

Successful Manipulation of the Quality and Quantity of Fat and Carbohydrate Consumed by Free-Living Individuals Using a Food Exchange Model^{1,2}

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Abstract

Our objective in this study was to develop and implement an effective intervention strategy to manipulate the amount and composition of dietary fat and carbohydrate (CHO) in free-living individuals in the RISCK study. The study was a randomized, controlled dietary intervention study that was conducted in 720 participants identified as higher risk for or with metabolic syndrome. All followed a 4-wk run-in reference diet [high saturated fatty acids (SF)/high glycemic index (GI)]. Volunteers were randomized to continue this diet for a further 24 wk or to 1 of 4 isoenergetic prescriptions [high monounsaturated fatty acids (MUFA)/high GI; high MUFA/low GI; low fat (LF)/high GI; and LF/low GI]. We developed a food exchange model to implement each diet. Dietary records and plasma phospholipid fatty acids were used to assess the effectiveness of the intervention strategy. Reported fat intake from the LF diets was significantly reduced to 28% of energy (%E) compared with 38%E from the HM and LF diets. SF intake was successfully decreased in the HM and LF diets to $\leq 10\%$ E compared with 17%E in the reference diet ($P = 0.001$). Dietary MUFA in the HM diets was $\sim 17\%$ E, significantly higher than in the reference (12%E) and LF diets (10%E) ($P = 0.001$). Changes in plasma phospholipid fatty acids provided further evidence for the successful manipulation of fat intake. The GI of the HGI and LGI arms differed by ~ 9 points ($P = 0.001$). The food exchange model provided an effective dietary strategy for the design and implementation across multiple sites of 5 experimental diets with specific targets for the proportion of fat and CHO. *J. Nutr.* 139: 1534–1540, 2009.

Introduction

Metabolic syndrome affects as many as 25% of the U.K. population (1) and confers substantial risk of cardiovascular disease. Evidence exists that the amount and quality of dietary fat can modify features of metabolic syndrome, including blood lipids (2), insulin resistance (3), hypertension, and endothelial function (4–7). A key scientific and public health question is whether reducing intakes of saturated fats (SF)¹⁰ via low-fat, high-

carbohydrate (CHO) diets, or by moderate-fat diets in which SF are substituted with monounsaturated fatty acids (MUFA), have differential effects on risk factors for metabolic syndrome.

Controlled intervention trials in this area are difficult to perform, not least because of the challenge in designing a dietary manipulation strategy to simultaneously address the quality and type of fat and CHO. Few long-term studies have achieved good

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University of Reading: Julie Lovegrove (principal investigator), Christine Williams (coinvestigator), Rachel Gitau (local coordinator). University of Surrey: Bruce Griffin (principal investigator), Margaret Griffin (local coordinator), Nicola Harman (lead for lipid subclasses). The RISCK study was a randomized, controlled, parallel trial, performed in free-living participants at 5 UK centers (University of Reading, Imperial College London, Kings College London, University of Surrey, and the MRC HNR).

¹⁰ Abbreviations used: CHO, carbohydrate; %E, percent of energy; GI, glycemic index; HM/HGI, high-monounsaturated fat/high-glycemic index diet; HM/LGI, high-monounsaturated fat/low-glycemic index diet; HS/HGI, high-SF/high-glycemic index diet; LF, low fat; LF/HGI, low-fat/high-glycemic index diet; LF/LGI, low-fat/low-glycemic index diet; MRC HNR, Medical Research Council Human Nutrition Research; MUFA, monounsaturated fatty acid; NDNS, National Diet and Nutrition Survey; NSP, nonstarch polysaccharide; SF, saturated fat.

compliance with such extensive dietary changes in free-living individuals. This article describes the food exchange model, which was developed and used successfully to implement the dietary manipulation required for the RISCK study. It details the steps taken including an assessment of typical U.K. dietary intakes, consideration of the availability and ease of substitution of study foods with a desirable nutrient composition into an individual's habitual diets, and the development of dietary guidelines that were sustainable over the duration of the study, while allowing some flexibility for an individual's food preferences and lifestyle. The effectiveness of the dietary strategy is also assessed through analysis of dietary records and measurement of plasma phospholipid fatty acid status.

