

Replacement of digestible by resistant starch lowers diet-induced thermogenesis in healthy men

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The present study describes the effect of replacement of digestible starch by resistant starch (RS) on diet-induced thermogenesis (DIT), postprandial glucose and insulin responses, and colonic fermentation. Ten healthy males consumed three test meals, consisting of diluted, artificially-sweetened fruit syrup and either 50 g raw potato starch (550 g RS/kg), or 50 g pregelatinized potato starch (0 g RS/kg) or 30 g pregelatinized potato starch plus 20 g lactulose (670 g indigestible disaccharide/kg). The meals were served in the morning after an overnight fast. Each volunteer consumed each meal twice on six separate days in random order. Metabolic rate was measured by indirect calorimetry in the fasting state for 15 min and postprandially for 5 h. Shortly before and hourly up to 7 h after consumption of the test meal, end-expiratory breath samples were obtained for H₂ and CH₄ analysis. Shortly before the meal and 30, 60, 180, and 300 min postprandially, blood samples were taken for glucose and insulin analyses. Postprandial increases in glucose and insulin levels were proportional to the amount of digestible carbohydrate in the meal. Breath H₂ and CH₄ concentrations indicated that the pregelatinized starch was not fermented and that lactulose was fermented rapidly. Fermentation of the raw starch started only 6 to 7 h after consumption, resulting in a rise in breath H₂ but not in CH₄. The replacement of 27 g digestible starch by RS in a single meal lowered DIT by on average 90 kJ/5 h, as could also be calculated by assuming that RS does not contribute to DIT. The ingestion of lactulose resulted in a substantial rise in DIT which was most probably caused by its fermentation.

Diet-induced thermogenesis: Glucose: Insulin: Resistant starch

Diet-induced thermogenesis (DIT) depends on the composition of the meal, including the type of carbohydrate (Sharief & MacDonald, 1982; Schwarz *et al.* 1989). About 60–70% of DIT comprises the obligatory energy costs of ingesting, digesting, absorbing and metabolizing the food consumed. The rest of the DIT can be ascribed to a facultative thermogenic effect of the food (Himms-Hagen, 1989). A meal with a high content of rapidly digestible and absorbable carbohydrate, resulting in relatively high postprandial blood glucose concentrations and hence relatively high insulin responses, may increase the facultative part of the DIT via stimulation of the sympathetic nervous system (Landsberg & Young, 1983).

Resistant starch (RS) is not absorbed in the small intestine of healthy humans (Englyst *et al.* 1992). Therefore, RS consumption represents a lower net energy intake compared with an identical amount of digestible starch. This might be of benefit for the obese. Furthermore, because of its indigestible character, RS consumption may result in lower

postprandial glucose and insulin responses compared with digestible starch consumption which in turn could lower the DIT. A lower DIT after ingestion of RS would counteract the advantage for obese people when consuming RS instead of digestible starch.

Since RS is not absorbed in the small intestine it enters the colon where it can be fermented, resulting in the production of H_2 , short-chain fatty acids (SCFA), CO_2 and in some people also CH_4 (Cummings & MacFarlane, 1991). Parts of these products are absorbed from the colon, and H_2 , CH_4 and CO_2 are partly excreted in breath. The percentage of people producing CH_4 (as measured by CH_4 in end-expiratory air) varies from 20 to 70% in different studies (Pitt *et al.* 1980; Bjørneklett & Jenssen, 1982; McNamara *et al.* 1986; Segal *et al.* 1988; Gibson *et al.* 1990). It could be suggested that the absorption and metabolism of fermentation products contribute to the DIT.

In the present study the effect of replacing digestible starch with resistant starch on DIT, postprandial glucose and insulin responses, and colonic fermentation (as measured by H_2 and CH_4 in end-expiratory air) was investigated. To assess the impact of fermentation products generated from RS, lactulose, an indigestible disaccharide that is rapidly fermented in the colon, was studied as well.

