

## Influence of the amount of starch on the glycaemic index to rice in non-insulin-dependent diabetic subjects

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To test whether the glycaemic index is altered by the amount of carbohydrate, meals containing 25 and 50 g carbohydrate as white rice and white bread were taken by seven non-insulin-dependent diabetic subjects. The glycaemic indices to parboiled white rice corresponding to 25 and 50 g carbohydrate were similar, being 55 (SE 10) and 60 (SE 8) respectively. The incremental areas of blood glucose (above basal) to parboiled white rice were significantly lower than to white bread after both an amount corresponding to 25 g carbohydrate (85 (SE 24)  $\text{mM} \times 180 \text{ min}$  *v.* 181 (SE 55)  $\text{mM} \times 180 \text{ min}$ ;  $P < 0.01$ ) and to 50 g carbohydrate 226 (SE 29)  $\text{mM} \times 180 \text{ min}$  *v.* 423 (SE 76)  $\text{mM} \times 180 \text{ min}$ ;  $P < 0.01$ ). Similar insulin response areas to 25 g carbohydrate given as parboiled white rice and white bread were found, whereas 50 g carbohydrate as white bread caused a significantly higher insulin response area than parboiled white rice ( $P < 0.05$ ).

In conclusion, the glycaemic index of parboiled white rice is not affected by the amount of carbohydrate ingested, at least under the present study conditions.

**Non-insulin-dependent diabetes mellitus: Glycaemic index: Rice**

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Post-prandial blood glucose and insulin responses to starch-rich foods vary considerably in both normal and diabetic subjects (Coulston *et al.* 1980; Crapo *et al.* 1980, 1981; Bantle *et al.* 1983; Jenkins *et al.* 1983, 1984; Hermansen *et al.* 1986, 1987; Hollenbeck *et al.* 1986; Rasmussen *et al.* 1989). Due to the large variation of glycaemic responses between individuals, the results are often difficult to interpret and to use in practical diet planning for diabetic subjects. Jenkins *et al.* (1981) introduced the concept of glycaemic indexing (GI), classifying the glycaemic response to 50 g carbohydrate (CHO) of a particular food as a percentage of a standard meal of 50 g CHO as glucose, in order to reduce inter-person variation (Wolever *et al.* 1989, 1990). Thus, the GI has been postulated to be an easy and practical implement for diabetic subjects to control post-prandial hyperglycaemia by selecting foods of low GI. Recent reports have found improved metabolic control and decreased plasma triacylglycerol levels after 2–3 weeks of switching from high to low GI foods in insulin-dependent diabetes mellitus (IDDM) and non-insulin-dependent diabetes mellitus (NIDDM), suggesting that the concept of the 'GI' tested in acute conditions is valid on a chronic basis (Fontvieille *et al.* 1988; Jenkins *et al.* 1988). The question arises if the GI of a food depends on the amount of CHO. In other words if smaller amounts of food than 50 g CHO behave in a similar way with respect to the GI. This is a problem of daily life as mixed meals seldom contain more than 20–25 g CHO derived from a single

Table 1. *Clinical information for seven non-insulin-dependent diabetic patients*  
(Mean values with their standard errors)

Patient no.	Sex	Age (years)	Body mass index (kg/m <sup>2</sup> )	Duration of diabetes (years)	Fasting blood glucose (mmol/l) for meals:			
					A	B	C	D
1	F	63	32.6	6	7.7	8.8	6.1	4.9
2	F	62	25.4	2	7.9	7.4	6.8	6.9
3	M	58	27.3	8	6.6	7.2	9.0	8.4
4	F	62	25.0	2	9.3	7.5	6.7	7.3
5	F	63	35.9	14	7.5	8.1	8.0	8.1
6	M	64	24.5	2	7.1	7.0	5.7	5.8
7	F	64	31.6	7	6.5	6.9	5.9	6.1
Mean		62	28.9	6	7.5	7.6	6.9	6.8
SE		1	1.8	2	0.4	0.3	0.5	0.5

Meal A, 50 g white bread; meal B, 30 g rice; meal C, 100 g white bread; meal D, 60 g rice.

carbohydrate source. Furthermore, comparison of glycaemic responses is difficult due to varying amounts of starch ingested by the participants; e.g. 22 g CHO (Heinonen *et al.* 1985), 39 g CHO (Arends *et al.* 1987), 111 g CHO (Wolever *et al.* 1988). To see if the amount of CHO influences the GI, the glycaemic response of parboiled rice and white bread to 25 g and 50 g CHO was studied in seven NIDDM subjects.