Methods

Study participants and design

The RISCK study was a randomized, controlled, parallel trial performed in free-living participants at 5 U.K. centers (University of Reading, Imperial College London, Kings College London, University of Surrey, and the Medical Research Council Human Nutrition Research [MRC HNR]). The study was approved by the South East Multi-Center Research Ethics Committee (ref: MREC04/MRE01/2). Written informed consent was obtained from all participants. The study design has been described elsewhere (8). In brief, a total of 720 participants selected on the basis of their increased risk or presence of metabolic syndrome were recruited. All participants followed a 4-wk run-in period during which they were prescribed a high-SF, high-glycemic index (GI) (HS/HGI) reference diet. A minimization procedure (controlling for age, gender, waist, and HDL cholesterol) was then used to assign participants to the reference diet for a further 24 wk or to 1 of 4 isoenergetic dietary prescriptions [high MUFA/high GI (HM/HGI), high MUFA/low GI (HM/LGI), low fat (LF)/high GI (LF/HGI), and LF/LGI]. The target intake for total fat was 38% of energy (%E) in the high-SF and both high-MUFA diets and 28%E in the 2 LF diets, which also had a higher target CHO intake (55 vs. 45%E). The 4 intervention diets were designed to reduce SF intake to 10%E, where protein and alcohol were unaltered and the remaining energy was derived from CHO. Participants attended the study centers for 2 half-days of measures after the run-in period and after the 24-wk intervention. The main outcome was a measure of insulin sensitivity with secondary outcomes, including a range of cardiovascular risk markers, the results of which will be reported elsewhere.

Overall dietary intervention strategy

A fundamental tenet of the study was to prescribe appropriate isoenergetic substitutions for the individual dietary regimens while allowing participants to eat ad libitum. To facilitate the required dietary changes and maximize compliance, specific study foods that were components of household meals or recipes were collected by each participant fortnightly from respective study centers in sufficient quantities for the whole household.

Strategy for achieving target fat intakes

A RISCK food exchange model was designed based on data from the National Diet and Nutrition Survey (NDNS) (9) and had a similar format to a strategy used previously (10). The survey showed dietary fat accounted for ~34%E in the mean U.K. diet including alcohol consumption. Through the removal of the major exchangeable dietary sources of fat, it was calculated that ~42% of total dietary fat was accessible (Table 1). The model requires exchangeable fat to be replaced by study foods with a specific fatty acid profile (Tables 2–4). Specially formulated fat spreads, cooking and baking fats, and mayonnaises were provided by Unilever Food and Health Research Institute (Unilever R&D) (Table 5). The products were provided to participants in plain packaging identifiable by a code and were accompanied by specifically chosen commercially available snack foods to replace those normally consumed. The remaining fat replacement was achieved through an appropriate exchange of full-fat or low-fat dairy products.

Strategy for achieving target CHO intakes

To maintain the energy content of the prescribed diet, changes in the amount of CHO were also necessary. The reference diet (HS/HGI) and the HM diets required participants to consume an extra ~4%E from fat compared with the U.K. mean intake; therefore, CHO of equivalent %E was removed daily (Tables 2 and 3). The target for the LF diets required a 6%E reduction in fat compared with the mean U.K. intake; therefore, the energy from the fat removed from the diet was replaced by adding at least 1 extra CHO portion (Table 4).

An exchange system was used to manipulate GI and participants were provided with CHO foods either high or low in GI to substitute into their habitual diet in a similar manner to previous studies (11). Breakfast cereals were provided by Cereal Partners UK, Grampian, Weetabix Ltd., and Sainsbury's Supermarkets Ltd. The impact of these dietary exchanges was modeled on intakes of major contributors to CHO intake derived from the latest NDNS survey in men 35–49 y old (9) and published GI values (12). These data suggest that it is possible to decrease GI by ~13 points in the LF/LGI diet and by ~11 points in the HM/LGI diet. The lower decrease in the high-MUFA diet reflects the limitations imposed by a reduced CHO intake overall.

Appropriate foods to include in the exchange system were identified using GI data derived principally from independent testing undertaken at MRC HNR using the methodology previously described (13). In general, foods included in the exchange model fell within the standard definition of high- and low-GI products with values either >70 or <55.

