1H-MRS in the in vivo mouse heart: a comparison between PRESS and STEAM
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Target audience - This work is relevant to the field of myocardial metabolism in general, and to those interested in strategies for (mouse) cardiac MR spectroscopy in particular.

Purpose - Cardiac lipid metabolism has become an important area of research, particularly due to the increasing prevalence of cardiovascular diseases associated with obesity and diabetes. Proton MR spectroscopy (1H-MRS) allows for the noninvasive detection of myocardial metabolites such as myocardial triglyceride (TG). So far, its application in preclinical rodent studies has been limited [1]. For 1H-MRS of brain, liver, and skeletal muscle, two different single-voxel single-shot localization techniques are commonly used: STEAM (stimulated echo acquisition mode) and PRESS (point resolved spectroscopy). While STEAM may benefit from a reduced minimal echo time (TE) and the absence of refocusing pulses, it suffers from 50% signal reduction and is more susceptible to motion artifacts compared to PRESS. The small (~100 mg), fast-beating (400-600 min−1) mouse heart imposes challenges regarding optimization of sensitivity and minimization of motion artifacts. The aim of this work is to compare the suitability of PRESS and STEAM for 1H-MRS of the in vivo mouse heart. Also, mouse myocardial water and TG T1 relaxation times at 9.4 T were determined.

Methods - We implemented ECG-triggered, respiratory-gated PRESS and STEAM sequences on a horizontal-bore 9.4 T MR system (Bruker BioSpin) equipped with a quadrature-driven birdcage coil (Ø 35 mm) for RF transmission and signal reception. PRESS parameters were TE = 9.1 ms; 0.4 ms Hermite-shaped 90° excitation pulse (bandwidth 33 ppm); 0.9 ms Mao-type 180° refocusing pulses (bandwidth 15.5 ppm). STEAM parameters were TE = 3.6 ms; TM = 3.8 ms; 0.4 ms Hermite-shaped 90° pulses (bandwidth 33 ppm). To maintain steady state of magnetization, dummy pulses were performed during respiratory gates [2]. Water suppression was performed with a CHESS module prior to the localization sequence. In anesthetized male C57BL/6 mice (n = 12), cardiac 1H MR spectra (NA = 256) were obtained from a 4 μL voxel positioned in the end-diastolic interventricular septum using PRESS and STEAM at TR = 2 seconds. Series of 32 water spectra were acquired in the same voxel, and quantified using jMRUI to assess signal stability via the standard deviation (SD) of the signal amplitude and zero-order phase. Finally, in a subset of mice (n = 6), saturation recovery experiments were performed using PRESS to determine in vivo myocardial water and TG methylene T1 values.

Results - Water signal amplitude stability was comparable for PRESS and STEAM (SD 9.3 ± 6.4 % vs. 7.8 ± 7.2 %, respectively). Zero-order phase was slightly more stable for PRESS than for STEAM (SD 13.4 ± 6.3 ° vs. 20.2 ± 9.8 °; P < 0.05). Water line width was smaller for PRESS than for STEAM (28.8 ± 4.3 Hz vs. 38.0 ± 6.8 Hz; P < 0.001), and SNR of the water signal was almost two-fold higher for PRESS than for STEAM (99.7 ± 13.7 vs. 51.1 ± 12.1; P < 0.001). In water-suppressed spectra, peaks from several metabolites could be distinguished, although small peaks such as for creatine methyl protons (Cr-CH3; 2.99 ppm) were obscured by noise in STEAM spectra (Figure 1). The amplitudes of the dominant TG methylene (TG-CH2; 1.28 ppm) signals, determined from PRESS and STEAM spectra, showed a good correlation (r = 0.92; P < 0.001, Figure 2). T1 relaxation times were 1.69 ± 0.24 s for mouse myocardial water and 0.62 ± 0.19 s for TG-CH2 (Figure 3).

Discussion - Here, we demonstrated the feasibility to acquire 1H MR spectra in the in vivo mouse heart using both PRESS and STEAM. Water signal stability was slightly better for PRESS than for STEAM. Evidenced by a good correlation between PRESS and STEAM measurements, both methods can be used to quantify mouse myocardial TG content. However, the two-fold higher SNR for PRESS compared to STEAM with the current parameter settings makes PRESS superior to STEAM for in vivo detection of myocardial metabolites, particularly for low-concentration metabolites such as creatine. The T1 value for mouse myocardial water at 9.4 T is in agreement with a previous report using MRI to determine myocardial water T1 [3], demonstrating the effective maintenance of steady state of magnetization with dummy pulses during respiratory gates. Importantly, with this approach, we were able to estimate the T1 for mouse myocardial TG at 9.4 T. In future research, these T1 values can be used to convert semi-quantitative measures of myocardial TG content to absolute values of myocardial TG levels.


Figure 1. Water-suppressed STEAM (top) and PRESS (bottom) 1H MR spectra from the in vivo mouse heart.

Figure 2. Correlation between PRESS and STEAM for myocardial TG methylene signal amplitude.

Figure 3. Saturation recovery curves for in vivo mouse myocardial water and triglyceride TG obtained with PRESS-localized acquisitions.