# Resistant starch: the effect on postprandial glycemia, hormonal response, and satiety[1–3]

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**ABSTRACT** The effect of resistant starch (RS) on postprandial plasma concentrations of glucose, lipids, and hormones, and on subjective satiety and palatability ratings was investigated in 10 healthy, normal-weight, young males. The test meals consisted of 50 g pregelatinized starch (0% RS) (S) or 50 g raw potato starch (54% RS) (R) together with 500 g artificially sweetened syrup. After the R meal postprandial plasma concentrations of glucose, lactate, insulin, gastric inhibitory polypeptide (GIP), glucagon-like peptide-1, and epinephrine were significantly lower compared with after the S meal. Moreover, subjective scores for satiety and fullness were significantly lower after the R meal than after the S meal. Differences in GIP, texture, and palatability may have been involved in these findings. In conclusion, the replacement of digestible starch with RS resulted in significant reductions in postprandial glycemia and insulinemia, and in the subjective sensations of satiety. *Am J Clin Nutr* 1994;60:544–51.

**KEY WORDS** Potato starch, appetite, palatability, lactate, insulin, gastric inhibitory polypeptide, catecholamines

## Introduction

Until recently, starch was believed to be 100% digested in the small intestine—independent of the source, type, and preparation of the starch. However, within the past 10 y it has been found that, despite the fact that pancreatic $\alpha$-amylase is present in the gut in ample amounts (1), a fraction of the ingested starch passes undigested to the large bowel (2–4). Here the starch undergoes a more or less complete fermentation, resulting in the production and uptake of short-chain fatty acids (acetic acid, butyric acid, and propionic acid) (5, 6). This fraction of the starch has been named resistant starch (RS) and has been defined by the European FLAIR Concerted Action on Resistant Starch (EURESTA) as "the sum of starch and products of starch hydrolysis not absorbed in the small intestine of healthy individuals."

The amount of RS present in starch-rich foods depends on several factors, ie, the source, ripeness, processing, preparation, and storage of the foods. It has been shown that starch from white bread, porridge oats, and cornflakes is almost completely digested in the small intestine (4) whereas native starch from banana and uncooked potato is highly resistant to hydrolysis in vitro (7) and in vivo (8). When the potato is cooked the starch granules gelatinize and become readily digestible, whereas cooling of the potato reverses this gelatinization process and renders ≈12% of the starch resistant to small intestinal digestion (9).

The importance of RS to human health, ie, diabetes, overweight, cardiovascular diseases, or cancer, is still not known. However, because RS—unlike nonresistant starch—is not digested and therefore not absorbed as glucose in the small intestine of healthy humans (8, 9), a reduction in both postprandial glycemia and insulinemia can be expected after the intake of RS compared with digestible starch. Such an effect of RS may be beneficial in the control of diabetes. Moreover, RS may through mechanisms similar to those exerted by soluble dietary fiber influence the amount and rate of absorption of other nutrients in the diet, ie, glucose and fat, which may be beneficial in the control of glycemia or lipidemia.

The potential use of RS as a weight-reducing agent may also be of interest because the energy value of 1 g RS, including the contribution from fermentation products, has been estimated to be only 9.0–9.8 kJ/g, ie, half the value of digestible starch (10). It is therefore tempting to suggest RS as a weight-loss agent; however, the impact of RS on macronutrient balance and appetite control may be less beneficial to weight regulation. This hypothesis is based on the recently developed concepts on the regulation of energy and macronutrient balance. Thus, there seems to be a close regulation between the body's macronutrient stores and the three macronutrients (fat, protein, and carbohydrate) in the diet (11, 12). The maintenance of the body's relatively small carbohydrate stores seems especially crucial to overall energy balance (11). Moreover, recent studies have shown that the control of appetite may be influenced by the amount of available carbohydrate in the diet (13, 14) and that consumption of a certain amount of carbohydrate (or protein) in a meal may be necessary to achieve satiation (15). Increasing the amount of RS in the diet with a resultant decreased amount of absorbed carbohydrate and a decreased carbohydrate-to-fat ratio in the diet may thus lead to

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[2] Supported by grants from The Danish Research and Development Programme for Food Technology 1990–1994 and the Danish Medical Research Council grant 12-9537-3.