Implementation of the diets

After standardized training, study dietitians or researchers gave one-on-one dietary advice at the beginning of both the run-in and the intervention diet. At the initial dietary instruction, the investigator assessed the habitual diet of the participant through discussion and reference to a previously completed diet diary and used this to tailor individual advice. Participants were provided with a booklet to supplement the verbal information, which included clear tables of foods to be substituted, target intakes, and advice on food choices when eating out. Asian men and

TABLE 1 The RISCK food exchange model: removal of the major exchangeable sources of dietary fat, based on NDNS data

	Energy		Fat		SF		MUFA		PUFA		CHO	
	Male	Female	Male	Female	Male	Female	Male	Female	Male	Female	Male	Female
NDNS, %E from			33.7	33.9	12.6	12.8	11.3	11.0	5.9	6.1	44.6	46.6
NDNS intake	<i>kJ/d</i>		<i>g/d</i>									
Added fats- oil	376	263	10.0	7.0	0.9	0.7	3.9	2.7	4.7	3.3	0	0
Added fats-spreads	387	261	10.4	6.8	3.9	2.6	3.5	2.2	2.1	1.2	0	0
All milk	485	410	4.3	3.7	2.6	2.1	1.2	0.8	0.3	0.3	11	8.1
Cheese	290	205	5.2	3.7	3.3	2.3	1.2	0.8	0.1	0.03	0	0
Biscuits, cakes, buns, pastries	582	477	6.1	4.3	2.6	1.9	2.0	1.4	0.7	0.5	19.3	14.2
Total of foods	2120	1616	36.0	25.5	13.3	9.6	11.8	7.9	7.9	5.3	30.3	22.3
Intake less fats, milk, and cheese	7558	5212	50.5	35.9	19.2	13.7	17.3	12.3	7.3	5.8	244.7	180.7

TABLE 2 Replacement of exchangeable sources of dietary fat with study foods: diet HS/HGI

Diet HS/HGI	Amount		Energy		Fat		SF		MUFA		PUFA		CHO	
	Male	Female	Male	Female	Male	Female	Male	Female	Male	Female	Male	Female	Male	Female
	g/d		kJ/d		g/d									
Study spread, high SF	20	15	590	444	16.0	12.0	7.7	5.8	4.5	3.2	4.0	3.0	0	0
Study cooking fat	11	6	405	221	11.0	6.0	5.2	2.9	3.7	2.0	2.0	1.1	0	0
Cheese (full fat)	17	12	293	207	5.8	4.1	3.7	2.6	1.6	1.1	0.2	0.2	0.02	0.01
Milk (whole)	230	190	635	525	9.0	7.4	5.5	4.6	2.5	2.1	0.2	0.2	11.0	9.1
CHO portion	-45	-30	-442	-295	-0.9	-0.6	-0.2	-0.1	-0.2	-0.1	-0.2	-0.2	-22.2	-14.8
Study snack (mean of 2)	20	20	433	433	5.7	5.7	3.6	3.6	1.6	1.6	0.2	0.2	12	12
Intake less fats, milk, and cheese			7558	5212	50.5	35.9	19.2	13.7	17.3	12.3	7.3	5.8	244.7	180.7
Total			9472	6747	97.1	70.5	44.7	33.1	31.0	22.2	13.7	10.3	245.5	187.0
%E					38.6	39.3	17.8	18.5	12.3	12.4	5.4	5.7	40.7	43.5

women, a population group predisposed to greater metabolic risk, were a specific target group for RISCK and therefore a separate booklet was developed based on an Asian diet. Participants were advised to keep to the targets set without changing other components of the diet.

Due to the length of the study, specific advice was given for eating during holidays and special occasions (such as Christmas) and no outcome measurements were scheduled immediately after such events.

Dietary fat advice

High-SF diet. For all participants during the run-in and those randomized to the HS/HGI intervention, 2 solid fats were provided: a spread and the other to use in all cooking and baking. Because there was no liquid oil, a salad dressing recipe was suggested using crème fraîche. Participants were asked to replace all dairy products with full-fat options and to use the minimum weekly target for cheese. A choice of 2 snacks (chocolate wafer biscuit or all-butter shortbread) was provided and advice given to replace 1 habitually eaten snack each day.

High-MUFA diets. For participants receiving the HM diets, oil for cooking and salad dressings, study spread, and baking fat were provided. Participants were also asked to replace all dairy products with low-fat varieties. A choice of 2 snacks were provided (muesli bar containing nuts or high-MUFA crisps) to replace 1 snack each day. In addition, participants were asked to eat 1 tablespoon (15 mL) of the provided mayonnaise or 2 tablespoons (30 mL) of hazelnuts per day to increase MUFA intakes. They were also given mixed nuts to snack on during the day. Advice for holidays or eating out included selection of olives, avocados, and nuts over pastry-based snacks and crisps, vegetable-based sauces over cheese- or cream-based sauces, and selection of olive oil-based dressings, sauces, or dips.