SUBJECTS AND METHODS

The study was carried out at the Department of Human Nutrition, Wageningen Agricultural University.

Subjects

Ten apparently healthy (as assessed by a medical questionnaire) male students, aged 24 (SD 2, range 20–26) years, mean body weight (in bathing trunks) 72.2 (SD 8.6, range 61.0–91.1) kg, mean height 1.83 (SD 0.08, range 1.72–1.99) m, mean body mass index 21.5 (SD 1.5, range 19.4–23.5) kg/m^2 , with no history of gastrointestinal diseases or diabetes mellitus and not using a special diet or medication, participated in the study.

Before entering the study the volunteers were screened for CH_4 in their breath on three separate days. A subject was classified as a methane producer when at least two of three end-expiratory breath samples had a methane concentration ≥ 3 ppm after subtracting the CH_4 concentration of the ambient air (McNamara *et al.* 1986; Nagengast *et al.* 1988; Rumessen, 1992). Three out of the ten subjects were found to be methane producers.

Experimental meals

Table 1 shows the composition of the experimental meals. For meal A, 50 g raw potato starch (supplied by the Institut National de la Recherche Agronomique, Nantes, France) containing 550 g RS/kg (type 2, i.e. raw starch granules, RS_2) as measured *in vitro* according to the procedure of Englyst *et al.* (1992) was used. For the control meal (B), 50 g pregelatinized potato starch (Institut National de la Recherche Agronomique, Nantes, France) containing 0 g RS/kg (Englyst *et al.* 1992) was used. As a reference (meal C) we used lactulose (Sirupus Lactulosi; Pharmachemie B.V., Haarlem, The Netherlands), an indigestible disaccharide (4-O- β -D-galactopyranosyl-D-fructose, $C_{12}H_{22}O_{11}$) that is rapidly and extensively fermented in the colon. However, because of its laxative effect only 20 g lactulose was served in a meal. Therefore we added 30 g pregelatinized potato starch to meal C to have equal amounts of 50 g carbohydrate in all meals. The raw and pregelatinized potato starch were both in a dry powdered form and lactulose was available as a syrup, containing 667 g lactulose, 110 g galactose, 10 g glucose, 10 g fructose and 60 g lactose/l. The starches and lactulose syrup were added to 125 ml concentrated fruit syrup (Irma A/S, Rødovre, Denmark) containing 23 g glucose, 35 g fructose and 9 g sucrose/l.

Table 1. *Composition of the experimental meals*

	Meal		
	A Raw potato starch	B Pregelatinized potato starch	C Lactulose plus pregelatinized starch
Total starch (g)	40.7*	46.5†	27.9†
Rapidly digestible starch (g)	3.0*	44.0†	26.4†
Slowly digestible starch (g)	10.6*	2.6†	1.5†
Resistant starch (g)	27.1*	—	—
Total mono- + disaccharides (g)	8.4‡	8.4‡	34.1‡§
Digestible mono- + disaccharides (g)	8.4‡	8.4‡	14.1‡§
Lactulose (resistant disaccharide) (g)	—	—	20.0§
Total carbohydrate (g)	49.1	54.9	62.0
Digestible carbohydrate (g)	22.0	54.9	42.0
Resistant carbohydrate (g)	27.1	—	20.0
Gross energy (kJ)	830	930	1008

* From raw potato starch.

† From pregelatinized potato starch.

‡ From concentrated fruit syrup.

§ From lactulose syrup.

|| Calculated as total starch (g) × 17.2 kJ plus total mono- + disaccharides (g) × 15.5 kJ (Passmore & Eastwood, 1986).

Tap water was added to reach a final volume of 500 ml. The raw and pregelatinized potato starches differed in water content (166 and 49 g/kg respectively) so that meals A and B differed slightly in total starch content (Table 1).