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Received November 2, 1993.

Accepted for publication May 9, 1994.

 

TABLE 1
Subject characteristics[1]

| | Denmark ($n$ = 5) | Italy ($n$ = 5) | All ($n$ = 10) |
|---|---|---|---|
| Age (y) | 25.6 ± 1.9 | 21.2 ± 0.7 | 23.4 ± 1.2 |
| Height (m) | 1.80 ± 0.01 | 1.78 ± 0.02 | 1.79 ± 0.01 |
| Weight (kg) | 71.2 ± 3.0 | 74.0 ± 3.5 | 72.6 ± 2.2 |
| BMI[2] | 22.0 ± 1.1 | 23.2 ± 0.6 | 22.6 ± 0.6 |
| Fat mass (%) | 18.6 ± 2.4 | 20.8 ± 1.3 | 19.7 ± 1.3 |

[1] $\bar{x}$ ± SEM.
[2] In kg/m$^2$.

a reduction in the satiating power of the diet. This may result in overconsumption and subsequent weight gain. However, if RS has a "dietary-fiber-like" positive effect on satiety (16–18) an increase in the amount of RS in the diet may be beneficial to weight maintenance.

The purpose of the present study was to investigate the acute effects of RS vs digestible nonresistant starch on postprandial changes in glycemia, hormonal response, and subjective sensations of hunger and satiety. The two test starches were given in as pure a form as possible to clarify the role of the starches per se without the interference from other nutrients.

## Subjects and methods

The experiment was performed as a joint study within the EURESTA framework. Exactly the same experiment was conducted at two centers: the Research Department of Human Nutrition, The Royal Veterinary and Agricultural University, Denmark, and the Department of Human Nutrition, University of Pavia, Italy.

### *Subjects*

In each center five healthy male subjects (20–31 y of age, normal-weight, nonsmokers, not elite athletes) with no history of obesity or diabetes, participated in the study (**Table 1**). Females were not included to avoid possible differences due to the menstrual cycle. The subjects' energy needs during the study were determined by using WHO tables according to age, weight, height, and sex. The multiplication factor 1.78 was used to account for medium physical activity level of the subjects (19). The body composition of the subjects was estimated by the bioimpedance method by an Animater (HTS Engineering Inc, Odense) in Denmark and BIA 109 (RJl Systems, Detroit) in Italy. Fat-free mass was calculated by using the equation of Deurenberg et al (20). The study was approved by the Municipal Ethical Committee of Copenhagen and Frederiksberg to be in accordance with the Helsinki-II declaration and all subjects gave written consent after the experimental procedure had been explained to them.

### *Diets*

The two test meals consisted of 50 g raw potato starch (54.1% RS) (R) or 50 g pregelatinized potato starch (100% digestible) (S) mixed into 500 mL diluted artificially sweetened fruit syrup (**Table 2**). The syrup was based on apple, grape, red current, elderberry, black current, and cherry and commercially purchased from Irma, Denmark. The starches were produced and

TABLE 2
EURESTA reference starch materials[1]

| Food | DM | Calculated starch values | | | |
|---|---|---|---|---|---|
| | | TS | RDS | SDS | RS |
| | % | % by wt | | | |
| Raw potato starch | 83.4 | 81.3 | 6.0 | 21.2 | 54.1 |
| Pregelatinized potato starch | 95.1 | 92.9 | 87.9 | 5.2 | — |

[1] DM, dry matter; TS, total starch; RDS, rapidly digestible starch; SDS, slowly digestible starch; RS, resistant starch. Analyzed from the Dunn Clinical Nutrition Centre. Methods described by Englyst et al (21) and Englyst and Cummings (22).

supplied from l'Institut National de la Recherche Agronomique (INRA), Nantes, France. Both starches appeared as a white flour. Just before consumption the starch was slowly poured into the syrup while mixing it with a hand mixer at the slowest speed to avoid an increase in temperature of the mixture. Several other ways of administration were tested before deciding on these test meals. However, the pregelatinized starch could not be mixed into other liquids without producing a very unpleasant, inedible substance. Because heating would affect the physical properties of the raw starch, a cold-meal preparation had to be used. The starches could thus not be baked into a bread or used in a pasta. The best compromise for these two starches was therefore to mix them with the syrup. For the R meal this resulted in a drinkable mixture whereas the S meal was a porridge-like gel to be eaten by spoon. Although the syrup was sweetened with artificial sweeteners it contained some fruit sugars naturally occurring in the berries and fruits (**Table 3**).