LF diets. Participants following the LF diets were asked to replace all dairy products with low-fat varieties, given advice on low-fat cooking and baking, and provided with a choice of 3 low-fat snacks (muesli bar

containing dried fruit, jaffa cakes, or iced gems) to replace habitually eaten snacks such as chocolate or crisps. Participants were asked to make low-fat choices while on holiday or eating out.

Dietary CHO advice

All participants during the run-in diet and those assigned to the high-GI intervention diets were asked to avoid eating pasta, which has a low GI, and were supplied with a choice of high-GI staple foods. These included breakfast cereals, standard sliced whole-meal and white bread, potatoes (especially old potatoes), and Sainsbury's easy-cook basmati rice. Participants following the low-GI diets were asked to avoid eating potatoes and were given a choice of low- or relatively low-GI staples, including all types of pasta, lower GI breakfast cereals, and bread including Tesco Jumbo Porridge Oats and Multigrain Batch Bread. Additional lower GI alternatives, sweet potatoes, egg noodles, and crisp breads were also recommended.

Assessment of dietary intake

On 4 occasions (screening, end of run-in, 12 wk, and 24 wk), participants completed an unweighed 4-d diet diary for 3 wk and 1 weekend day. Portion sizes were estimated using published tables (14).

MRC HNR's in-house database of the nutrient composition of foods consumed in the U.K. diet, DIDO (Diet In, Data Out), was used to calculate specific macronutrient and micronutrient intake. This was based on McCance and Widdowson's 'The Composition of Foods' (15) with additional data obtained from manufacturers. GI values for CHO-containing foods have been incorporated into a newly developed database at MRC HNR, DINO (Diet In, Nutrients Out) for the analysis of dietary GI.

Assessment of plasma phospholipid fatty acid status

Fasted plasma phospholipid fatty acids were measured at the University of Reading. Fatty acids were solvent extracted and isolated using a sep-pac C18 column (Waters Associates) (16). The lipids were then trans-

TABLE 3 Replacement of exchangeable sources of dietary fat with study foods: diets HM/HGI and HM/LGI

Diet HM/HGI and HM/LGI	Amount		Energy		Fat		SF		MUFA		PUFA		CHO	
	Male	Female	Male	Female	Male	Female	Male	Female	Male	Female	Male	Female	Male	Female
	g/d		kJ/d		g/d									
Study spread, high MUFA	20	15	592	444	16.0	12.0	3.0	2.3	9.5	7.2	3.4	2.5	0	0
Study oil, high MUFA	11	6	407	222	11.0	6.0	1.7	0.9	8.4	4.6	1.0	0.5	0	0
Cheese (half-fat)	17	12	186	131	2.6	1.8	1.6	1.1	0.7	0.5	0.1	0.1	0.02	0.01
Milk (skimmed)	230	190	318	262	0.2	0.2	0.1	0.1	0.0	0.0	0.0	0.0	11.5	9.5
CHO portion	-45	-30	-442	-295	-0.9	-0.6	-0.2	-0.1	-0.2	-0.1	-0.2	-0.2	-22.2	-14.8
Study snack (mean of 2)	27	27	492	494	4.7	4.7	0.6	0.6	3.4	3.4	0.7	0.7	16.9	16.9
Study mayonnaise	20	12	592	355	16.0	9.6	2.3	1.4	12.5	7.5	1.2	0.7	0	0
Intake less fats, milk, and cheese			7558	5212	50.5	35.9	19.2	13.7	17.3	12.3	7.3	5.8	244.7	180.7
Total			9703	6825	100.1	69.6	28.3	20.0	51.6	35.4	13.5	10.1	250.9	192.3
%E					38.8	38.4	11.0	11.0	20.0	19.5	5.2	5.6	40.6	44.2

TABLE 4 Replacement of exchangeable sources of dietary fat with study foods: diets LF/HGI and LF/LGI