The meals were prepared freshly every day just before consumption. Meal A was a liquid suspension in which the raw starch tended to sink to the bottom of the glass. Meal B was viscous and had to be eaten with a spoon. Meal C was less viscous than B and could be drunk as meal A. Because of these clear differences in viscosity the subjects were able to distinguish between the meals although they did not know which viscosity corresponded to which type of meal.

Experimental design

On a measurement day, subjects were picked up at home by car after an overnight fast. After voiding and weighing, the subjects rested on a bed in a semi-supine position and after a period of about 15 min in which the metabolic rate stabilized, resting metabolic rate (RMR) was measured for 45 min. Then the subjects consumed one of the experimental meals within 10 min and postprandial energy expenditure (PEE) was measured for 5 h. During the metabolic rate measurements the subjects watched video movies. They were allowed to go to the toilet if necessary. All urine produced during the metabolic rate measurements was collected for N determination, necessary to estimate the amount of protein oxidation. Shortly before and 30, 60, 180 and 240 min after consumption of the meal, blood samples were taken by venepuncture for determination of glucose and insulin concentrations. Immediately after the meal the subjects judged the palatability of the meals using visual analogue scales. Shortly before and every hour for 7 h after consuming the meals a questionnaire asking for gastrointestinal complaints was filled in and end-expiratory breath samples were taken for measuring H₂ and CH₄ concentrations. Every time a breath sample was taken a sample of the ambient air was taken as well. After the

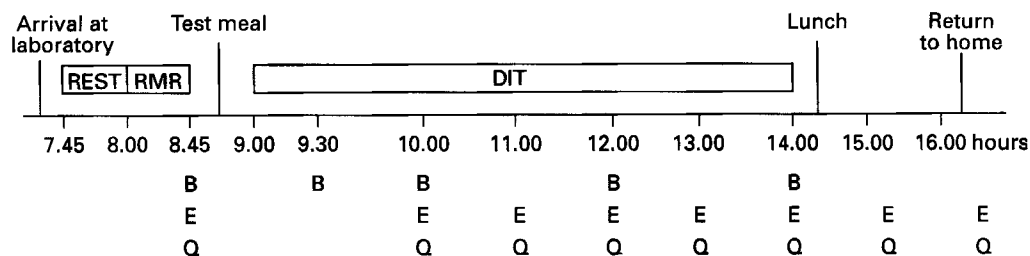


Fig. 1. Time schedule for a measurement day. RMR, resting metabolic rate; DIT, diet-induced thermogenesis; B, blood sample; E, end-expiratory breath sample; Q, questionnaire on gastrointestinal complaints.

metabolic rate measurements were completed (after 5 h) the subjects were served a standardized lunch consisting of two white rolls and one roll with raisins, together with 20 g margarine, 24 g raw (i.e. salted, dried and smoked) ham, 20 g cooked ham, 250 ml partly skimmed milk and coffee or tea (total meal: 3.11 MJ; carbohydrate 43% energy intake, fat 41% energy intake, protein 16% energy intake, and 3.9 g dietary fibre). Two more breath samples were collected at 6 and 7 h after the test meal. Fig. 1 shows the flow diagram of an experimental day. On most measurement days, two subjects were measured simultaneously.

Each subject consumed every type of meal twice. Since a period of at least 6 d separated each two successive measurement days of a subject, carry-over effects were not expected nor was an effect of the order of the meals. To be sure, the order of the meals was random but different for every subject. All measurements were completed within 2.5 months. On the 3 d before the measurements a standardized diet consisting of ordinary foods and containing carbohydrate 60% energy intake, fat 28% energy intake, protein 12% energy intake, and 3.5 g dietary fibre/MJ was provided. The amount of food was attuned to individual energy needs as based on World Health Organization (WHO) energy requirement formulas (WHO, 1985); a suitable activity factor was assessed by asking the subjects about their sporting activities. At 2 d before the measurements the subjects abstained from strenuous physical activities. Throughout the experimental period the subjects kept a diary in which they noted any deviations from the study protocol, gastrointestinal complaints, illnesses and medication taken.

