Localized H¹-MRS of the human frontal white matter at 3 T: Metabolite Concentrations and Relaxation Times

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Introduction

Differences in metabolite concentrations e.g. between groups of psychiatric patients and healthy controls obtained from usually T1 and/or T2 weighted spectra could also be due to differences in T1 or T2 relaxation time. In our study we investigated absolute metabolite concentrations from N-acetyl aspartate (NAA), total creatine (tCr) and choline compounds (tCho) in the frontal white matter with simultaneously determined T1 and T2 relaxation times by acquiring spectra with varying TE and TR.



Figure 1: Sample spectra (TE = 30 ms; TR = 6000 ms), fit and position of the single voxel in the frontal white matter

Methods

In vivo proton spectroscopy has been performed on 13 healthy volunteers (7 female/5 male) aged between 26 and 44 years, mean age: 37.5 ± 7.6 years. All experiments were performed on a 3 T Siemens TRIO (Siemens Medical Solutions, Erlangen, Germany). Spectra were taken from a left frontal white matter location (Fig. 1). Water suppressed localized spectroscopy was performed using a PRESS sequence. The voxel size was 10x40x10 mm³. For T2 quantification 4 measurements of nearly fully relaxed spectra at TR = 6 s and echo times TE = 30, 80, 200 and 420 ms with 40 averages were performed (4 min each). A progressive saturation experiment was performed to determine the T1 relaxation times. Spectra at 7 different TR were acquired, TR = 1.5, 2, 2.5, 3, 4, 5, 6 s and TE = 30 ms. The acquisition time was kept constant at 4 min by varying the number of acquisitions from 160 at TR = 1.5 s to 40 at TR = 6 s. Metabolite signal amplitudes were quantified by time-domain fitting using the nonlinear AMARES algorithm of the jMRUI software package [1]. For each spectrum of the time-series 18 Gaussian spectral components were used to fit the contribution of the metabolites NAA, tCre and tCho. T2 and T1 relaxation times were calculated monoexponentially using a two-parameter least-squares fit in Origin:

 $S(TE) = S'(TE)/f(T1,TR,TE) = S_0 \cdot exp(-TE/T2)$ with $f(T1,TR,TE) = 1 \cdot exp(-TR/T1)$ $S(TR) = S_{\infty} \cdot (1 \cdot exp(-TR/T1))$

For absolute metabolite quantification we evaluated the biexponential water-decay by measuring the fully relaxed water signal as an internal standard as it is described in [2] with TR = 10 s, TE = 30, 80, 276, 552, 1500 ms. Concentrations were calculated as follows [3]:

$$C_{\text{NAA}} = C_{\text{H20}} \bullet (S_{\text{NAA}}/S_{\text{H20}}) \bullet 2/3 \qquad \qquad C_{\text{tCr}} = C_{\text{H20}} \bullet (S_{\text{tCr}}/S_{\text{H20}}) \bullet 2/3 \qquad \qquad C_{\text{tCbo}} = C_{\text{H20}} \bullet (S_{\text{tCbo}}/S_{\text{H20}}) \bullet 2/9$$

 C_i is the averaged concentration of water/metabolite *i* in the measured voxel, S_i the amplitude of the corresponding signal *i*. The metabolite signals were corrected for both T1 and T2 and S_{H20} only for T2. The water content of white matter was assumed to be 0.71 or 39.4 mmol/g wet weight. Concentrations were corrected for partial volume effects.

Results

In vivo T1 and T2 relaxation times and absolute concentrations of NAA, tCre and tCho in frontal white matter were determined for 13 subjects. The estimated averaged relaxation times and concentrations and the corresponding standard deviations (SD) are shown in table 1.

Table 1: Average absolute concentrations, T1, T2 relaxation times and standard deviations (n=13)

	NAA	tCr	tCho
Concentration [mmol/kg]	11.24	7.40	2.35
SD _{conc}	± 0.68	± 0.61	± 0.16
T1 [ms]	1333	1399	1132
SD _{T1}	± 180	± 235	±191
T2 [ms]	342	193	240
SD _{T2}	± 28	± 19	± 30

Discussion

Our findings are in reasonable agreements to the literature [4-6]. The relaxation time standard deviations of up to 17 % might not be solely due to fit errors but possibly also to an influence of dietary or further unknown covariates on relaxation times, which are not receiving attention today. Based on previous observations at 1.5T [7] this ongoing study was set up to investigate the amount of alcohol, water and caffeine consumption as a

covariate on the concentration, microstructure or relaxation times of the observed metabolites. Today the number of studied subjects and the variance in the covariates does not yet allow any significant conclusions.

References

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