



# A comparison of the effect of free access to reduced fat products or their full fat equivalents on food intake, body weight, blood lipids and fat-soluble antioxidants levels and haemostasis variables

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**Objectives:** To compare the effects of free access to reduced fat products or their full fat equivalents on fat and energy intake, body weight, plasma lipids and fat-soluble antioxidants concentrations and haemostasis variables.

**Design:** A multicentre open randomised controlled trial in which intervention and control groups were followed in parallel for six months. Volunteers had free access to 44 different foods either in reduced fat or full fat version, covering between 30 and 40% of energy intake. The remainder of energy intake was covered by foods bought in regular shops.

**Setting:** Zeist, Wageningen and Maastricht, The Netherlands.

**Subjects:** Two hundred and forty-one non-obese healthy volunteers who had no intention to lose weight.

**Main outcome measures:** Food intake, body weight, plasma lipid, vitamin E,  $\beta$ -carotene, lycopene and fibrinogen concentrations, plasma factor VII clotting activity, and plasminogen-activator-inhibitor-I antigen level.

**Results:** One hundred and three volunteers in the full fat group and 117 volunteers in the reduced fat group completed the study. Energy and fat intake from the free access products was lower in the reduced fat group, but no difference in energy and fat intake of other products occurred. Body weight, energy-, fat- and vitamin E intake and percentage of energy derived from fat decreased in the reduced fat group. No other statistical significant intervention effects were observed. Blood lipid concentrations, factor VII activity and plasminogen-inhibitor-activator-1 level were reduced after consumption of reduced fat products.

**Conclusions:** When subjects without intention to lose weight limit fat intake by switching from *ad libitum* consumption of full fat products to reduced fat products body weight gain is prevented, and fat and energy intake are reduced. Such a switch may have beneficial effects on biochemical cardiovascular risk factors. We concluded that reduced fat products will help in a population strategy aimed at preventing overweight and obesity, they will also be effective in maintaining a lower body weight after slimming. *Ad libitum* consumption of reduced fat products will be ineffective for those individuals that want to reduce body weight because they are currently overweight or obese.

**Sponsorship:** Unilever

**Descriptors:** fat reduction; body weight; blood lipids; haemostasis; antioxidants; food intake; intervention study

## Introduction

During the last decade the prevalence of overweight and obesity has shown an alarming increase in Britain and the USA (Office of Population Censuses and Surveys, 1984; White *et al*, 1993; Kuzcmarski *et al*, 1994). Obesity and overweight are major sources of ill-health in affluent societies. Both conditions are associated with higher mortality from coronary heart disease and stroke. Also morbidity and mortality from gallbladder disease, diabetes, respiratory diseases and some cancers is increased (National Research Council, 1989).

Obesity and overweight seem to be associated with a diet rich in fat and a sedentary lifestyle (Romieu *et al*, 1988; Klesges *et al*, 1992). Fat-rich diets appear to permit passive overconsumption of energy due to their high energy density, and, possibly, relatively low satiating action (Cotton *et al*, 1994). Lower rates of energy expenditure at work and at home, characteristic of the modern lifestyle, may also contribute toward an enhanced storage of dietary energy as body fat.

It is now recognized that it is very difficult to treat obesity and overweight with long term success (Council on Scientific Affairs, 1988). Therefore mass prevention of unhealthy weight gain becomes very important. Health professionals have recommended for years a reduction in dietary fat intake. This advice has led to a growing public concern with the adverse effects on health of a high fat

