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Replacement of dietary fat by sucrose or starch: Effects on 14 d *ad libitum* energy intake, energy expenditure and body weight in formerly obese and never-obese subjects

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OBJECTIVE: To investigate the impact of a high-sucrose diet *vs* a high-starch and a high-fat diet on 14 d *ad libitum* energy intake, body weight, energy expenditure and sympathoadrenal activity.
MEASURMENTS: Food intake; body weight and composition (bioelectrical impedance); 24 h energy expenditure, substrate oxidation rates, spontaneous physical activity, heart rate and appetite sensations in a respiration chamber (VAS scores); plasma catecholamine concentration and blood pressure.
SUBJECTS: Twenty normal-weight, healthy women, 9 post-obese (body mass index (BMI): 22.9 ± 0.7 kg/m^2) and 11 closely matched controls (BMI: 22.6 ± 0.4 kg/m^2).
RESULTS: Average 14 d *ad libitum* energy intake was 13% and 12% lower on the starch diet compared with the sucrose and fat diets, respectively ($P < 0.05$). In both post-obese and normal-weight subjects, body weight and fat mass decreased significantly on the starch diet (by 0.7 ± 0.2 kg and 0.4 ± 0.1 kg, respectively, $P < 0.05$). No changes were observed on the fat or sucrose diets. After 14 d on the sucrose diet, 24 h energy expenditure as well as postprandial plasma adrenaline and noradrenaline concentrations, were significantly increased compared with the other two diets. Overall satiety and palatability ratings were also highest on the sucrose diet.
CONCLUSION: Intake of a 14-d *ad libitum* high-starch diet decreased energy intake and body weight compared with a high-fat or high-sucrose diet. The increased energy expenditure observed on the sucrose-rich diet can probably be explained both by the increased intake of energy and fructose (mainly from sucrose) on this diet.

Keywords: recommended diet; post-obese; indirect calorimetry; macronutrient balance; sympathetic nervous system; appetite ratings

Introduction

The prevalence of obesity is high and still increasing in the Western world.[1,2] Two major reasons are the decrease in the level of physical activity as well as a dietary consumption pattern favouring a too high fat intake.[2,3] The latter trend is in striking contrast to the dietary recommendations which suggest that fat intake is kept below 30% of total energy (E%) and carbohydrate intake between 55 E% and 60 E%.[4] Furthermore, it is recommended to keep sucrose intake below 10 E%.[4] In practice, however, this advice may be difficult to combine with the recommendations of a low fat intake. Thus a certain level of sweetness of the diet may be necessary to achieve the recommended carbohydrate-rich diet.[5] Data from several food surveys have shown a negative relation between daily fat intake (E%) and sucrose intake (E%).[6–8] Furthermore, cross-sectional and epidemiological studies (excluding the studies with obvious underreporting of energy intake) have shown a negative association between sucrose intake and obesity.[9,10] A high sucrose intake seems therefore to be associated with a low dietary fat intake and a low prevalence of obesity and *vice versa*.

The above types of studies suffer, however, from their observational nature and the shortcomings of dietary records, with the ensuing risk of macronutrient-specific underreporting. Although actively debated, the role of sucrose in the development of obesity is therefore still not clarified.[11–13] The purpose of the present study was to investigate the *ad libitum* energy intake, changes in body weight, 24 h energy expenditure and sympathoadrenal activity when replacing dietary fat with sucrose or starch during a 14 d period. Normal-weight subjects with or without a history of obesity were included in the study.

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Methods

Subjects

Twenty healthy, normal-weight women, 9 post-obese (PO) and 11 controls (C), closely matched for age, weight, height, fat mass, and fat-free mass participated in the study (Table 1). The PO women had a family history of obesity (at least one obese parent or sibling), had been more than 10% overweight (average ± s.e.m; 44 ± 10%) and had been weight-stable for at least 2 months. None had undergone surgical operations to become normal-weight. As assessed by 7 d weighed food records (performed 1–4 weeks before the first test diet), habitual energy and fat intake was lower and carbohydrate intake higher in PO than in C ($P < 0.05$) (Table 1). The study was approved by the Municipal Ethical Committee of Copenhagen and Frederiksberg to be in accordance with the Helsinki-II declaration. All subjects gave written consent after the experimental procedure had been explained to them.

Experimental design

Each subject completed the three 14 d dietary periods, a sucrose-rich (sucrose), a starch-rich (starch) and a fat-rich (fat). The order of the periods differed, but subjects in the PO and C groups were 'paired' (except for two controls) so that the diet order was similar in the two groups (see appendix 1). Each subject was measured at the same time in her menstrual cycle. Before each experimental period, subjects were given a standardized, weight-maintenance diet for 3 d. The third day was spent in a respiration chamber (day 0). Subjects were not allowed to engage in strenuous physical activity on the day before the respiration chamber stay. After the standard diet, the experimental diets were supplied in *ad libitum* amounts (see below) to be consumed at home for 14 d. On day 14, the subjects spent another day in the respiration chamber. In the morning on day 15, blood samples were taken in the fasted state and postprandially after breakfast and lunch. Body weight and composition were measured before and after each stay in the respiration chamber (days 1 and 15). At least 2 weeks and no more than 6 weeks separated the dietary periods. The subjects were instructed not to change their physical activity pattern during or between the three experimental diets. Furthermore, they were told not to weigh themselves during the experimental period. Data on blood concentrations of glucose, lipids and different hormones will be published separately.

Table 1 Subject characteristics[a]

	Post-obese (PO) women (n = 9)	*Control (C) women (n = 11)*
Age (y)	39 ± 3	38 ± 3
Height (m)	1.67 ± 0.01	1.66 ± 0.01
Weight (kg)	63.9 ± 1.8	61.9 ± 1.2
Body mass index (kg/m^2)	22.9 ± 0.7	22.6 ± 0.4
Maximal weight (kg)	90 ± 4	63 ± 1
Fat mass (kg)	16.7 ± 1.2	17.0 ± 0.8
Fat mass (%)	27.5 ± 1.6	28.4 ± 1.0
Fat-free mass (kg)	47.2 ± 1.0	44.9 ± 0.8
Fat-free mass (%)	72.5 ± 1.6	71.5 ± 1.0
Reported habitual diet[b]		
Energy (MJ)	7930 ± 725	10 084 ± 559*
Carbohydrate (E%)	54.7 ± 2.4	45.5 ± 2.6*
Fat (E%)	29.0 ± 1.6	35.7 ± 1.1*
Protein (E%)	15.5 ± 1.1	13.7 ± 0.6
Alcohol (E%)	1.0 ± 0.5	5.2 ± 2.0
Sucrose (E%)	10.4 ± 4.6	8.4 ± 1.6
Dietary fiber (g)	22 ± 3	21 ± 2

Data are means ± s.e.m. [a] Measured on day 1 of the first of three *ad libitum* diets. [b] From 7 d weighed food records. E%: Energy-percent. * Post-obese *vs* controls, $P < 0.05$ by unpaired *t*-test.

Diets

The planned macronutrient composition of the sucrose and starch diet was similar with 59 energy-percent (E%) carbohydrate, 28 E% fat, and 13 E% protein, while the fat diet contributed 45–50 E% fat, 37–42 E% carbohydrate and 13 E% protein. Sucrose contributed 23 E% on the sucrose diet and 2 E% on the starch and fat diets. (Energy contribution from carbohydrate is expressed as metabolizable monosaccharide equivalents, with 17 kJ/g. Energy contribution from fat and protein was set at 38 kJ/g and 17 kJ/g, respectively). Dietary fiber amounted to 2.0 g/MJ on the sucrose and fat diet and 3.6 g/MJ on the starch diet. The ratio between polyunsaturated and saturated fatty acids (P/S-ratio) was 0.4 on the fat diet and 0.7 on both the sucrose and starch diets.

The diets were *ad libitum* up to a ceiling of 15 MJ/d for the first five subjects and 18 MJ/d for the last 15 subjects. The change was made after one of the first C subjects had been able to consume all of the 15 MJ/d version (starch diet). Thereafter no subject consumed all the foods delivered (18 MJ/d), but one more C and five PO consumed more than 15 MJ/d (on average 15.6 MJ/d). The C subject was on the sucrose diet, while the PO's were on the fat and/or sucrose diet. The subjects also received two extra sandwiches of 0.5 MJ/d at the beginning of each dietary period, and each day a piece of fresh fruit to be eaten if desired. The subjects collected the food at the department twice a week. At the same time, they returned all left-overs (including packing) for weighing and recording by the dietician. The subjects were told to eat as much as they liked until they were pleasantly satisfied.

A 4 d rotating menu was given for each diet. Breakfast, lunch, dinner and snacks contributed 25%, 30%, 35% and 10%, respectively, of the daily energy load. Each meal had the same energy composition as the whole diet. The three diets contained similar food items and dishes, whenever possible (see 'menu 1' in appendix 2). This was done both to hide the different energy compositions from the subjects and to eliminate differences in energy intake due to

the different palatability of the diets. During the planning of the diets, great care was taken to make all the meals as palatable as possible, taking into consideration that a major part of the meals would be frozen, thawed and often heated before consumption. When preparing the different foods and dishes (for example, sandwiches, warm dishes, pasta or rice salads), an even distribution of all the ingredients within the dish was aimed at. Most of the meals consisted of 2–3 different items (for example, dinner on the sucrose diet = pizza + sweet drink). In order to achieve the correct energy composition in each meal and over the day, the subjects were very carefully instructed in how to consume the prepacked foods (for example, if half the pizza was eaten, half of the drink should be drunk). After the dietary periods, the actual amount consumed by each subject was recorded and the energy content and composition subsequently calculated by a dietician. A multiple-choice questionnaire was given to the subjects after the dietary periods to investigate whether they had identified the macronutrient composition of the three diets.

