Human bile phosphatidylcholine contributes to $^{31}$P MRS hepatic signal at 2.06 ppm.

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Purpose/Introduction

$^{31}$P-MRS provides unique information on hepatic energy metabolism in vivo. Alternations in phosphodiester (PDE) signals have been associated with alcoholic, viral and cholestatic etiologies [1]. Main contributors to PDE signal are glycerophosphocholine (GPC) and glycerophosphoethanolamine (GPE). An additional resonance at 2.06 ppm assigned to phosphoenolpyruvate (PEP) [2,3] can be separated from PDE resonances at 3T with proton decoupling [3,4] and at 7T without proton decoupling [5]. Contribution of phosphatidylcholine (PtdC, part of lecithin) which is dominant metabolite in bile [6] to this signal is under discussion [4,7].

The purpose of this study was to assess possible contribution of PtdC to signal at 2.06 ppm by in vitro measurements of test object solutions (PEP and PtdC) and by $^{31}$P 3D MRSI in vivo measurements including signal from liver and gall bladder.

Subjects and Methods

4 cylindrical tubes filled with inorganic phosphate (Pi), phosphocreatine (PCr), PEP and PtdC were placed in the plastic box filed with water. Single shot non-localized FID acquisition (Fig.1 middle-top) and 2D CSI (16x16x16, TR 4s, TA 20min) phantom data (Fig.1) were acquired on a 7T MR system (Siemens) using double-tuned surface coil ($^1$H/$^{31}$P) (RAPID Biomedical GmbH, Rimpar, Germany), with a diameter of 10 cm.

In vivo hepatic $^{31}$P 3D MRSI data acquired at 3T (n=30, 13x13x13, TR 1s, TA 34min)[8] and 7T (n=5, 12x12x12, TR 1.5s, TA=21min)[5] were retrospectively analyzed for the presence of gall bladder on localizer images (Fig. 2 middle). PDE region of representative spectra from gall bladder and from liver tissue were fitted in jMRUI.

Results

Based on in vitro phantom measurement, following chemical shifts were observed: 0ppm - PCr, 3.16 ppm - Pi, -1.32ppm - PEP, 2.13ppm – PtdC (Fig.1). These shifts are in good agreement with previous in vitro results [7,9] and indicates that rather PtdC then PEP resonates at 2ppm.

10 of 35 in vivo data sets included signals from gall bladder. PDE signal was 3.9 fold higher (p= 0.007) in gall bladder spectra (PDE 142.6 ± 34.1 a.u.) than in liver tissue (PDE 36.3 ± 11.8 a.u.).

7T data (Fig.2) allowed good separation of PDE components with clearly visible dominant signal at 2.06ppm (Fig. 2 right).

Discussion/Conclusion

Based on both phantom and in vivo data we can suggest phosphatydicholline (lecithin) from bile rather than phosphoenolpyruvate contributes to $^{31}$P MR hepatic signal at 2.06ppm. Further studies should investigate potential use of this signal for metabolic studies of the liver and bile ducts.

Further-on, findings of altered PDE signals, especially when not idealy resolved, should take into account possible MRS contamination by hepatic bile or by gall bladder signals.

References

[7] Ijare et al. ISMRM 2011

Fig.1 Phantom measurement including 4 samples filled with Pi, PCr,PEP and PtdC

Non-localized spectrum is displayed middle-top and representative spectra left and right.

Fig.2 Example of 7T in vivo $^{31}$P 3D MRSI measurement including both liver and gall bladder data. Note strong signal of PtdC at 2ppm in gall blader and its surrounding (right).