

^{13}C Spectroscopic Imaging of Glycogen in Rat Brain

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INTRODUCTION

Chemical shift imaging (CSI) of ^1H and ^{31}P nuclei is widely used to image metabolite signals. ^{13}C NMR has the unique ability to obtain metabolic information from the rate of label incorporation into amino acids^{1,2} and glycogen³. The localization of brain glycogen signals is critical to avoid contamination from the more concentrated glycogen in skeleton muscle. Previously, we demonstrated the feasibility to localize the rapidly relaxing signal of glycogen using a T_1 -optimized outer volume suppression (OVS) scheme³. Our aim of the present study was to implement a 1D ^{13}C CSI pulse sequence based on a 3D OVS for *in vivo* brain glycogen distribution measurement in rat and to obtain a visual confirmation of the performance of the 3D OVS localization.

PULSE SEQUENCE AND METHODS

The pulse sequence was designed based on a previous 3D OVS method² and performed at 9.4T, as in Choi et al.³. The required phase encoding gradient was applied after the adiabatic pulse (90° in Fig. 1), which introduced an additional delay prior to data acquisition (T_{pe} in Fig. 1). With the gradient capable to switch 300 mT/m in 500 μs , a spatial resolution of 1.5 mm was possible when with 3-cm FOV and 20 phase encoding steps. The imaging sequence was extensively tested on several suitable phantoms. For *in vivo* studies, eight SD male rats were prepared and maintained brain glucose at steady-state with continuous ^{13}C glucose infusing for at least four hours as previously described⁴.

RESULTS AND DISCUSSION

Due to the short T_2^* of glycogen in several ms, the effect of introducing T_{pe} on the quantified glycogen signals was evaluated in oyster glycogen phantom up to 1 ms. Over this range, the signal loss was less than 20% and only 10% at the selected T_{pe} of 0.4 ms (not shown). A phantom containing glucose and glycogen resulted that in the inhomogeneous RF field of the surface coil, the profile shape of glycogen in phase-encoding direction mimicked that of glucose very well (not shown), which allowed to assess major concentrations differences *in vivo*. Imaging glycogen illustrated intense signal close to the surface coil ascribed to muscle glycogen (Fig. 2) that was well-suppressed when 3D OVS was applied (Fig. 3). In conclusion, *in vivo* 1D ^{13}C CSI of brain glycogen is feasible using 3D OVS localization despite of higher muscle glycogen. It also visually confirmed the performance of 3D OVS localization. Lastly, brain glycogen presented a profile comparable to brain glucose at steady state, which indicated brain glycogen distribution was similar to brain glucose.

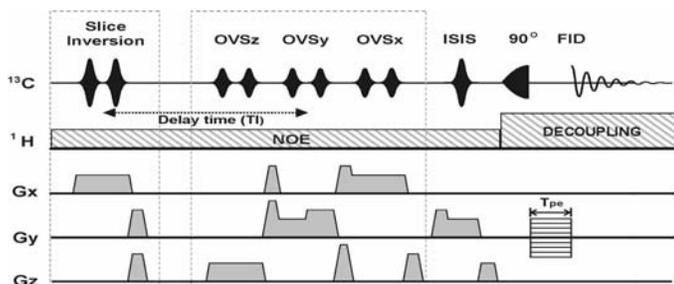


Figure 1 Pulse sequence diagram for 1D CSI based on the three-dimension OVS localization method². A traditional phase-encoding step (y) is added with a time delay, T_{pe} , right after the excitation pulse and before FID acquisition to obtain a 1D CSI map.

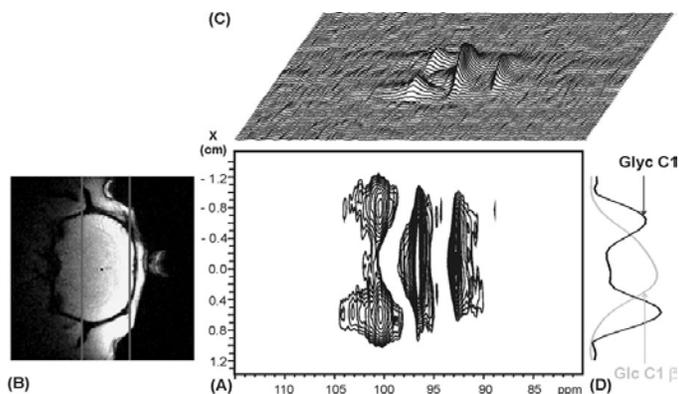


Figure 2. *In vivo* brain glycogen distribution acquired without 3D OVS localization. A 1D CSI map of glycogen and glucose (A) was acquired with only 1D ISIS, phase encoding along x direction and 3-mm spatial resolution between two grey lines in B. The top stacked plots (C) are the projection over x. The plots at right side (D) are projection profiles for Glc C1 β (grey) and Glyc C1 (black). Glycogen signal in the brain was clearly much lower than glycogen in the muscle (D).

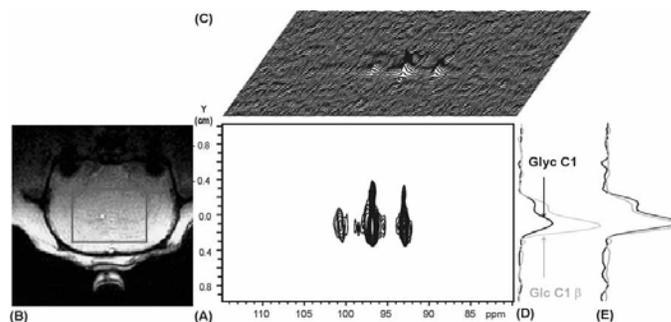


Figure 3 *In vivo* brain glycogen distribution acquired with 3D OVS localization (Fig. 1). A 1D CSI map of glycogen and glucose (A) was obtained from the VOI as shown in B and $nt = 512$. Spatial resolution is 1.5 mm. C has a series of projecting profiles over the spatial dimension (Y). D contains two sums over Glyc C1 (black) and Glc C1 β (grey), respectively. In E, Glyc C1 profile (black) was normalized to Glc C1 β (grey).

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