intake. The perceived health benefits of a lower fat intake has promoted a proliferation on the market of reduced fat products. It is assumed that consumption of such products will be beneficial to the consumer. However, no data have been published of a randomized controlled trial in free-living subjects of the effects of free access to such products for a prolonged period of time on food intake, body weight and biological variables. Strictly controlled laboratory studies using covert manipulations of the fat content of the diet have shown that fat and energy intake are reduced when the fat content of food is reduced (Lissner *et al*, 1987; Kendall *et al*, 1991). The simple question whether a voluntary switch from commercially available full fat products to their reduced fat counterparts helps consumers in reducing energy and fat intake and body weight has not yet been answered for individuals outside a strictly controlled laboratory setting. We here report the results of an efficacy study of reduced fat products using a study set-up resembling the situation consumers face in real life. We investigated effects on food intake and body weight and on a number of biological variables, potentially, related to risk on cardiovascular disease. Our hypothesis was that free access to reduced fat products would cause a significant decrease in energy and fat intake and body weight and would produce a more favourable blood lipid profile in comparison to free access to full fat products.

## Subjects and methods

### Subjects

This six month parallel comparison study was carried out simultaneously at three different research centres in Zeist, Wageningen and Maastricht in the Netherlands and coordinated by the Unilever Research Laboratorium in Vlaardingen, the Netherlands. Two hundred and forty-one non-obese volunteers were randomly assigned to a reduced fat products or a full fat products group. As the products were clearly labelled as such, volunteers were aware of their group assignment. It should be clear that we deliberately did not choose a blinded situation as we wanted to obtain results generalisable to the situation consumers face when they are offered reduced fat or full fat products in regular shops. Eligible subjects had stable weights, normal eating habits (no slimming or medically prescribed diet) and were healthy as assessed by a medical examination and routine clinical chemistry. Serum cholesterol levels at entry were below 8 mmol/L, age was between 19 and 55 y. Half of the volunteers were aged between 19 and 36 y and the other half were older. The body mass index for the younger groups was at entry between 20 and 28 and for the older group between 24 and 31 kg m<sup>-2</sup>. Exclusion criteria were intensive exercise (> 7 h/w) and excessive alcohol consumption (> 21 alcoholic drinks/w for females; > 28 alcoholic drinks/w for males). Each research facility recruited about 80 eligible subjects by advertisement in regional newspapers. Volunteers were randomized over the full fat and reduced fat groups matched for mean age, body mass index, sex distribution, type of household and eating behaviour characteristics. All subjects gave their written informed consent for participation and the study protocol was approved by the Institutional Review Board of each centre.

### Experimental procedures

At each research centre a small realistic supermarket provided full fat commercial products and their reduced

fat alternatives. The available products, 44 for each treatment, fitted in the normal diet of the volunteers and included items as frying fat, desserts, hard and cream cheeses, sausages, patés, salads, dressings, deep frozen meals and pizzas, frozen fish fingers, cheese spreads, butter, margarines, frozen meat-based snacks, cookies and cakes. The full fat and reduced fat products were screened prior to the start of the study on sensory similarity, consumers' acceptance and nutrient composition. The products differed primarily in fat content (reduced fat products contained on average 52% less fat than the full fat products) and not in the other macronutrients. Forty-two products were commercially available products. Full fat and reduced fat cookies and cakes were prepared by the Unilever Research Laboratorium. The macronutrient content was derived from the manufacturer's information and the Dutch food composition table (Voorlichtingsbureau voor de Voeding, 1993) and checked by chemical analysis.

The volunteers visited the supermarket generally once a week. They were allowed to take as many products as they wished, and instructed not to share the products with their family or friends. The dieticians interviewed the volunteers after each visit to check compliance with the instructions. Products were free of charge, but volunteers had to make the effort to come shopping in the research centres. In addition to the products provided free of charge, volunteers bought other products, such as fruits, vegetables, snacks, drinks, meats, bread etc. in regular shops. Volunteers were free in their choice of these products. The experimental procedures have been described in detail previously (Hof van het *et al*, 1997). The study was designed to have a power of 90% to detect with a confidence of 95%, assuming a drop-out rate of 20%, differences between both groups in energy intake, fat intake and fat energy percentage of 1.4 MJ/d (325 kcal/d), 19 g/d and 3.4%, respectively.

