Effects of isocaloric exchange of dietary sucrose and starch on fasting serum lipids, postprandial insulin secretion and alimentary lipaemia in human subjects

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- 1. Fasting serum cholesterol and triglyceride, and post prandial insulin secretion and lipaemia were measured in human subjects in a metabolic ward, who were given an ordinary diet (diet 1) in which the sucrose was isocalorically replaced by starch (diet 2) or vice versa. The subjects were nine healthy normolipaemic adult males. In eight of these subjects the effect of sucrose caloric reduction (diet 3) on fasting serum lipids was also studied.
- 2. When starch replaced sucrose, there were no significant differences in fasting serum lipid concentrations or immunoreactive insulin or in the insulin response and alimentary lipaemia after a standard mixed breakfast.
- 3. Serum triglyceride concentration fell and cholesterol concentration rose during the period of sucrose (and calorie) restriction.
- 4. After lunch and supper on the first two diets (when different carbohydrates were given) the lipaemic response was larger and the insulin response smaller after meals containing sucrose.
- 5. Thus, there was no difference between concentrations of fasting serum lipids when starch replaced sucrose at 23 % total calories, but the concentrations of serum triglycerides were higher after individual mixed meals containing sucrose.
- 6. There were no significant differences in the fatty acid patterns of serum lipids on the different diets.

In a field study we found that fasting serum triglyceride concentrations fell in healthy male volunteers on a sucrose-restricted diet. It seemed, however, that the mechanism for this reduction may have been negative calorie balance in the low-sugar group because they lost weight at the same time (Mann, Truswell, Hendricks & Manning, 1970). The metabolic ward experiments reported here were designed to examine this possibility.

In addition, we hoped to clarify the rather conflicting reports on the effects of different dietary carbohydrates on fasting serum lipids. Some workers have reported elevated serum lipid concentrations in normolipaemic people when sucrose was exchanged for starch (Macdonald & Braithwate, 1964; Macdonald, 1965a; Antar & Ohlson, 1965; Hodges, Krehl, Stone & Lopez, 1967; Szanto & Yudkin, 1969; Akinyanju, Qureshi, Salter & Yudkin, 1968; Nestel, Carroll & Havenstein, 1970), while others have reported little or no difference (Keys, Anderson & Grande, 1960; Lees, 1965; Grande, Anderson & Keys, 1965; Macdonald, 1965b; Antonis, Iles & Pilkington, 1968; Dunnigan, Fyfe, McKiddie & Crosbie, 1970). However, in only a few studies has sucrose been given in proportions normally eaten (Keys et al. 1960; Grande et al. 1965; Dunnigan et al. 1970).

Szanto & Yudkin (1969) found elevated fasting insulin levels and an abnormal

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insulin response to glucose in some individuals on a diet very high in sucrose (438 g/d). Dunnigan et al. (1970) reported no abnormality of insulin response to glucose when comparing diets containing sucrose (20% of total calories) or starch. We have therefore studied the insulin response to the physiological stimulus of a normal meal with diets varying in carbohydrate composition, but containing sucrose in the ordinary range of intake (Walker, Holdsworth & Walker, 1971).

Alimentary lipaemia is cleared more rapidly when glucose rather than sucrose is included in a mixed meal (Mann, Truswell & Pimstone, 1971). It seemed that the greater insulin secretion stimulated by glucose potentiated the clearing factor, adipose tissue lipoprotein lipase. We looked to see whether the same phenomenon occurs following individual meals during the course of the diets in the present experiments. If there were large secretions of insulin on the starch diet, the rate of clearing of endogenous triglyceride could be increased and fasting levels affected.

Finally, we wished to see whether the changes in the fatty acid patterns of serum lipids which some workers have reported on high-carbohydrate diets when dietary sucrose and starch were exchanged (Macdonald & Braithwaite, 1964; Kuo & Bassett, 1965) would be found when carbohydrates were exchanged at ordinary intake levels.

