

A 7 Day Low v High Glycaemic Index Diet Reduces Liver Fat Content

S Bawden^{1,2}, M Stephenson³, K Hunter⁴, M Taylor⁵, PG Morris², L Marciani¹, IA Macdonald⁶, GP Aithal¹, and PA Gowland²

¹NIHR Nottingham Digestive Diseases Biomedical Research Unit, Nottingham University Hospitals NHS Trust and University of Nottingham, Nottingham, United Kingdom, ²Sir Peter Mansfield Imaging Centre, University of Nottingham, Nottingham, United Kingdom, ³Agency for Science, Technology and Research, Singapore, ⁴Unilever Discover, Bedfordshire, United Kingdom, ⁵Faculty of Human Nutrition, University of Nottingham, United Kingdom, ⁶School of Life Sciences, University of Nottingham, United Kingdom

Background: Glycaemic index (GI) is a way of ranking carbohydrates according to the postprandial effect on blood glucose levels and has received increasing interest in recent years [1, 2]. Low glycaemic index diets have been considered potentially beneficial in diabetes, coronary heart disease and obesity [3] and cohort studies have shown correlations between dietary glycaemic index and non-alcoholic fatty liver disease (NAFLD) [4]. ¹H MRS provides a powerful, well validated method of measuring liver fat fractions non-invasively with numerous benefits over biopsies [5]. In this study, the effects of 7 day low (LGI) v high (HGI) glycaemic index diet on hepatic lipid stores were investigated using ¹H MRS.

Study design: After obtaining ethical approval, 8 healthy males were recruited following informed consent (sedentary, non-smokers, no metabolic disorders, Age=20.1±0.4 yr, BMI=23.0±0.9 kg m⁻²). Subjects were investigated using a two way randomized cross over study design with a 7 day HGI v LGI dietary intervention and >4 week washout (isocaloric, Fat =28%, Protein=12%, CHO = 61%, [6]) (Fig 1). Subjects were provided with all foods for the diet week and underwent pre and post diet study test days.

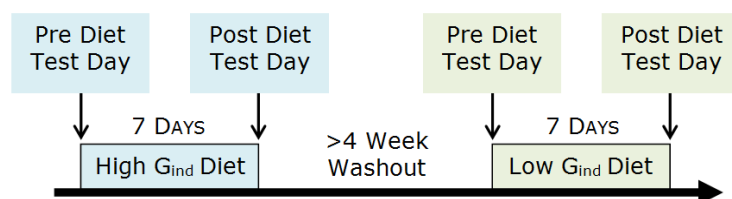


Figure 1. Study Design Overview.

Test Day: During the test day subjects arrived at the test centre between 7:30 and 8:30am and were fasting from 10pm the previous evening. ¹H MRS were acquired for fasted liver lipid measurements, after which subjects consumed either a HGI or LGI test breakfast. Blood samples were taken regularly for the following 300 minutes to confirm glycaemic response and a final ¹H MRS scan was taken at the end of the test day at t = 360 minutes.

MR Protocol: All measurements were performed on a Philips Achieva 3T scanner and ¹H MRS acquired using a Philips XL torso coil. Scout images were obtained and used for voxel placement (30x30x30mm). ¹H MRS were obtained using a respiratory triggered, water suppressed, PRESS sequence with varying TE (BW=2 kHz, samples=1024, TR=5000ms, NSA=40, TE=40ms, 50ms, 60ms, 80ms, total scan ~10 mins). A water unsuppressed spectrum was acquired with half the averages as an internal reference peak to determine lipid fat fractions. Spectra were phase corrected and the area of the main CH₂ peak (~1.3 ppm) and water peak were quantified using a peak fitting algorithm. Fat fractions were calculated using standard formula described previously [7] and corrected for T₂ relaxation using the spectra acquire at varying TE

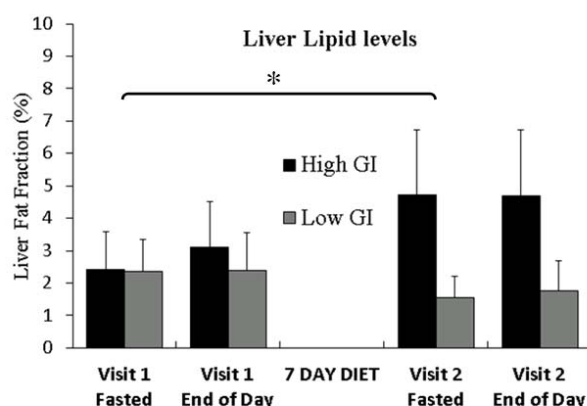


Figure 2. Changes in Liver Lipid levels *P<0.05

Result: Blood samples confirmed a high and low glycaemic response for respective test meals. On the pre diet test day, fasted liver fat fractions were consistent for both HGI and LGI arms (HGI = 2.4 ± 1.2 %; LGI = 2.4 ± 1.0 %; P = 0.82) and end of day lipid levels were within error of fasted levels (FIG 2). Following the 7 day diet, fasted liver fat fractions increased for HGI (4.7 ± 2.0 %) compared with LGI (1.6 ± 0.7 %) reaching statistical significance (two way F-test, P < 0.05) and this effect was consistent across the test day.

Conclusions: This study used a simple MRS protocol to show the beneficial impact on liver lipid levels of a LGI compared with HGI diet after only 7 days. This is consistent with other cohort studies [4] and has important applications in the prevention and control of metabolic disease. Further studies should explore longer term interventions and patient groups.

Acknowledgments: We wish to thank Unilever and BBSRC for funding this study and the NIHR NDD BRU for continuing support.

1.Jenkins D.J.A. *et al.*, Am J Clin Nutr (1981) 34, 362-366; 2.Foster-Powell K. *et al.*, Am J Clin Nutr (2002) 76, 5-56; 3.Jenkins D.J.A. *et al.*, Am J Clin Nutr (2002) 76, 266s-273s; 4.Valtueña S. *et al.*, Am J Clin Nutr (2006) 84, 136-142; 5.Hamilton G. *et al.*, Nmr in Biomedicine (2011) 24, 784-790; 6.Morgan L.M. *et al.*, Brit J Nutr (2012) 108, 1286-1291; 7.Stephenson M.C. *et al.*, NMR in Biomedicine (2013) 26, 1518 - 1526;