### Measures

Total food intake was assessed just before the run-in period, and after 2–4 w, three months and at the end of the actual study. At each point in time, food intake including the intake derived from the products provided by each centre, using household measures to estimate quantities, was recorded for three days (one day in the weekend). Macronutrient and vitamin E intake were calculated using the Dutch food composition table (Voorlichtingsbureau voor de Voeding, 1993) and data of the chemical analyses of the full fat and reduced fat products. Fasting venous blood samples were taken before, after two and four months and at the end of the study. At the same day of blood sampling, body weight was assessed with the subjects in underwear and after voiding. Serum total cholesterol, HDL-cholesterol, triacylglycerol levels were determined using commercial test kits. LDL-cholesterol level was calculated using the Friedewald formula (Friedewald *et al*, 1972). For the Zeist cohort only, serum Lp(a) was assessed by a rate immunonephelometric assay. Haemostasis variables were also measured only for the Zeist cohort: fibrinogen was measured using a clotting rate method, factor VII clotting activity was measured with a chromogenic method (Chromogenix AB, Möndal, Sweden) and plasminogen-activator-inhibitor-1 antigen was measured by ELISA (Coalize PAI-1/Innotest PAI-1, Chromogenix AB, Möndal, Sweden). Also, only in the Zeist cohort plasma levels of vitamin E, beta-carotene and lycopene were determined by HPLC using UV detection. Body composition

was assessed at the start and end of the study using the deuterium dilution technique (Van Marken Lichtenbelt *et al*, 1994).

### Statistical analysis

Intervention effects were evaluated for individuals who completed the whole trial using analysis of variance with sex and intervention as factors when assumptions of normality were met. To determine the intervention effect the changes between baseline values and values at each point in time during the trial were compared between both groups (reduced fat–full fat) for a statistically significant (confidence of 95%) difference. There was great similarity in intervention effects throughout the trial, therefore most analyses are reported only for the changes between start and end (six months) of the study. Differences in dichotomized variables (education, restrained eating behaviour, type of household) at start between the full fat and reduced fat group were assessed by Fisher's exact test. Differences in blood lipids, body weight, and body fat content between initial low and high fat consumers were assessed by analysis of covariance, using sex, age and total energy intake as covariates. Intervention effects were also evaluated using initial body mass index or fat consumption as a covariate. SAS computer software was used for all statistical calculations (Statistical Analysis System Institute, 1987).

## Results

### Volunteers

Table 1 shows characteristics of the volunteers at the start of the study. Twenty-one subjects, seventeen in the full fat and four in the reduced fat group, did for various reasons not complete the study. These subjects did not differ in entry characteristics from the volunteers who completed the study. As expected there were no significant differences in entry characteristics between full fat and reduced fat groups at the start, neither for the male or female subgroup. We divided the total population at entry to the study in initial low (<35% energy from fat) or high ( $\geq 35\%$ ) fat consumers as we hypothesised that intervention effects might differ between these groups. We observed that energy intake adjusted for age and sex was lower in the initial low fat consumers (Table 2). Body weight and body fat content were significantly lower in the initial low fat consumers compared to the high fat consumers in contrast to plasma HDL-cholesterol level and the LDL/HDL-cholesterol ratio.

### Energy, macronutrient and vitamin E intake

Table 3 shows energy and vitamin E intake, the proportion of energy derived from the various macronutrients, alcohol and the various fatty acid classes in both groups before and at the end of the trial for the total group and for groups

**Table 1** Mean  $\pm$  s.d. baseline values of descriptive characteristics, sociodemographic characteristics and energy and fat intake of volunteers in reduced fat and full fat group who completed the study, separately for males and females

