

Winter Meeting, 4-5 December 2018, Optimal diet and lifestyle strategies for the management of cardio-metabolic risk

The effect of dietary carbohydrate manipulation on low-density lipoprotein-cholesterol and its associated cardiometabolic risk

D. McCullough^{1,2}, T. Harrison¹, K.E. Lane¹, L.M. Boddy², C.E. Stewart², K.J. Enright¹, F. Amirabdollahian³, M.A. Schmidt⁴ and I.G. Davies¹

¹School of Sport Studies, Leisure and Nutrition, Liverpool John Moores University, Liverpool, L17 6BD,

²School of Sports and Exercise Sciences, Liverpool John Moores University, Liverpool, L3 3AF,

³School of Health Sciences, Liverpool Hope University, Liverpool, L16 9JD and

⁴Advanced Pattern Analysis and Countermeasures Group, Research Innovation Centre, Colorado State University,

Fort Collins, USA

Cardiometabolic (CM) risk is typically increased with elevated low-density lipoprotein-cholesterol (LDL-C) and insulin resistance (IR)^(1,2). A low carbohydrate, high fat (LCHF) diet has been shown to increase LDL-C albeit improving other CM risk factors such as high-density lipoprotein-cholesterol and triglycerides⁽³⁾. There are several subclasses of LDL, in which some may be more atherogenic such as small-dense LDL (sdLDL)⁽⁴⁾. Few studies have compared a LCHF diet to a high carbohydrate, lower fat diet under *ad libitum* conditions, particularly their effect on sdLDL:LDL-C ratio. The current feasibility study intends to address such gap. Furthermore, to the authors' knowledge, the effect of the new reduced sugar UK Eatwell guide on CM health is yet to be investigated. Therefore, the aims of this investigation was to measure the effect of a low carbohydrate (LC) diet vs. a high carbohydrate (HC) diet on LDL-C, sdLDL-C and IR in 16 (9 males, 7 females) healthy Caucasian adults aged 19-64.

The study received ethical approval from Liverpool John Moores University Research Ethics Committee (16/ELS/029) and was registered with ClinicalTrials.gov (Ref. NCT03257085). Participants were randomly assigned to either a HC diet (the UK Eatwell guidelines; ≥50 % of energy from carbohydrates) (n = 8, 5 males, 3 females), or a LC diet (consume <50 g/day of carbohydrates) (n = 8, 4 males, 4 females) for 8 weeks. At 0, 4 and 8 weeks blood was collected after a 12 hour fast, processed for plasma and stored at -80°C. Plasma was analysed by an automated chemistry analyser (Daytona, Randox Laboratories Ltd, UK) for LDL-C, sdLDL-C and glucose levels. Insulin levels were measured using immunoassay technology (Randox Evidence Investigator™ Metabolic Syndrome Arrays I). The homeostatic model assessment (HOMA) was used to calculate IR. Statistical analysis was undertaken using IBM SPSS 24®. Normally distributed data underwent a 2 × 3 mixed ANOVA to investigate significant differences for effect of time and interaction effect. Spearman's correlation was used to analyse the association between variables.

LDL-C non-significantly (P = 0.141) increased by 0.22 (mmol/L) within the LC group whereas the HC group remained unchanged. Within the LC group sdLDL-C levels decreased by 0.14 (mmol/L); however, sdLDL-C in the HC group increased by 0.07 (mmol/L) resulting in a significant interaction effect (P = 0.026). The ratio of sdLDL:LDL-C therefore decreased by 0.06 in the LC group and increased by 0.01 in the HC group resulting in a significant interaction effect (P = 0.003). HOMA significantly improved (P = 0.008) similarly in both groups but the change in HOMA was only significantly (P = 0.008) associated with the change in sdLDL: LDL-C within the LC group.

In conclusion, the study provided preliminary evidence showing that a LC diet may improve CM health via positive changes in LDL composition with an associated reduction in IR. Although the HC diet improved IR, the unfavourable changes in LDL size may indicate only a partial improvement in CM health. Further research is required on how dietary carbohydrate manipulation can improve LDL composition and overall CM health. The use of sdLDL:LDL-C ratio may be of more importance when assessing improvements in CM health compared to LDL-C alone.

- 1. Fulcher J, O'Connell R, Voysey M, et al. Lancet, 2015; 385, 1397-405.
- Gast KB, Tjeerdema N, Stijnen T, et al. PloS one. 2012 28, 7(12):e52036.
 Mansoor N, Vinknes KJ, Veierød MB et al. Brit J Nutr. 2016 115, 466–479.
- 3. Mansoor N, Vinknes KJ, Veierod MB *et al. Brit J Nuir.* 2016 115, 44. 4. Hirayama S & Miida T. *Clin Chim Acta*. 2012 **414**, 215–224.

https://doi.org/10.1017/S002966511900020X Published online by Cambridge University Press