METHODS

Subjects

The subjects were all normolipaemic men aged 30–40 years who had been admitted to hospital earlier for non-metabolic conditions such as cerebral vascular accident and nerve palsy. They had normal reactions to tests of liver function and their erythrocyte sedimentation rates were normal by the time the diets were commenced. They had been on the usual ward diets before admission to the metabolism ward and then, during the first 2 weeks of the adjustment period, were started on either diet 1 or 2 so that each individual's calorie requirement could be determined. Each subject remained in the metabolism ward for 8 weeks. All were ambulant and allowed ordinary ward activities during this period. They received physiotherapy and occupational therapy during their stay in the metabolism ward. Informed consent was obtained from each subject and all co-operated well. Subject 9, however, did not wish to continue in the study after the period on diet 2.

Diets

The subjects were given three diets and each dietary period was of 14 d duration. Diet I (see Table I) was intended to represent a fairly average Western diet in proportions of proximate food constituents and amounts of dietary sucrose (Morris, Marr, Heady, Mills & Pilkington, 1963; Papp, Padilla & Johnson, 1965). In diet 2 the sucrose of diet I was isocalorically replaced by two complex carbohydrates, potato and rice, so that the amount of total carbohydrate remained unchanged. Thus the breakfast meal on the two diets was identical. The lunch and supper meals were isocaloric, but at lunch and supper on diet 2, potato and rice respectively, rather than sucrose, were the principal sources of carbohydrate. In diet 3 the total calorie

content of the diet was reduced by eliminating the sucrose from diet I and not replacing it with other food constituents.

Minor changes from the values shown in Table 1 were made to suit the individual daily calorie requirement of each volunteer by altering the bread intake. Individual total calorie intakes ranged from 2150 to 2440 kcal (8996–10 209 kJ) per d.

Table 1. Composition of diet 1, intended to represent an average Western daily diet in proportions of proximate food constituents and amount of dietary sucrose

				Total carbo-		
		Protein	Fat	hydrate	Sucrose	Calories
		(g)	(g)	(g)	(g)	(kcal)
Breakfast						_
Jungle Oats	300 g*	6.0	3.0	29.1		165
Bread	60 g	6∙0	1.2	28.8	_	146
Butter	15 g		12.0			114
Milk for day	500 ml	17.5	18.2	24.2		325
Clear tea						
Mid-morning						
Clear tea					A THE MAN	
Bread	30 g	3.0	0.6	14.4		73
Marmite (yeast extract)		_				
Lunch						
Chicken	100 g†	31.6	3.4			166
Carrots	100 g‡	0.9	0.5	7.1		31
Butter	25 g		20.0			190
Bread	60 g	6.0	1-2	28.8		146
Sugar	70 g			70.0	70.0	266
Clear tea						
Mid-afternoon						
Clear tea				_		
Bread	30 g	3.0	0.6	14'4	-	73
Marmite						
Supper						
Bread	60 g	6∙0	1.3	28.8		146
Tomato	100 g§	1.6		7.0	_	33
Egg, hard-boiled	(one)	6.5	5.8	0.4		81
Butter	то д		8∙o		****	76
Sugar	70 g	_		70.0	70.0	266
Clear tea		-		-		-
Total daily calories ((kcal)		-			2297 (9611 kJ)
Quantity of each cor	nstituent (g)	88·1	75.7	323.3	140.0	
Percentage of total of	daily calories	15	30	55	23	-
* Cool	ked. † W	hite meat, co	oked.	‡ Tinned.	§ Raw.	

Food tables were used to determine the composition of most of the foods used. However, as most of the values in the available tables are not for South African foods and in view of the variable moisture content of food, particular care was taken with the carbohydrate foods involved in the exchange – rice, potato and sugar. Constant carbohydrate sources were used throughout the series of experiments (Tastic rice, Maggi potato flakes and Hulett's white sugar). The moisture content of both rice and potato was checked and remained constant at approximately 9% of undried weight.

The sugar was found to have no appreciable amount of water. The caloric values of these three carbohydrates were determined in duplicate by bomb calorimetry and all were found to be 3.8 kcal/g dry weight. The moisture content of the bread chosen (approximately 40% of undried weight) varied little for 3 d after baking. The moisture content of other breads appeared to vary considerably, which could have caused variation in daily calorie intake. Standard foods were used to make up the remainder of the diet and were the same in all three diets. The same menu was given each day during the dietary period.