	Males		Females	
	Reduced fat	Full fat	Reduced fat	Full fat
N	59	52	58	51
Age (y)	35.5 $\pm$ 10.8	35.6 $\pm$ 9.6	36.0 $\pm$ 11.8	36.0 $\pm$ 10.9
Body weight (kg)	81.5 $\pm$ 8.8	82.7 $\pm$ 9.5	68.4 $\pm$ 7.0	70.6 $\pm$ 7.0
Body height (m)	1.81 $\pm$ 0.075	1.82 $\pm$ 0.058	1.66 $\pm$ 0.051	1.68 $\pm$ 0.068
Body mass index (kg/m <sup>2</sup> )	24.9 $\pm$ 2.3	24.9 $\pm$ 2.2	24.7 $\pm$ 2.0	25.0 $\pm$ 2.0
Restrained eaters (%)	6.8	7.7	17.2	11.8
Type of household				
1 person (%)	8.5	9.6	8.6	17.6
2 person (%)	20.3	26.9	19.0	19.6
other (%)	71.2	63.5	72.4	62.8
Education				
$\leq 12$ y (%)	39.0	40.4	62.1	62.8
$> 12$ y (%)	61.0	59.6	37.9	37.2
Energy intake (MJ/d)	11.8 $\pm$ 3.3	11.8 $\pm$ 2.6	8.5 $\pm$ 2.1	8.9 $\pm$ 2.2
Fat intake (en%)	34.1 $\pm$ 5.4	34.5 $\pm$ 6.1	35.4 $\pm$ 5.4	36.7 $\pm$ 6.4

**Table 2** Cross sectional analyses of differences in energy intake, body weight, body fat content, blood lipids levels in subjects with a high or low fat intake at entry to the study

	Initial low fat intake (n = 110)	Initial high fat intake (n = 110)	Difference (95% confidence interval)	P-value
Fat energy %	30.7 $\pm$ 3.6	39.6 $\pm$ 4.1	8.5 (7.4–9.5) <sup>a</sup>	0.001
Energy intake (MJ/d)	10.1 $\pm$ 2.9	10.5 $\pm$ 3.2	1.0 (0.3–1.7) <sup>b</sup>	0.004
Body weight (kg)	75.0 $\pm$ 10.5	76.6 $\pm$ 10.2	2.4 (0.2–4.6) <sup>a</sup>	0.03
Body fat (%)	26.3 $\pm$ 7.9	29.9 $\pm$ 6.9	2.1 (0.9–3.3) <sup>a</sup>	0.001
Body mass index (kg/m <sup>2</sup> )	24.6 $\pm$ 2.3	25.2 $\pm$ 1.9	0.4 (–0.1–0.9) <sup>a</sup>	0.13
Total cholesterol (mmol/L)	5.64 $\pm$ 1.14	5.74 $\pm$ 1.15	0 (–0.3–0.3) <sup>a</sup>	0.98
LDL-cholesterol (mmol/L)	3.59 $\pm$ 1.00	3.82 $\pm$ 1.04	0.15 (–0.1–0.3) <sup>a</sup>	0.24
HDL-cholesterol (mmol/L)	1.45 $\pm$ 0.38	1.38 $\pm$ 0.35	–0.11 (–0.19–0.02) <sup>a</sup>	0.02
LDL/HDL-cholesterol ratio	2.67 $\pm$ 1.05	2.97 $\pm$ 1.20	0.34 (0.08–0.06) <sup>a</sup>	0.009
Triglycerides (mmol/L)	1.33 $\pm$ 0.69	1.22 $\pm$ 0.56	–0.09 (–0.26–0.08) <sup>a</sup>	0.29
Mean $\pm$ s.d.				

<sup>a</sup>Difference adjusted for sex, age and energy intake.

<sup>b</sup>Difference adjusted for sex and age.