#### SUBJECTS AND METHODS

##### *Subjects*

Seven NIDDM subjects (five females and two males), fully informed of the experimental nature of the investigation, were studied. The NIDDM subjects were all treated by diet, six received sulphonylurea and two in addition received metformin (subjects nos 3 and 5). The study had the approval of the local ethical committee. Clinical information relating to the participants is given in Table 1. All diabetics were free of neuropathy, nephropathy and retinopathy (except simplex retinopathy in subject no. 5).

##### *Experimental protocol*

Each subject consumed the four meals in random order within a 4-week period. Studies were performed on an outpatient basis after a 12 h overnight fast. At 08.00 hours on the day of the experiment a catheter was inserted into an antecubital vein to ensure blood samples. Blood samples were collected at -30, -15, 0, 15, 30, 45, 60, 90, 120, 180 and 240 min. The test meals were served at 08.30 hours and ingested continuously over a 10 min period. Tap water (250 ml) was taken with the meals. The participants took their usual medication at the start of the meal. The patients were asked to micturate before meal intake and the glucose loss was measured in the urine collected during the test period (0-240 min).

##### *Meals*

The composition of the test meals is given in Table 2. The parboiled white rice (raw weight 30 and 60 g) was cooked for 12 min before ingestion and, like the corresponding amount of white bread (50 and 100 g), taken *per se*. The approximate content of carbohydrate, protein and fat was calculated according to Helms (1980).

Table 2. *Meal composition: content of nutrients of the ingested rice (raw weight) and white bread*

(250 ml tap water was taken with each meal)

	Wt (g)	Fat (g)	Protein (g)	Carbohydrate (available) (g)
Parboiled white rice	30	0.1	2.2	24
	60	0.2	4.4	48
White bread	50	0.8	3.9	25
	100	1.6	7.8	50

*Analytical techniques*

Plasma and urinary glucose were measured by the glucose oxidase (EC 1.1.1.34) method. Haemoglobin A<sub>1c</sub> (HbA<sub>1c</sub>) values were determined by a commercial kit (Bio-Rad, Richmond, CA, USA; normal values 3.5–5.5%). Serum insulin levels were determined by specific radioimmunoassay.

*Statistical methods*

The post-prandial blood glucose and insulin-response areas above basal were calculated geometrically (Jenkins *et al.* 1981). Blood glucose values below basal values were excluded. The basal glucose concentration was defined as the mean of values obtained at 15 min and 0 min before meal intake. Results are expressed as means with their standard errors. Statistical analysis and multiple comparisons between response areas were made by analysis of variance and Student's *t* test for paired data. The level of statistical significance was set to 0.05.

## RESULTS

*NIDDM*

Identical HbA<sub>1c</sub> values (%) were present before the test meals: 30 g rice 6.4 (SE 1.5), 60 g rice 6.5 (SE 6.7), 50 g white bread 6.7 (SE 2.3) and 100 g white bread 6.6 (SE 2.9) respectively. No difference in mean fasting blood glucose values between the four meals were seen (Table 1). During the observation period no glucosuria was detected in any of the patients. The post-prandial blood glucose and insulin responses are shown in Fig. 1. Mean blood glucose and insulin-response areas to the four test meals over 180 min are depicted in Fig. 2. Post-prandial blood glucose response areas after 25 and 50 g CHO as parboiled white rice (85 (SE 24) mm × 180 min and 226 (SE 29) mm × 180 min) were significantly lower when compared with the corresponding meals of white bread (181 (SE 55) mm × 180 min (*P* < 0.01) and 423 (SE 76) mm × 180 min (*P* < 0.01) respectively). Similar GI of parboiled white rice after 25 g CHO (55 (SE 10)) and 50 g CHO (60 (SE 8)) were found. There was no difference in insulin-response areas after 25 g CHO as parboiled white rice (2075 (SE 730) μU/ml × 180 min) and white bread (2310 (SE 724) μU/ml × 180 min), whereas the insulin-response area to 50 g CHO as white bread (4440 (SE 714) μU/ml × 180 min) was significantly higher than that to parboiled white rice (2502 (SE 737) μU/ml × 180 min; *P* < 0.05).