The standard diet contributed 13 E% protein, 37 E% fat, 50 E% carbohydrate (9 E% sucrose), 2.9 g/MJ dietary fiber and had a P/S-ratio of 0.40. The diet was prepared according to each subjects' individual energy needs (adjusted to the nearest 0.5 MJ) determined from WHO-tables according to age, weight, height, and gender.[14] To allow for a slightly higher level of physical activity outside the respiration chambers a surplus of 0.5 MJ/d was given during the first two days of the standard diet. The subjects were instructed to adhere strictly to the diet. In case they could not consume all foods, they had to bring the left-overs to the department for weighing and recording. The same amount was deducted during the following two dietary periods. The computer database of foods from the National Food Agency of Denmark (Dankost 2.0) was used in the calculations of energy and nutrient intake of the diets.[15]

Anthropometric measurements

Body weight was measured (blinded for the subjects) in the morning of days 1 and 15 after 10 h fasting and after voiding. The same digital scale was used each time (Seca model 707, Copenhagen, Denmark). Body composition was subsequently estimated by electrical bioimpedance using an Animeter (HTS-Engineering Inc, Odense, Denmark). Fat mass (FM) and fat-free mass (FFM) were calculated using the equations by Heitmann.[16] Blood pressure was measured using an automatically inflating cuff (UA-743, A & D Company Ltd, Tokyo, Japan).

Respiration chamber

Two open-circuit respiration chambers were used to measure 24 h energy expenditure (EE) and substrate oxidation rates. The chambers have been described in detail elsewhere.[17,18] Protein oxidation was calculated from 24 h urinary nitrogen excretion. Energy expenditure and oxidation of lipid and carbohydrate were calculated using the equations of Brouwer.[19] Heart rates and electrocardiograms were continuously monitored by a telemetry system (Dialogue 2000, Danica Electronics, Denmark). Heart rate, blood pressure and catecholamines were measured to estimate sympathetic nervous activity. Spontaneous physical activity (SPA) was assessed by two microwave radars (Zettler GHz-Doppler Mime 15, Munich, Germany). Subjects were kept under surveillance by a laboratory technician in the daytime and by trained medical students during the night time.

A fixed protocol for the chamber measurements was followed for all subjects. The subjects arrived at the department at 22.00 h on the evening before the measurements. The next morning they were awakened at 07.45 h, they measured (rectally) their body temperatures, they voided and anthropometric measurements were subsequently performed. The 24 h measurement started at 09.00 h and finished the next morning at 09.00 h. Basal metabolic rate (BMR) was measured on the second day from 08.00–09.00 h. Diet-induced thermogenesis (DIT) was measured on the first day from 06.00–22.00 h, where the subjects were sedentary:

$$\text{DIT (kJ)} = \text{EE from 06.00-22.00 h (kJ/4 h)} - (\text{BMR(kJ/h)*4 (h)})$$

$$\text{DIT (\%)} = \frac{\text{DIT (kJ)}}{\text{Energy consumed at dinner (kJ)}} \times 100$$

Three meals were given during the stay, breakfast at 09.00 h, lunch + snack at 13.00 h and dinner at 18.00 h. The meals were eaten within one hour. The amount of food in the meals was fixed on day 0, but *ad libitum* on day 14. The menu given on day 13 (the day before the chamber stay) and day 14 (in the chamber) was always menu 'day 1'. Two bicycle bouts of 75 watt and 15 min duration were performed at 11.00 h and 16.00 h and two periods of walking (forward and back in the chamber 25 times) at 9.30 h and 14.30 h. Otherwise the subjects could read, write, phone, watch television or listen to the radio. Consumption of water, tea, coffee and smoking (only 'light'cigarettes) during the day was allowed, but the total amount drunk and smoked during the first stay was noted and repeated during the ensuing five respiration chamber stays. Three PO and one C subject smoked on average 10.3 ± 1.7 and 11 cigarettes/d, respectively. The influence of smoking on O_2 consumption and CO_2 production in the chambers has previously been analyzed. Five Prince Light cigarettes were combusted, leading to a CO_2 production of 0.31 l/cigarette and an O_2 consumption of 0.29 l/cigarette. For all smokers, O_2 consumption and CO_2 production were corrected for these values in the subsequent analyses.

Every hour from 09.00 h (fasting) until 23.00 h, subjects recorded their sensations of hunger, fullness,

desire to eat and prospective food consumption on 10 cm visual analogue scales (VAS) with words anchored at each end, expressing the most positive (for example, good, pleasant) or the most negative rating (for example bad, unpleasant).[20] Overall palatability, taste, aftertaste, smell, and visual appeal of the 3 test meals were also recorded by the subjects ($n = 8$ PO, $n = 10$ C).[20]

Blood sampling

On day 15 the subjects left the respiration chamber at 09.00 h. After voiding and weighing, the subjects lay on a bed in the supine position and a Venflon catheter (Viggo, Gothenborg, Sweden) was inserted into an antecubital vein in the arm. After 10 min rest, a fasting blood sample was taken. Breakfast was eaten at 10.00 h and lunch at 14.00 h. Blood samples were taken after 15, 30, 60, 120 and 240 min from initiation of breakfast and lunch. The subjects rested in the supine position for 10 min before each blood sample. During the day the subjects could sit, walk quietly or go to the toilet. The amount of food given for breakfast and lunch was similar to the amounts consumed on day 14 in the chamber. Coffee, tea and water consumption as well as smoking (two PO and one C) were allowed, but the amount and time were copied from the first respiration chamber stay. The type of activity during the day was noted. Smoking has been found to increase sympathetic nervous activity and catecholamine concentrations.[21] However, since the number, type and timing of cigarettes smoked was identical on the three blood sampling days, the effect was assumed to be the same on the three occasions.

Blood sampling was not possible for one PO subject. In order to keep the groups matched, one control subject was therefore removed from the analyses of blood parameters. One PO subject developed a fever in the evening on day 14 of the sucrose diet. Blood sampling was therefore postponed by a few days on the *ad libitum* diet until the fever had disappeared (day 17).

Laboratory analyses

Blood was sampled without stasis through the indwelling catheter. Blood for determination of plasma catecholamines was collected in iced tubes containing ethylene glycol-bis(β-aminoethyl ether)-N,N,N′,N′-tetraacetic acid (EGTA) and glutathione. Within 30 min, samples were centrifuged for 10 min at 3000 *g* and 4°C and the plasma stored at −80°C until determination by an electrochemical detection method.[22] Urinary nitrogen concentration was measured using a nitrogen analyzer (NA 1500, Carlo Erba Strumentazione, Milan, Italy). The energy content of 10 MJ versions of each diet was analyzed from duplicate collections of one day (standard diet) or 7 d menus (experimental diets). The diets were mixed, homogenized, freeze-dried and ground before determination by adiabatic bomb calorimetry (IKA, Janke & Kunkel GmbH, Staufen, Germany).

Statistical analyses

All results are given as means ± s.e.m. Initial group differences were tested by a *t*-test (Table 1). Other differences between the three diets and two groups were tested by parametric analysis of variance (ANOVA) ('split-plot' design) using the GLM procedure in SAS (SAS Institute, Cary, NC, USA). Factors were group, diet and group*diet, using subject (group) as error term for group effects. In case of significance a *t*-test on least squares means (for unbalanced designs) was used to test for differences between groups or diets. Differences in postprandial catecholamine responses were tested by an analysis of variance (ANOVA) with time, diet, group, diet*group, time*group, diet*time, and diet* time*group as factors and subject (group) as error term for group effects. The areas under the curves (d-AUC) were calculated separately for each subject as the difference between the integrated area of the response curve and the rectangular area determined by the basal values. Negative areas were included. Where data adjustment was performed, this was done between groups on each of the three diets separately, as described by Ravussin and Bogardus.[23] For 24 h energy expenditure adjustments for differences in fat-free mass were thus performed using the equation: $EE_{adj} = EE_{act} + \alpha (FFM_{mean} - FFM_{act})$, where EE_{act} is the measured unadjusted EE, FFM_{mean} the total group mean ($n = 20$), FFM_{act} the actual FFM, α the slope and β the intercept derived from linear regression analysis between FFM and EE in both groups (on each diet): $EE = \beta + \alpha \times FFM$. Simple linear regression analyses were performed within each diet ($n = 20$). The significance level was set at $P < 0.05$. Statgraphics Software version 4.2 (Graphic Software Systems, Inc, Rockville, MD, USA) and the Statistical Analysis Package were used in the statistical calculations.

Results

***Ad libitum* energy and macronutrient intake**

Average 14 d energy intake (EI) for all subjects was lowest on the starch diet (9.1 ± 0.4 MJ/d) compared with both the sucrose (10.3 ± 0.4 MJ/d) and fat diet (10.2 ± 0.4 MJ/d) ($P < 0.05$) (Figure 1). Compared with data from 7 d food records, PO consumed more on the fat ($P < 0.05$) and sucrose diets ($P < 0.001$), but the same on the starch diet. Conversely, C consumed less on the starch diet ($P < 0.05$), but the same on the fat and sucrose diets compared with 7 d food records.