**Table 3** Food intake before and after six months in the study in the reduced fat products and full fat products group, in total and separately for initial high fat and initial low fat consumers

Variable	Reduced fat products (n = 117)		Full fat products (n = 103)		Intervention effect (95% confidence interval)	P values
	Before	Six months	Before	Six months		
Energy (MJ/d)	10.2	10.3	10.4	11.3	-0.9 (-1.6--0.2)	0.014
Initial high fat <sup>a</sup>	10.5	10.2	10.5	11.4	-1.3 (-2.3--0.3)	0.009
Initial low fat <sup>b</sup>	9.9	10.3	10.2	11.1	-0.5 (-1.5--0.5)	0.36
Fat (en%)	35	32	36	41	-7 (-9--6)	0.001
Initial high fat	39	35	40	43	-7 (-9--5)	0.001
Initial low fat	31	32	30	39	-8 (-10--6)	0.001
Saturated fat (en%)	13	13	14	17	-4 (-5--3)	0.001
Initial high fat	15	14	15	18	-4 (-5--3)	0.001
Initial low fat	12	12	12	17	-5 (-6--4)	0.001
Monounsaturated fat (en%)	13	12	13	14	-2 (-3--1)	0.001
Initial high fat	15	12	15	15	-2 (-3--1)	0.001
Initial low fat	12	11	11	14	-3 (-4--2)	0.001
Polyunsaturated fat (en%)	6	6	6	7	-0.3 (-1.0--0.3)	0.29
Initial high fat	7	6	7	7	-0.6 (-1.5--0.3)	0.16
Initial low fat	5	6	5	6	-0.2 (-1.0--0.6)	0.68
Protein (en%)	15	16	15	14	2 (1-3)	0.001
Initial high fat	15	17	15	14	2 (1-3)	0.001
Initial low fat	16	17	15	14	2 (1-3)	0.001
Carbohydrate (en%)	46	47	46	41	6 (5-7)	0.001
Initial high fat	42	46	42	40	6 (4-8)	0.001
Initial low fat	49	49	50	43	6 (4-8)	0.001
Alcohol (en%)	4	3	4	3	-0.1 (-0.1--0.8)	0.28
Initial high fat	3	3	3	3	-0.5 (-1.6--0.6)	0.39
Initial low fat	4	3	5	3	-1.2 (-1.2--1.8)	0.73
Vitamin E (mg/d)	9.6	6.8	9.2	10.8	-4.3 (-6.0--2.7)	0.001
Initial high fat	10.8	7.0	10.0	11.6	-5.6 (-8.2--3.0)	0.001
Initial low fat	8.5	6.7	8.2	9.9	-3.4 (-5.4--1.4)	0.001

<sup>a</sup>There are 54 initial high fat consumers in the reduced fat group and 56 in the full fat group.

<sup>b</sup>There are 63 initial low fat consumers in the reduced fat group and 47 in the full fat group.

subdivided according to fat consumption at entry. There was a significant intervention effect on energy, total fat, fatty acids and vitamin E intake. Energy, total fat, saturated fat, monounsaturated and polyunsaturated fatty acid intake increased significantly in the full fat group compared to the reduced fat group. As a consequence there were significant differences in the proportion of energy derived from total fat, the various fatty acid classes, carbohydrates and proteins between both groups. There were no differences between both sexes in these effects. The free access to reduced fat products induced a decrease in energy intake and in the percentage of energy from fat; this effect was most pronounced for those individuals that were classified as high fat consumers at entry. None of the intervention effects was significantly different between the initial low and high fat consumers. The experimental products provided on average 37% of energy intake in the full fat group and 30% in the reduced fat group. Mean fat and energy intake from free access products was 70 g/d and 4.2 MJ/d in the full fat group and 37 g/d and 3.1 MJ/d in the other group. Mean fat and energy intake from other foods was not different between both groups, 63 g/d and 7.1 MJ/d.