Diet LF/HGI and LF/LGI	Amount		Energy		Fat		SF		MUFA		PUFA		CHO	
	Male	Female	Male	Female	Male	Female	Male	Female	Male	Female	Male	Female	Male	Female
	g/d		kJ/d						g/d					
Study spread, low fat	20	15	355	266	5.5	4.1	1.2	0.9	2.8	2.1	1.5	1.1	0.0	0.0
Study oil	4.5	4.5	166	166	4.5	4.5	0.5	0.5	2.5	2.5	1.5	1.5	0.0	0.0
Cheese (half-fat)	17	12	186	131	2.6	1.8	1.6	1.1	0.7	0.5	0.1	0.1	0.02	0.01
Milk (skimmed)	230	190	318	262	0.2	0.2	0.1	0.1	0.0	0.0	0.0	0.0	11.5	9.5
Study snack (mean of 3)	28	28	460	460	1.9	1.9	0.6	0.6	1.1	1.1	0.5	0.5	21.8	21.8
CHO portion, e.g. slice bread	45	30	442	295	0.9	0.6	0.2	0.1	0.2	0.1	0.2	0.2	22.2	14.8
Intake less fats, milk, and cheese			7558	5212	50.5	35.9	19.2	13.7	17.3	12.3	7.3	5.8	244.7	180.7
Total			9484	6792	66.1	49.0	23.4	17.0	24.6	18.6	11.1	9.2	300.2	226.8
%E					26.2	27.2	9.3	9.4	9.8	10.3	4.4	5.1	49.7	52.4

methyated and quantified by GC using a CPSil 88 column (Chrompak) using a method adapted from Indu and Ghafoorunissa (17). Mean inter-assay and intra-assay CV were 7.2 and 3.2%, respectively.

Statistical analysis

For dietary data, a repeated-measures ANOVA adjusted for gender and baseline value was used. Between-diet differences were assessed by Tukey's multiple range test. For phospholipid fatty acids, an ANCOVA model was used. Transformations were used to stabilize the variance of the residuals where necessary. Once outcome measures had been transformed, outliers were defined according to their end of run-in and end of intervention (transformed) measures, as well as the difference between their (transformed) measures. In each case, outliers were defined as points $>2.5 \times$ interquartile range (IQR) away from the median. Center and ethnicity together with the end of run-in measure and the minimization variables [baseline waist, age, log(HDL), and gender] were included in the model. A global test of between-diet differences (4 degrees of freedom) provided the main *P*-value. Only if this *P*-value was <0.05 were further comparisons made.

Results

Of the 720 randomized participants, 24% dropped out [105 before and 67 during or after attending the visit (wk 0) after run-in]. Of those who withdrew prior to the visit following the run-in

period, approximately one-third (32/105) described the run-in HS/HGI diet as the primary or contributing factor compared with approximately one-sixth (9/67) of participants who attended the visit following the run-in. A similar number of participants dropped out in each of the dietary treatments after completing the run-in (HS/HGI, 12; HM/HGI, 12; HM/LGI, 14; LF/HGI, 16; and LF/LGI, 13).

Dietary records of habitual food intake were completed by 517 participants and 481 participants completed both the run-in and 24-wk diet diary. To assess dietary data quality, estimated energy requirements were calculated using the Institute of Medicine equations (18) based on a low active population. Reported habitual energy intakes were $-15.7 \pm 19.8\%$ (mean \pm SD) lower than estimated energy requirements. This discrepancy was of a similar magnitude during the run-in period ($-13.8 \pm 19.8\%$) but greater at the end of the intervention period ($-20.1 \pm 19.1\%$).

Participants' reported habitual intakes (including alcohol) were representative of the U.K. population (9) (Table 6). During the intervention period, there were substantial and significant differences in nutrient intakes between the groups that were consistent with dietary intervention targets and were maintained throughout the 24-wk intervention period (Table 7). Total fat intake from the LF diets was successfully reduced to 28%E compared with 39%E in the reference diet ($P = 0.0001$) and there were compensatory significant increases in CHO in the LF groups ($P = 0.0001$). In the high-MUFA and LF diets, SF intake was reduced to $\leq 10\%$ E compared with 17%E in the reference diet ($P = 0.0001$). Dietary MUFA in the HM diets was higher

TABLE 5 Composition of fat sources used for intervention diets¹

	Fatty acid composition								
	Study spread			Baking/cooking		Study		Mayonnaise	
				product		oil			
	HS	HM	LF	HS	HM	HM	LF	HM	LF
	<i>g/100 g</i>								
Total FA	80.0	80.0	27.4	99.5	99.0	100.0	100.0	79.1	29.8
Total SF	38.7	15.1	6.0	47.6	19.9	15.2	11.4	6.2	2.7
16:0	12.3	7.8	2.73	15.5	9.5	11.2	7.4	4.0	1.7
18:0	12.1	4.0	1.7	15.8	5.4	2.9	2.8	1.4	0.6
Total <i>cis</i> -MUFA	22.5	47.7	14.1	33.6	55.1	75.9	55.0	50.0	18.7
18:1	20.93	46.6	13.7	32.7	53.6	74.6	53.9	48.3	18.1
Total <i>cis</i> -PUFA	19.8	17.0	7.3	18.2	23.8	8.78	33.2	22.9	8.3
18:2(n-6)	17.0	14.5	6.2	15.3	20.0	8.20	28.6	16.3	5.9
18:3(n-3)	2.8	2.5	1.1	2.9	3.8	0.6	4.6	7.0	2.5
Total <i>trans</i> -FA	0.2	0.3	0.1	0.3	0.3	0.1	0.4	0	0

¹ 16:0, palmitic acid; 18:0, stearic acid; 18:1, oleic acid; 18:3(n-3) α -linolenic acid.