Each test day was preceded by 3 d on an identical carbohydrate-rich diet (60% of energy as carbohydrate, 28% as fat, and 12% as protein, and 3.5 g dietary fiber/MJ), prepared at the departments from food items according to each subjects' individual energy requirements, and adjusted to the nearest 0.5 MJ. The subjects were instructed to adhere strictly to the diet. If subjects

TABLE 3
Carbohydrate and energy contents in the two test meals (50 g potato starch + 500 mL diluted fruit syrup)[1]

| | Test meal | |
|---|---|---|
| | R | S |
| Total starch in 50 g potato starch (g) | 40.7 | 46.5 |
| RS (g) | 27.1 | 0.0 |
| RDS (g) | 3.0 | 44.0 |
| SDS (g) | 10.6 | 2.6 |
| Fruit sugars from syrup (g) | 8.4 | 8.4 |
| Glucose (g) | 2.9 | 2.9 |
| Fructose (g) | 4.4 | 4.4 |
| Sucrose (g) | 1.1 | 1.1 |
| Total digestible carbohydrate (g)[2] | 22.0 | 54.9 |
| Total energy (kJ)[3] | 367 | 917 |

[1] R, raw potato starch; S, pregelatinized potato starch; RS, resistant starch; RDS, readily digestible starch; SDS, slowly digestible starch.
[2] Not taking fermentation into consideration.
[3] 16.7 kJ/g.

could not consume all the food, they had to bring the leftovers to the departments for weighing and registration. The same food was deducted from the diet during the following preexperimental periods. The subjects were instructed to abstain from strenuous physical activity for the 2 d before the test days. Together with the standard diet this should ensure equally filled glycogen stores and similar macronutrient balance on the 2 test days. The computer databases of foods from the National Food Agency of Denmark (Dankost) and Italy (INN) were used in the calculations of energy and nutrient composition of the test diets.

*Experimental protocol*

The two test meals were given in a crossover design on separate days with ≥ 1 wk and no more than 6 wk separating the test days. On the test day the subjects arrived at the institute at 0800, with a minimum of physical activity, by car, bus, or train after having fasted for 12 h from the evening before. After subjects voided and were weighed (to the nearest 100 g), bioimpedance was measured. The subjects then rested in the supine position on a bed covered with an antidecubitus mattress with slight elevation of the head. A Venflon catheter (Viggo, Gothenborg, Sweden) was inserted in an antecubital arm vein. After a 10-min rest a fasting blood sample was taken and after a further 20-min rest resting metabolic rate was measured by indirect calorimetry by using a ventilated hood. A second fasting blood sample was taken hereafter and the test meal was then served and consumed within 10 min. Exactly the same time was spent on the two test meals for each individual subject. Postprandial energy expenditure was measured for 5 h (1000–1500 h), and blood samples were taken 15, 30, 45, 60, 75, 90, 105, 120, 150, 180, 210, 240, and 300 min after the meal was consumed. During the postprandial measurements the subjects were allowed to watch light entertainment movies, and to have a break of a maximum of 5 min after 2 and 4 h. During the break the subjects could sit, walk quietly, or go to the toilet. The exact time schedule was noted and repeated on the following test days. Water consumption during the test period was allowed, but the total amount consumed was noted and repeated on the second test day.

Immediately before and every 30 min after the meal, questionnaires to assess hunger, satiety, fullness, and prospective consumption were filled out by each subject. Ratings were made on 100-mm visual analogue scales (VAS) with words anchored at each end, expressing the most positive (ie, good, pleasant) or the most negative ratings (ie, bad, unpleasant) (23). Immediately after the test meals the palatability, taste, aftertaste, texture, and visual appeal of the two test meals were recorded by the subjects using VAS scores. Data on energy expenditure are being published separately (A Tagliabue, A Raben, ML Heijnen, P Deusenberg, E Pasquali, A Astrup, unpublished observations, 1994).