Methods

RMR and DIT were measured by indirect calorimetry with a ventilated-hood system as described in detail elsewhere (Weststrate, 1993). Metabolic rate was calculated using the formula of Jéquier *et al.* (1987). DIT was calculated by subtracting RMR from PEE.

Plasma glucose was measured enzymically by the combined activities of hexokinase (EC 2.7.1.1) and glucose-6-phosphate dehydrogenase (EC 1.1.1.49) (Abbott Spectrum High Performance Diagnostic System; Abbott Laboratories, North Chicago, IL, USA). Serum insulin was measured by immunoassay with a commercial test combination (Boehringer Mannheim GmbH, Germany).

Urinary N was determined by the Kjeldahl method with a Kjeltic autosampler system 1035 analyser (Tecator, Hoganas, Sweden).

End-expiratory breath samples were collected in plastic syringes of 60 ml (Plastipak, Becton Dickinson, Dublin, Ireland). Within 2 h of collection, a 20 ml portion of each sample was used to measure H_2 with an electrochemical measurement cell (Exhaled Hydrogen Monitor; Gas Measurement Instruments Ltd., Renfrew, Scotland). The measurement cell was calibrated twice daily with 105 ppm H_2 in N_2 gas (Intermar B.V., Breda, The Netherlands). The remaining portion of the breath sample was used for a

duplicate methane determination by gas chromatography (Hewlett Packard, model 427, Stimadzu, Chromatopac C-R3A). For calibration (once daily), 5 ppm and 29 ppm CH₄ in N₂ gases were used (Intermar B.V., Breda, The Netherlands).

Statistical analysis

Results are expressed as means with their standard errors. First the means of the duplicates per subject per type of meal were calculated, then the mean and standard error per type of meal. The significance of the differences between the three meals regarding glucose, insulin and DIT was assessed by analysis of variance with type of meal as fixed factor and subject as random factor (thus taking the intrinsic individual levels into account). In general, adding the order of the meals to the model did not contribute to the model. In the case of a statistically significant effect (P value < 0.05) in the analysis of variance, group means were compared by pairwise Student's t tests. Because of the small numbers of CH₄ (n 3) and non-CH₄ producers (n 7) the differences between the meals regarding H₂ and CH₄ excretion in breath were only described and not statistically evaluated. The statistical analysis package SAS, release 6.07 (Statistical Analysis Systems Institute Inc., Cary, NC, USA), was used to perform the statistical analyses.

Ethical considerations

The experimental design and possible discomforts were explained to the subjects before written informed consent was obtained. The experimental protocol was approved by the Medical-Ethical Committee of the Department of Human Nutrition of the Wageningen Agricultural University.

RESULTS

On one of the measurement days, lactulose was added wrongly to one meal and omitted wrongly from another. Inspection of the diaries revealed that one subject was taking antibiotics on his last measurement day. The results of these volunteers on those three experimental days were excluded from the statistical analysis.

The meals were acceptable to the volunteers but in general palatability was regarded as suboptimal. With increasing viscosity (raw starch $<$ lactulose $<$ pregelatinized starch) the meals were less appreciated.

Few gastrointestinal complaints were reported after consumption of the raw and pregelatinized starch meals. One subject reported abdominal complaints starting in the evening of a measurement day on which he had consumed a raw starch meal. Consumption of the lactulose meal resulted in increased flatulence and intestinal rumbling in most subjects, indicative of fermentation of lactulose by the intestinal bacteria. The discomfort caused by the reported intestinal complaints was described as light in most cases and sometimes as moderate.