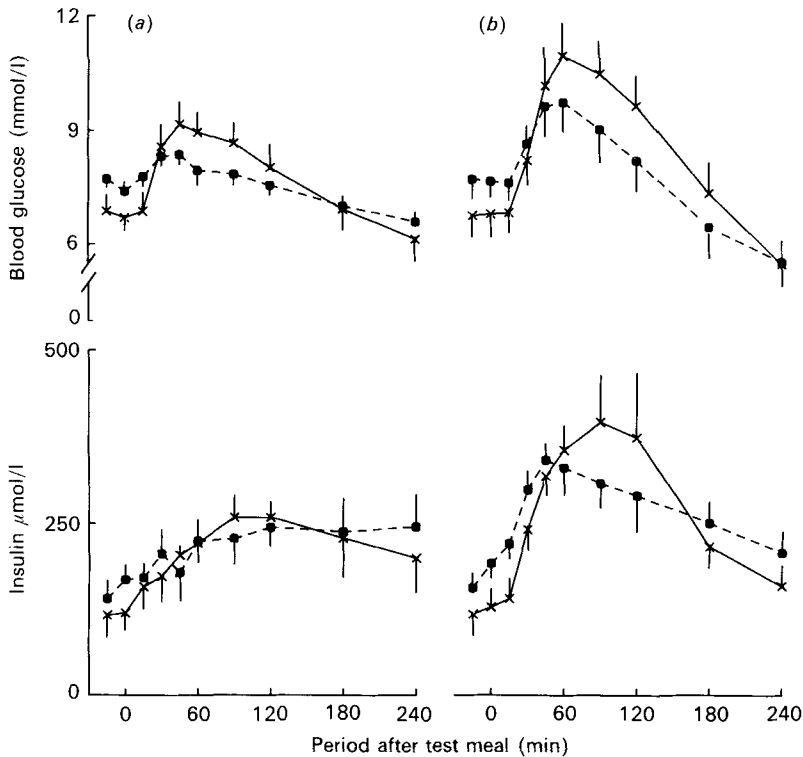


Fig. 1. Mean blood glucose and plasma insulin variations observed after (a) 30 and (b) 60 g parboiled white rice (raw weight) (■) and the corresponding amount of starch as 50 and 100 g white bread (×) in seven non-insulin-dependent diabetic patients. Points are means with their standard errors represented by vertical bars for seven subjects.

#### DISCUSSION

Recent studies have demonstrated that starch-rich foods are characterized by distinctive glycaemic responses in normal and NIDDM subjects (Coulston *et al.* 1980; Crapo *et al.* 1980, 1981; Bantle *et al.* 1983; Jenkins *et al.* 1983, 1984; Hermansen *et al.* 1986, 1987; Hollenbeck *et al.* 1986; Rasmussen *et al.* 1989). The glycaemic responses to different starch-rich foods are compared with white bread and expressed as GI (Jenkins *et al.* 1984). Normally amounts of 50 g carbohydrate are compared. The potential value of these findings in practical diet management of post-prandial hyperglycaemia in diabetic subjects is uncertain, however, as meals of 50 g CHO of a single food are seldom ingested. The present study was designed to determine if the GI of a food is modified when only half the amount of starch (25 g CHO) is ingested in NIDDM patients. We found a similar GI of parboiled white rice to 25 g CHO (55 (SE 10)) and 50 g CHO (60 (SE 8)) in seven NIDDM subjects. The low GI of parboiled white rice found in our study group corroborates previous results (Wolever *et al.* 1986). Jenkins *et al.* (1984) found that 25 g CHO as white bread constituted blood-glucose-response areas of 48 (SE 4)% of that of 50 g CHO as white bread in a mixed group of tablet- and insulin-treated NIDDM subjects. The proportional increase in post-prandial blood glucose response to white bread is not surprising since the blood glucose response to oral glucose and white bread is similar (Bantle *et al.* 1983), demonstrating no delay in the post-prandial absorption of glucose from a starch-rich food such as white bread. The proportional blood glucose increase to a low GI food such as parboiled white rice was identical to that of white bread at different levels of starch intake.