The macronutrient composition of the ingested food (delivered minus left-overs) on the three diets was very similar to the planned macronutrient composition. Thus on the fat, starch and sucrose diet the intake

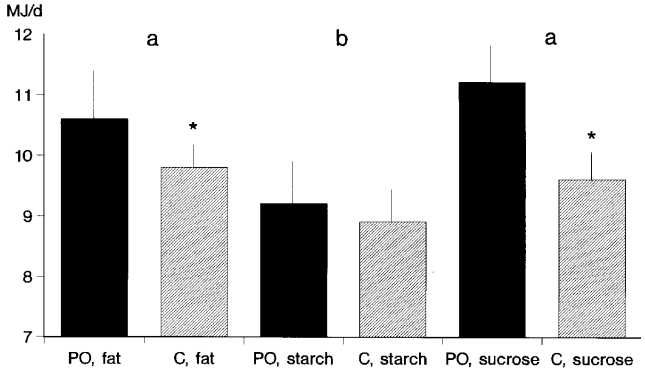


Figure 1 Mean (±s.e.m.) 14 d *ad libitum* energy intake in post-obese (PO, $n=9$) and matched controls (C, $n=11$) on a fat-rich, a starch-rich and a sucrose-rich diet. Significant by ANOVA, $P<0.05$: Diet × group interaction and diet effect. Different letter: Significant difference between diets, $P<0.05$. *: Differences between PO and C, $P<0.05$.

of carbohydrate averaged 40.8, 59.1 and 58.6 E% ($P<0.0001$), of sucrose 2.2, 2.6 and 23.2 E% ($P<0.0001$), of fat 46.1, 28.0 and 28.6 E% ($P<0.0001$) and of protein 13.1, 13.4, and 13.2 E% ($P<0.05$), respectively (Table 2). The weight of foods consumed was significantly higher on the sucrose diet (1512 ± 60 g/d) compared with the starch diet (1411 ± 55 g/d) ($P<0.05$) and lowest on the fat diet (1228 ± 51 g/d) ($P<0.05$ *vs* sucrose or starch) (Table 2). Compared with the C group the PO subjects consumed significantly more energy on the sucrose and fat diets (Figure 1). Compared with the controls, the PO subjects consumed significantly more (in grams) carbohydrate and sucrose on the sucrose diet (Table 2). No other significant group differences were observed.

The pattern of *ad libitum* 24 h energy and macronutrient intake during the respiration chamber stay (day 14) showed similar differences as during the previous 13 d period (Table 3). However, the C group consumed approximately 1 MJ/d more on the sucrose diet ($P<0.05$), and approximately 0.5 MJ/d less on the fat and starch diets ($0.05<P<0.1$). PO only consumed less on the starch diet (approximately 1.2 MJ/d, $P=0.07$) when in the chamber (Table 2 and 3).

The questionnaire given to the subjects after the dietary periods revealed that a majority of the subjects were able to tell whether the diets had a low, medium or high content of fat, carbohydrate, protein, dietary fibre, sucrose or artificial sweetener. Interestingly, however, several subjects (especially in the PO group) thought that the sucrose diet was also rich in fat.

Food analyses of the fat, starch, sucrose and standard diet showed a 4.3%, 2.3%, 0.8% and 2.7% higher energy content, respectively, compared with the calculated energy content. These values were considered within the range of uncertainty in recording of food consumption. The calculated values were therefore not corrected.

Table 2 Average daily food and macronutrient intake during 14 d *ad libitum* intake of a diet rich in fat, starch or sucrose

		Fat	*Starch*	*Sucrose*	*ANOVA P-value*		
					d × g	*diet*	*group*
Energy, kJ/d	PO	10 684 ± 721[a]	9229 ± 606[b]	11 166 ± 615[a]	0.05	0.0001	0.21
	C	9803 ± 388*	8934 ± 473	9576 ± 491*			
Energy-%:							
Carbohydrate	PO	41.1 ± 0.7[a]	59.1 ± 0.2[b]	58.7 ± 0.2[b]	0.84	0.0001	0.60
	C	40.6 ± 0.6	59.1 ± 0.3	58.6 ± 0.2			
Sucrose	PO	2.2 ± 0.2[a]	2.3 ± 0.2[a]	23.4 ± 0.2[b]	0.06	0.0001	0.99
	C	2.2 ± 0.1	2.7 ± 0.2	23.0 ± 0.2			
Fat	PO	45.9 ± 0.7[a]	27.9 ± 0.2[b]	28.6 ± 0.1[b]	0.90	0.0001	0.48
	C	46.3 ± 0.6	28.1 ± 0.2	28.6 ± 0.1			
Protein	PO	13.1 ± 0.1[a]	13.4 ± 0.1[b]	13.1 ± 0.1	0.66	0.02	0.46
	C	13.2 ± 0.1	13.4 ± 0.1	13.3 ± 0.1			
Carbohydrate, g/d	PO	259 ± 19[a]	320 ± 20[b]	385 ± 21[c]	0.05	0.0001	0.17
	C	233 ± 7	310 ± 16	330 ± 17*			
Sucrose, g/d	PO	14 ± 1[a]	13 ± 1[a]	153 ± 8[b]	0.01	0.0001	0.17
	C	13 ± 1	14 ± 1	130 ± 7*			
Dietary fiber, g/d	PO	23 ± 2[a]	32 ± 2[b]	22 ± 1[a]	0.57	0.0001	0.24
	C	21 ± 1	31 ± 2	19 ± 1			
Fat, g/d	PO	129 ± 8[a]	68 ± 5[b]	84 ± 5[c]	0.19	0.0001	0.28
	C	120 ± 6	66 ± 3	72 ± 4			
P/S-ratio	PO	0.40 ± 0.01[a]	0.71 ± 0.00[b]	0.69 ± 0.00[c]	1.00	0.0001	0.93
	C	0.40 ± 0.01	0.71 ± 0.00	0.69 ± 0.01			
Protein, g/d	PO	82 ± 6[a]	73 ± 5[b]	87 ± 5[a]	0.09	0.0001	0.28
	C	76 ± 3	71 ± 4	75 ± 4			
Weight of food, g/d	PO	1302 ± 91[a]	1443 ± 84[b]	1624 ± 90[c]	0.16	0.0001	0.21
	C	1169 ± 52	1385 ± 76	1420 ± 72			
Energy density, kJ/g	PO	8.2 ± 0.1[a]	6.4 ± 0.1[b]	6.9 ± 0.1[c]	0.15	0.0001	0.57
	C	8.4 ± 0.1	6.5 ± 0.1	6.7 ± 0.1			

Data are means ± s.e.m. PO: Post-obese ($n=9$). C: Controls ($n=11$). d × g = diet × group interaction. Results in the same row with different superscript are significantly different (per diet, $n=20$) ($P<0.05$). * PO *vs* C, $P<0.05$.

Table 3 Energy and macronutrient intake and oxidations on day 14 of 14 d *ad libitum* intake of a diet rich in fat, starch or sucrose

					ANOVA P value		
		Fat	*Starch*	*Sucrose*	*d × g*	*diet*	*group*
BMR adjusted[1], kJ/h	PO	271 ± 8	270 ± 5	277 ± 7	0.29	0.68	0.17
	C	268 ± 7	262 ± 4	260 ± 3			
DIT, %	PO	9.6 ± 1.6	12.0 ± 1.9	10.3 ± 0.8	0.70	0.09	0.44
	C	11.4 ± 1.5	13.9 ± 2.1	10.2 ± 0.7			
24 h NP-RQ adjusted[2]	PO	0.843 ± 0.006[a]	0.865 ± 0.006[b]	0.914 ± 0.009[c]	0.68	0.0001	0.93
	C	0.842 ± 0.004	0.871 ± 0.004	0.911 ± 0.005			
24 h energy							
Expenditure, kJ/d	PO	8354 ± 151[a]	8190 ± 183[a]	8770 ± 129[b]	0.14	0.002	0.09
	C	8087 ± 124	7994 ± 163	8194 ± 174			
Intake, kJ/d	PO	10 543 ± 753[a]	8073 ± 745[b]	11 276 ± 940[c]	0.06	0.0001	0.45
	C	9282 ± 345	8309 ± 342	10 517 ± 456			
Balance, kJ/d	PO	2188 ± 682[a]	−117 ± 718[b]	2506 ± 842[c]	0.07	0.0001	0.72
	C	1194 ± 376	314 ± 268	2323 ± 328			
24 h carbohydrate							
Oxidation, g/d	PO	197 ± 16[a]	219 ± 15[b]	301 ± 26[c]	0.27	0.0001	0.50
	C	176 ± 6	228 ± 10	278 ± 13			
Intake, g/d	PO	259 ± 21[a]	281 ± 25[b]	391 ± 32[c]	0.10	0.0001	0.53
	C	225 ± 8	294 ± 12	366 ± 15			
Balance, g/d	PO	63 ± 12[a]	62 ± 17[a]	90 ± 13[b]	0.56	0.0001	0.73
	C	49 ± 5	66 ± 7	88 ± 8			
24 h fat							
Oxidation, g/d	PO	90 ± 7[a]	84 ± 7[b]	54 ± 11[c]	0.24	0.0001	0.84
	C	95 ± 4	74 ± 4	54 ± 4			
Intake, g/d	PO	126 ± 8[a]	59 ± 6[b]	85 ± 7[c]	0.06	0.0001	0.37
	C	112 ± 5	60 ± 2	79 ± 4			
Balance, g/d	PO	36 ± 14[a]	−25 ± 12[b]	31 ± 18[a]	0.07	0.0001	0.73
	C	17 ± 8	−14 ± 6	25 ± 7			
24 h protein							
Oxidation, g/d	PO	72 ± 6[a]	56 ± 4[b]	74 ± 6[a]	0.04	0.0001	0.35
	C	67 ± 2*	57 ± 2	64 ± 4*			
Intake, g/d	PO	81 ± 6[a]	63 ± 3[b]	89 ± 8[c]	0.12	0.0001	0.42
	C	71 ± 3	64 ± 3	83 ± 4			
Balance, g/d	PO	9 ± 3[a]	8 ± 3[a]	15 ± 4[b]	0.27	0.0001	0.87
	C	4 ± 3	7 ± 1	18 ± 2			

Data are means ± s.e.m. BMR: Basal metabolic rate (day 15, 8–9 am). DIT: Diet-induced thermogenesis (6–10 pm). NP-RQ: non-protein respiratory quotient. PO: Post-obese ($n = 9$). C: Controls ($n = 11$). d × g = diet × group interaction. [1]Adjusted for differences between groups in fat-free mass. [2]Adjusted for differences in energy balance and 14 d weight changes. Results in the same row with different superscript are significantly different (per diet, $n = 20$) ($P < 0.05$). *PO *vs* C, $P < 0.05$.

