludes most blood vessels), and mean T2 value of the whole liver region (excluding blood vessels) were then calculated.

Results and Discussion
All patients tolerated the examination well as all the data was acquired under respiratory triggering. Water T2 measured in liver tissue using SE-EPI and PRESS are shown in figure 1. There is good correlation between MRI data (including vessels) and MRS T2 data, whereas the MRS systematically measured a longer T2 compared to the peak (mode) histogram T2 which reflects solely tissue T2. There is also significant variation between subjects which may be a result of iron deposition or fibrosis, or a combination of both. However, we are currently blinded to the liver biopsy data and can draw no conclusions about this spread in the data at present. The phantom data (figure 2) showed good correlation between MRI and MRS (R=0.9993); good agreement was found between imaging and spectroscopy for short T2 (<50 ms; range similar to liver tissue), however, as T2 increases there is a trend for the spectroscopy to measure T2 shorter than imaging. Therefore, the in vivo slight increase in T2 measured from spectroscopy is most likely due to vessel contamination of the voxel (as reflected by the closer agreement when vessels are included in the estimation of the T2 parameter). Although every effort was taken to place the spectroscopy voxel away from large blood vessels, the large voxel size of MRS, 30x30x30 mm3 will result in the ‘tissue’ sample containing some blood vessels which have a corresponding long T2 (50-80 ms) which may explain the longer T2 measured when comparing the MRS T2 with the peak (but not mean) of the MRI T2 histogram. The main limitation of this study is that we are unable to compare the T2 measurements for exactly the same tissue regions as the imaging technique has slice gaps of 4 mm (to cover the whole liver region in 9 slices) and some blood vessels in the T2 maps appear bright and others dark depending on the orientation of the vessel in the slice. The close correspondence between MRS and MRI also suggests that 4 echo times are sufficient for measuring T2 using spectroscopy when calculating individual T2-corrected fat fractions [4].

Conclusions
There is considerable variation in T2 of the liver of patients with chronic liver disease and therefore liver fat fractions measured using MRS require individual T2 decay corrections. There is good agreement between liver water T2 measured using MRS data acquired at 4 echo times and SE-EPI data, suggesting that T2 can be accurately assessed from multiple TE MRS data (for MRS T2 decay correction) using just 4 echo times.

References

Acknowledgements
This work was funded by a Strategic Funding Initiative from the Biomedical Research Committee at the University of Nottingham.

Figure 1. Chronic liver disease patient data comparing T2 measurements made using MRI and MRS. Dashed line is line of identity.

Figure 2. Phantom data comparing T2 measurements made with MRI and MRS. Solid line is line of identity.