A check was made on the daily calorie intake from each diet by homogenizing all the food eaten on one day of each dietary period and then carrying out bomb calorimetry in duplicate on samples of the homogenate. The total calorie contents of diets 1, 2 and 3 measured by this method were 2513, 2498 and 1802 kcal (10514, 10452 and 7540 kJ) per d respectively. For all three diets these values were somewhat higher than the values calculated from food tables – 2297 kcal (9611 kJ) for diets 1 and 2 and 1765 (7384 kJ) for diet 3. However, the agreement between the measured calorie content of diets 1 and 2 was very satisfactory.

I. Macdonald (1970, personal communication) pointed out that for strict accuracy the protein which was present in the rice and potato of diet 2 should have been replaced during the period on diet 1. By Kjeldahl analysis the potato flakes were found to contain 6.88 mg nitrogen/g and the rice 9.79 mg nitrogen/g. This meant that the potato provided 3.1 g protein ($N \times 6.25$) and the rice 4.4 g protein per d. As the protein intake was not marginal, it seemed unlikely that this small amount would be significant; nevertheless the last three subjects were given protein supplements during the period on diet 1 in the form of Casilan (Glaxo), 3.4 g at lunch and 4.6 g at supper.

Diets 1 and 2 were given to the subjects in a random order but diet 3 was always given last. The experiments were all carried out between January and September 1970 in the Groote Schuur Hospital metabolism ward.

Procedures

Subjects were weighed daily under standard conditions on a platform scale throughout the experiment.

Fasting blood samples were taken on days 0, 3, 6, 9, 12, 13 and 14 of each dietary period, day 0 corresponding to day 14 of the previous diet. Serum triglyceride was measured on each of the samples, and serum cholesterol on the last three samples of each dietary period. Fatty acid patterns of serum total lipids and of serum triglyceride were determined on one of the samples from day 12, 13 or 14 of each dietary period, depending upon the day on which most serum was available.

In subjects 4, 5, 7, 8 and 9, patterns of insulin secretion and triglyceride clearance after meals were also studied. On random days during the last 4 d of diets 1 and 2, blood was taken before breakfast, lunch and supper and then again at 0.5 h intervals for 2.5 h after the completion of the meal. Serum triglyceride, serum insulin and blood sugar were measured on each of these samples. These breakfast, lunch and supper experiments were carried out on different days.

Analytical methods

Standard techniques were used for the measurement of serum triglyceride (Van Handel & Zilversmit, 1957; Young & Eastman, 1963), cholesterol (Abell, Levy, Brodie & Kendall, 1952; Anderson & Keys, 1956) and blood sugar (Hoffman, 1937). Serum insulin was assayed by the method described by Morgan & Lazarow (1963). The glycaemic stimulus and lipaemic response to a meal were calculated and expressed as mg-h and the insulin response was microunit-h as previously described from this laboratory (Mann *et al.* 1971).

The fatty acid patterns of serum total lipids and serum triglycerides were determinded by gas-liquid chromatography (James & Martin, 1956) of the methyl esters (Stoffel, Chu & Ahrens, 1959). Triglycerides were first separated by thin-layer chromatography (Wagener, 1965).

Both the Wilcoxon test and Student's t test were used to determine the significance of differences between body-weight, serum cholesterol and serum triglyceride concentrations on the different diets. (In fact the same P values were obtained by the use of these two different tests.) The significance of differences between lipaemic response, glycaemic stimulus and insulin response to meals on diets \mathbf{I} and $\mathbf{2}$ were determined by Student's t test.

RESULTS

Body-weight

Mean body-weights were virtually the same at the end of dietary periods 1 and 2 (Table 2). The mean weight for the last 3 d on diet 2 was, however, significantly lower than on diets 1 and 2 (P < 0.05 in both instances).