Body weight and composition

There were no significant differences between the 2 groups in changes of body weight or composition (Figure 2). Compared to a change of 0.0 kg, total body weight decreased on the starch diet by 0.7 ± 0.2 kg ($P < 0.05$), but was unchanged on the fat (-0.3 ± 0.3 kg) and sucrose diet (0.2 ± 0.2 kg). The changes were significantly different between the starch and sucrose diets ($P < 0.05$) (Figure 2). Fat-free mass did not change significantly on the starch and fat diet, but tended to increase on the sucrose diet (0.3 ± 0.1 kg) ($P = 0.08$). Again the difference between the starch and sucrose diet was significant ($P < 0.05$) (Figure 2). Fat mass decreased on the starch diet (-0.4 ± 0.1 kg) ($P < 0.05$), but was unchanged on the sucrose and fat diet (Figure 2). There were no differences in fat mass changes between the three diets.

Energy expenditure (EE)

Before the three experimental diets 24 h EE adjusted for group differences in FFM (day 0) was similar between groups and diets (average PO: 8271 kJ/d, C: 8192 kJ/d). After the intervention periods, however, 24 h EE adjusted for differences between groups in FFM and EI was 3% and 4.5% higher on the sucrose diet (8453 ± 128 kJ/d) compared with the fat diet (8207 ± 99 kJ/d) and starch diet (8082 ± 121 kJ/d) ($P < 0.05$), respectively (Figure 3). This was mainly due to an increase in the PO group (8625 ± 112 kJ/d) compared with the C group (8311 ± 162 kJ/d) (Figure 3). The diet differences persisted after removing the PO subject who developed a fever on the sucrose diet from the analysis (8600 ± 123 kJ/d, $n = 8$). Basal metabolic rate adjusted for differences in FFM between groups was not significantly different between groups or diets (Table 3). Diet-induced thermogenesis (6–10 pm) expressed in percent of EI at dinner was also not significantly different between diets or groups (Table 3). However, when comparing 24 h EE with EI over the whole day, some differences appeared. Using 24 h EE on the starch diet as baseline, we observed an increase in 24 h EE of 4.6% on the sucrose diet and of 1.5% on the fat diet ($P < 0.05$),

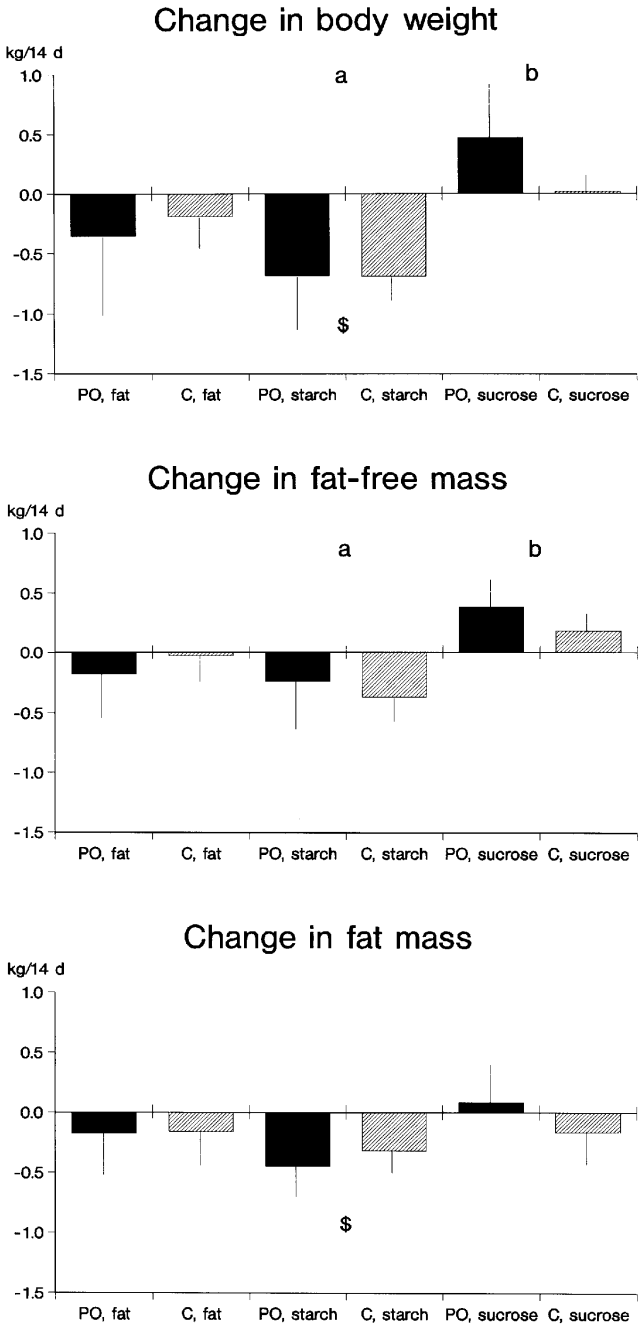


Figure 2 Mean (±s.e.m.) changes in body weight, fat-free mass and fat mass in 9 post-obese (PO) and 11 matched controls (C) after 14 d *ad libitum* of a fat-rich, a starch-rich and a sucrose-rich diet. Significant by ANOVA, $P<0.05$; Changes in body weight and fat-free mass: diet effect. Different letter: difference between diets, $P<0.05$. $: Different from 0.0 kg ($n=20$), $P<0.05$. There were no differences between PO and C.

thus a three-fold difference between the two diets. Expressed in relation to the increase in EI on the diets (starch diet still used as baseline) the increase in 24 h EE amounted to 14.0% (PO: 18.1% and C: 9.0%) on the sucrose diet, but to only 7.6% (PO: 5.4% and C: 8.5%) on the fat diet.

On the starch diet 24 h energy balance was close to 0 kJ/d (120 ± 348 kJ/d), while it was positive on both the fat (1642 ± 377 kJ/d) and sucrose diets

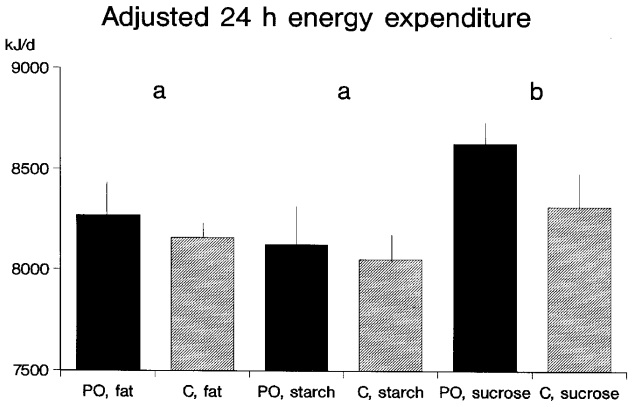
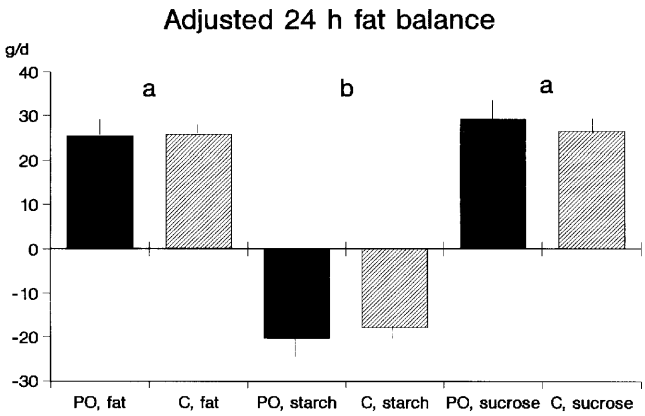


Figure 3 Mean (±s.e.m.) 24 h energy expenditure (adjusted for differences between groups in energy intake and fat-free mass) in a respiration chamber after 14 d *ad libitum* intake of a fat-rich, a starch-rich and a sucrose-rich diet. Subjects were 9 post-obese (PO) and 11 matched controls (C). Significant by ANOVA, $P<0.05$; Adjusted 24 h energy expenditure: diet effect. Different letter: Difference between diets, $P<0.05$.

(2405 ± 421 kJ/d) ($P<0.05$ between all three diets) (Table 3). Simple regression analyses between 24 h EE and EI on day 14 showed a significant correlation only on the sucrose diet ($r=0.69$, $P=0.0008$). The regression line ($y=\beta+\alpha*x$) was 24 h EE (kJ/d) = 6484 (kJ/d) + 0.18 * 24 h EI (kJ/d). On the two other diets, the correlation was not significant (fat: $r=0.38$, $P=0.09$, starch: $r=0.37$, $P=0.11$). When analyzing the groups separately, a significant correlation was also observed for C on the starch diet ($r=0.64$, $P=0.03$).

Macronutrient oxidation rates

On the standard diet, before the experimental diets, 24 h non-protein respiratory quotient (NP-RQ) was similar between groups and diets (average NP-RQ = 0.843). After the three *ad libitum* diets NP-RQ was highest on the sucrose diet (0.913 ± 0.021) compared with the starch diet (0.868 ± 0.016) and lowest on the fat diet (0.842 ± 0.015) ($P<0.05$). There were no differences in NP-RQ between the two groups, even after adjusting for differences in 14 d body weight changes and energy balance (Table 3). Carbohydrate oxidation was highest on the sucrose diet and lowest on the fat diet compared with the starch diet ($P<0.05$) (Table 3). Conversely, fat oxidation was highest on the fat diet and lowest on the sucrose diet compared with the starch diet ($P<0.05$) (Table 3). There were no differences in carbohydrate or fat oxidation between the two groups. Protein oxidation however, was significantly increased in PO compared with C on the fat and sucrose diet ($P<0.05$) (Table 3). A negative fat balance was observed on the starch diet compared with sucrose and fat diets ($P<0.05$), while carbohydrate and protein balances were more positive on the sucrose diet compared with the other two diets ($P<0.05$) (Table 3). These differences persisted after adjusting for differences between groups in 24 h energy balance (Figure 4).