#### Body weight, blood variables

Table 4 shows absolute values and differences between both groups for body weight, blood lipids, fibrinogen and plasminogen-inhibitor-activator-1 antigen levels, factor VII clotting activity and plasma vitamin E, beta-carotene and lycopene levels. There was a significant intervention effect on body weight at two and six months. Weight remained stable in the reduced fat group and it increased significantly by about 1 kg in the full fat group. Average body weight

changed gradually in the control group from 76.7 kg to 77.3, 77.5 and 77.8 kg at two, four and six months respectively. Mean body weight changed in the reduced fat group from 75.0 kg to 75.2, 75.2 and 75.4 kg at two, four and six months respectively. The difference between both groups in the change of fat mass (0.61 kg) was of borderline significance ( $P=0.052$ ). Sex, initial body mass index or fat consumption did not significantly affect the weight changes.

In general, total cholesterol, LDL-cholesterol, HDL-cholesterol and triacylglycerol levels were lower during the study in the reduced fat group compared to the full fat. The intervention effect was, however, not significant. There was also no systematic intervention effect or sex by intervention interaction effect on the LDL to HDL ratio, serum Lp(a) and triglyceride levels, plasma levels of fibrinogen and plasminogen-inhibitor-activator-1 antigen, factor VII clotting activity and plasma vitamin E, beta-carotene and lycopene levels.

We observed that for a number of variables the intervention effect was not too different from the effect that would have been statistically significant. Significant intervention effects would have been obtained in the current study when blood lipid reductions had been 0.17 mmol/L (observed 0.16 mmol/L), 0.15 mmol/L (observed 0.10 mmol/L), 0.05 mmol/L (observed 0.03 mmol/L) and 0.14 mmol/L (observed 0.07 mmol/L) for serum total cholesterol, LDL-cholesterol, HDL-cholesterol and triacylglycerol concentrations, respectively. Also for factor VII activity and plasminogen-inhibitor-activator-1 antigen level the significant effect was not too different from the observed effect, respectively 6.7% (observed 5%) and 18 ng/mL (observed 10 ng/mL).

**Table 4** Body weight, blood lipids and hemostatic variables, plasma vitamin E,  $\beta$ -carotene and lycopene concentrations (Mean  $\pm$  s.e.m.) in the reduced fat products and full fat products group at the start and end of the study

	<i>N</i>	<i>Start</i>	<i>Six months</i>	<i>Intervention effect</i> (95% confidence interval)
Body weight (kg)				
Reduced fat	117	75.0 $\pm$ 0.95	75.4 $\pm$ 0.97	-0.72 (-1.35-0.10) <sup>a</sup>
Full fat	103	76.7 $\pm$ 1.01	77.8 $\pm$ 1.04	
Total cholesterol (mmol/L)				
Reduced fat	117	5.70 $\pm$ 0.11	5.61 $\pm$ 0.10	-16 (-0.33-0.016)
Full fat	103	5.68 $\pm$ 0.11	5.75 $\pm$ 0.10	
LDL-cholesterol (mmol/L)				
Reduced fat	117	3.70 $\pm$ 0.10	3.68 $\pm$ 0.09	-0.10 (-0.25-0.05)
Full fat	103	3.71 $\pm$ 0.11	3.79 $\pm$ 0.08	
HDL-cholesterol (mmol/L)				
Reduced fat	117	1.40 $\pm$ 0.03	1.34 $\pm$ 0.03	-0.03 (-0.08-0.03)
Full fat	103	1.43 $\pm$ 0.04	1.40 $\pm$ 0.04	
LDL-cholesterol/HDL-cholesterol				
Reduced fat	117	2.86 $\pm$ 0.11	3.02 $\pm$ 0.13	0.004 (-0.14-0.15)
Full fat	103	2.77 $\pm$ 0.10	2.92 $\pm$ 0.11	
Triacylglycerol (mmol/L)				
Reduced fat	117	1.34 $\pm$ 0.06	1.30 $\pm$ 0.07	-0.07 (-0.22-0.08)
Full fat	103	1.20 $\pm$ 0.06	1.24 $\pm$ 0.06	
Lp(a) ( $\mu$ g/mL)				
Reduced fat	40	23.2 $\pm$ 4.6	23.8 $\pm$ 4.7	0.2 (-1.4-1.8)
Full fat	36	20.4 $\pm$ 4.6	20.8 $\pm$ 4.4	
Fibrinogen (g/L)				
Reduced fat	40	2.83 $\pm$ 0.10	3.03 $\pm$ 0.07	0.04 (-0.19-0.27)
Full fat	36	2.91 $\pm$ 0.08	3.06 $\pm$ 0.09	
Factor VII (%)				
Reduced fat	40	115 $\pm$ 3.3	112 $\pm$ 4.1	-5 (-11.6-1.6)
Full fat	36	99 $\pm$ 6.0	101 $\pm$ 4.5	
Plasminogen-inhibitor-activator-1 (ng/mL)				
Reduced fat	40	62.0 $\pm$ 9.5	60.2 $\pm$ 6.0	-10 (-27-7)
Vitamin E ( $\mu$ g/mL)				
Reduced fat	40	11.2 $\pm$ 0.45	11.0 $\pm$ 0.48	-0.2 (-0.9-0.5)
Full fat	36	11.0 $\pm$ 0.38	11.0 $\pm$ 0.30	
$\beta$ -Carotene ( $\mu$ g/mL)				
Reduced fat	40	0.30 $\pm$ 0.02	0.29 $\pm$ 0.02	0.01 (-0.03-0.05)
Full fat	36	0.32 $\pm$ 0.02	0.30 $\pm$ 0.02	
Lycopene ( $\mu$ g/mL)				
Reduced fat	40	0.26 $\pm$ 0.02	0.31 $\pm$ 0.02	0.06 (0.00-0.010)
Full fat	36	0.28 $\pm$ 0.02	0.26 $\pm$ 0.02	

