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Dietary carbohydrate intake, visceral adipose tissue and associated markers of cardiometabolic risk

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Risk of cardiometabolic (CM) disease is characterised by elevated visceral adipose tissue (VAT) and a number of associated biomarkers[1]. Some dietary carbohydrates (CHO) have been found to contribute to VAT accumulation[2]. Little is known about the impact of following a low-carbohydrate diet versus a high-carbohydrate diet on VAT, adiponectin (ADPN), leptin (LEPT) and leptin:adiponectin ratio (LAR). The aim of this investigation was to assess the impact of dietary carbohydrates (CHO) on VAT and emerging CM risk markers in a sample of 10 healthy normal-weight and overweight Caucasian adults aged 32–60 (80 % male) at increased CM risk[3]. This pilot 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 allocated to one of two groups and asked to either consume <50 g/d of dietary CHO (low-carb (LC)) or to follow the UK dietary guidelines and obtain >50 % energy from CHO (high-carb (HC)) for a duration of 8 weeks. VAT was analysed via bioelectrical impedance (SECA mBCA 515). Blood plasma samples were collected at baseline (BL), interim point (IP) and endpoint (EP) after a 12-hour overnight fast, immediately processed and frozen at -80°C. Thawed plasma samples were analysed via immunoassay technology (Randox Evidence Investigator™ Metabolic Syndrome Arrays I and II) for ADPN and LEPT levels. Statistical analysis was undertaken using IBM SPSS 24®.

Parametric data was analysed via two-way mixed ANOVA; non-parametric data was analysed via Mann-Whitney U test and Friedman test. Average daily carbohydrate intake in the LC group was 44·2 g at IP and 48·9 g at EP.

There were no significant differences between groups at any time point for ADPN, LEPT, LAR or VAT and no significant interactions for time or group*time for ADPN, LEPT or LAR. However, in the LC group VAT decreased significantly between baseline and endpoint by 15 % ($p = ·015$) Over the course of the intervention ADPN and LEPT decreased non- significantly (by 4 % and 70 % respectively) in the LC group, whilst increasing non-significantly in the HC group (9 % and 65 % respectively). LAR increased in the HC group throughout the study, whilst LAR in the LC group decreased albeit not significantly.

	VAT (litre)						ADPN (ng/mL)			LEPT (ng/mL)			LAR		
	BL		IP		EP		Median			Median			Median		
	M	SD	M	SD	M	SD	BL	IP	EP	BL	IP	EP	BL	IP	EP
LC	4·1[a]	1·2	3·8	1·3	3·5[a]	1·2	8·9	8·6	8·5	3·96	1·64	1·20	0·45	0·19	0·14
HC	2·7	0·1	1·6	0·3	2·5	0·1	11·3	13·4	12·3	0·97	1·1	1·60	0·07	0·07	0·46

ADPN = adiponectin, BL = baseline, EP = endpoint, HC = high-carbohydrate, moderate fat diet, IP = interim point, LAR = leptin:adiponectin ratio, LEPT = leptin, LC = low-carbohydrate, high-fat diet, VAT = visceral adipose tissue, [a]$p = ·015$. NB: interquartile ranges not provided for median values due to missing data.

Higher LAR has been found to be a marker of increased CM risk[4]. In conclusion, while the significant reduction in VAT in the LC group corresponds with the reduction of LAR further evidence is required to corroborate these findings. Previous evidence for LC is supportive for improved CM health from various biomarkers[5]; LAR should be considered as a useful endocrine addition for future LC studies.

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