*Laboratory analyses*

Blood was sampled without stasis through the indwelling antecubital cannula by using iced syringes. Plasma glucose and lactate were analyzed by standard enzymatic methods (24). Plasma glycerol was analyzed after trichloracetic acid precipitation, essentially as described by Chernick (25). Blood for determination of plasma catecholamines was collected in tubes containing ethylene glycol-bis(β-aminoethyl ether)-*N,N,N′,N′*-tetraacetic acid (EGTA) and glutathione. Samples were immediately centrifuged for 10 min at 3000 × *g* and 4 °C and the plasma stored at −80 °C until determination of catecholamines by a radioenzymatic method (26). Immunoreactive insulin concentrations were measured in plasma with radioimmunoassay kits purchased from Novo, Copenhagen. Gastric inhibitory polypeptide (GIP) and glucagon-like peptide-1 (GLP-1) were determined by radioimmunoassay on plasma extracted with ethanol as previously described (27–29). Samples for the GIP and GLP-1 analyses could not be taken from the Italian subjects. The starch analyses were performed at the Dunn Clinical Nutrition Centre (United Kingdom) by using the methods described by Englyst et al (21) and Englyst and Cummings (22).

*Statistical analyses*

All results are given as means ± SEM. Data are presented as changes from basal fasting concentrations by using the second fasting blood sample as the basal concentration. Responses to the two test meals were compared by parametric analysis of variance (ANOVA) for repeated, paired measures with time and diet as factors and with subject nested into diet. The key results are presented with $F$ or $t$ values and degrees of freedom. Areas under the curves (AUCs) for the 5-h measurement periods 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 (= net response). A paired $t$ test was used in the comparisons between two means on the same subject. Regression analyses were performed on the differences between the S and R meals (referred to as S−R) for the 5-h AUCs and means for the VAS scores, peak/nadir values, and △peak/△nadir values. This was done to account for the data being paired. The level of significance was set at $P < 0.05$. *Statgraphics* software version 4.2 (Graphic Software Systems, Inc, Rockville, MD) and the *Statistical Analysis Package* (SAS Institute, Cary, NC) were used in the statistical calculations.

## Results

*Glucose and lactate*

Fasting plasma glucose concentrations averaged 4.97 ± 0.08 mmol/L before the S meal and 4.91 ± 0.07 mmol/L before the R meal (NS). A significant interaction between diet and time was observed after the test meals with glucose concentrations increasing nine times as much after the S meal as after the R meal (△peak concentrations: S, 3.07 ± 0.29 mmol/L after 30 min; R, 0.36 ± 0.13 mmol/L after 15 min) ($F_{[12,216]} = 25.2$, $P < 0.0001$) (**Fig 1**). The 5-h AUCs averaged 79.53 ± 27.26 mmol·min/L after the S meal and −42.02 ± 9.51 mmol·min/L after the R meal ($t_9 = 4.51$, $P = 0.0015$). The AUCs for the first 2 h also resulted in a negative area for the R meal (−13.37 ± 6.71 mmol·min/L) compared with the S meal (140.00 ± 21.53 mmol·min/L) ($t_9 = 7.11$, $P < 0.0001$).

Plasma lactate concentrations increased after both test meals with a peak after 45 min for the S meal (1.36 ± 0.09 mmol/L), and after 15 min for the R meal (1.18 ± 0.10 mmol/L) (interaction diet-time: $F_{[12,216]} = 25.2$, $P < 0.0001$) (Fig 1). The increase from basal was twice as high after the S (0.52 ± 0.06 mmol/L) than after the R meal (0.24 ± 0.06 mmol/L). Five-hour AUCs averaged 6.9 ± 10.0 mmol·min/L after the S meal and −26.3 ± 8.9 mmol·min/L after the R meal ($t_9 = 3.70$, $P = 0.005$).