Postprandial plasma glucose rose to a peak value 30 min after consumption of the meals and returned to baseline levels within 3 h. The peak values differed significantly between the meals ($P < 0.0001$): the mean changes from baseline were 3.9 (SE 0.5), 2.5 (SE 0.5) and 0.5 (SE 0.2) mmol/l for the pregelatinized starch, lactulose and raw starch meals respectively. At 1 h after consumption the plasma glucose concentration was 1.5 (SE 0.4) mmol/l higher after the pregelatinized meal than after the raw starch meal and 1.3 (SE 0.3) mmol/l higher than after the lactulose meal ($P < 0.0001$). At 3 h after consumption the plasma glucose concentration was 0.4 (SE 0.2) mmol/l higher after the raw starch meal than after the pregelatinized meal and 0.3 (SE 0.2) mmol/l higher than after the lactulose meal ($P < 0.001$).

Postprandial serum insulin rose to a peak value 30 min after consumption of the meals and returned to baseline levels within 3 h. The peak value after consumption of the raw

Table 2. Amount of hydrogen expired during 7 h after consumption of a meal containing 50 g raw potato starch, 50 pregelatinized potato starch or 20 g lactulose plus 30 g pregelatinized potato starch by methane (n = 3) and non-methane producers (n = 7)*

(Mean values with their standard errors†)

	Area below the hydrogen curve (ppm × 7 h)				Area below the methane curve (ppm × 7 h)	
	Non-CH ₄ producers		CH ₄ producers		CH ₄ producers	
	Mean	SE	Mean	SE	Mean	SE
Meal:						
Raw starch (A)	63	12	23	6	449	186
Pregelatinized starch (B)	43	10	28	7	400	164
Lactulose (C)	511	57	271	140	592	256
Mean difference‡						
A-B	20	14	-5	1	49	51
C-B	468	60	243	134	192	101
A-C	-448	53	-248	135	-143	71

* For details of diets and procedures, see Table 1 and pp. 424-427.

† First the means of the duplicates per subject were calculated, then the mean and SE per meal.

‡ Values were calculated from the differences on each individual separately for each pair of diets.

starch meal was lower ($P < 0.01$) than after the pregelatinized starch meal (19 mU/l) and the lactulose meal (8 mU/l), the changes from baseline being 37 (SE 18), 36 (SE 7) and 7 (SE 3) mU/l for the pregelatinized starch, lactulose and raw starch meals respectively. At 1 h after consumption the serum insulin concentration was 36 (SE 13) mU/l higher after the pregelatinized starch meal than after the raw starch meal and 27 (SE 8) mU/l higher than after the lactulose meal ($P < 0.0001$).

After consumption of lactulose the H₂ concentration in end-expiratory air rose rapidly to a peak value 3 h after consumption of the meal, both in non-CH₄ and CH₄ producers. The total area below the H₂ curve, as a measure of the total amount of H₂ expired during the experimental period, was larger after consumption of the lactulose meal than after consumption of the other two meals, both for CH₄ and non-CH₄ producers (Table 2). However, the amount of H₂ produced by CH₄ producers after consumption of lactulose was about half the amount produced by non-CH₄ producers. In CH₄ producers breath CH₄ concentration also tended to increase after lactulose consumption. Pregelatinized potato starch seemed to be fully absorbed and not fermented since the H₂ and CH₄ concentrations in end-expiratory air up to 7 h after consumption of the meal were not different from the fasting values, both in CH₄ and non-CH₄ producers. Up to 5 h after ingestion of the raw starch meal in non-CH₄ producers, and up to 6 h in CH₄ producers, breath H₂ concentration did not differ from that after consumption of the pregelatinized starch meal. At 6 to 7 h after consumption of the raw starch meal, however, breath H₂ concentration started to rise: mean difference 7 (SE 2) ppm at 6 h and 12 (SE 5) ppm at 7 h in non-CH₄ producers, and 5 (SE 1) ppm at 7 h in CH₄ producers. No difference was found in breath CH₄ excretion after consumption of the pregelatinized and raw starch meals (Table 2).

RMR was similar before every meal: 5.0 (SE 0.2), 5.0 (SE 0.2) and 4.8 (SE 0.2) kJ/min for the raw starch, pregelatinized starch and lactulose meals respectively.

