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Impact of the amount and type of fat and carbohydrate on vascular function in the RISCK study

Tom Sanders[1], Fiona Lewis[1], Gary Frost[2], Louise Goff[2] and Phil Chowienczyk[1]
[1]*King's College London, London, UK and* [2]*Imperial College London, London, UK*

RISCK is a multi-centre randomised controlled dietary intervention in subjects at increased risk of metabolic syndrome[(1)]. The results are reported of measurements of vascular function from a substudy conducted on cohorts recruited at King's College London and Imperial College London. Altered vascular function is one of the hallmarks of metabolic syndrome[(2)]. It is currently believed that the impaired vascular function is a consequence of increased production of reactive oxygen species that decrease the bioavailability of NO, a potent anti-atherogenic molecule with vasodilator, anti-platelet, anti-leucocyte and anti-proliferative actions[(3)]. Following a 1-month run-in period on a diet high in SFA, measurements were made of: endothelial function using the flow-mediated dilatation (FMD) technique; endothelial-independent vasodilation following 25 μg sublingual glycerol trinitrate (GTN); aortic stiffness using the carotid–femoral pulse wave velocity (PWV); vascular tone using the digital volume pulse stiffness index (SI). Further measures were carried out following a 6-month dietary intervention with a SFA-rich diet similar to the run-in diet, a high-MUFA diet or a low-fat (LF) diet. The target intake for total fat was 38% energy (%E) for the SFA and MUFA diets and 28%E for the LF diets. The MUFA and LF diets were designed to reduce dietary SFA to 10%E with a planned MUFA intake of 18%E on the MUFA diet. The study had statistical power to detect a change of 1% in FMD. The results are shown in the Table.

	SFA		MUFA		LF	
	Mean	SD or 95% CI	Mean	SD or 95% CI	Mean	SD or 95% CI
FMD (%)	(*n* 29)		(*n* 44)		(*n* 37)	
Baseline	6.6	2.2	6.9	2.3	6.6	2.2
Follow-up	6.9	2.3	6.7	2.4	6.7	2.4
Change	0.3	−0.5, 1.1	−0.2	−1.0, 0.5	0.1	−0.4, 0.7
GTN (%)	(*n* 29)		(*n* 44)		(*n* 36)	
Baseline	11.3	3.2	11.4	3.1	11.6	4.4
Follow-up	11.1	3.6	11.7	3.2	11.3	3.8
Change	−0.2	−1.4, 0.9	0.3	−0.8, 1.4	−0.3	−1.7, 1.1
PWV (m/s)	(*n* 27)		(*n* 42)		(*n* 36)	
Baseline	8.2	1.6	7.7	1.8	7.6	1.3
Follow-up	8.0	1.5	7.8	1.5	7.5	1.3
Change	−0.2	−0.6, 0.2	0.2	−0.2, 0.5	−0.1	−0.4, 0.3
SI (m/s)	(*n* 27)		(*n* 41)		(*n* 35)	
Baseline	7.9	1.6	8.1	2.0	8.1	2.3
Follow-up	8.5	2.0	8.1	2.1	7.5	1.9
Change	0.6	−0.2, 1.3	−0.1	−0.7, 0.5	−0.6	*−1.2, 0.1

Value was significantly different from that for the SFA treatment (Bonferroni's multiple comparison test): $*P<0.05$.

The dietary intervention did not affect endothelium-dependent or -independent vasodilatation of the brachial artery as assessed by ultrasound. This finding suggests that the diets did not affect the production of NO. Large-artery stiffness as measured by PWV was not affected by treatment. However, there was a borderline-significant ($P=0.05$) difference between the SFA diet and the LF diet on SI, which although it is correlated with PWV is also an indicator of small-vessel reactivity. The findings of the present study do not support previous conclusions that a diet high in SFA impairs FMD by about 50% compared with high-MUFA or LF diets in healthy subjects.

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