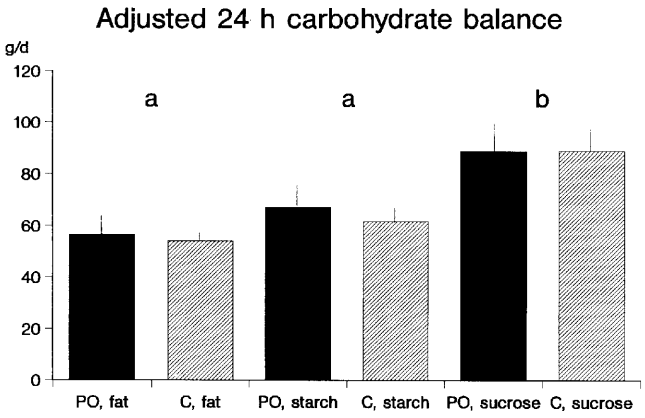
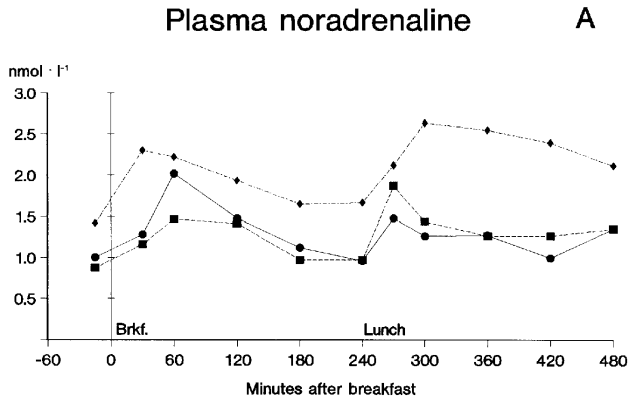


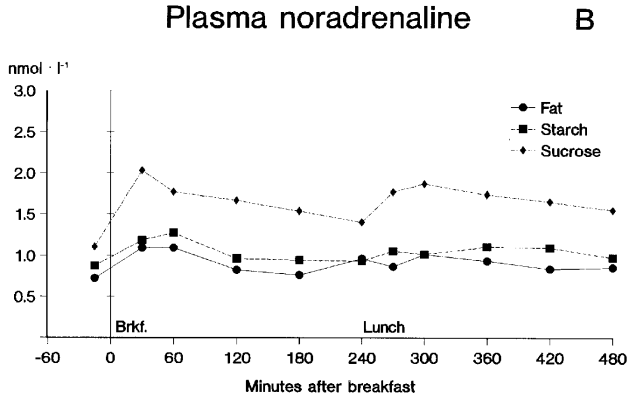
Figure 4 Mean (±s.e.m.) 24 h fat and carbohydrate balances (adjusted for differences in energy balance within diets) in a respiration chamber after 14 d *ad libitum* intake of a fat-rich, a starch-rich and a sucrose-rich diet. Subjects were 9 post-obese (PO) and 11 matched controls (C). Significant by ANOVA, $P < 0.05$; Both fat and carbohydrate balance: diet effect. Different letter: Difference between diets, $P < 0.05$.

Plasma noradrenaline (NA) and adrenaline (A)

On day 15, fasting and total postprandial NA were higher on the sucrose diet compared with the fat and starch diet ($P < 0.05$) (Figure 5). A higher concentration was observed for PO compared with C both on the sucrose and fat diets ($P < 0.05$) (Figure 5). The area under the curve after subtracting fasting values (d-AUC) was 2–3 times larger on the sucrose diet compared with the fat and starch diets ($P < 0.05$). There were no significant differences between groups in d-AUC's (Figure 5). This was also true after adjusting for group differences in energy balance (day 14).

After 14 d on the *ad libitum* diets, fasting as well as total postprandial A concentrations were significantly higher on the sucrose diet compared with the starch and the fat diet ($P < 0.05$) (Figure 6). The d-AUC's were higher on the sucrose diet ($P < 0.05$) and tended to be likewise after the starch diet ($P = 0.055$) compared with the fat diet. There were no differences between the groups in d-AUC's, even after adjusting for differences between groups in energy balance (day 14) (Figure 6).





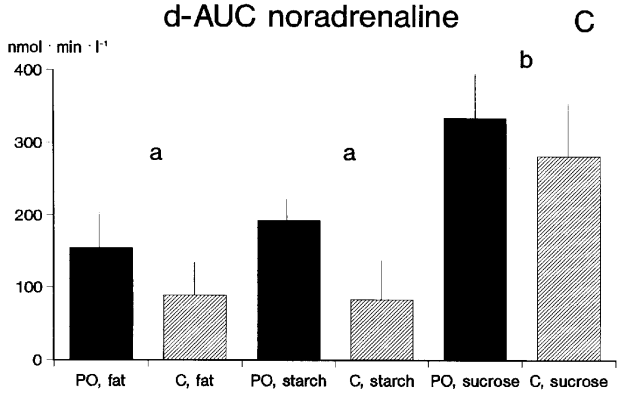


Figure 5 Mean plasma noradrenaline concentrations in : A, 8 post-obese (PO) and B, 10 controls (C) after 14 d *ad libitum* intake of a fat-rich, a starch-rich and a sucrose-rich diet. Brkf. = Breakfast. Significant by ANOVA, $P < 0.05$: diet*time, diet (sucrose > fat = starch) and time. Within each diet: Significant difference between PO and C on the fat and sucrose diets, $P < 0.05$. C, mean (±s.e.m.) areas under the curves above fasting levels (d-AUC). Significant by ANOVA, $P < 0.05$: Diet effect. Different letter: difference between diets, $P < 0.05$.

There were no correlations (for each diet separately, $n = 20$) between energy balance (day 14) or energy intake in breakfast and lunch (day 15) and d-AUC for NA or A. For both NA and A, the diet differences in d-AUC's persisted after dividing the d-AUC's by the amount of kJ consumed during breakfast and lunch on day 15 (data not shown).

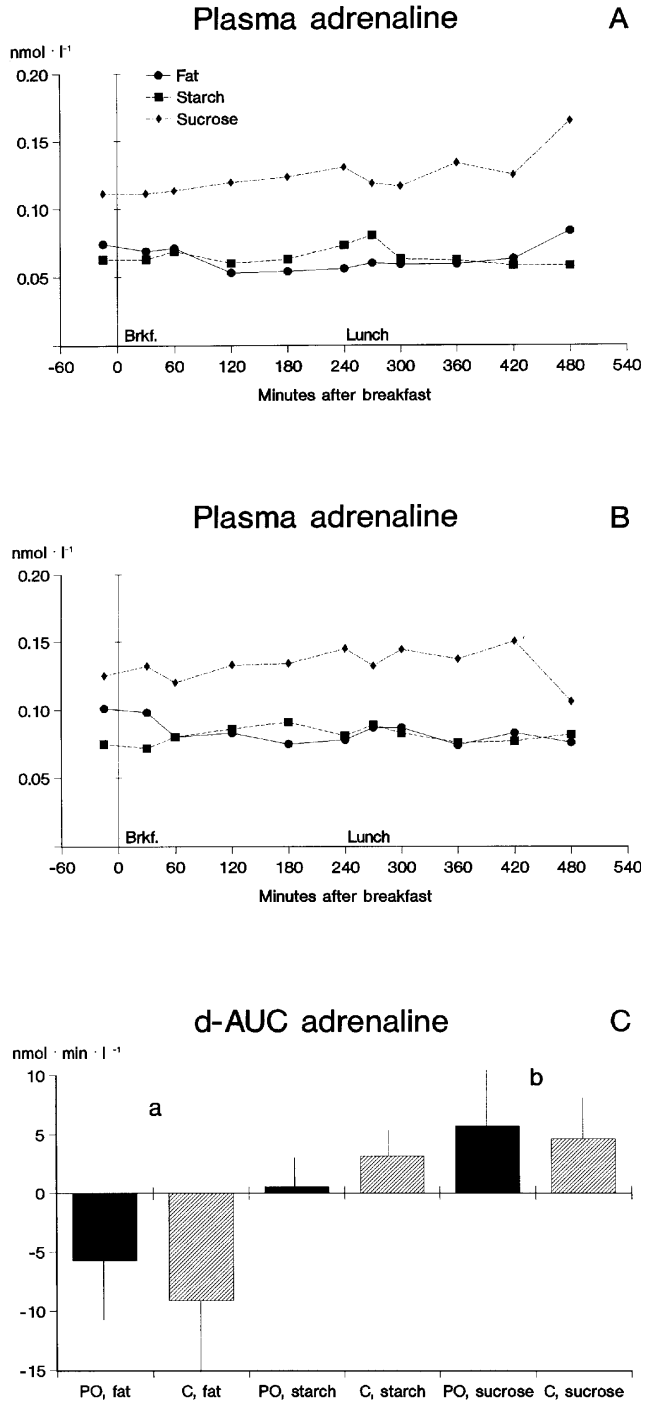


Figure 6 Mean plasma concentrations in: A, post-obese (PO) and B, 10 controls (C) after 14 d *ad libitum* intake of a fat-rich, a starch-rich and a sucrose-rich diet. Brkf. = Breakfast. Significant by ANOVA, $P < 0.05$: time*group, diet (sucrose > fat = starch) and time. C, mean (± s.e.m.) areas under the curves above fasting levels (d-AUC). Significant by ANOVA, $P < 0.05$: Diet effect. Different letter: difference between diets, $P < 0.05$.

Spontaneous physical activity (SPA)

On the standard diet, PO had significantly lower 24 h SPA values than C before all three diets (6.1 ± 0.2% *vs* 7.6 ± 0.3%) ($P < 0.05$). After 14 d on the diets, significantly lower values were still observed in PO ($P < 0.05$) and there were no differences between the diets (Table 4).

Heart rate and blood pressure

Heart rate in the chamber on day 0 was not different between diets or groups (average for PO: 70.6 b/min and C: 69.3 b/min). On day 14 a diet*group interaction was found ($P < 0.05$) due to an increase on the sucrose diet in the PO subjects compared with the C subjects (Table 4). When the PO subject who had a slight fever was removed from the analyses, this diet*group interaction disappeared, however.

