Fat contents of human liver, pancreas and kidney

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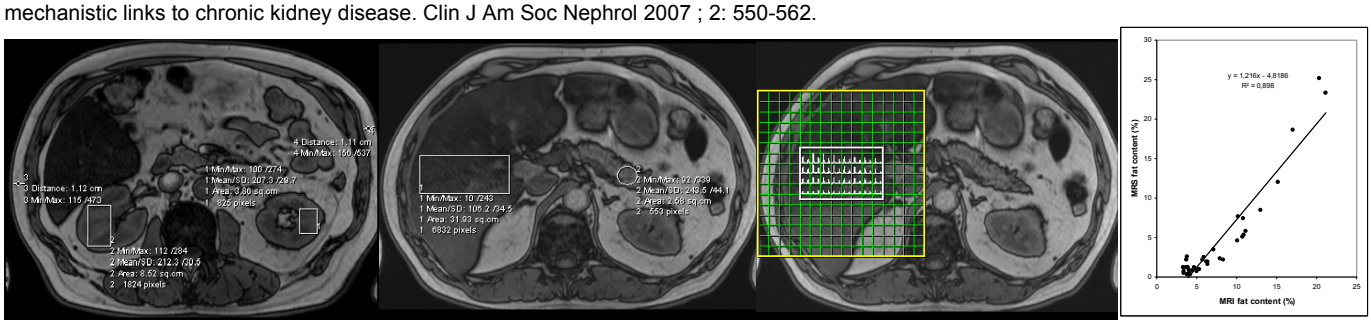
Purpose Experimental data indicate that renal lipid accumulation plays a role in the pathogenesis of renal disease (**1**). Whereas liver steatosis has been quantified in multiple MRI studies, and pancreatic lipomatosis (**2-4**) and muscle fattening (**2**) in some, quantitative documentation of the content of fat in human kidneys *in situ* appears to be lacking entirely. The purpose of this study was to quantify the lipid contents of kidney, liver, and pancreas tissue in volunteers with diverse body weights, and to assess the relationship in fat contents between the different organs.
Methods Multivoxel MR spectroscopy and a previously (**5**) validated gradient echo MRI adaptation of Dixon's two-point technique (**6**) were used to quantify liver fat in 36 healthy adults (Fig. A-D). The body mass index (BMI) of the volunteers ranged from 20 to 42.9 kg/m^2 with a mean value of 27.5 kg/m^2 (24 man,12 women; mean age = 44 years; range: 22 – 64 years). In short, breath-hold T_1 weighted dual flip angle gradient echo MR images were acquired with 6 mm slice thickness, section gap 0 mm, matrix 256x160 and a repetition time (TR) of 155 ms, and TEs of 2.4 ms (OP) and 4.8 ms (IP). Flip angles of 20° and 70° were used to generate intermediate weighted and T_1-weighted images, respectively. Images were corrected for T_2^* decay: $S_{corrected} = S\,e^{\frac{\tau}{T2^*}}$,where τ is the echo time difference between IP and OP images, and *S* represents the signal intensity in a defined region of interest (ROI). Under these conditions τ = 2.4 ms combined with T_2^* = 19.44, calculated from the mean spectral line width of the water peak in human liver measured by MRS in the 36 volunteers, gave a correction factor of 1.13 for S_{ip} relative to S_{op}. An algorithm, modified to prevent occasional mix-ups of water and fat signals (**5**), was used for estimating liver fat content.
Results After correction of all MRI data for a systematic overestimation inherent to the MRI method (Fig.D), the mean fat content of liver, pancreas and kidney was 4.4%, 4.0% and 0.8%, respectively. BMI, a measure of overall fat, correlated significantly with the amount of subcutaneous fat, liver fat content and pancreas fat content (r=0.77, r=0.52 and r=0.35, respectively; *P*<0.05). The amount of subcutaneous fat also correlated significantly with liver fat content and pancreas fat content (r=0.45 and r=0.44, respectively; *P*<0.01). Kidney fat content correlated with none of the other parameters, indicating that renal lipid accumulation, unlike the coupled accumulations of fat in liver and pancreas (r=0.43; *P*<0.01), is not observed in obese healthy subjects.
Discussion Our observation of significant correlation between the liver and pancreas fat contents according to MRI (r=0.43; P<0.01) is in disagreement with two recent studies that, however, included smaller numbers of volunteers than our study (17 and 15 vs. 36) (**4,7**). The significant correlations of BMI with both liver and pancreas fat content in this study are in agreement with a previous report (**7**). Why would obese subjects tend to accumulate fat in liver and pancreas and not in the kidney, despite the probability that obesity and the metabolic syndrome are involved with initiation of chronic kidney disease (**1,7**)? Our hypothesis is that due to specific requirements such as albumine loading needed to facilitate the uptake of fat into kidney tissue, obese but otherwise healthy subjects may not offer circumstances allowing for the accumulation of fat in kidney.
References **1.** Bobulescu IA Debree M, Zhang J, et al. Effect of renal lipid accumulation on proximal tubule Na+/H+ exchange and ammonium secretion. Am J Renal Physiol 2008;294:F1315-F1322. **2.** Kovanlikaya A, Mittelman SD, Ward A, et al. Obesity and fat quantification in lean tissues using three-point Dixon MR imaging. Pediatr Radiol 2005;35:601-7. **3.** Raeder H, Haldorsen IS, Ersland L, et al. Pancreatin lipomatosis is a structural marker in nondiabetic children with mutations in carboxyl-ester lipase. Diabetes 2007;56:444-9. **4.** Schwenzer NF, Machann J, Martirosian P, et al. Quantification of pancreatic lipomatosis and liver steatosis by MRI: comparison of in/opposed phase and spectral-spacial excitation techniques. Invest Radiol 2008;43:330-7. **5**.Irwan R, Edens MA, Sijens PE. Assessment of the variation in fat content in normal liver using a fast MR imaging method in comparison with the results obtained by spectroscopic imaging. Eur Radiol 2008;18:806-813. **6.** Dixon WT. Simple proton spectroscopic imaging. Radiology 1984;153:189-94. **7**. Wahba IM, Mak RH. Obesity and obesity-initiated metabolic syndrome: mechanistic links to chronic kidney disease. Clin J Am Soc Nephrol 2007 ; 2: 550-562.



A B C D

Figures: Regions of interest on MRI (OP series with 70º pulse angle and TE=2.4 ms) for A) kidneys , B) liver and pancreas and C) the corresponding MRS result for the same liver VOI. The MRS results were used to correct all MRI-determined fat contents according to the equation given in D

Table: Correlation (r_s) between measures of obesity, and MRI fat measurements in kidney, liver and pancreas after correction by MRS

(n=36)	Mean ± sd	BMI	Subc.fat	Kidney fat	Liver fat	Pancreas fat
BMI	27.5 ± 5.2	-------	0.765**	0.264	0.517**	0.349*
Subcut. Fat (cm)	1.4 ± 0.8	0.765**	-------	0.256	0.447**	0.442**
Liver fat (%)	4.4 ± 5.8	0.517**	0.447**	0.231	-------	0.428**
Pancreas fat (%)	4.0 ± 4.5	0.349*	0.442**	0.081	0.428**	-------
Kidney fat (%)	0.8 ± 1.0	0.264	0.256	-------	0.231	0.081

* = p<0.05, ** = p<0.01.