

Fat suppression for 1H MRSI at 7T using a spectrally selective adiabatic inversion pulse

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Introduction: ¹H Magnetic resonance spectroscopic imaging (¹H MRSI) is a useful technique for measuring metabolite levels in the brain. Some of the main metabolites of interest are Choline (Cho), Creatine (Cr) and N-Acetyl-Aspartate (NAA). ¹H MRSI at higher fields, such as 7T, offers the advantages of increased SNR and spectral separation. However, it suffers signal losses from increased B₁ inhomogeneity (40% B₁ variation from center to periphery of head at 7T), B₀ inhomogeneity and chemical shift localization (CSL) errors. These factors place the added requirements of B₁-immunity, robustness to B₀ shifts and conservation of metabolite signal on any 7T fat-suppression scheme. One technique available to suppress fat is the use of an inversion pulse before the 90° excitation pulse in a PRESS sequence so that species with short T₁'s such as fat pass through their null when excitation occurs [1]. Metabolites have longer T₁'s than fat resulting in a partial loss on the order of 50% of metabolite signal at 7T. However, the increased spectral separation at 7T makes it possible to design a spectrally selective adiabatic inversion pulse that only inverts lipid frequencies, leaving most of the metabolites untouched. Such a pulse was designed and inserted into a standard PRESS sequence.

Method: An adiabatic hyperbolic secant pulse with a bandwidth (BW) of 573 Hz and duration of 30 ms was designed. The pulse duration had to be long enough to achieve transition bands narrow enough to avoid inverting NAA while staying below the RF peak amplitude limit for our 7T magnet (approx 17 μT). The pulse BW had to be large enough to invert a range of lipid frequencies as well as those shifted due to B₀ inhomogeneity. Since the pulse is not spatially selective, it is not subject to CSL errors. At 7T, NAA (2.0 ppm) and lipids (1.3 ppm) are separated by 210 Hz. The final pulse we designed has a transition width of approx 110 Hz. Thus with this pulse design, it is possible to selectively and adiabatically invert fat without affecting NAA. The inversion pulse was inserted into a standard PRESS sequence right before the CHESS water suppression pulses. The pulse was overdriven by a factor of 2.8. This is more than sufficient to invert lipids in the presence of the 40% B₁ variation measured at our 7T magnet. Optimum suppression was achieved when the inversion pulse was placed 300 ms before the 90° pulse. Figure 1 shows A) the adiabatic selective 180° inversion pulse, B) the spectral profile for the inversion pulse, and C) the gradient and RF waveforms for the final PRESS sequence with the adiabatic fat-selective inversion pulse.

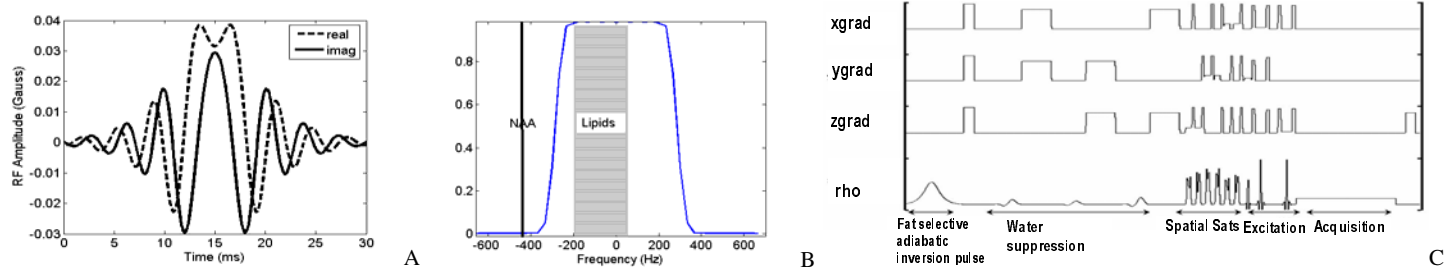


Figure 1: Simulations for 7T fat selective inversion pulse: (A) real and imag components of RF waveform, (B) spectral profile and (C) gradient and RF waveforms for PRESS sequence with fat selective inversion recovery.