DIT rose rapidly after consumption of the meals, started to decrease within 1 h and levelled off after 2 h. Mean DIT, total DIT and DIT as a percentage of RMR after the

Table 3. *Effects of meals containing raw potato starch, pregelatinized potato starch or lactulose on diet-induced thermogenesis (DIT; calculated as postprandial energy expenditure minus resting metabolic rate (RMR)) in young men†*

(Mean values with their standard errors‡ for ten subjects)

	DIT					
	kJ/min		kJ		% RMR	
	Mean	SE	Mean	SE	Mean	SE
Meal:						
Raw starch (A)	0.1*	0.1	36*	23	2.6*	1.5
Pregelatinized starch (B)	0.4	0.1	125	16	8.6	1.2
Lactulose (C)	0.5	0.1	164	18	11.5	1.4
Analysis of variance:						
Meal	$P < 0.0001$		$P < 0.0001$		$P < 0.001$	
Subject	NS		NS		NS	
Mean differences§						
A - B	-0.3	0.1	-90	30	-6.0	2.0
C - B	0.1	0.1	39	22	2.9	1.6
A - C	-0.4	0.1	-128	20	-8.9	1.4

NS, not significant.

* Mean values were significantly different from the pregelatinized starch and lactulose meals, $P < 0.05$ (Student's *t* test).

† For details of diets and procedures, see Table 1 and pp. 424-427.

‡ First the means of the duplicates per subject were calculated, then the mean and SE per meal.

§ Values were calculated from the differences on each individual separately for each pair of diets.

pregelatinized starch and lactulose meals were similar; DIT after the raw starch meal was significantly lower, both in terms of magnitude and duration (Table 3).

DISCUSSION

To our knowledge the influence of RS consumption on DIT has not been reported before. We found a DIT of 125 (SE 16) kJ in 5 h after consumption of a meal containing 1000 g digestible carbohydrates/kg (B), i.e. 14% of the gross energy content of the meal. Based on the fact that the raw starch meal (A) contained 550 g resistant starch/kg and 450 g digestible carbohydrate/kg, an expected theoretical value of DIT of about 50 kJ in 5 h can be calculated. The observed value of 36 (SE 23) kJ was well within the 95% confidence limits. The somewhat lower value could be due to a lower digestibility *in vivo* compared with the digestibility determined *in vitro* by the Englyst method (Englyst *et al.* 1992). DIT after 2 h was 83 (SE 6) kJ for the pregelatinized starch, whereas 35 (SE 8) kJ was found for the raw starch. The latter value compares well with the expected theoretical value of 33 kJ. It can be concluded that consumption of a meal in which part of the digestible starch is replaced by RS lowers DIT during the first 5 h after the meal to the extent expected based on the amount of indigestible carbohydrate in the meal.

Possibly the DIT after consumption of RS is postponed: SCFA produced on colonic fermentation of RS could add to the DIT. However, since the fermentation of the type of RS used (type 2, RS₂, raw starch granules) started only 6 to 7 h after consumption, as indicated by the H₂ concentration in end-expiratory air (a finding that is in accordance with other studies; Olesen *et al.* 1992), we cannot confirm this with our current experimental design in which DIT was measured for the first 5 h after the meal. Since RS fermentation

most probably occurs gradually the possible 'SCFA-effect' will be too small to be measured by indirect calorimetry. The delayed RS_2 fermentation compared with lactulose could mean that RS_2 is a less suitable substrate for colonic fermentation, or that RS_2 is fermented at a different site in the colon compared with lactulose. In addition, the time to pass through the digestive tract could be longer for RS_2 than for lactulose. It can be calculated that an intake of 27 g RS/d would reduce the daily energy expenditure of these subjects by approximately 0.7%.