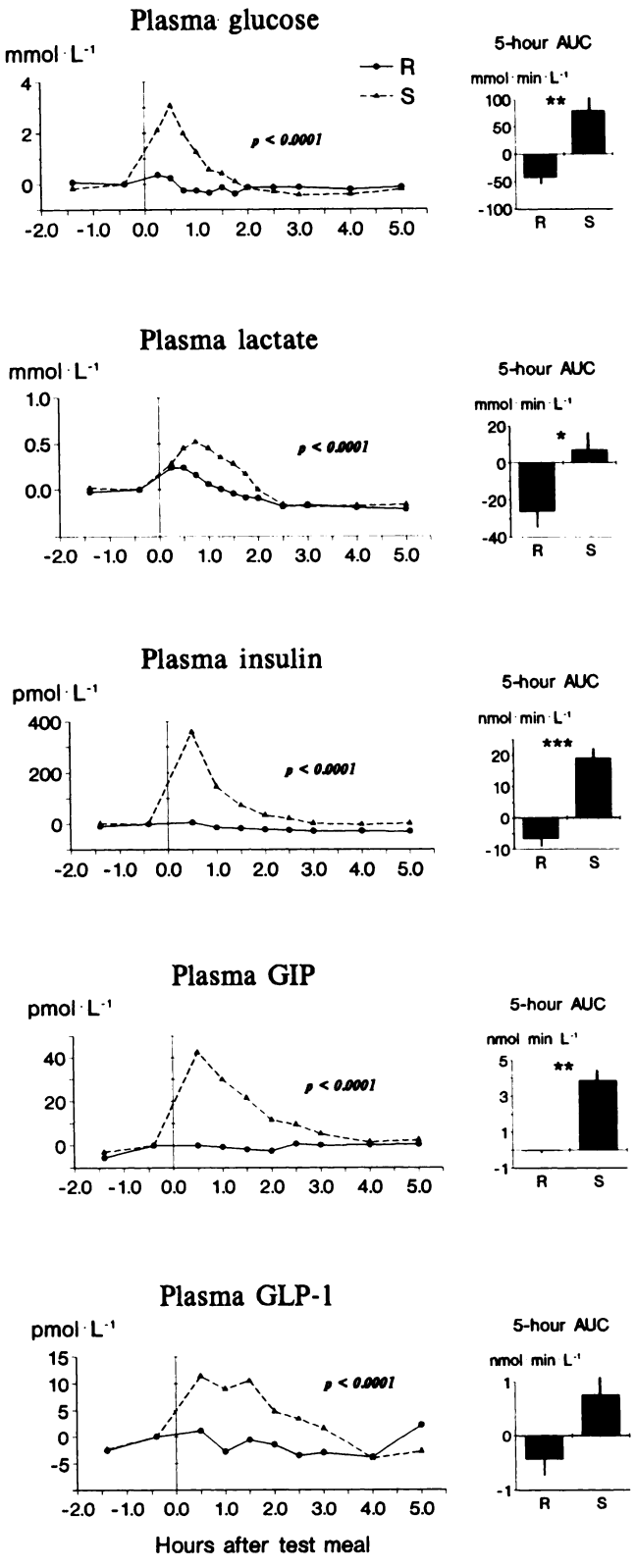


FIG 1. Change in plasma concentrations of glucose, lactate, insulin, gastric inhibitory polypeptide (GIP), and glucagon-like peptide-1 (GLP-1) after a raw potato starch meal [54% resistant starch (RS)] (R) and a pregelatinized potato starch meal (0% RS) (S) in 10 healthy, normal-weight, male subjects. All data are mean (±SEM), expressed as differences from fasting concentrations. Left panel (ANOVA): all (time effect: $P < 0.0001$); plasma glucose (diet effect: $F_{[1,18]} = 25.1$, $P < 0.0001$; interaction between diet and time: $F_{[12,216]} = 25.2$, $P < 0.0001$); plasma lactate (diet effect: $F_{[1,18]} = 36.7$, $P < 0.0001$; interaction between diet and time: $F_{[12,216]} = 25.2$, $P < 0.0001$); plasma insulin (diet effect: $F_{[1,18]} = 39.9$, $P < 0.0001$; interaction between diet and time: $F_{[8,143]} = 15.1$, $P < 0.0001$); plasma GIP ($n = 5$) (diet effect: $F_{[1,8]} = 27.3$, $P < 0.0001$; interaction between diet and time: $F_{[8,63]} = 14.6$, $P < 0.0001$); plasma GLP-1 ($n = 5$) (interaction between diet and time: $F_{[8,57]} = 4.7$, $P < 0.0001$). Right panel: areas under the curves (AUC). *** $P < 0.05$, ** $P < 0.01$, * $P < 0.001$.

