

## Short-term Dietary Effects on Liver Lipids Measured With 3T MRS

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### Introduction

Fatty liver disease has become increasingly prevalent, yet is not well understood [1]. Previous studies have suggested that a simple-sugar diet versus a complex carbohydrate containing diet may influence lipogenesis and thus, liver lipids [2]. <sup>1</sup>H MRS of the liver offers a noninvasive means to measure hepatic lipid content and has been used in studying fatty liver disease [3]. The ability to non-invasively monitor the liver fat in response to dietary changes, exercise, and other therapeutic interventions would be quite valuable. We performed MR imaging and spectroscopy at 3T to assess their ability to monitor interventions while assessing a short-term change in liver lipids following two seven-day diets – one high in complex carbohydrates and the other high in simple sugars and expected to be lipogenic [2].

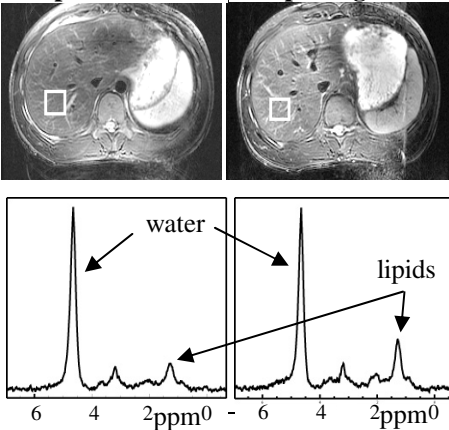
### Methods

MR imaging and spectroscopy were performed in 6 male volunteers at the same time of day at baseline and after each of two successive, seven-day diet periods. Four subjects received a complex carbohydrate diet for the first period, then an isocaloric, simple sugar-based diet (expected to be lipogenic) for the second period. Two subjects received the diets in reverse order. All subjects were carefully monitored in a metabolic ward for the duration of the study. MR anatomic imaging and spectroscopy were acquired on a GE 3T scanner. <sup>1</sup>H MRS was obtained in a 20cc voxel in the liver, placed to avoid blood vessels and away from the edges of the liver in all dimensions. The proton MR spectroscopy was acquired using a 64 acquisition time series of PRESS single voxels (20cc, TR/TE=2500/37). Unsuppressed water spectra with 8 acquisitions also were acquired at each location. Each spectrum was Fourier transformed, baseline subtracted, and phase and frequency corrected. Any data with artifacts were removed [4] and the remaining spectra were averaged for each location. Peaks attributed to CH<sub>2</sub> and CH<sub>3</sub> portions of lipids, ranging from 0.8 – 2.7ppm, were integrated. The unsuppressed water peak at 4.6ppm was also integrated. The total lipid to unsuppressed water peak ratio was computed for the three timepoints.

### Results

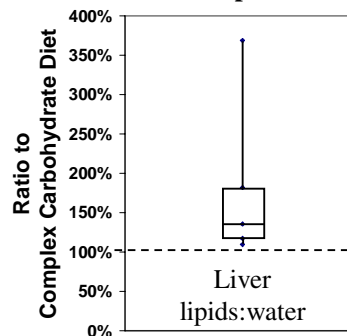
Figure 1 shows representative MR images and liver spectra, illustrating higher liver lipid levels after the simple-sugar based diet. All subjects had a higher level of liver lipids after the simple-sugar diet versus the complex-carbohydrate diet (median lipid/water=0.039 vs. 0.017, respectively, p=0.031, Wilcoxon Sign-Rank Test) (Figure 2). There was variability in the amount of increase, ranging from 110% to 370% of the complex-carbohydrate diet values. There was no correlation between the magnitude of the increase in liver lipids on the simple sugar-diet and the baseline liver lipid levels.

### Complex Carb Diet Simple-sugar Diet



**Figure 1** – MRI showing MRS location (top) and averaged spectrum (bottom) from a subject after the complex-carbohydrate diet (left) and after the simple-sugar based diet (right). Note the higher lipid peak in the spectrum shown on the right (lipids:water = 138% of the complex-carbohydrate diet).

### Effect of Simple-Sugar Diet on Liver Lipids



**Figure 2** – Liver lipids: water after a seven-day simple-sugar diet. Results are graphed as a percent of values after a seven-day complex-carbohydrate diet. The box indicates the median, first and third quartiles, with the bars indicating the extreme values.

### Discussion

This pilot study demonstrated a significantly higher level of liver lipids after a simple-sugar based diet as compared to after a complex carbohydrate-based diet. This was consistent in all six subjects studied. In conclusion, dietary carbohydrate quality can have significant effects on liver fat which can be non-invasively detected by <sup>1</sup>H MRS after periods as short as seven days.

**References** 1. Neuschwander-Tetri BA, Caldwell SH. *Hepatology* 2003;37(5):1202-1219. 2. Faeh D, et al. *Diabetes* 2006;54:1907-1913. 3. Longo R, et al. *Invest Radiol* 1993; 28(4): 297-302. 4. Noworolski SM, et al. *ISMRM* 2005;1:336.

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