<sup>a</sup>  $P = 0.023$ .

## Discussion

We here report data of a 6 months randomized trial in normal weight and moderately overweight adults of the efficacy of free access to reduced fat products to decrease energy and fat intake and to improve blood lipid profile. The study's most significant results are that free access to reduced fat products versus their full fat equivalents prevents weight gain and reduces energy and fat intake and increases the percentage of energy derived from carbohydrates, especially for those individuals that have a high habitual fat consumption. The effects observed were relatively small. We observed that consumption of reduced fat products did lower, but not significantly, blood levels of lipids, factor VII activity and plasminogen-inhibitor-activator-1 antigen level.

Dietary guidelines in affluent countries universally emphasize a reduction in the intake of dietary fat, in particular of saturated fatty acids. In spite of these recommendations, average fat consumption in for example the USA and the UK, but also in Germany and the Netherlands is still exceeding what is recommended.

This study suggests that *part* of a population strategy to reduce the increasing prevalence of overweight and obesity involves a switch from the consumption of full fat products to their reduced fat alternatives. This is particularly true

when the full fat products are rich in saturated or trans fatty acids. In addition, stimulating an increased energy use in daily life will also be important, perhaps even more important than limiting fat intake, in the prevention of weight gain as was recently suggested (Prentice & Jebb, 1995).

In recent years reduced fat products have flooded the markets in Europe and Northern America. Laboratory investigations of the effect in humans of reducing the dietary fat content on spontaneous energy and fat intake have shown a decreased energy and fat intake (Lissner *et al*, 1987; Kendall *et al*, 1991). Due to the strictly controlled nature of these studies, most of them have been limited to relatively small groups and have been of relatively short duration, in contrast to this study. Moreover most studies did not measure blood variables and used a *covert* manipulation of the fat content of the diet, a situation consumers do not readily encounter in practice as most reduced fat products are clearly labelled as such. Weight loss in subjects participating in 12 months field trials of low-fat diets have produced a body weight lowering of between 1.5 and 2.5 kg, which is in good agreement with the findings of this study (Sheppard *et al*, 1991; Lee-Han *et al*, 1988). However, these trials are not comparable to our study as subjects were intensively counselled to reduce their fat intake. In our trial we obtained estimates of potential

effects on body weight, food intake and a number of plasma parameters if individuals without the deliberate intention to lose weight would voluntarily decide to substitute a number of the full fat products they usually consume for reduced fat alternatives.