*Insulin, GIP, and GLP-1*

Fasting plasma insulin concentrations averaged 53.7 ± 5.0 pmol/L before the S meal and 75.4 ± 10.0 pmol/L before the R meal (NS). A significantly different response pattern was observed after the two test meals. Compared with fasting concentrations plasma insulin increased by a factor of six to 355.4 ± 68.5 pmol/L after the S meal, but to only 84.6 ± 7.2 pmol/L after the R meal (interaction diet-time: $F_{[8,143]} = 15.14$, $P < 0.0001$) (Fig 1). The AUCs averaged 19.1 ± 4.0 nmol·min/L after the S meal and −6.6 ± 1.2 nmol·min/L after the R meal ($t_9 = 4.90$, $P = 0.0008$). Also, the AUCs for the first 2 h resulted in differences between the S (18.4 ± 2.8 nmol·min/L) and the R meals (−1.4 ± 1.1 nmol·min/L) ($t_9 = 5.34$, $P = 0.0005$).

Plasma GIP showed the same response pattern as plasma insulin (Fig 1). Thus compared with fasting values, GIP increased by a factor of 10 after the S meal whereas no increase was observed after the R meal (interaction diet-time: $F_{[8,63]} = 14.6$, $P < 0.0001$). The AUC S−R for GIP and lactate were positively correlated ($r = 0.91$, $P = 0.03$).

Plasma GLP-1 increased significantly after the S meal whereas no changes were observed after the R meal (interaction diet-time: $F_{[8,57]} = 4.7$, $P = 0.0002$) (Fig 1). The difference in the AUCs after the R and the S meals was nearly significant ($t_9 = 2.56$, $P = 0.06$). The AUC S−R for GLP-1 was negatively correlated with GIP ($r = -0.94$, $P = 0.02$) and lactate ($r = -0.88$, $P = 0.052$) and △peak S−R for GLP-1 negatively correlated with GIP ($r = -0.91$, $P = 0.03$).

*Triglycerides (TG) and glycerol*

Plasma triglyceride concentrations showed a similar response pattern after the two test meals (**Fig 2**). Thus, TG decreased slightly after both meals with a nadir after 2.5 h and returned to baseline after 5 h (time effect, $F_{[9,135]} = 4.1$, $P < 0.001$). Triglyceride concentrations were significantly correlated with insulin for the S−R AUCs ($r = 0.65$, $P = 0.04$) and the S−R △peak/△nadir values ($r = 0.83$, $P = 0.006$).

A tendency to a different response pattern for plasma glycerol was found after the two test meals. Thus plasma glycerol decreased after the S meal to a nadir after 1.5 h whereas no decrease was observed after the R meal (interaction diet-time: $F_{[8,144]} = 1.9$, $P = 0.06$) (Fig 2). After 5 h glycerol concentrations had increased by 59.3 mmol/L for the S meal and by 39.8 mmol/L for the R meal compared with fasting concentrations. The AUCs were not significantly different after the two test meals. The AUC S−R for glycerol was negatively correlated with glucose ($r = -0.75$, $P = 0.01$), whereas peak S−R for glycerol was positively correlated with peak S−R triglyceride ($r = 0.72$, $P = 0.02$).

