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 phosphodiesters (PDE) signals have been associated with alcoholic, viral and cholestatic etiologies [1]. Main contributors to PDE signal are glycerophosphocholine (GPC) and glycerophosphoetanolamine (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.

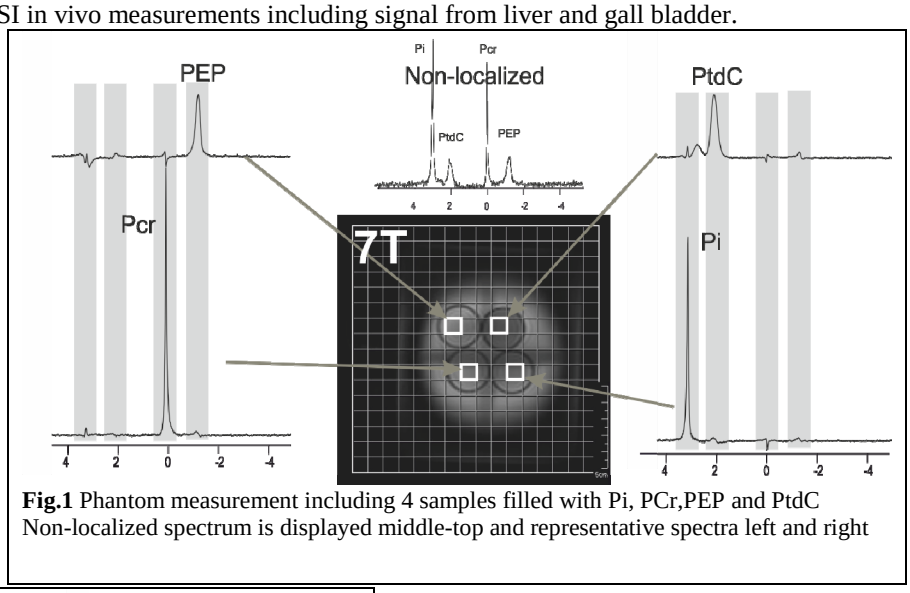

Fig.1 Phantom measurement including 4 samples filled with Pi, PCr,PEP and PtdC Non-localized spectrum is displayed middle-top and representative spectra left and right

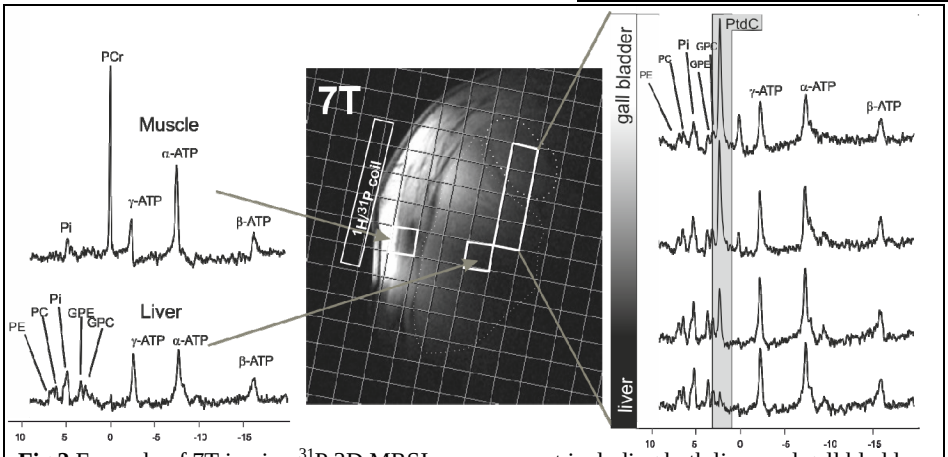

Fig.2 Example of 7T in vivo ^{31}P 3D MRSI measurement including both liver and gall bladder data. Note strong signal of PtdC at 2ppm in gall blader and its surrounding (right).

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 phosphatydilcholine (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

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