TABLE 6 Reported habitual dietary intakes of RISCK participants¹

Nutrient	Males	Females
<i>n</i>	216	301
Energy, ² MJ/d	9.7 \pm 2.5	7.6 \pm 1.7
Fat, %E	34.7 \pm 6.4	35.8 \pm 5.9
SF, %E	12.8 \pm 3.3	13.1 \pm 3.6
MUFA, %E	11.6 \pm 2.7	11.7 \pm 2.4
(n-6) PUFA, %E	5.4 \pm 2.0	5.7 \pm 2.0
(n-3) PUFA, %E	0.7 \pm 0.3	0.8 \pm 0.4
CHO, %E	44.3 \pm 7.4	45.2 \pm 7.4
GI	62.0 \pm 3.9	61.1 \pm 4.2
NSP, g/d	18.0 \pm 6.9	15.8 \pm 5.1
Protein, %E	16.2 \pm 2.9	16.4 \pm 3.0

¹ Values are means \pm SD.

² % total energy including alcohol (%E).

(~17%E) than both the reference (12%E) and LF (10%E) diets ($P = 0.0001$). There was an inadvertent increase in (n-6) PUFA intake in the high MUFA diets; this was partially due to a higher PUFA content of the mayonnaise than originally modeled and also to the increase in consumption of nuts in these 2 groups. The intake of (n-3) PUFA was similar across all groups (data not shown). Reported energy intake from the LF groups was slightly lower than by the other groups ($P = 0.001$). The intervention strategy produced a significant 9-point GI difference between the high- and low-GI diet groups, but nonstarch polysaccharides (NSP) intake did not differ between the HM/HGI and HM/LGI or the LF/HGI and LF/LGI groups.

There were significant changes in the profile in the proportions of plasma phospholipid fatty acid classes over the 24 wk of intervention (Fig. 1). The changes in %SF and %MUFA differed between the diets ($P \leq 0.04$). Changes for both HM diets were

similar as were changes for the HS/HGI and both LF diets. After intervention, the combined HM groups had lower plasma phospholipid %SF than the combined LF groups ($P \leq 0.03$) and higher %MUFA by 0.69% ($P = 0.0001$). The dietary interventions did not affect other fatty acid classes [(n-3) PUFA, (n-6) PUFA, and *trans* FA]. These data provide further evidence that dietary objectives for RISCK were met.

Discussion

To our knowledge, the RISCK study implemented changes to dietary fat and CHO on a scale not attempted in any other randomized controlled trial in free-living individuals outside of “supermarket” models (19).

The intervention strategy was successful in attaining 5 dietary regimens that were clearly characterized in terms of total fat and

TABLE 7 Energy, fat, SF, MUFA, PUFA, CHO, NSP daily intakes, and GI during run-in, mid-intervention, and after 24 wk of dietary intervention along with planned target intakes¹