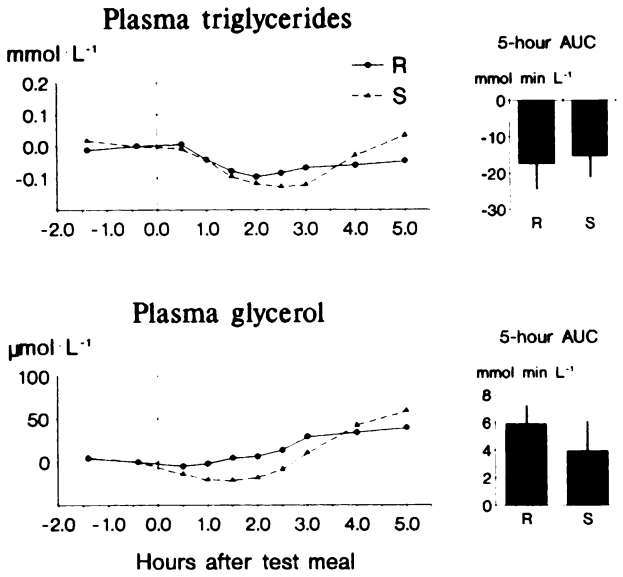


FIG 2. Change in plasma concentrations of triglycerides and glycerol after a raw potato starch meal (54% resistant starch [RS]) (R) and a pregelatinized potato starch meal (0% RS) (S) in 10 healthy, normal-weight, male subjects. All data are mean (±SEM) expressed as changes from fasting concentrations. Left panel (ANOVA): all (time effect: $P < 0.001$); plasma glycerol (interaction between diet and time: $F_{[8,144]} = 1.9$, $P = 0.064$). Right panel: areas under the curves (AUC).

*Epinephrine and norepinephrine*

No significant differences were observed in plasma norepinephrine (NE) after the two test meals (**Fig 3**). However, a significantly different response pattern for E was found after the two meals. Thus, no changes in plasma E concentrations were observed after the R meal, whereas E increased significantly from 2 to 4 h after the S meal (interaction diet-time: $F_{[9,144]} = 2.68$, $P = 0.0064$) (Fig 3). The differences in AUCs were not significantly different for E or NE.

*Satiety scores*

Significant differences were found for satiety and fullness (interaction diet-time: $F_{[11,198]} = 1.95$, $P = 0.03$ and $F_{[11,198]} = 2.30$, $P = 0.012$) after the two test meals (**Fig 4**). Thus the subjects felt more satisfied and more full after the S meal than after the R meal.

Simple regression analysis showed no correlations between any of the mean satiety scores and the blood indexes when 5-h AUCs were used. When peak and nadir values were included, mean hunger S−R was significantly correlated with △peak S−R for lactate ($r = 0.68$, $P = 0.03$) whereas peak GIP S−R was correlated with peak fullness ($r = -0.87$, $P = 0.057$) and with nadir prospective food consumption ($r = 0.88$, $P = 0.046$).

*Evaluation of the test meal*

The subjects found that the S meal looked less appetizing ($t_9 = 3.52$, $P < 0.001$), had a more unpleasant taste ($t_9 = 2.84$, $P < 0.05$), and had a firmer texture ($t_9 = -5.38$, $P = 0.0004$) than the R meal (**Table 4**). They did not find that the aftertaste or overall palatability were different between the meals. Simple regression analysis on the S−R scores showed correlations between aftertaste and mean satiety ($r = 0.65$, $P = 0.04$), hunger ($r = -0.64$, $P = 0.049$), and fullness ($r = 0.62$, $P = 0.054$).

## Discussion

Marked differences in plasma concentrations of substrates and hormones as well as in palatability and satiety scores were observed after the two test meals. Overall, the S meal greatly stimulated plasma concentrations of glucose, insulin, and gastrointestinal hormones whereas no or only a modest stimulation of these indexes was observed after the R meal.

Plasma glucose increased nine times more after the S meal than after the R meal. This was not entirely unexpected on the basis of the analytical data on digestibility of the two test starches. However, the differences in postprandial glycemia were somewhat larger than would be expected from the clinical analyses. Thus, the ratio between the peak glucose response after the R and S meal was 1:9 (0.36/3.07 mmol/L), whereas the ratio between the amount of readily digestible carbohydrate was 1:5 (11.4/54.4 g) (Table 3). The reason for this difference is not readily apparent but may be connected with differences in the stimulation of gastrointestinal factors and insulin after the two meals.

Despite the minor increase in plasma glucose after the R meal, the increase in plasma lactate concentration amounted to 50% of the increase after the S meal. This may be due to the fructose present in the test meal (4.4 g) or to a part of the glucose being converted to lactate via nonoxidative pathways in the splanchnic region (30).

