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The RISCK study: impact of the amount and type of dietary fat and carbohydrate on the plasma phospholipid-fatty acid status of free-living individuals

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The metabolic syndrome (MS) affects $\leq 25\%$ of the UK population⁽¹⁾ and confers substantial risk of CVD. A key scientific and public health question is whether reducing intakes of SFA via low-fat–high-carbohydrate (CHO) diets, or by moderate-fat diets in which SFA are substituted with MUFA, have differential effects on risk factors for MS. Moreover, there is a further question to be addressed in relation to the type of dietary CHO, with current interest in the role of foods with a low glycaemic index (GI; LGI).

RISCK is a multi-centre randomised controlled dietary intervention study of 720 subjects at increased risk of MS. The study design has been previously reported⁽²⁾. After a run-in period of 4 weeks following the reference diet (high-SFA (HS)–high-GI (HGI) containing 38% energy (%E) fat, 18%E SFA, 10%E MUFA, 6%E PUFA) subjects were randomised to either the reference diet or one of four isoenergetic diets for 24 weeks (high-MUFA (HM)–HGI or HM–LGI) containing 38%E fat, 10%E SFA, 20%E MUFA, 6%E PUFA, or low-fat (LF)–HGI or LF–LGI containing 28%E fat, 10%E SFA, 11%E MUFA, 6%E PUFA). Total plasma phospholipid-fatty acid concentrations were determined at baseline and at the end of the study by GC analysis.

The total plasma phospholipid-fatty acid analysis was performed as a compliance measure to monitor the intake of dietary fat in the five intervention groups. Changes in the fatty acid profiles from the baseline visit to the follow-up visit were consistent with dietary prescription. The raised plasma phospholipid-MUFA levels (predominantly oleic acid) in the HM dietary groups appeared to increase at the expense of SFA. Changes in SFA and MUFA were driven by changes in palmitic and oleic acid respectively, which were the primary dietary sources of MUFA and SFA. No significant changes in other fatty acid classes such as *n*-3 PUFA, *n*-6 PUFA and *trans*-FA were observed.

Table. Plasma phospholipid-SFA and -MUFA levels (% total fatty acids) at baseline and follow-up

	HS–HGI		HM–HGI		HM–LGI		LF–HGI		LF–LGI	
	Median	IQR	Median	IQR	Median	IQR	Median	IQR	Median	IQR
SFA										
<i>n</i>		78		106		103		102		109
Baseline	44.3	43.4, 45.4	44.7	43.5, 45.9	45.0	43.7, 45.7	44.6	43.2, 45.6	44.6	43.2, 45.6
Follow-up	44.3	43.3, 45.5	44.4	43.2, 45.5	44.4	43.3, 45.5	44.7	43.7, 45.6	44.7	43.5, 45.8
Change	0.19	–0.15, 0.53	–0.40	–0.72, –0.09	–0.42	–0.73, –0.12	0.18	–0.13, 0.49	–0.19	–0.49, 0.12
					<i>P</i> = 0.039*, <i>P</i> = 0.022†					
MUFA										
<i>n</i>		82		107		112		110		119
Baseline	11.3	10.4, 12.5	11.6	10.4, 12.5	11.4	10.6, 12.0	11.5	10.7, 12.5	11.3	10.6, 12.6
Follow-up	11.2	10.5, 12.5	12.4	11.0, 13.6	12.3	11.5, 13.1	11.8	10.7, 12.6	12.0	10.9, 12.8
Change	0.07	–0.22, 0.36	0.82	0.56, 1.09	0.88	0.61, 1.15	0.18	–0.04, 0.39	0.27	0.04, 0.49
					<i>P</i> = 0.0000*, <i>P</i> = 0.0000†					

Mean change and 95% CI (IQR) calculated on transformed scale, but expressed as change from median value at baseline; outliers first removed; Transformations: cubed (*n*-6 PUFA), log(*n*-3 PUFA). *From ANCOVA of transformed baseline measures on transformed follow-up measures adjusted for gender, centre, ethnicity and baseline waist, (log)HDL, age; outliers first removed.†Also adjusted for weight change.

There was clear concordance between the plasma phospholipid-fatty acid levels and reported dietary intake data providing supporting evidence that the dietary fatty acid manipulation in the RISCK study was successfully achieved.

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1. Tonkin R (2003) The X factor: obesity and the metabolic syndrome. <http://www.the-ba.net/NR/rdonlyres/9DE73A6C-C61F-4007-9373-2BCBFC08F92B/0/Report.pdf>
2. Jebb SA, Frost G, Griffin BA, Lovegrove JA, Moore C, Sanders T & Williams CM (2007) *Nutr Bull* 32, 154–156.