Hahn T₂ Relaxation Times of the Neurochemical Profile at 14.1T in the in vivo Rat Brain

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Introduction

The field strength of MR scanners is steadily increasing, with an increasing number of scanners for animal research being installed at B_0 of 9.4 and 11.7 T. We have recently taken delivery and installed a horizontal-bore scanner operating at 14.1 Tesla. Since no relaxation theory can quantitatively account for the complex interactions within *in vivo* tissue [1], the relaxation times need to be determined experimentally at this new field strength [2, 3]. The current study carries a two-fold purpose: first, to measure the Hahn T_2 of uncoupled and coupled spin resonances of cerebral metabolites in rat brain *in vivo* at 14.1T; second, to compare these results with those obtained in a previous study performed at 9.4 T [3].

Methods

Experiments. MRS was performed on a 14.1T/26 cm magnet (Varian/Magnex). In vivo spectra were acquired in five rats, from a 70 μl VOI centered in the hippocampus, using a novel pulse sequence (SPECIAL, [4]). Spectra were measured at eleven echo times, ranging from 2.8 to 300 ms. Data Analysis. To assess the T_2 of coupled spin resonances, the J-modulation of the lineshapes was taken into account by using the LCModel software [5] with TE-specific basis-sets to analyze spectra at each TE. Basis-sets were generated with density matrix simulations, using published values of coupling constants J and chemical shifts δ [6]. To assess the quality of the T_2 fit, the coefficient of determination (R^2) was calculated.

Results

Good agreement between the *in vivo* water-suppressed ¹H MR spectra of and LCModel fit was observed at all TEs (Fig. 1). The T₂ of singlets and coupled spin resonances of cerebral metabolites ranged from 72 ms (glutamate) to 148 ms (total choline) (Table). The T₂s at 14.1 T were slightly shorter than the T₂s measured in a previous study at 9.4 T [3], in particular for NAA(CH₃), tCho and Ins (Fig. 2).

Discussion

The current findings are in agreement with previous studies [1, 2], where a decrease in T_2 was observed with increasing field strength. In the current study, the comparison between T_2 of cerebral metabolites measured at 9.4 and 14.1 T shows that T_2 change of NAA singlet is larger than that of tCr (Fig. 2). De Graaf et al. [2] reported a small change between the T_2 values of NAA singlet and tCr at 9.4T and 11.7T which were measured by LASER type of sequence, thus reducing the contribution of chemical exchange and diffusion.

Metabolite	T ₂ (ms) @ 14.1 T	R²
tCho	148 ± 14	0.958
NAA(CH ₃)	142 ± 7	0.988
NAA_m	122 ± 15	0.929
tCr(CH ₃)	107 ± 2	0.998
tCr(CH ₂)	98 ± 2	0.996
Ins	97 ± 6	0.983
Gln	95 ± 13	0.943
GSH	87 ± 13	0.948
Tau	82 ± 7	0.965
Glu	72 ± 8	0.958

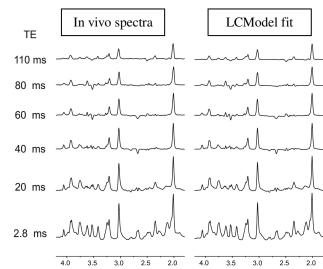


Fig. 1. In vivo spectra (gf = 0.12) and LCModel fit at TE ranging from 2.8ms to 110ms.

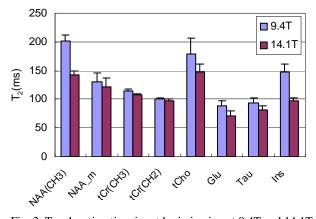


Fig. 2. T_2 relaxation time in rat brain in vivo at 9.4T and 14.1T.

The large T₂ change in NAA singlet in present study might be

due to the larger contribution of chemical exchange of acidic protons in the molecule and diffusion effects in the Hahn echo sequence with increasing field strength. It should be noted that both at 9.4 and 14.1 T, data acquisition (sequence, TEs and VOI) and data analysis (LCModel fit with TE-specific basis-sets) were the same. For metabolite quantitation, the decrease in T_2 with increasing field strength should be taken into account when performing MRS experiments at long TE, such as, for instance, in editing techniques. However, we conclude that as at 9.4 Tesla, the T_2 of coupled spin systems appears similar and comparable to that of tCr. It is therefore postulated that the similarity of T_2 persists at lower field strengths, which permits to validate previous quantification strategies for spectral editing based on comparison with internal reference signals such as the aspartyl NAA or the Cr methyl resonance.

References

[1] Michaeli S et al., Magn Reson Med. 2002. [2] de Graaf RA et al., Magn Reson Med. 2006. [3] Xin L et al., NMR Biomed. 2007. [4] Mlynarik V et al., Magn Reson Med. 2006. [5] Provencher SW, Magn Reson Med 1993. [6] Govindaraju V et al., NMR Biomed. 2000.

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