The effects of the intervention in this study on weight gain were relatively small. A recent prospective study, however, showed that relatively small weight gains, namely between 5 and 11 kg over a period of 14 y (average less than 1 kg per year) were associated with a significant increased risk for coronary heart disease in women aged 18 y and older (Willett *et al*, 1995). Whether the small weight gain prevented in this study can be translated to a larger weight gain of for example 5–10 kg in a longer period of 5–10 y remains to be investigated.

Apart from the effects on food intake we assessed effects on blood lipids and fat-soluble antioxidant levels and haemostasis variables. It has been estimated that iso-energetic exchange of saturated dietary fat for carbohydrates would reduce total cholesterol, LDL-cholesterol and HDL-cholesterol levels (Mensink & Katan, 1992). Therefore, one of the adverse effects of introducing reduced fat products in the diet at the expense of full fat products might be a reduction in HDL-cholesterol levels. We observed a lowering of both LDL- and HDL-cholesterol concentrations in subjects consuming reduced fat products. The LDL to HDL-cholesterol ratio, an important predictor of coronary heart disease (Stampfer *et al*, 1991), was not adversely affected by the treatment. A cross-sectional analysis of the total study population prior to the start of the study revealed an inverse association between HDL-cholesterol levels and the percentage of energy from fat, indicating that in affluent societies in a free living situation a lower fat intake might not necessarily be associated with reduced HDL-cholesterol levels, possibly due to a lower body weight. Indeed we found that body weight, adjusted for sex, age and energy intake was about 2.4 kg lower at entry to the study in subjects with a lower dietary fat intake, confirming data by Romieu *et al* (Romieu *et al*, 1988).

Levels of Lp(a), which has been proposed to be an independent risk factor for coronary heart disease (Schaefer *et al*, 1994), were not adversely influenced by the reduction in dietary fat intake. Increased fibrinogen, plasminogen-inhibitor-activator-1 antigen levels and factor VII clotting activity may increase the risk of coronary heart disease (Kannel *et al*, 1987; Broadhurst *et al*, 1990; Hamsten *et al*, 1987). Markmann *et al* (1993), observed that prolonged consumption of a low fat high fibre diet reduced these variables. We also observed lower factor VII activity and plasminogen-inhibitor-activator-1 antigen levels in subjects consuming reduced fat products.

The significant decrease in vitamin E intake in the reduced fat group was striking. The most important sources of vitamin E in the Dutch diet are vegetable oils, dressings and soft margarines (Kistemaker & van den Berg, 1993). A reduction in the fat content of those products generally causes a reduction in the dietary vitamin E intake, which poses the question of fortification of such products with vitamin E. Adequate intake of vitamin E might be important in lowering the risk of coronary heart disease (Stampfer *et al*, 1993).

## Conclusions

This study suggests that a switch from full fat products to reduced fat alternatives will help prevent body weight gain

in individuals who do not have the intention to change their body weight and will help to prevent overconsumption of dietary fat and energy. Such a switch may also have a small beneficial effect on cardiovascular risk factors. Our data suggest that a substitution of reduced fat products for full fat products will be helpful in a population strategy for the prevention of obesity. A voluntary switch to these products without severely reducing energy intake as well will not be effective in reducing body weight in those individuals already overweight or obese. Reduced fat products can provide a nutritionally advantageous alternative for full fat products, in particular when these full fat products are rich in saturated or trans fatty acids. Supplementation with vitamin E of reduced fat products to the level occurring in the full fat equivalents may further increase the nutritional benefits of such products.

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