Table 2. Mean body-weight, serum cholesterol and triglyceride concentrations of the eight subjects (for the last 3 d on each diet) on diets 1, 2 and 3

0	,	10	5	,	
			Weight (kg)	Cholesterol (mg/100 ml)	Triglyceride (mg/100 ml)
				, ,	()
			Diet 1: b	asal + sugar	
Mea	n		59.2	188.8	97.8
Rang	ge	4	7.2-84.3	140-259	67-128
SEM			3.65	14.68	9.95
			Diet 2: ba	asal + starch	
Mea	n		59.1	187.4	98-4
Rang	ge	4	6.6-84.5	142-228	65-127
SEM			3.65	11.26	9.95
			Diet	3: basal	
Mea	n		57.8	204.3	84.1
Rang	ge	4	4.7-83.5	162-253	67-107
SEM		•	4.08	10.26	4.45

Serum triglyceride

There were no significant differences between fasting serum triglyceride concentrations on diets 1 and 2 whether considering daily triglyceride values, mean of all values while on the diets or mean of the last three values while on the two diets

(Table 2). However, the mean for the last 3 d on diet 3 was significantly lower than the mean for the last 3 d on diets 1 (P < 0.02) and 2 (P < 0.05). The fall in triglyceride concentrations which occurred on diet 3 was confined to those subjects whose fasting serum triglyceride concentrations on diets 1 and 2 were greater than 90 mg/100 ml (Table 3).

Serum cholesterol

There were no significant differences between concentrations on diets 1 and 2 (Table 2) but the mean value was significantly higher at the end of the period on diet 3 (P < 0.05).

From the mean values given in Table 4 it appears that replacement of the small amount of protein during the period on diet 1 made no difference to the mean bodyweight, or serum cholesterol and trigylceride concentrations on diets 1 and 2.

Table 3. Mean serum triglyceride concentration (mg/100 ml) (for the last 3 d on each diet) on diets 1, 2 and 3 for each of the subjects

Subject no.	Diet 1: basal+sugar	Diet 2: basal+starch	Diet 3: basal
I	113	127	96
2	128	125	107
3	67	65	67
4	102	100	83
	121	116	88
5 6	91	92	76
7	83	83	82
8	77	79	76
9	163	156	-

Table 4. Mean values for body-weight and serum lipid concentrations on diets 1 and 2, for the subjects for whom protein of potato and rice was not replaced while on diet 1 (subjects 1-6) and for those for whom it was replaced (subjects 7-9)

	Die	t I: basal+suga	ır	Diet 2: basal + starch			
Subjects	Weight (kg)	Cholesterol (mg/100 ml)	Triglyceride (mg/100 ml)	Weight (kg)	Cholesterol (mg/100 ml)	Triglyceride (mg/100 ml)	
1-6 7-9	61·2 54·0	203·2 180·0	103·7 107·7	61·1 54·1	200·5 179·7	104·1 106·0	

Fatty acid pattern of serum lipids

It can be clearly seen from Table 5 that there were no significant differences between fatty acid patterns of fasting serum triglycerides in dietary periods 1 and 2. The slightly lower myristic and higher linoleic acid concentrations on diet 3 were not statistically significant. Likewise there were no differences in the fatty acid patterns of fasting serum total lipids between the three diets.

Glycaemic stimulus, insulin response and lipaemic response after breakfast, lunch and supper on diets 1 and 2

The glycaemic stimulus, insulin and lipaemic responses to the breakfast, lunch and supper on diets 1 and 2 are shown in Table 6.

After the breakfast on the two different diets (when the stimulus was the same) the total serum insulin responses were almost identical. Similarly, the glycaemic stimulus of the meal and the lipaemic response were similar on the two different diets.

Table 5. Fatty acid composition (% by weight of total fatty acids) of serum triglycerides of the subjects on diets 1, 2 and 3

Fatty	Diet 1: basal+sugar		Diet 2: basal+starch		Diet 3: basal	
acid	Mean	SD	Mean	SD	Mean	SD
C14:0	3.4	1.10	3.3	1.13	2.1	0.20
C16:0	31.0	1.60	31.7	1.81	32.1	0.92
C16:1	6.3	1.44	6·4	1.24	6∙0	1.22
C18:0	5.0	o·98	5.4	1.18	5:3	o∙66
C 18: 1	43.6	2-43	43.4	3.51	44.3	2.03
C 18:2	8-6	1.45	8.8	1.21	6.1	1.03
Others	2.0	1.76	1.1	1.02	1.0	0.72

Table 6. Glycaemic stimulus, insulin response and lipaemic response of five subjects to each of the meals on diets 1 and 2

	Glycaemic stimulus (mg-h)*		Insulin response (microunit-h)*		Lipaemic response (mg-h)*	
	Diet 1	Diet 2	Diet 1	Diet 2	Diet 1	Diet 2
Breakfast						
Mean	21.30	23.20	62.30	61.70	18.10	15.20
SEM	2.24	3.14	6.49	5.12	4.66	3.72
Lunch						
Mean	26·80	43.60	49.00	68.70	33·8o	23.90
SEM	6.66	5.84	8.44	8.27	7.68	4.87
Supper						
Mean	29.10	31.25	35.90	47.90	7.10	3.60
SEM	4.68	6.98	4.16	14.00	5.47	2.23

^{*} See Mann et al. (1971) for methods of calculation.