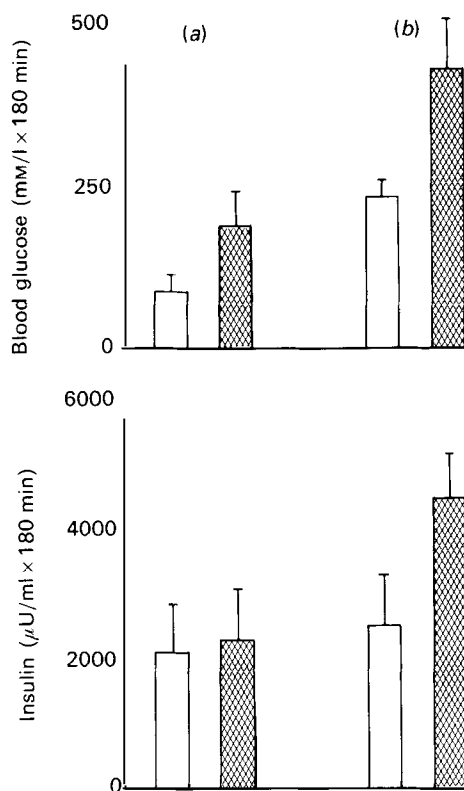


Fig. 2. Mean blood glucose and plasma insulin-response areas in seven non-insulin-dependent diabetic patients to (a) 25 g and (b) 50 g carbohydrate as parboiled white rice (□) and white bread (▨). Values are means with their standard errors represented by vertical bars for seven subjects.

This implies that the factor responsible for the low glycaemic response of rice, e.g. delayed gastric emptying, decreased access of digestive enzymes to the starch or unabsorbed starch, is not affected by the level of starch intake.

The finding that the insulin-response areas to 25 g CHO as parboiled white rice and white bread were similar at different blood glucose rises could appear puzzling, as a major determinant of insulin secretion is the rate of change in the splanchnic blood glucose level. However, we have also found similar blood-glucose-response areas to meals of spaghetti (Rasmussen *et al.* 1990) taken *per se* and as part of mixed meals, although the insulin response areas differed significantly. These results underline the low insulin sensitivity in NIDDM subjects, emphasizing that a major determinant of the post-prandial blood glucose rise in NIDDM subjects is the nature of the CHO eaten. Coulston *et al.* (1987) and Laine *et al.* (1987) have questioned these findings as these authors found similar plasma glucose and insulin responses to mixed meals derived from CHO of low, intermediate or high GI in NIDDM subjects. The discrepancy in results might be explained by the relatively high content of monosaccharides (400 g/kg) in the mixed meals taken in the latter two studies. In addition, all diabetic patients (Coulston *et al.* 1987; Laine *et al.* 1987) had high fasting blood glucose levels (> 9 mm) which may dilute differences among glycaemic responses after starch-rich foods due to the enhanced glucose clearance by muscle and kidneys. Thus, in IDDM subjects we have previously found significantly subdued glycaemic responses at a pre-prandial blood glucose of 9 mm compared with a level of 6 mm

(Rasmussen & Hermansen, 1991). In our groups the fasting blood glucose levels were close to normal, and we found no difference in the amount of glucose loss in the urine during the study period.

In conclusion, our results indicate that the GI of starch-rich food is not affected by the amount of starch ingested in NIDDM patients.

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