No significant differences in fasting diastolic or systolic blood pressure were observed in the morning after the standard diet (day 1) or after the *ad libitum* diets (day 15) (Table 4).

Appetite and palatability ratings

Comparison of mean appetite ratings (from 09.00–23.00 h) on day 14 showed that the subjects felt less desire to eat (prospective consumption) and greater fullness on the sucrose diet compared with the fat diet ($P < 0.05$) (Table 5). Furthermore, the subjects felt more satisfied on the sucrose diet compared with both the fat and starch diet ($P < 0.05$) (Table 5). On all three diets PO felt less hungry, more satisfied and less desire to eat that C ($P < 0.05$) (Table 5). When dividing each of the four appetite scores by the amount of kJ consumed over the day, the results changed slightly (Table 5). The subjects felt less hungry and less desire to eat on the sucrose diet compared with the other two diets ($P < 0.05$) (Table 5), but satiety and fullness were now significantly higher on the starch diet compared with the other two diets ($P < 0.05$) (Table 5). An overall group difference was still observed.

Average subjective ratings of breakfast, lunch and dinner showed that the subjects estimated overall palatability to be the best on the sucrose diet compared with the other two diets ($P < 0.05$) (Table 6). There were no other significant diet differences. Between the groups, PO found overall palatability and taste of the diets better than C ($P < 0.05$) (Table 6).

Discussion

***Ad libitum* 14 d EI**

The present study showed that a diet composed according to the Nordic nutrition recommendations (<30 E% fat, 55–60 E% carbohydrate, > 3 g/MJ/d dietary fiber) resulted in a significantly lower energy intake and a small but significant reduction in body weight and fat mass after 14 d *ad libitum* intake in both PO and normal subjects than a sucrose-rich or fat-rich diet. These findings are therefore in accordance with previous studies of similar or longer duration in normal-weight and overweight subjects showing a spontaneous reduction in EI and[a] weight loss on an *ad libitum* starch-rich, fiber-rich diet.[24–26]

Table 4 Fasting and/or 24 h heart rate, spontaneous physical activity (SPA) and blood pressure (BP) after 14 d *ad libitum* intake of a diet rich in fat, starch or sucrose

					ANOVA P-value		
		Fat	*Starch*	*Sucrose*	*d × g*	*diet*	*group*
24 h SPA, %	PO	5.9 ± 0.2	6.3 ± 0.3	6.3 ± 0.4	0.66	0.32	0.03
	C	7.4 ± 0.4*	7.5 ± 0.4*	7.5 ± 0.5*			
24 h heart rate, b/min	PO	70 ± 3	69 ± 3	75 ± 4	0.04	0.08	0.57
	C	70 ± 2	69 ± 2	69 ± 3*			
Fasting heart rate, b/min	PO	64 ± 3	63 ± 3	69 ± 4	0.03	0.15	0.23
	C	62 ± 8	61 ± 2	61 ± 2*			
Fasting systolic BP, mmHg	PO	111 ± 5	109 ± 3	114 ± 8	0.12	0.71	0.55
	C	113 ± 4	119 ± 5	114 ± 6			
Fasting diastolic BP, mmHg	PO	67 ± 3	68 ± 3	71 ± 4	0.99	0.12	0.79
	C	66 ± 3	66 ± 3	70 ± 4			

Data are means ± s.e.m. PO: Post-obese ($n = 9$). C: Controls ($n = 11$). d × g = diet × group interaction. Morning fasting, day 15. * PO *vs* C, $P < 0.05$.

Table 5 Appetite sensations† on day 14 after 14 d *ad libitum* intake of a diet rich in fat, starch or sucrose

					ANOVA P-value		
		Fat	*Starch*	*Sucrose*	*d × g*	*diet*	*group*
Hunger, cm	PO	2.7 ± 0.4	2.6 ± 0.4	2.1 ± 0.4	0.59	0.07	0.007
	C	4.2 ± 0.3*	4.1 ± 0.4*	4.0 ± 0.4*			
Satiety, cm	PO	7.2 ± 0.4[a]	7.3 ± 1.1[a]	7.9 ± 0.4[b]	0.54	0.02	0.01
	C	5.9 ± 0.3*	6.1 ± 1.1*	6.3 ± 0.4*			
Prospective consumption, cm	PO	3.3 ± 0.4[a]	3.0 ± 0.4	2.5 ± 0.5[b]	0.46	0.005	0.05
	C	4.4 ± 0.4*	4.1 ± 0.4*	3.9 ± 0.4*			
Fullness, cm	PO	6.7 ± 0.7[a]	6.8 ± 0.6	7.5 ± 0.6[b]	0.60	0.03	0.13
	C	5.8 ± 0.4	6.0 ± 0.3	6.2 ± 0.4			
Hunger, cm* min/kJ	PO	0.22 ± 0.03[a]	0.29 ± 0.06[a]	0.17 ± 0.04[b]	0.75	0.0001	0.009
	C	0.39 ± 0.03*	0.42 ± 0.04*	0.33 ± 0.04*			
Satiety, cm* min/kJ	PO	0.61 ± 0.07[a]	0.83 ± 0.11[b]	0.63 ± 0.07[a]	0.05	0.0001	0.15
	C	0.55 ± 0.05	0.63 ± 0.05*	0.51 ± 0.03*			
Prospective consumption, cm* min/kJ	PO	0.27 ± 0.03[a]	0.33 ± 0.05[a]	0.19 ± 0.04[b]	0.44	0.0001	0.04
	C	0.40 ± 0.04*	0.41 ± 0.04*	0.32 ± 0.04*			
Fullness, cm* min/kJ	PO	0.57 ± 0.08[a]	0.80 ± 0.13[b]	0.60 ± 0.08[a]	0.11	0.0001	0.30
	C	0.53 ± 0.05	0.62 ± 0.05	0.50 ± 0.03			

† Means ± s.e.m. of hourly registrations (09.00–23.00 h = 840 min) using 10 cm visual analogue scales in a respiration chamber. *Ad libitum* meals were served at 09.00 h, 13.00 h and 18.00 h. PO: Post-obese ($n = 9$). C: Controls ($n = 11$). d × g = diet × group interaction. Results in the same row with different superscript are significantly different (per diet, $n = 20$) ($P < 0.05$). *PO *vs* C, $P < 0.05$.

Table 6 Subjective evaluation of a fat-rich, a starch-rich and a sucrose-rich diet†

					ANOVA P-value		
		Fat	*Starch*	*Sucrose*	*d × g*	*Diet*	*Group*
Overall palatability, cm	PO	2.9 ± 0.8[a]	2.5 ± 0.6[a]	1.6 ± 0.4[b]	0.73	0.04	0.01
(O: Appetizing)	C	4.5 ± 0.5*	4.4 ± 0.6*	4.0 ± 0.6*			
Taste, cm	PO	2.8 ± 0.8	2.0 ± 0.5	1.8 ± 0.6	0.76	0.33	0.04
(O: Good)	C	3.9 ± 0.5*	3.8 ± 0.5*	3.7 ± 0.6*			
Smell, cm	PO	3.5 ± 0.7	3.1 ± 0.6	2.3 ± 0.5	0.79	0.07	0.07
(O: Appetizing)	C	4.6 ± 0.5	4.3 ± 0.5	4.0 ± 0.6			
Visual appeal, cm	PO	3.5 ± 0.8	3.2 ± 0.5	2.1 ± 0.5	0.78	0.08	0.06
(O: Appetizing)	C	4.6 ± 0.6	4.5 ± 0.6	4.0 ± 0.6			
Aftertaste, cm	PO	8.1 ± 0.6	8.3 ± 0.6	8.4 ± 0.6	0.11	0.70	0.15
(O: Much)	C	7.5 ± 0.4	7.5 ± 0.6	6.8 ± 0.7			

† Means ± s.e.m. Average for three *ad libitum* meals during a 24 h respiration chamber stay on day 14 of the *ad libitum* diets ($n = 8$, PO, $n = 10$, C). d × g = diet × group intervention. Results in the same row with different superscript are significantly (per diet, $n = 20$), $P < 0.05$. *PO *vs* C, $P < 0.05$.

Substituting a large part of the starch with sucrose (approximately 20 E%) did not result in a similar decrease in EI or body weight. On the other hand, neither body weight nor fat mass increased on this or on the fat diet. Thus 14 d energy balance was apparently maintained on these two diets, while the subjects were not able to maintain EI at a level corresponding to EE on the starch diet.

The reason for the reduction in EI on the starch- and fiber-rich diets must be either an increased satiating power or reduced palatability compared with the sucrose and fat diets. In comparison with a fat-rich

diet, a carbohydrate-rich diet may induce greater satiety due to its larger carbohydrate content.[13,27,28] However, since the amount of energy from carbohydrate was similar on the sucrose and starch diets the reason for the different EI between these two diets must be looked for in the type of carbohydrate and/or the different food items used on the diets. It is likely that the higher dietary fiber intake on the starch diet (approximately 30 g/d) induced a greater satiation compared with the sucrose diet (approximately 20 g/d).[29,30] This was found to be the case when comparing satiety and fullness per kJ consumed on the two diets (Table 5). Although the energy density of the starch diet (6.5 kJ/d) was quite similar to the energy density on the sucrose diet (6.8 kJ/d), the weight consumed was lower on the starch diet compared with the sucrose diet. The volume of the starch diet (not indicated by the energy density values) may, however, have been larger, giving rise to greater stomach filling and satiety and therefore lower EI compared with the other two diets.