Although the difference was not statistically significant, it is striking that DIT after consumption of 20 g (indigestible) lactulose plus 30 g pregelatinized (digestible) starch was larger than after consumption of 50 g pregelatinized starch. Based on the amount of digestible starch a DIT of 97 kJ in 5 h was expected. However, 164 (SE 18) kJ was found. Thus 66 kJ of the observed DIT was caused by the lactulose. Of this 3.3 kJ/g lactulose, 1.2 kJ can be ascribed to heat of fermentation (Livesey, 1992) and 1.8 kJ to the metabolism of the rapidly absorbed (McNeil *et al.* 1978; Ruppin *et al.* 1980; Pomare *et al.* 1985; Scheppach *et al.* 1991; Peters *et al.* 1992) SCFA (Smith & Bryant, 1979; Stryer, 1988) formed during the rapid fermentation of lactulose, as found in this and other studies (Bjørneklett & Jenssen, 1982; Florent *et al.* 1985; Würsch *et al.* 1989; Cloarec *et al.* 1990; Rumessen *et al.* 1990). The combustion of part of the H_2 and CH_4 produced upon lactulose fermentation might account at least partly for the remaining gap of 0.3 kJ, although the efficiency of the conversion of fermentable carbohydrate to the combustible gases H_2 and CH_4 might be as low as the equivalent of 0.02 kJ gas (breath + flatus) per kJ carbohydrate fermented (Ruppin *et al.* 1980). Although these calculations seem to fit the data nicely, it should be kept in mind that large variations were found in DIT and, in view of the breath H_2 concentrations, probably not all lactulose was fermented within the 5 h period after consumption.

Ritz *et al.* (1993) found a significant increase in CO_2 production when 20 g lactulose was added to a standardized glucose load of 50 g. This excess CO_2 probably arose from colonic fermentation of lactulose and from addition to the fuel mix of SCFA, especially acetate, produced during lactulose fermentation. To be precise, the calculation of energy expenditure based on indirect calorimetry should be corrected for the CO_2 produced during colonic fermentation. However, for several reasons it is very hazardous to estimate the proportion of exhaled CO_2 that arises from lactulose fermentation. First, the end products of colonic fermentation depend on the composition of the bacterial flora, so that there are several fermentation equations possible. Second, it is not known what proportion of the CO_2 produced during fermentation is absorbed, and third, it is not known what proportion of the absorbed CO_2 can eventually be measured in breath. It can be calculated that energy expenditure is overestimated by a maximum of 0.9% when no correction is made for the amount of CO_2 produced during fermentation of lactulose. Since this is only a small error, especially in view of the large variation inherent in the ventilated-hood method, that would not alter the conclusion, no such correction was made when presenting the results of the present experiment.

Not surprisingly, postprandial plasma glucose and serum insulin levels were proportional to the amount of digestible carbohydrate in the meals, as found by others as well (Collings *et al.* 1981; Jenkins *et al.* 1987; Holm *et al.* 1988, 1989; Bornet *et al.* 1989; Holm & Björck, 1992). However, plasma glucose and serum insulin responses after the raw starch meal were smaller than expected from the amount of rapidly digestible carbohydrate as was found by Raben *et al.* (1994) as well. Raben *et al.* (1994) suggested that the lack of increase in gastric inhibitory polypeptide (GIP) and glucagon-like peptide-1 (GLP-1) that they found after a raw starch meal might explain the difference in insulin response between the meals, beyond what can be expected from the glucose-stimulated insulin secretion (GIP and GLP-1 are

both potent stimulators of insulin secretion). Possibly the decrease in insulin response after consumption of the raw starch meal explains to a substantial extent the reduction in DIT found.

In conclusion, consumption of a meal in which part of the digestible starch is replaced by resistant starch was found to lower DIT during the first 5 h after the meal to the extent that would be expected based on the amount of indigestible carbohydrate in the meal. This outcome might be explained by the observation that the resistant starch used was not fermented within 5 h after consumption as evidenced by unchanged H_2 and CH_4 concentrations in end-expiratory air.

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