

Measurement of the T_1 of ^{31}P -Metabolites at 7 Tesla in the Human Heart

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PURPOSE: This work aims to measure the T_1 of ^{31}P in PCr and ATP in the human heart with high precision. Specifically, we test the hypothesis that the T_1 of these metabolites decreases with field strength.

METHODS: Sequence. A Look-Locker Inversion Recovery Chemical Shift Imaging sequence incorporating a gap for magnetization recovery was implemented on a Siemens 7T scanner. One repeat of the sequence is shown in Fig. 1. This module is repeated for each point in a 3D phase encoding scheme to create a complete 4D (3xspatial + TI) dataset. The experiment was repeated with inversion at two frequencies to cover the full range of metabolite chemical shift. The inversion pulse was a 29.7ms HS8 with $R=18$, the excitation pulse was taken from [1] but with reduced duration (2.9T/7T) and centred at a frequency 586 Hz from PCr to ensure uniform flip angle for all the relevant metabolites.

Acquisition. Pilot data were acquired from the calf of 2 volunteers (both male) each with 5 repetitions having inversion centred at frequencies of: 836, 47, -566, -1339 and -1589Hz.

The first TI was 40ms with readout at 600ms intervals thereafter. Excitation was nominally 15° , matrix size and FOV were $8 \times 8 \times 8$ and $200 \times 200 \times 200 \text{mm}^3$. Cardiac data were then acquired from healthy volunteers (5m, $34 \pm 7\text{y}$) using similar methods, the only differences being that $R=24$ and only 2 repetitions were performed with offset frequencies of 774Hz, -827Hz. Localisation used a 10cm loop ^1H T/R coil (RapidBiomed) to acquire transverse, pseudo-long-axis images followed by a short axis stack of cardiac-gated gradient echo images. Having optimised coil placement relative to the heart, the ^1H coil was replaced with an identically positioned 10cm loop ^{31}P T/R coil. The ^{31}P data collection required 40min (calf), 80min (heart).

Fitting. For each subject a single voxel was chosen and all the data from all the experiments were simultaneously fitted in the time domain using a Matlab Bloch equation simulator to model the exact sequence used for acquisition. Using Isqcurvefit, the following 22 parameters were simultaneously fit per subject and voxel: T_1 Pi, T_1 PCr, T_1 γ -ATP, T_1 α -ATP, T_1 β -ATP, T_2 Pi, T_2 PCr, T_2 γ -ATP, T_2 α -ATP, T_2 β -ATP, T_2^* global, M_0 Pi, M_0 PCr, M_0 γ -ATP, M_0 α -ATP, M_0 β -ATP, Δf Pi, Δf γ -ATP, Δf α -ATP, Δf β -ATP, Phase, and B_1 excitation scaling factor. This fitting approach is advantageous because it automatically handles the partial inversion/saturation expected due to the relatively weak B_1^+ available whilst minimising the number of parameters. Fitting required $\sim 30\text{min}$ on a desktop PC and was robust even with data too noisy to fit individual spectra.

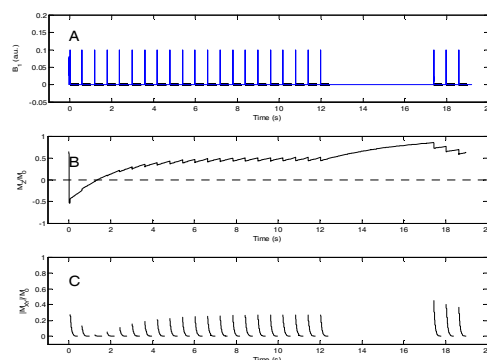
RESULTS: T_1 values (ignoring CK flux) for relevant metabolites are shown in the table and compared with literature values.

DISCUSSION: For calf muscle, our T_1 values agree well with those recently reported [2]. The exception to this is our longer γ -ATP T_1 ($4.12 \pm 0.15\text{s}$ vs. $3.3 \pm 0.2\text{s}$), which we believe is due to differences in acquisition and a decision in both cases to neglect the effects of CK flux. This agreement in the calf validates our acquisition and fitting methods. At 7T, we found that ^{31}P T_1 in the heart is shorter than in calf muscle at 7T. It is also clear that these T_1 values are shorter than at lower field strengths (see literature data in table). This trend is consistent with animal studies [3]. Yet, all these values ignore the effects of CK flux. When we included that effect in our model, assuming minimally that the intrinsic T_1 of γ -ATP is the same as that of β -ATP, and k_f of 0.26s^{-1} (leg), 0.32s^{-1} (heart) [5], we found the intrinsic T_1 of PCr to be $8.4 \pm 0.7\text{s}$ (leg) and $6.7 \pm 2.1\text{s}$ (heart). This indicates that the CK flux and the short T_1 of γ -ATP drives 53% of apparent PCr relaxation in the calf and 58% in the heart at 7T. Hence, while the apparent T_1 of ATP and PCr clearly decreases with field strength, and while the intrinsic T_1 of ATP decreases with field strength, it is not yet possible to determine if the intrinsic T_1 of PCr increases or decreases. Given the large chemical shift anisotropy of ATP [2], we may well expect that the reducing intrinsic T_1 of ATP dominates the trends in T_1 of PCr.

CONCLUSION: Our new method accurately measures the T_1 of ^{31}P -containing metabolites in the heart and leg. The shorter apparent values of T_1 contribute to the improved data quality at 7T. CK flux contributes strongly to the measured T_1 of PCr and γ -ATP.

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T_1	Calf Muscle 7T (this work)	Calf muscle 7T from [2]	Cardiac Muscle 7T (this work)	Cardiac Muscle 3T from [4]
Pi	$6.65 \pm 0.23\text{s}$	No value	No value	No value
PCr	$3.96 \pm 0.07\text{s}$	$4.0 \pm 0.2\text{s}$	$2.81 \pm 0.47\text{s}$	$5.8 \pm 0.5\text{s}$
γ -ATP	$4.12 \pm 0.15\text{s}$	$3.3 \pm 0.4\text{s}$	$1.86 \pm 0.15\text{s}$	$3.1 \pm 0.6\text{s}$
α -ATP	$1.70 \pm 0.02\text{s}$	$1.8 \pm 0.1\text{s}$	$1.34 \pm 0.09\text{s}$	No value
β -ATP	$1.42 \pm 0.12\text{s}$	$1.8 \pm 0.1\text{s}$	$0.98 \pm 0.15\text{s}$	No value