Another major reason for the higher energy intake on the sucrose diet may be the quite large amounts of sucrose-containing drinks (fruit syrup and soft drinks) on this diet. Since fluids in general have been shown to be less efficient in increasing satiety and suppressing food intake compared with solid foods,[31,32] this may also explain the difference in EI between the two carbohydrate-rich diets. Still, as mentioned above, overeating did not take place on the sucrose diet – a finding similar to a previous study in humans.[33] In contrast to these studies in humans, overeating has been observed in rats on a sucrose-rich diet.[34]

The subjects liked the sucrose diet better than the starch diet, in spite of the sucrose diet being much higher in sucrose content (23 E%) than the subjects' habitual diet (8.4–10.4 E%) and the Danish population average (10 E%).[6] Increased palatability and thereby stimulation of appetite can therefore also explain why EI was higher on the sucrose diet compared with the starch diet.[35] Still, it cannot explain why EI was also high on the fat diet. From short-term studies in humans we might have expected EI to be lower on the sucrose diet compared with the fat diet.[36] That this was not the case may be due to the fact that the subjects in the present study did not overeat on the fat diet. Thus, it has previously been shown that EI and body weight increases on a fat-rich diet (45–60 E% fat) when consumed *ad libitum* for 7–14 d.[37,38] A difference in energy density of the diets used in the different studies cannot explain these discrepancies. In the study by Lissner *et al*[37] energy density in the high-fat diet (45–50 E%) was 8.0 kJ/g *vs* 6.7 kJ/g in the medium-fat (30–35 E%) diet and in the study by Stubbs *et al*,[38] energy density amounted to 7.0 kJ/g on a 60 E% fat, 5.6 kJ/g on a 40 E% fat and 4.8 kJ/g on a 20 E% fat diet. These differences in energy density are therefore very similar to the differences in energy densities in the present study. It is possible that the palatability of the fat diet used here was poorer compared with the diets used in other studies. A more plausible explanation is, however, that our subjects were more diet-concerned and/or better at detecting fat in the diet. This would result in a reduced tendency to overconsume on the fat diet.

Compared with C, PO consumed approximately 1.6 MJ/d more on the sucrose diet and approximately 0.9 MJ/d more on the fat diet. A plausible explanation for this is that PO subjects are more prone to overeat on energy-dense diets than C subjects. This may contribute to the PO's greater susceptibility to obesity.

The fact that the PO subjects reported greater satiety, fullness and less hunger and prospective consumption, compared with C on each of the three *ad libitum* diets in the chamber, cannot be explained by their increased energy intake only, since this was not the case on the starch diet. In general, however, we suspect that obesity-prone and diet-concerned subjects are less likely to report their true sensations (unconsciously or consciously) or actually have different appetite sensations than normal subjects. If the latter is the case it is apparently not macronutrient-specific, as judged from the results in the present study.

Compared with their recorded habitual diet the PO subjects consumed ca. 3 MJ/d more during the fat and sucrose diet (Tables 1 and 2). Since this occurred without a concomitant increase in body weight, an underreporting of almost 30% must have taken place during the PO's recording of habitual food intake. This should be considered in future studies of habitual diet in such study groups. In contrast to this, a minor overreporting was observed in the controls (400 kJ/d = 4%) when comparing with their energy intake on the fat and sucrose diets where no weight changes occurred.

24 h EE

A significantly increased 24 h EE was observed after 14 d on the sucrose diet compared with both the fat and starch diets (Figure 3). This was particularly so in PO. In contrast, the estimate of diet-induced thermogenesis in the chambers (6–10 pm) was not different between diets or groups. This may, however, be due to the difficulties of picking up small meal-induced differences in EE in a respiration chamber because of the generally non-resting situation here compared with, for example, a ventilated hood system. Thus, when using the whole 24 h measurement period, the difference in 24 h energy expenditure expressed as a percentage of the difference in 24 h EI (compared with the starch diet), was twice as high (14.0%) on the sucrose diet as on the fat diet (6.7%) indicating less storage and larger dissipation of the extra energy consumed on the sucrose diet. Again this was much more pronounced in PO (18%) than in C (9%).

Several previous short- and long-term studies support the concept of energy expenditure being increased by a high intake of sucrose. Recently, Horton *et al*[39] demonstrated that only 75–85% of excess energy was stored when overfeeding subjects for 14 d with mainly mono- and disaccharides (+50% of requirements) compared with 90–95% storage during fat supplementation. An extra stimulation of diet-induced thermogenesis after sucrose or fructose ingestion compared with glucose or starch has previously been reported.[40–42] The reason for this is most likely to be ongoing gluconeogenesis as well as higher costs of glycogen deposition after fructose ingestion (3.5–4.5 mol ATP/mol) than after glucose ingestion (2.5 mol ATP/mol).[40] Thus, obligatory thermogenesis is increased after fructose ingestion. Diet-induced thermogenesis after carbohydrate ingestion consists of both an obligatory and a facultative thermogenesis. The latter is probably due to an insulin-mediated stimulation of the sympathetic nervous system, although it is difficult to distinguish between this effect and a secondary effect of insulin mediated via the stimulation of intracellular glucose (or other substrate) metabolism.[40] Since fructose does not stimulate insulin secretion to any major extent, facultative thermogenesis after fructose ingestion would be expected to be negligible. However, thermogenesis after oral or intravenous fructose, has actually been found to be suppressed by 40% by β-adrenergic blockade (propranolol),[40] indicating that fructose also activates β-adrenoceptor-sensitive substrate cycles.[43] In the present study both postprandial NA and A concentrations were significantly increased after the sucrose diet compared with the other two diets, even when taking the different energy intake on the day into account. Sympathetic nervous activity and most likely also EE, was therefore especially stimulated on this diet. Increased postprandial NA concentration and diet-induced thermogenesis have previously been found after intake of a sucrose- and fructose-rich meal compared with a starch-rich meal.[41] We therefore believe that the increased 24 h EE on the sucrose diet was due not only to an increased energy intake but also to an increased intake of carbohydrate, especially sucrose and fructose.

Not only 24 h EI and EE, but also 14 d *ad libitum* EI was higher (+16%) in PO than in C on the sucrose diet. Despite this, the PO group did not gain weight on the sucrose diet. This must therefore relate to increased free-living EE (diet-induced thermogenesis and/or physical activity) in the PO group or to this group being especially sensitive to changes in dietary carbohydrate. The latter would be in line with previous studies of PO subjects.[18,44]

24 h macronutrient balance

On day 14 in the respiration chambers, carbohydrate oxidation was higher and fat oxidation lower on the sucrose diet leading to a positive fat balance compared with the starch diet. A similarly positive fat balance was observed on the fat diet, although energy intake (on day 14) was about 1 MJ/d lower than on the sucrose diet. On neither diet, however, did the positive fat balance observed on day 14 lead to fat storage over the 14 d period. Energy balance was on average 1.6 MJ on the fat diet and 2.4 MJ/d on the sucrose diet ($P < 0.05$), thus more positive on the sucrose diet (by 0.8 MJ/d). It is strange then, that the subjects did not gain weight on this diet as 0.8 MJ/d * 14 d = 11,200 kJ would correspond to 300 g fat mass stored. No increase in fat mass was observed on the sucrose diet ($= -0.05$ kg, ns). The most likely reason for the positive fat and energy balance in the chamber is therefore the restriction of physical activity here compared with the free-living situation. In support of this is a recent study of young men showing that EE in real life (measured by doubly-labelled water) was 2.7 MJ/d higher compared with in a respiration chamber.[45] This value corresponds to the positive 24 h energy balance found on the sucrose and fat diets after 14 d, taking the gender difference into account. We are aware, however, that by doing the above calculations we may be working beyond the limits of detection of the bioimpedance methods.

Surprisingly, no differences in 24 h macronutrient oxidation rates or balances were found between the PO and C subjects after 14 d on either of the three diets. From previous studies such a difference would, however, have been expected, especially on the fat-rich diet.[44,46,47] In these studies, however, the measurements were performed after 0–3 d controlled diet, while the present measurements were performed after 14 d on the diets. Thus it may be that the substrate oxidation capacity of the subjects in the present study had adapted to the different macronutrient composition after 14 d exposure to the diets, resulting in no detectable group differences.

Conclusion

In conclusion, a reduction in energy intake, body weight and fat mass was observed when normal-weight subjects with or without a history of obesity consumed a starch- and fiber-rich diet *ad libitum* for 14 d. In contrast to this, no significant changes in either of these parameters were observed on the sucrose-rich diet, however, 24 h EE and sympatho-adrenal activity was increased, with no untoward effects on blood pressure, compared with the starch-rich and fat-rich diet. This can probably be explained by both the increased amount of energy and the increased amount of carbohydrate, especially fructose (alone and from sucrose) consumed on the sucrose diet. Studies of longer duration, including both

normal, obesity-prone and obese subjects are, however, still needed to evaluate the long-term health consequences of a high sucrose content in the diet.

