IN VIVO PROTON MAGNETIC RESONANCE SPECTROSCOPY OF HUMAN ADIPOSE TISSUE FATTY ACIDS: A FEASIBILITY STUDY

J. Lundbom¹, A. Hakkarainen¹, M-R. Taskinen², and N. Lundbom¹

¹Helsinki Medical Imaging Centre, Hospital District of Helsinki and Uusimaa, Helsinki, Finland, ²Department of Internal Medicine, Hospital District of Helsinki and Uusimaa, Helsinki, Finland

Introduction

Adipose tissue fatty acid composition has been shown to reflect well the overall fatty acid profile of the long term diet and is commonly used to estimate the proportions of saturated (SAFA), monounsaturated (MUFA) and polyunsaturated fatty acids (PUFA) in the human diet (1). Human adipose tissue fatty acids can be studied in vivo with Carbon-13 nuclear magnetic resonance spectroscopy, which has been shown to be applicable for studies of dietary influence on adipose tissue (2). The method is, however, not readily available in clinical imagers. Recently, *in vivo* proton magnetic resonance spectroscopy (MRS) has been used to study lipids in skeletal muscle and liver in connection with type 2 diabetes (3). Although the spectrum quality allowed the quantitation of intracellular and extracellular lipids, their composition was not determined. We evaluated the feasibility of using proton MRS to study the fatty acid composition of adipose tissue *in vivo*. The subcutaneous adipose tissue is a very homogeneous and stationary region and should thus suits well for in vivo proton MRS.

Measurements

The MRS proton spectrum of the subcutaneous adipose tissue from the waist of 4 volunteers were measured on a clinical 1.5 T whole body imaging system (Magnetom Avanto, Siemens, Erlangen, Germany) (FIG. 1.). The PRESS sequence was used to measure the proton spectra, with the parameters: TR = 1500 msec; TE = 135 msec; 1024 data points over a spectral width of 1000 kHz and 128 acquisitions.

Three cooking oils (olive, rapeseed and sunflower) of with different known concentrations of MUFA and PUFA fats were also measured with the same imager to validate the findings. The oils were measured with a PRESS sequence with TE/TR 30/1500. The proportion of MUFA and PUFA protons in the oils was calculated from their known compositions.

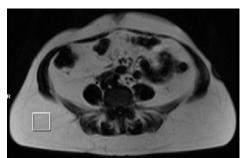


FIG. 1. Localization of the VOI in adipose tissue.

The results suggest that the *in vivo* proton spectrum of adipose tissue enables the calculation of the SAFA, MUFA

interventions on the fat composition of human adipose tissue, which in turn has implications for the development of metabolite syndrome and atherosclerotic vascular disease

(4,5). Benefits of proton MRS over carbon-MRS is good availability and a well defined volume of interest, which also enables the study of visceral adipose tissue. Further

refinement of the method requires measurement of the T_1

and T₂ relaxation times of the different proton resonances.

and PUFA proportions. The method will have applications in studying long term effects of dietary and pharmacological

Results

The spectra from the volunteers showed that a sufficiently narrow line-width is achieved for separation of the methylene (CH₂ at 1.4 ppm) and methyl (CH₃) at 1.0 ppm) peaks, and a sufficient signal-to-noise ratio is achieved for the detection of (HC=CH at 5.3 ppm) and (C=C-CH₂-C=C at 2.8 ppm) proton resonances. An in vivo proton spectrum of subcutaneous adipose tissue is shown in Fig. 2, along with relevant fatty acid peak assignments as in (3). The water signal at 4.8 ppm is not present in the long echo time spectra, although it can be seen at very short echo times (30 msec). The areas of the peaks at 5.3 ppm and 2.8 ppm in the cooking oils were approximated with the jMRUI v.2.2 software and were taken to represent the total amount of double bonds (tDB) and methylene interrupted double bonds (MeDB), respectively. The calculated PUFA/(MUFA+PUFA) ratio correlated positively with the measured (MeDB / tDB) ratio with $R^2 = 0.9935$.

Discussion

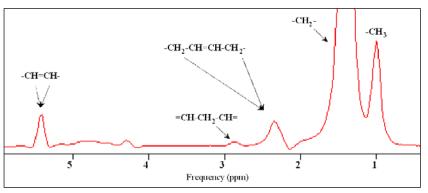


FIG. 2. In vivo proton spectrum of adipose tissue.

References:

- 1. van Staveren WA, Deurenberg P, Katan MB, Burema J, de Groot LCPGM & Hoffmans MDAF. Am J Epidemiol 1986; 123: 455-463.
- 2. Thomas EL, Frost G, Barnard ML, Bryant DJ, Taylor-Robinson SD, Simbrunner J, Coutts GA, Burl M, Bloom SR, Sales KD, Bell JD. Lipids 1996; **31**: 145-151.
- 3. Szczepaniak LS, Babcock EE, Schick F, Dobbins RL, Garg A, Burns DK, McGarry JD and Stein DT. Am J Physiol 1999; 276: E977-E989.
- Vessby B, Uusitupa M, Hermansen K, Riccardi G, Rivellese AA, Tapsell LC, Nälsén C, Berglund L, Louheranta A, Rasmussen BM, Calvert G D, Maffetone A, Pedersen E, Gustafsson I-B, Storlien LH. Diabetologia 2001; 44: 312-19.
- 5. Frank B. Hu FB, Willett WC. JAMA 2002; 288: 2569-2578.