	HS/HGI	HM/HGI	HM/LGI	LF/HGI	LF/LGI	<i>P</i> -value ³
<i>n</i>	85	111	116	115	121	
Energy, MJ/d						
Run-in	8.35 ± 1.95	8.58 ± 1.97	8.66 ± 2.41	8.51 ± 2.14	8.78 ± 2.05	
Mid	8.30 ± 2.28 ^a	7.98 ± 1.94 ^a	8.51 ± 2.25 ^a	7.67 ± 2.33 ^b	8.04 ± 2.05 ^a	0.001
Final	8.19 ± 2.17 ^a	7.99 ± 2.01 ^a	8.41 ± 2.19 ^a	7.66 ± 2.33 ^b	7.46 ± 1.75 ^b	
Fat, ² %E						
Run-in	37.8 ± 5.3	38.9 ± 6.0	36.7 ± 5.1	37.9 ± 4.4	38.0 ± 5.5	
Mid	39.2 ± 5.7 ^a	36.3 ± 6.7 ^{ab}	34.4 ± 5.4 ^b	27.8 ± 5.7 ^c	27.2 ± 5.6 ^c	<0.0001
Final	37.7 ± 5.5 ^a	36.5 ± 6.9 ^{ab}	34.5 ± 5.7 ^b	27.4 ± 6.5 ^c	26.1 ± 6.2 ^c	
Target	38	38	38	28	28	
SF, %E						
Run-in	16.7 ± 2.9	16.9 ± 3.4	16.0 ± 2.8	16.4 ± 2.7	16.5 ± 3.1	
Mid	17.0 ± 3.0 ^a	9.6 ± 2.5 ^b	9.2 ± 2.6 ^{bc}	9.0 ± 2.6 ^{bc}	8.3 ± 2.3 ^c	<0.0001
Final	16.2 ± 2.9 ^a	9.9 ± 2.5 ^b	9.0 ± 2.5 ^{bc}	9.0 ± 2.7 ^{bc}	8.2 ± 2.5 ^c	
Target	18	10	10	10	10	
MUFA, %E						
Run-in	11.6 ± 2.1	11.9 ± 2.3	11.1 ± 2.0	11.7 ± 1.8	11.6 ± 2.1	
Mid	11.9 ± 2.3 ^a	16.7 ± 4.3 ^b	15.6 ± 3.7 ^b	10.1 ± 3.0 ^c	10.2 ± 2.6 ^c	<0.0001
Final	11.7 ± 2.5 ^a	16.4 ± 4.7 ^b	15.7 ± 3.8 ^b	9.9 ± 3.1 ^c	9.6 ± 2.9 ^c	
Target	12	20	20	11	11	
PUFA, %E						
Run-in	5.6 ± 1.5	5.9 ± 1.7	5.6 ± 1.5	5.8 ± 1.4	5.9 ± 2.2	
Mid	6.1 ± 1.5 ^a	6.7 ± 1.9 ^a	6.3 ± 1.5 ^a	5.5 ± 1.9 ^b	5.4 ± 1.5 ^b	<0.0001
Final	5.8 ± 1.7 ^a	6.8 ± 1.9 ^b	6.6 ± 1.9 ^b	5.3 ± 1.7 ^a	5.1 ± 1.8 ^a	
Target	6	6	6	6	6	
CHO, %E						
Run-in	42.8 ± 6.6	42.5 ± 6.8	43.7 ± 7.0	42.6 ± 5.9	43.2 ± 6.1	
Mid	40.9 ± 7.7 ^a	44.6 ± 8.1 ^b	45.6 ± 6.4 ^b	50.3 ± 8.2 ^c	51.7 ± 6.6 ^c	<0.0001
Final	41.5 ± 7.4 ^a	44.6 ± 7.1 ^b	45.5 ± 7.1 ^b	50.2 ± 8.2 ^c	51.8 ± 7.9 ^c	
Target	45	45	45	55	55	
GI						
Run-in	64.1 ± 4.0	63.4 ± 3.9	63.3 ± 3.4	63.7 ± 3.6	63.2 ± 3.3	
Mid	64.1 ± 3.5 ^a	63.7 ± 3.8 ^a	55.1 ± 3.8 ^b	64.1 ± 3.5 ^a	56.1 ± 3.6 ^b	<0.0001
Final	64.6 ± 3.7 ^a	63.2 ± 3.7 ^a	55.0 ± 3.7 ^b	64.8 ± 3.8 ^a	56.0 ± 3.5 ^b	
Target	64	64	53	64	51	
NSP, g/d						
Run-in	16.2 ± 4.8	17.2 ± 6.1	17.5 ± 6.0	17.8 ± 5.6	18.6 ± 6.0	
Mid	15.4 ± 4.1 ^a	17.2 ± 5.4 ^{ab}	19.1 ± 5.7 ^{bc}	18.9 ± 7.6 ^{bc}	20.3 ± 6.1 ^c	<0.0001
Final	15.5 ± 5.0 ^a	17.6 ± 5.9 ^{ab}	19.3 ± 6.2 ^b	18.4 ± 6.7 ^b	18.8 ± 6.0 ^b	

¹ Values are means ± SD. Means in a row with superscripts without a common letter differ, $P < 0.05$.

² Includes alcohol.

³ Repeated-measures ANOVA adjusted for gender and baseline value.