After lunch, however, on the two different diets when the stimuli differed, the glycaemic stimulus was significantly greater after the starch meal (containing potato) than after the sugar meal (P < 0.05) and similarly the total insulin response was significantly greater (P < 0.001). Total lipaemic response to the meal was significantly lower after the starch meal than after the meal on the sugar diet (P < 0.05). A smaller total lipaemic response reflected the more rapid clearing of ingested triglyceride. This was particularly evident from the individual triglyceride values for each sampling period.* All five subjects in whom these tests were carried out showed similar patterns.

After the supper meal on the two different diets, glycaemic stimulus and insulin response were once again higher after the starch meal than after the sugar meal. This time the differences were not statistically significant; only three of the five subjects showed this pattern. The lipaemic responses to both meals were very small after the supper meal on both diets. Only 10 g butter were given with these meals.

^{*} These results are available from the Editorial Office.

The insulin response seemed to be related to the glycaemic stimulus in that there was no significant difference between ratios of insulin response to glycaemic stimulus on either diet when different glycaemic stimuli were obtained with different meals. The mean ratios were $2.48 \ (\pm 0.38)$ on diet 1 and $2.21 \ (\pm 0.29)$ on diet 2. Fasting serum insulin concentrations did not differ significantly on diets 1 and 2 (3.6 ± 0.68) and 4.0 ± 0.71 microunits per ml respectively).

DISCUSSION

This study has shown that, when sucrose (in amounts commonly eaten) was isocalorically replaced by complex carbohydrates, there was no significant change in the fasting serum lipid levels of normolipaemic adult males. Failure to have replaced, during the period on diet 1, the small quantity of protein present in the rice and potato of diet 2 for some subjects appears to have made no difference to fasting serum cholesterol and triglyceride concentrations. However, when the sucrose was eliminated from the diet and the 532 kcal (2226 kJ) therefrom not replaced, certain changes were noted. Mean serum triglyceride concentrations had started to fall by the 9th day after the commencement of this reduced-calorie diet. The mean triglyceride concentrations for the last 3 d on diet 3 were significantly lower than on the other two diets. This change in serum triglyceride only took place in those subjects whose fasting levels were greater than 90 mg/100 ml. On the other hand, serum cholesterol concentrations for the last 3 d were significantly increased on the reducedcalorie diet when compared with the values on diets 1 and 2, between which they did not differ significantly. These serum lipid changes coincided with the significant weight reduction which occurred during the third dietary period.

The fall in serum triglyceride concentration on diet 3, after values had remained unchanged on diets 1 and 2, supports the suggestion that changes in calorie balance can explain the observed differences in fasting serum lipids when complex carbohydrate replaces sucrose (Antonis et al. 1968; Porte, Bierman & Bagdade, 1966). Several of the subjects in the present study felt that they were being given too much to eat when switched from diet 1 to diet 2. Starch-containing foods are more bulky than sucrose and it is probably difficult for an individual voluntarily to replace all his dietary sucrose with starch.

It has been claimed that substitution of sucrose for starch causes elevation of serum lipids when the dietary fat is saturated (Stoffel et al. 1959; Macdonald, 1967; Antar, Little, Lucas, Buckley & Csima, 1970). However, in the present experiments most of the fat (provided as butter) was saturated, yet fasting serum lipids were not affected by sucrose, possibly because the sucrose was given in more physiological amounts.

