Characterization of the downfield part of the human cerebral ^{1}H MR spectrum at 3 T

R. Kreis[1], C. Boesch[1], P. Vermathen[1]
[1]Dept. of Clinical Research, University Berne, Berne, Switzerland

Introduction

The downfield region (5 to 10 ppm) of ^{1}H-MR spectra from human brain is still poorly characterized. MRS studies in humans yielded exact resonance positions for phenylalanine (Phe, 7.37 ppm)[1], homo-carnosine (Cs, 7.05, 8.02 ppm)[2], and histidine (His, 7.07, 7.83 ppm)[3], but also showed that from these, only Cs contributes considerably to spectra of healthy controls. A further undisputed component is the N-H proton of NAA at ~7.9 ppm. Other potentially visible compounds include glutamine (6.8 ppm), creatine (6.8 ppm), tryptophane (7.3 ppm), and ATP (8.22, 8.45 ppm). Macromolecular vs. metabolic contributions have been distinguished in a preliminary study[4]. It yielded controversial results w.r.t. the unassigned peak at 7.3 ppm, which seemed to originate mostly from macromolecules based on its saturation recovery behavior, but had a TE-dependence[3] of a small molecule. While all these characterizations have been performed at 1.5 T, animal experiments conducted at higher fields[5-7] proved potential for further resonances, particularly exchangeable amid protons. This motivated an evaluation of the downfield region in humans at the new clinical field strength of 3 T.

Methods

Spectra were recorded on clinical 3T (Trio, Siemens) and 1.5T (Signa, GE) MR scanners using standard quadrature head coils. Data acquisition was performed with short TE PRESS sequences (20-120 ms TE, 0.5-5 s TR, 16 step phase rotation, water presaturation, outer volume saturation) from a large supraventricular volume (70 cm^3). Results from six healthy subjects are presented. Each figure contains mean spectra for three subjects.

Results

The field dependence for the downfield part of cerebral short TE spectra is illustrated in Fig. 1. The spectral features are better resolved at 3 T than at 1.5 T. The TE-dependence of the downfield part is presented in Fig 2, which contains spectra obtained for 5 different TEs (20, 40, 60, 80, 120 ms) at 1.5 T (Fig. 2a) and 3 T (Fig 2b). The TR dependence at 3 T is shown in Fig. 3 (TR 0.5, 1, 2, 5s). Saturation recovery results for 1.5 T have been presented earlier[4].

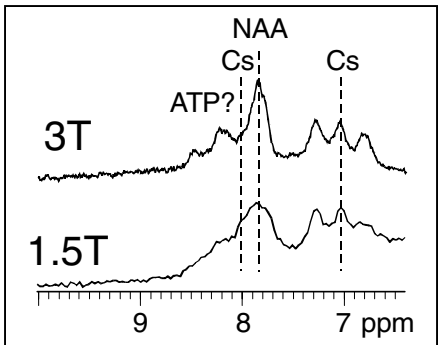

Fig. 1: 1.5 T vs. 3 T (20ms TE, 2s TR)

Discussion & Conclusion

Similar spectral features are present at 3 and 1.5 T. All peaks are better resolved at 3 T. In particular, the peaks at 8.2 and 8.45 ppm, tentatively assigned to ATP, are much better discerned from background at 3 T and can even be detected at long TE. Long TE spectra are quite different at the two field strengths. The peak at 7.3 ppm shows a striking field dependence. Its T_2 seems to shorten dramatically when going from 1.5 T to 3 T, such that at 3T only the Cs peaks remain for TE > 80 ms. The short T_2 of this peak concords with the assignment to a macromolecule – consistent with the short T_1 found at 1.5 T. Why this peak has a much longer T_2 at lower fields (Fig 2a, Ref 3) remains speculation at present (intermediate size molecule or effect of exchange or superposition of signals?). However, it appears that 3T offers a flatter baseline to detect Phe in PKU patients at longer TE.

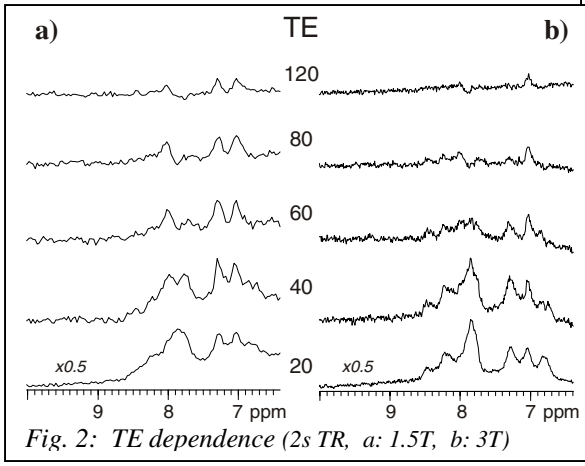

Fig. 2: TE dependence (2s TR, a: 1.5T, b: 3T)

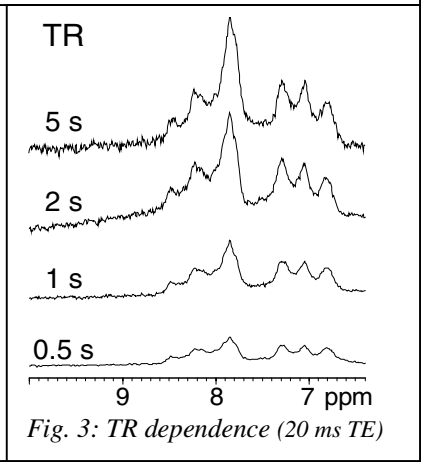

Fig. 3: TR dependence (20 ms TE)

References

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Supported by the Swiss National Foundation (31-103938).