

Assessment of compressed sensing for high resolution *in vivo* mouse cardiac ^{23}Na chemical shift imaging

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Target Audience: MR spectroscopists, small animal preclinical researchers, preclinical cardiac researchers

Purpose: To evaluate the application of compressed sensing (CS) to high-resolution murine ^{23}Na cardiac chemical shift imaging (CSI), and analyze the impact of signal to noise ratio (SNR) and acceleration factor (R) on reconstruction fidelity.

Methods: CS reconstruction was applied as described previously¹. A noise free synthetic phantom approximated myocardial CSI in mice (Fig. 1a). Gaussian noise was added to vary the SNR (SNR=20, 10, 5) and k-space was undersampled (R=10, 5, 3.3, 2.5, 2). Reconstructed spectra were fitted in the time domain (in-house software). Uniformly sampled *in vivo*, cardiac gated, short axis, single slice, 2D CSI data were acquired (25 mm FOV, 3 mm slice, 36x36 PE steps, TR \approx 125 ms, TE=0.760 ms, 5 avg) in wild-type C57/B16 mice as described previously². These data were undersampled retrospectively and reconstructed as for the synthetic phantom.

Results: Synthetic phantom data demonstrated accurate signal reconstruction for R<10 and SNR>5 (Fig. 1b). For R=10, CS reconstruction results in underestimation of signal amplitude >5 %. Similarly reconstructed signal amplitudes are underestimated for SNR<5. Increasing acceleration factor also results in a linear scaling of signal amplitude relative to the fully sampled data. CS reconstruction of ROI4 (Fig 1a) caused a consistent underestimation of signal amplitude principally due to artefacts in the reconstruction of the compartment boundaries contributing significantly to the compartment volume. When the data are normalized to the signal from ROI1, all reconstructions return errors <5 % except for ROI4, SNR=5 or R=10. Both amplitude and phase of the *in vivo* data were accurately reconstructed by CS for acceleration factors up to R=5 (Fig 1c). There was a linear scaling of the CS reconstructions relative to the fully sampled data such that the reconstructions underestimated signal amplitude; the scaling increased with R (Fig 1d).

Discussion: ^{23}Na CSI is challenging due to the relative low MR sensitivity of the nucleus, and the extremely short T2. Coupled with the small size of the mouse heart and high heart rates (~400-600 bpm), ^{23}Na CSI demands long acquisition times. The CS reconstruction tested here offers the opportunity to dramatically reduce the acquisition time without sacrificing either accuracy or SNR. Normalization of the signal to an internal concentration reference phantom removes any issues arising from linear scaling of data as a result of CS reconstruction.

Conclusion: Applying compressed sensing to high resolution *in vivo* mouse cardiac ^{23}Na CSI is feasible. The dramatically reduced acquisition time of a prospectively undersampled CSI opens up the possibility to include ^{23}Na CSI as part of a comprehensive MR myocardial phenotyping protocol or to study the progression of the disruption of ion homeostasis, and oedema, immediately following ischaemic insult.

References: 1. Geethanath et al., Radiology, **262**, 985-994 (2012); 2. Maguire et al., in Proc ISMRM 2013, 1361.

Funding: British Heart Foundation (FS/11/50/2903), MRC/EPSC (G0600829).

Figure 1. (a) Structure of synthetic phantom. (b) Plot of error in CS reconstruction of ROI1 relative to noise free reconstruction with R=1. (c) Magnitude image of CS reconstructed *in vivo* CSI (R=5) with contours for heart, chest wall and concentration reference phantom (CR). (d) Plot of amplitudes from normalized fully sampled *in vivo* CSI versus amplitudes of the CS reconstructed data (R=5).