The increase in serum cholesterol during the third dietary period was unexpected. Several workers have demonstrated elevated serum cholesterol concentrations during acute starvation (Bloom, Azar & Clark, 1966; Ende, 1960), with the highest values 72 and 96 h after the commencement of starvation. Others (Kaminen & Miettinen, 1963) found no significant increase in serum cholesterol over the same time-interval

when giving a 500 kcal diet to overweight subjects. On more physiological weight-reducing diets, most investigators have found that weight loss was associated with a fall in serum cholesterol concentration (Walker, Lawry, Love, Mann, Levine & Stare, 1953; Joliffe, Rinzler & Archer, 1962; Greenberg & Elkins, 1964; Galbraith, Connor & Stone, 1966). But in three of the studies the diets had a fairly low total fat content (Walker et al. 1953; Joliffe et al. 1962; Greenberg et al. 1964) and a high proportion of polyunsaturated fat. Other investigators have reported no change in serum cholesterol concentrations on weight-reducing diets (Moore, Fryer, Young & Maynard, 1953; Moore, Young & Maynard, 1954; Caldwell, Watson, Green, Florin, Braun & Bierenbaum, 1963). Two of them gave 80 g fat/d (Moore et al. 1953; Moore et al. 1954) and the third gave 40 g/d (Caldwell et al. 1963). Elevated serum cholesterol concentrations have been reported in anorexia nervosa (Blendis & Crisp, 1968).

In the present experiments total calories were reduced in diet 3, but the amount of fat given (which was nearly all saturated) remained unchanged, so that the proportion of total daily calories provided by fat actually increased from 30 to 39%. It may be that this is an important determinant, but further study is clearly required to determine the cause of the elevated cholesterol levels during this reduced calorie dietary period.

In this study there were no differences in the fatty acid patterns of serum trigly-cerides and total serum lipids on the different diets. We suggest that differences reported by other workers (Macdonald & Braithwaite, 1964; Kuo & Bassett, 1965) occur only when starch and sucrose are given and exchanged at very high proportions of total calories.

Measurement of the glycaemic stimulus, insulin response and lipaemic response to the meals on the different diets provided interesting results. Unfortunately these tests were carried out on only five of the nine subjects. They had to be carried out on selected subjects whose veins were not too difficult to puncture since a large number of venepunctures were required.

Fasting serum insulin concentrations did not differ significantly on diets 1 and 2, thus confirming the results of Dunnigan et al. (1970). Glycaemic stimulus, insulin response and lipaemic response to the breakfasts were virtually identical on the two different diets. The actual meal was the same on the two diets, thus showing that these responses to a physiological stimulus (such as breakfast) do not differ significantly when a diet containing an ordinary amount of sucrose is compared with one containing complex carbohydrates.

A different pattern was observed after lunch and supper when the effects of potato and rice were compared with those of sucrose. From our earlier study (Mann et al. 1971) and the results of other workers (Grodsky, Batts, Bennett, Vcella, McWilliams & Smith, 1963; Swan, Davidson & Albrink, 1966), it was expected that higher concentrations of serum insulin might be found after the complex carbohydrates, since these consist of polymers of glucose. After digestion, therefore, they would be expected to be stronger stimulators of insulin secretion than an isocaloric amount of sucrose, which is digested to glucose and fructose – the latter a very poor insulin stimulator. After the midday meals, when potato was compared with sucrose, this was in fact found to be

so. Significantly higher insulin concentrations were found on the starch diet. As in our previous study (Mann et al. 1971), the degree of lipaemia was significantly smaller after the starch meals when the insulin response was greater. This smaller lipaemic response after the starch meal appeared to be due to more rapid clearing of ingested fat, resulting from the higher levels of serum insulin, which potentiates clearing factor, lipoprotein lipase (Kessler, 1962, 1963; Nikkilä, 1969).

As in the previous experiment (Mann et al. 1971), the differences in insulin response appeared to be due to differences in glycaemic stimuli in that there was no significant difference between the ratios of insulin response to glycaemic stimulus on the two different diets when different stimuli were obtained.

At the evening meals with rice and sucrose, the mean values for glycaemic stimuli and insulin responses were once again higher after the starch meal than after the sucrose, but only three of the five subjects showed this pattern and the differences were not statistically significant. Lipaemia was very small after supper on both diets, because only 10 g butter were given.

It appears therefore that the differences in insulin secretion and triglyceride clearing after meals which occur when starch replaces sucrose in the diet are not sufficient to alter fasting serum lipid concentrations and fatty acid patterns when the sucrose is given at levels ordinarily consumed.

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