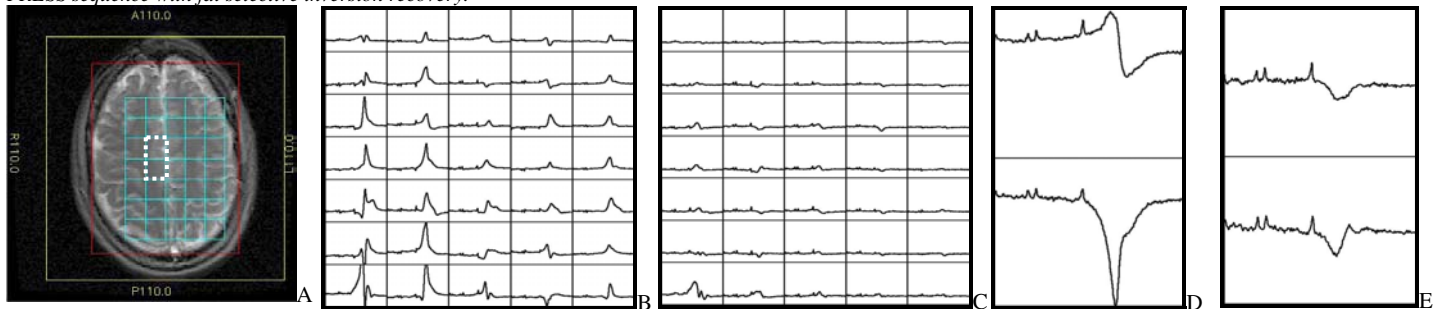


Figure 2: In vivo data from normal volunteer: (A) FOV, PRESS box, spectral grid location and two central voxels (dotted line), (B) spectral grid using standard GE PRESS, (C) spectral grid with fat-selective inversion recovery, (D) central voxels using standard GE PRESS (rephased so that metabolite peaks are upright) and (E) central voxels with fat-selective inversion recovery.

Results: Refer to Figure 2 for in vivo spectra from the brain of a normal volunteer scanned at 7T (GE Whole Body Magnet). Figure 2 A shows the ROI with 3.375 cc voxels within the selected PRESS box. For this experiment, a large PRESS box, with corners that are close to the edge of the brain, was placed near the top of the head, resulting in significant lipid contamination both in the center and edge voxels. In this way, the effectiveness of the pulses over a range of B₁ values could be tested (B₁ is highest at the isocenter of the coil). The metabolite map in Figure 2 B is obtained using the standard GE PRESS sequence (TE/TR = 90/3500 ms) and scaled to show the broad lipid peak. When the same region is excited with a PRESS sequence using the adiabatic fat-selective inversion pulse with an overdrive factor of 2.8 (TE/TR = 90/3500 ms), the metabolite map in Figure 2 C is obtained (scaled to the same magnitude as Figure 2 B). It is evident that fat has been significantly suppressed over the entire excited region (over a range of B₁). Figures 2 D & E show selected spectra for two central voxels (delineated by the dotted line in Figure 2 A) for the standard PRESS and PRESS using fat-selective adiabatic inversion respectively. Figures 2 D & E have been scaled to show the main metabolite peaks (Cho, Cre, NAA) more clearly and Figure 2 D has been phased so that the metabolite peaks are upright. These spectra show that fat has been significantly suppressed while not degrading the NAA signal (at 2 ppm).

Discussion: It can be seen from in vivo data that the adiabatic fat-selective inversion pulse successfully suppresses fat while not degrading signal from most of the metabolites of interest in the brain when compared to the standard GE PRESS sequence at 7T. Similar fat suppression may be achieved by using a non-selective adiabatic inversion pulse; however the lack of selectivity causes significant metabolite signal loss at 7T. The pulse will eventually be inserted into an interleaved adiabatic SPSP sequence (currently under development) so that, in addition to fat suppression, excitation is adiabatic and immune to CSL errors. This will allow for increased volumetric coverage without degraded metabolite signal for ¹H MRSI at 7T. Other options such as adiabatic inversion pulses with asymmetrical profiles [2] or selective adiabatic excitation pulses [3] may also be explored and compared to the selectivity, suppression efficiency and SAR levels of a standard hyperbolic secant inversion pulse.

References: [1] Spielman DM, et al. *J Magn Reson Imaging* 1992; May-Jun;2(3):253-62. [2] de Graaf R, Nicolay K. *Magn Reson Med* 1998; 40(5):690-696. [3] Rosenfeld D, et al. *Magn Reson Med* 1996 Sep;36(3):401-9.

Acknowledgements: Lucas Foundation, NIH RR09784, Prof. John Pauly