Introduction
In chronic hepatic encephalopathy (HE), increased brain ammonium is causing the accumulation of glutamine (Gln) and gradual release of osmolytes (myo-inositol, Ins, taurine, Tau). Metabolite properties in HE are relevant for neurometabolism. Then, DW-MRS data were acquired using localized STEAM-based spectroscopic pulse sequence. 2D-diffusion spectrum imaging (DSI) was performed with a 9.4T small-bore MRI system. The spectra were acquired in the hippocampus (2x2.8x2mm3) of control (n=5) and post-BDL for Glu, NAA, Ins and Tau. The derived diffusivity parameters before-surgery (Fig.4) were in good agreement with results in the healthy rodent brain (Table1).

Results
Glu, NAA, Ins, Tau and total creatine (tCr: Cr+PCr) signal decays were then fitted using three different approaches. First, Callaghan’s model of randomly oriented sticks (neurites or processes) was used for this fit. Secondly, a model based on the Monte Carlo simulations was applied. Thirdly, a model on the basis of a stick-fixed grid was considered. All models were used with the same goodness of fit. With metabolite diffusivity D=(Dn+Df)/2, the model showed a significant increase in intra-neurite/process diffusivity Df consistent with an increase in intra-neurite/process diffusivity Df. The apparent diffusivity D increased for Glu, NAA, Ins, Tau and tCr – consistent with an increase in Df, while there was no significant change in Dn. So far the effects of Gln accumulation on astrocytes morphology together with the molecular mechanisms behind this apparent osmoregulation are unclear. This suggests that Gln might affect astrocyte morphology and its molecular mechanisms as an osmoregulation, Dunja Simicic – 2014.

Discussion
So far, there are no reports on astrocytes morphology in Gln-overload astrocytes and the molecular mechanisms behind this apparent osmoregulation. In spite of this apparent osmoregulation, an increase in water apparent diffusion coefficient (ADC) has been sometimes observed in patients with chronic HE and has been associated with edema enlargement extracellular space (Fig.5). Therefore, astrocytes morphology in Gln-overload astrocytes and the molecular mechanisms behind this apparent osmoregulation need to be further investigated.

Acknowledgements

References


Results
The MSD of the stick model was calculated as follows:

\[ \text{MSD} = \sqrt{\sum_{i=1}^{N} (x_i - \bar{x})^2} \]

where \( x_i \) is the position of the i-th point and \( \bar{x} \) is the mean position.

The experiments were performed in a 9.4T small-bore MRI system. The data were acquired in the hippocampus (2x2.8x2mm3) of control (n=5) and post-BDL for Glu, NAA, Ins and Tau. The derived diffusivity parameters before-surgery (Fig.4) were in good agreement with results in the healthy rodent brain (Table1).

Figures

1. A: Top left: Photomicrograph of histological sections of Golgi-Cox staining and neuronal morphology analysis of pyramidal CA1 neurons. B: Top right: Representative ICV maps acquired in the hippocampus at 6 weeks post-BDL for Glu, NAA, Ins and Tau.

2. Representative ICV maps acquired in the hippocampus at 6 weeks post-BDL for Glu, NAA, Ins and Tau. The maps show a significant increase in intra-neurite/process diffusivity Df consistent with an increase in Df, while there was no significant change in Dn. So far, the effects of Gln accumulation on astrocytes morphology together with the molecular mechanisms behind this apparent osmoregulation are unclear. This suggests that Gln might affect astrocytes morphology and its molecular mechanisms as an osmoregulation, Dunja Simicic – 2014.

Figures

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