Introduction
At high magnetic field, $^1$H MR Spectroscopy benefits from an increase in sensitivity and spectral resolution by an intrinsic increase of the Signal-to-Noise Ratio and a higher chemical shift dispersion. At ultra-high magnetic fields such as 17.2 T, it is crucial to establish $T_1$ and $T_2$ relaxation times in order to optimize MRS acquisition parameters and to achieve proper quantification. Here, we present the measurement of $T_2$ relaxation times of more than 18 metabolites in the rat brain in vivo at 17.2T.

Methods

MRS Acquisitions. A total of 6 Dark-Agouti rats were studied under isoflurane anesthesia (1-2% in pure O2). Body temperature was monitored and maintained at 37°C ± 0.5°C. All Experiments were performed on a 17.2 T/26 cm Bruker BioSpec MRI scanner (Ettlingen, Germany) using a home-made 20 mm diameter single-loop surface coil transceiver. Anatomical images were acquired for positioning using a RARE sequence covering the entire brain. $^1$H MR Spectra were acquired with a LASER$^1$ sequence (TR=5s, 128 averages, 2048 points) from a volume of 50 μL (5x5x2mm$^3$) containing mostly cerebral cortex and contributions from the corpus callosum and the hippocampus. Local $B_0$ field homogenization on the same volume was done using mapshim and local-shim Bruker routines (water linewidth = 23 ± 3 Hz). $T_2$ weighting was introduced by increasing the echo time from 16.5 to 200 ms leading to a total of 12 different TE (16.5, 24, 35, 50, 60, 80, 100, 120, 140, 160, 180 and 200 ms). Water suppression was done using a WET module$^2$ with numerically optimized flip angles and delays. Metabolite-nulled spectra were acquired at TE=16.5 ms using a double inversion recovery scheme (TI1/TI2/TR = 2600/600/5000 ms).

Data Analysis. After removal of the residual water signal using the HLSVD$^3$ algorithm, each MR spectra was analyzed using LCModel$^4$ and a corresponding set of simulated spectra. Simulations were performed for all major brain metabolites using a spin simulation software developed by R.A. de Graaf, (MRRC, Yale School of Medicine) and Matlab (MathWorks, Natick, MA, USA). Due to their significant differences in $T_2$, signals from NAA-CH2 and NAA-CH3 as well as Cr-CH2 and Cr-CH3 were accounted separately. The line shapes of macromolecules were parameterized$^5$ and implemented in LCModel. $T_2$ relaxation times were calculated by fitting the metabolites concentrations to a mono-exponential decay function.

Results and Discussion

Figure 1 (Top) shows examples of our $T_2$-weighted MR spectra and their LCModel at the minimum TE of 16.5 ms, a moderate 60 ms TE and a long TE of 120 ms. As illustrated by Fig.1, the $J$-modulation of major brain metabolites such as glutamate (fig.2) and myo-inositol was adequately accounted for by our simulations. Figure 3 recapitulates the $T_2$ relaxation times calculated for metabolite and macromolecule resonance groups (see fig.1 “bottom” for macromolecule definition). Compared to $T_2$ values measured in the rat brain in vivo at lower magnetic field such as 11.7 Tesla, our values are shorter which is consistent with the established field-dependence of $T_2$ decay times due to increased microscopic susceptibility gradients as $B_0$ increases.

References