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References

1 Kuczmarski RJ, Flegal K, Campbell SM, Johnson CL. Increasing prevalence of overweight among US adults. *JAMA* 1994; **272**: 205–211.
2 Prentice AM, Jebb SA. Obesity in Britain: Gluttony or sloth? *Br Med J* 1995; **311**: 437–439.
3 Pietinen P, Vartiainen E, Männistös. Trends in body mass index and obesity among adults in Finland from 1972 to 1992. *Int J Obes* 1996; **20**: 114–120.
4 Nordic Committee on Foods, Nordic Council of Ministers. *Nordic Nutrition Recommendations* 2nd edn. Report 1989:2. Nordic Council of Ministers: Copenhagen 1989.
5 Drewnowski A. Human preferences for sugar and fat. In: Fernstrom JD, Miller GD, (eds). Appetite and body weight regulation: sugar, fat, and macronutrient substitutions. CRC Press, Boca Raton: 1994: 137–147.
6 Andersen NL, Fagt S, Groth MV, Hartkopp HB, Møller A, Ovesen L, Warming DL. *Danskernes Kostvaner 1995*. Hovedresultater. Levnedsmiddelstyrelsen, Sundhedsministeriet, publikation nr. 235, Quickly Tryk A/S, 1995.
7 Lewis CJ, Park YK, Dexter PB, Yetley EA. Nutrient intakes and body weights of persons consuming high and moderate levels of added sugars. *J Am Diet Assoc* 1992; **92**: 708–713.
8 Gibney M, Sigman-Grant M, Stanton JL, Keast DR. Consumption of sugars. *Am J Clin Nutr* 1995; **62(suppl)**: 178S–194S.
9 Romieu I, Walter CW, Stampfer MJ, Corditz GA, Sampson L, Rosner B *et al*. Energy intake and other determinants of relative weight. *Am J Clin Nutr* 1988; **67**: 406–412.
10 Bolton-Smith C, Woodward M. Dietary composition and fat to sugar ratios in relation to obesity. *Int J Obesity* 1994; **18**: 820–828.
11 Emmett PM, Heaton KW. Is extrinsic sugar a vehicle for dietary fat? *Lancet* 1995; **345**: 1537–1540.
12 Hill JO, Prentice AM. Sugar and body weight regulation. *Am J Clin Nutr* 1995; **62(suppl)**: 264S–274S.
13 Astrup A, Raben A. Carbohydrate and obesity. *Int J Obes* 1995; **19(suppl 5)**: S27–S37.
14 FAO/WHO/UNU. Energy and protein requirements. Report of a joint FAO/WHO/UNU Expert Consultation, 1985. *Tech Rept Ser* **No 74**. World Health Organization: Geneva.
15 Møller A. *Danish Food Tables* (Levnedsmiddeltabeller), 1989. Storkøkken-centeret, Levnedsmiddelstyrelsen.
16 Heitmann BL. Prediction of body water and fat in adult Danes from measurement of electrical impedance. A validation study. *Int J Obes* 1990; **14**: 789–802.
17 Astrup A, Thorbek G, Lind J, Isaksson B. Prediction of 24-h energy expenditure and its components from physical characteristics and body composition in normal-weight humans. *Am J Clin Nutr* 1990; **52**: 777–783.
18 Astrup A, Buemann B, Christensen NJ, Madsen J. 24-hour energy expenditure and sympathetic activity in post-obese women consuming a high-carbohydrate diet. *Am J Physiol* 1992; **262**: E282–E288.
19 Brouwer E. Report on subcommittee of constants and factors. In: Blaxter KL (ed). *Energy metabolism*. Proceedings of the 3rd Symposium on Energy Metabolism, EAAP Publ. 77. Academic Press: London, 1965, pp 441–443.
20 Hill AJ, Magson LD, Blundell JE. Hunger and palatability: tracking ratings of subjective experience before, during and after the consumption of preferred and less preferred food. *Appetite* 1984; **5**: 361–371.
21 Cryer PE, Haymond MW, Santiago JV, Shah SD. Noradrenaline and adrenaline release and adrenergic mediation of smoking-associated hemodynamic and metabolic events. *NEJM* 1976; **295**: 573–577.
22 Forster CD, Taylor JY, Macdonald IA. The assay of the catecholamine content of small volumes of human plasma. In: Rollema H, Westerink BHC, Drijfhout WJ (eds). *Monitoring molecules in neuroscience. Proceedings of the 5th International Conference on in vivo methods*. University Centre for Pharmacy, Groningen 1991, pp 173–174.
23 Ravussin E, Bogardus C. Relationship of genetics, age, and physical fitness to daily energy expenditure and fuel utilization. *Am J Clin Nutr* 1989; **54**: 968–975.
24 Sheppard L, Kristal AR, Kushi LH. Weight loss in women participating in a randomized trial of low-fat diets. *Am J Clin Nutr* 1991; **54**: 821–828.
25 Raben A, Jensen ND, Marckmann P, Sandström B, Astrup A. Spontaneous weight loss during 11 weeks' ad libitum intake of a low-fat/high-fiber diet in young, normal-weight subjects. *Int J Obes* 1995; **19**: 916–923.
26 Siggard R, Raben A, Astrup A. Weight loss during 12 weeks' ad libitum carbohydrate-rich diet in overweight and normal-weight subjects at a Danish work site. *Obes Res* 1996; **4**: 347–356.
27 Mayer J. Glucostatic mechanism of regulation of food intake. *NEJM* 1953; **249**: 13–16.
28 Raben A, Holst JJ, Christensen NJ, Astrup A. Determinants of postprandial appetite sensations: Macronutrient intake and glucose metabolism. *Int J Obesity* 1996; **20**: 161–169.
29 Burley VJ, Blundell JE. Action of dietary fiber on the satiety cascade. In: Kritchevsky D, Bonfield C, Anderson JW (eds). *Dietary fiber. Chemistry, physiology, and health effects*. New York, Plenum Press: 1990, pp 227–246.
30 Raben A, Christensen NJ, Madsen J, Holst JJ, Astrup A. Decreased postprandial thermogenesis and fat oxidation, but increased fullness after a high-fiber meal compared with a low-fiber meal. *Am J Clin Nutr* 1994; **59**: 1386–1394.
31 Mattes R. Dietary compensation by humans for supplemental energy provided as ethanol or carbohydrate in fluids. *Physiol & Behav* 1996; **59**: 179–187.
32 de Castro JM. The effects of the spontaneous ingestion of particular foods or beverages on the meal pattern and overall nutrient intake of humans. *Physiol & Behav* 1996; **53**: 1133–1144.
33 Schiffman SS, Warwich ZS, Mackey M. Sweetness and appetite in normal, overweight and elderly persons. In: Fernstrom JD, Miller GD (eds). *Appetite and body weight regulation: sugar, fat, and macronutrient substitutions*. CRC Press: Boca Raton, 1994, pp 99–112.
34 Hirsch E, Walsh M. Effect of limited access to sucrose on overeating and patterns of feeding., *Physiol Behav* **25**: 129–134.
35 Anderson GH. Sugars, sweetness, and food intake. *Am J Clin Nutr* 1995; **62(suppl)**: 195S–202S.
36 Green SM, Burley VJ, Blundell JE. Effect of fat- and sucrose-containing foods on the size of eating episodes and energy intake in lean males: Potential for causing overconsumption. *Eur J Clin Nutr* 1994; **48**: 547–555.

37 Lissner L, Levitsky DA, Strupp BJ, Kalkwarf HJ, Roe DA. Dietary fat and the regulation of energy intake in human subjects. *Am J Clin Nutr* 1987; **46**: 886–892.
38 Stubbs RJ, Harbron CG, Murgatroyd PR, Prentice AM. The effect of covert manipulation of the dietary fat and energy density on food intake and substrate flux in *ad libitum* feeding men. *Am J Clin Nutr* 1995; **62**: 316–329.
39 Horton TJ, Drougas H, Brachey A, Reed GW, Peters JC, Hill JO. Fat and carbohydrate overfeeding in humans: different effects on energy storage. *Am J Clin Nutr* 1995; **62**: 19–29.
40 Tappy L, Randin J-P, Felber J-P, Chiolero R, Simonson DC, Jéquier E, De Fronzo RA. Comparison of thermogenic effect of fructose and glucose in normal humans. *Am J Physiol* 1986; **250**: E718–E724.
41 Raben A, Kiens B, Richter EA. Differences in glycemia, hormonal response and energy expenditure after a meal rich in mono- and disaccharides compared to a meal rich in polysaccharides in physically fit and sedentary subjects. *Clin Physiol* 1994; **14**: 267–280.
42 Blaak EE, Saris WHM. Postprandial thermogenesis and substrate utilization after ingestion of different dietary carbohydrates. *Int J Obes* 1995; **19(suppl 2)**: O32.
43 Tappy L, Jéquier E. Fructose and dietary thermogenesis., *Am J Clin Nutr* 1993; **58**: 766S–770S.
44 Lean MEJ, James WPT. Metabolic effects of isoenergetic nutrient exchange over 24 hours in relation to obesity in women. *Int J Obes* 1988; **12**: 15–27.
45 Stubbs RJ, Ritz P, Coward WA, Prentice AM. Covert manipulation of the ratio of dietary fat to carbohydrate and energy density: effect on food intake and energy balance in free-living men eating ad libitum. *Am J Clin Nutr* 1995; **62**:330–337.
46 Astrup A, Buemann B, Christensen NJ, Toubro S, Raben A. Failure to increase lipid oxidation in response to increasing dietary fat content in formerly obese women. *Am J Physiol* 1994; **266**: E592–E599.
47 Raben A, Andersen HB, Christensen NJ, Madsen J, Holst JJ, Astrup A. Evidence for an abnormal postprandial response in women predisposed to obesity. *Am J Physiol* 1994; **267**: E549–E559.

Appendix 1 Diet order

	1st period	*2nd period*	*3rd period*
Post-obese (no)			
1, 3	A	B	C
4	C	B	A
5–7	B	A	C
2, 8, 9	C	A	B
Controls (no)			
11–13	A	B	C
14	C	B	A
15–17	B	A	C
18–21	C	A	B

A = Fat diet. B = Starch diet. C = Sucrose diet.

Appendix 2 Day 1 menu for each of the three *ad libitum* diets.

Meal	*Fat*	*Starch*	*Sucrose*
Breakfast	Sandwich-bread with margarine, 60% fat cheese and marmalade. Medium-fat milk.	Whole-meal sandwich with low-fat margarine and 30% fat cheese. Juice.	Low-fat sour-milk product with sugar-sweetened muesli. Fruit syrup.
Lunch	Salad with pasta, corn, peas, 60% fat cheese, dressing. Sandwich-bread with paté and cucumber.	Salad with pasta, corn, peas, dressing. Wholemeal sandwich with egg mayonnaise filling	Salad with rice, corn, green beans, peas, 30% fat cheese, dressing. Sandwich bread with tuna fish spread. Soft drink. Liquorice.
Dinner	Risotto with beef, sausages, vegetables and double cream. Pear.	Risotto with tender loin, vegetables and milk. Pear.	Pasta dish with vegetables and 45% fat cheese. Fruit syrup. Chocolate ('Raider') Pear.
Snack	Almond cake*.	Almond cake*.	Almond cake*.

*Different for each diet. %Fat = percentage of dry matter. The rest of the menus used can be given on request.