

³¹P MRSI of the healthy liver at 3T and 7T with AMESING-boosted SNR

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Purpose: As more people become infected with viral hepatitis or develop non-alcoholic steatohepatitis (NASH), there is a growing need for accurate and non-invasive means of quantifying fibrosis and of identifying the presence of inflammation. Several methods have been proposed, including serum assays, transient and MR elastography and ¹H- and ³¹P-MR Spectroscopy [1-3]. Especially the phosphomonoester/phosphodiester (PME/PDE) ratio and nicotinamide adenine dinucleotide phosphate (NADPH) over PME+PDE ratio may be of use for fibrosis staging and inflammation detection [4]. While most techniques applied thus far were based on single voxel acquisitions, we aim for incorporating spatial information in determining metabolite concentrations throughout the liver. As more intrinsic SNR is required for the relatively small voxels in MRSI, we investigate the gain in SNR when comparing a standard ³¹P liver setup at 3T with an SNR optimized setup at 7T. Moreover, the recently developed adiabatic multi-echo spectroscopic imaging sequence with spherical k-space sampling (AMESING) was applied to increase SNR even further [5] in the liver at 7T.

Methods: Five healthy volunteers were scanned with a 2D CSI adiabatic pulse acquire sequence in a fasting state at 3T and 7T. At 7T, 5 adiabatic refocusing pulses were added (i.e. AMESING). Data (1 FID and 5 echoes) were acquired in a 2D-CSI grid of 10×32 voxels of 10×10 mm with TR = 6s, ΔTE = 40 ms (7T) for a total acquisition time of 6:06 minutes per scan. Data were spatially Hamming filtered, apodized and zero-filled to 8192 datapoints. Phase (0th and 1st order for FID, 0th for echo) and baseline corrections were applied. SNR differences between 3T and 7T were assessed on exclusively the FID data, while the SNR gain of AMESING over only acquiring an FID at 7T was determined using the T2-weighted averaged sum spectra. SNR gains were calculated for the PDEs glycerol-3-phosphorylcholine (GPC) and glycerol-3-phosphorylethanolamine (GPE), the PMEs phosphorylcholine (PC) and phosphorylethanolamine (PE) and NADPH after T2* correction (using apodization with the minimum linewidth).

Results: The SNR gain of 7T over 3T ranged between 1.4 and 3.1 depending on metabolite, while applying the AMESING technique increased SNR further by 1.2 up to 1.6. Figs. 1A₁-B₁ portray the FIDs at 3T and 7T in a single volunteer and Fig. 1B₂ the corresponding 7T T2-weighted average sum spectrum. T2 values were calculated for 7T only and are given along with SNR values in table 1. SNR gain of AMESING for NADPH was calculated with a SNR weighting as T2 could not be calculated. Figures 2A-D show the mean signal intensities for the PMEs and PDEs at 7T versus TE. Importantly, at both 3T and 7T the NADPH peak was distinguishable from the α-ATP peak in the FID.

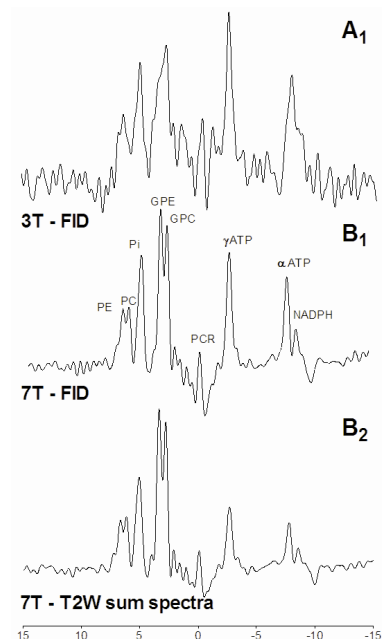


Fig. 1A-B. FID ³¹P MR Spectra at 3T and 7T (A₁,B₁) and corresponding GPC T2 weighted average sum spectrum (B₂).

	3T-FID	7T-FID	7T-T2W	T2 (ms)
GPC	21 ± 5	62 ± 22	100 ± 32	68.2 ± 5.1
GPE	22 ± 4	68 ± 20	106 ± 31	71.2 ± 4.5
Pi	20 ± 3	48 ± 18	62 ± 23	42.7 ± 8.2
PC	20 ± 4	31 ± 6	43 ± 11	51.1 ± 8.0
PE	20 ± 22	33 ± 4	41 ± 5	36.9 ± 5.1
NADPH	13 ± 6	16 ± 6	19 ± 7	-

Table 1. Mean SNR and T2 (ms) in 5 healthy volunteers

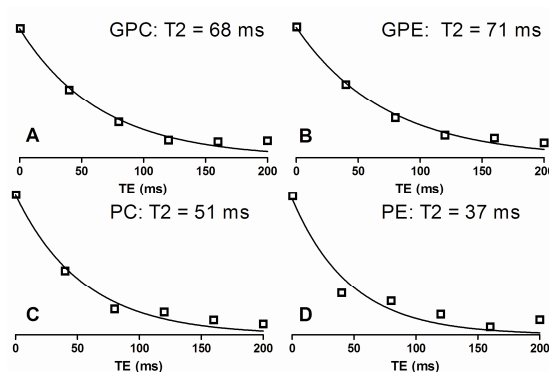


Fig. 2A-D. Signal decay versus TE for PDEs (2A-B) and PMEs (2C-D).

Discussion and Conclusion: A 1.3 to 3.1-fold SNR gain (statistically significant ($P < 0.05$) for all but NADPH) was observed when comparing a standard 3T ³¹P setup with a SNR optimized 7T setup, which can mostly be explained by the differences in field-strength (2.3), quadrature versus linear coil (1.4) and reduced T1 of ³¹P spins at higher fields. 2D AMESING can be used for localized adiabatic acquisition of ³¹P signal at 3T and 7T, while the addition of later echoes allows SNR boost and liver T2 calculations at 7T in a single measurement. Here we have demonstrated that 2D AMESING at 7T of the liver can increase SNR over conventional 3T setups and be used to map phospholipid and NADPH concentrations throughout the liver, which could play a role in the non-invasive separation of patients with inflammation or fibrosis from those without.

References:

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