In vivo measurement of hepatic lipid composition by proton magnetic resonance spectroscopy

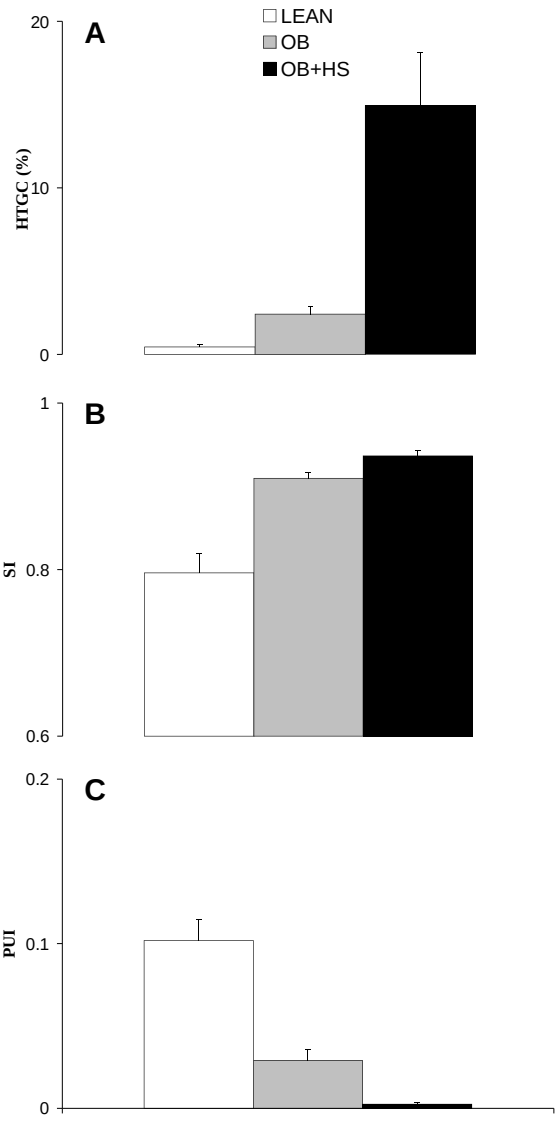
N.A. Johnson,[1] D.W. Walton,[2] T. Sachinwalla,[2] C.H. Thompson,[3] K. Smith,[1] P. Ruell,[1] S.R. Stannard[4] and J. George,[5] [1]Discipline of Exercise and Sport Science, The University of Sydney, East Street, Lidcombe, NSW 2141, Australia, [2]Department of Magnetic Resonance, Rayscan Imaging, 41-43 Goulburn Street Liverpool, NSW 2170, Australia, [3]Department of Medicine, Flinders University, Bedford Park, SA 5042, Australia, [4]Institute of Food, Nutrition and Human Health, Massey University, Palmerston North Private Bag 11222, New Zealand and [5]Storr Liver Unit Westmead Millennium Institute, The University of Sydney, Westmead Hospital, Westmead, NSW 2145, Australia.

Non-alcoholic fatty liver is frequently observed in obese individuals, yet the factors which predict its development and progression to liver disease are poorly understood. It has been proposed that an increase in hepatic saturated fatty acids and/or a decrease in polyunsaturated fatty acids predisposes to hepatic triglyceride accumulation and inflammation.

We assessed the efficacy of using proton magnetic resonance spectroscopy (^{1}H-MRS) to non-invasively measure hepatic lipid composition. Hepatic lipid unsaturation index (UI), a surrogate saturation index (SI) and polyunsaturation index (PUI) measured by ^{1}H-MRS were in agreement with those expected in oils of known composition. Hepatic triglyceride concentration (HTGC) and composition were then measured *in vivo* in healthy lean (LEAN) men (n = 10), obese men with normal HTGC (OB) (n = 8) and obese men with hepatic steatosis (OB+HS) (n = 9).

HTGC was significantly higher in OB+HS (14.9 ± 3.2 %) *versus* OB (2.4 ± 0.4 %) and LEAN (0.4 ± 0.1 %) (Figure *A*; $p < 0.01$ for both). SI was significantly higher in OB+HS (0.94 ± 0.01) and OB (0.91 ± 0.01) *versus* LEAN (0.80 ± 0.02) (Figure *B*; $p < 0.01$ for both). PUI was significantly lower in OB+HS (0.003 ± 0.001) and OB (0.029 ± 0.001) *versus* LEAN (0.102 ± 0.013) ($p < 0.01$), and significantly lower in OB+HS *versus* OB (Figure *C*; $p < 0.05$).

These findings demonstrate that hepatic lipid composition can be non-invasively measured by ^{1}H-MRS, and that obese men with and without hepatic steatosis exhibit a significant increase in SI and a decrease in PUI when compared with healthy lean men. Furthermore, obese men with hepatic steatosis showed specific depletion of polyunsaturated fatty acids (PUFAs) when compared with obese men with normal HTGC. These data are consistent with those derived by needle biopsy and histology by Araya *et al.* (2004). While a cause-effect relationship cannot be inferred from our cross-sectional research design, our finding is consistent with the postulation that dilution of hepatic lipid PUFAs promotes triglyceride accumulation by reducing triglyceride export and inhibiting lipid oxidation (Videla *et al.*, 2004). Further longitudinal research is required to confirm this hypothesis and the suggestion that this phenotype might further predispose to inflammation (Videla *et al.*, 2004).



Araya J, Rodrigo R, Videla LA, Thielemann L, Orellana M, Pettinelli P & Poniachik J. (2004) *Clinical Science*, **106:** 635-643.

Videla LA, Rodrigo R, Araya J & Poniachik J. (2004) *Free Radical Biology and Medicine*, **37:** 1499-1507.