CHO intakes, the composition of fat (high vs. low SF and high vs. low MUFA) and the composition of CHO (high vs. low GI). Specific dietary targets during the run-in and within each dietary prescription during the intervention period were largely achieved. The increases in MUFA intake from the HM diets and the differential in dietary GI between the high and low groups were slightly smaller than modeled. There was a small reduction in energy intake in the LF groups. In each case, the changes achieved are likely to represent realistic intakes using a substitution strategy based on participants' existing dietary habits. Another study implementing dietary fat manipulation in a free-living pan-European population also reported achieving similar dietary fat targets as the current study using a food exchange model (20).

It proved difficult to increase MUFA intake to 20%E using available foodstuffs. Nuts were an important food but contributed to the unintentional increases in (n-6) PUFA. Study-specific testing of a range of CHO foods allowed selection of foods that demonstrated real differences in GI for the high and low interventions. The study sought to maximize the differences in GI through specific food substitutions rather than broader dietary changes, e.g. inclusion of pulses. While this strategy may have placed some limitations on the extent of the GI manipulation, it minimized differences between the dietary regimens in other compositional aspects of the diet. Importantly, the GI intervention did not lead to differences in fiber intake, which have confounded the interpretation of some other GI interventions (21,22). Population-wide increases in dietary MUFA and reduction in GI intake will require substantial reformulation of foodstuffs and new product innovation with MUFA-rich and low-GI options.

While few foods had to be avoided and a wide range of alternative foods were provided, some participants underestimated the impact of having to substitute foods across such a large proportion of their total diet and dropped out at an early stage citing difficulty with following the prescribed diet (SF/HGI) as a primary reason for withdrawing during the run-in

period. This was a less common reason for dropping out of the study during the intervention period. Furthermore, a similar number of participants withdrew from each of the diet groups after the run-in, suggesting that each of the dietary prescriptions were equally acceptable.

The success of the RISCK strategy to implement such wide-ranging dietary changes was facilitated not only by the detailed modeling of each dietary prescription, but was also due to the practical aspects of delivering the intervention. The regular provision of specially formulated or selected foods ensured that participants had access to foods with the appropriate composition at no financial cost or added inconvenience. Regular visits to the study center helped to motivate the participants and find solutions to any problems in following the diets.

The staggering of participant recruitment meant that the intervention period of the study was conducted for over 25 mo. The logistical challenges (including delivery and storage arrangements) and costs of providing study foods were substantial. Vigilant recording of any changes in nutritional composition of the study foods added to the complexity of dietary coding.

This article describes a successful strategy for the design of 5 isoenergetic, experimental diets with specific targets for fat and CHO intakes and describes the process of implementation. It provides a comprehensive account of areas that need consideration in planning, executing, and evaluating the impact of dietary interventions.

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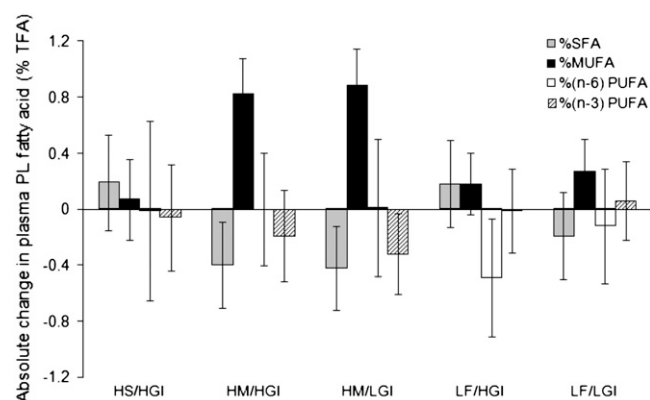


FIGURE 1 Changes in the profile of plasma phospholipid (PL) fatty acid classes [% total fatty acids (TFA)] from run-in to end of intervention (calculated on transformed scale but expressed as change from median value at run-in) (mean and 95% CI). The main *P*-values were: %SF, *P* = 0.039; %MUFA, *P* = 0.001; %(n-6) PUFA, *P* = 0.22; %(n-3) PUFA, *P* = 0.62. The HM/HGI (*n* = 106) and HMLGI (*n* = 103) groups combined had lower %SF than HS/HGI (*n* = 78) (*P* = 0.028) and LF/HGI (*n* = 102) and LF/LGI (*n* = 109) combined (*P* = 0.009) and higher %MUFA than HS/HGI (*n* = 82) (*P* = 0.0001) and LF/HGI (*n* = 110) and LF/LGI (*n* = 119) combined (*P* = 0.0001). Transformations used: %(n-6)PUFA cubed, %(n-3)PUFA log.

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