Also, plasma insulin increased markedly after the S compared with the R meal, producing a ratio between △peak values of 30:1 (182/6 pmol/L). This difference in insulin response is far greater

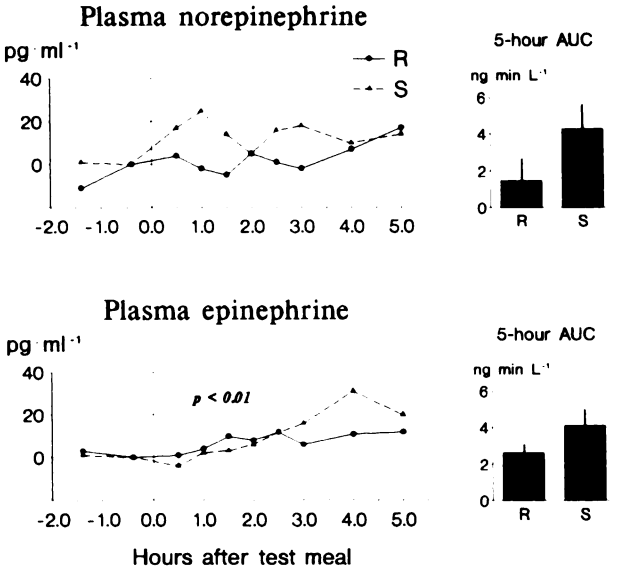


FIG 3. Changes in plasma concentrations of norepinephrine and epinephrine after a raw potato starch meal (54% resistant starch [RS]) (R) and a pregelatinized potato starch meal (0% RS) (S) in 10 healthy, normal-weight, male subjects. All data are mean (±SEM), expressed as differences from fasting concentrations. Plasma epinephrine (time effect: $F_{[9,144]} = 8.37$, $P < 0.0001$; interaction between diet and time: $F_{[9,144]} = 2.7$, $P = 0.0064$). Right panel: areas under the curves (AUC).

than would be expected from the difference in glucose load and glucose response after the two meals. In trying to explain this it may be useful to look at the changes in gastrointestinal hormones. Not only did the R meal result in a much lower blood glucose response compared with the S meal, but at the same time there seemed to be no effect on either the proximal (GIP) or distal (GLP-1) incretins in contrast with the increase in these hormones after the S meal. Both GIP and GLP-1 are known to be potent stimulators of insulin secretion (31). The large difference between the meals in these hormones may therefore explain the difference—beyond what can be expected from the glucose-stimulated insulin secretion—in insulin response between the two meals.

The subjective feelings of satiety and fullness were also significantly influenced by the digestibility of the starch. Thus, the pregelatinized and fully digestible potato starch meal resulted in greater feelings of satiety and fullness compared with the resistant and slowly digestible starch meal. Already, 1–1.5 h after the raw potato starch meal the subjective scores were back to fasting concentrations, whereas the satiating power of the S meal lasted 2.5–3 h postprandially.

It has previously been stated that changes in satiety after a carbohydrate load may be mediated through an effect of plasma glucose or hepatic glycogen concentration on specific glucosensitive cells in the brain (32) and studies have demonstrated a satiating effect of carbohydrate per se (14). We found no significant correlations between the changes in plasma glucose and in the four satiety scores. In a recent study from our department in which a high- and a low-fiber meal were given to 10 healthy subjects, the AUCs for glucose and satiety scores were not significantly correlated either (18). In the present study differences in hunger ratings between the two meals were in fact correlated with differences in Δpeak lactate concentration, which may reflect the rate of nonoxidative glucose disposal. In the present study carbohydrate was the only energy source in the test meals, but because an increased energy load in a meal has also been shown to result in an increased satiating power of the meal (33), it may be that the differences in satiety sensations were due to a different net energy load in the meals. A firmer conclusion may, however, require more direct measurements of glycogen stores or glucose disposal in order to explain changes in subjective satiety scores.

Gastrointestinal hormones have previously been connected with satiety and obesity (34, 35). In the present study some correlations were found between GIP and satiety scores but not between GLP-1 and satiety. The correlations found indicate that GIP may decrease satiety and increase hunger, which is in contrast with the previously suggested satiating power of GIP. In the present study, however, data on GIP were only available for five subjects. The correlations must therefore be considered with

TABLE 4
Subjective evaluation of the test meal[1]

| | R | S |
|---|---|---|
| Visual appeal (0: good, 10: bad) | 5.3 ± 0.7 | 8.1 ± 0.5[2] |
| Taste (0: pleasant, 10: unpleasant) | 6.0 ± 0.8 | 7.2 ± 0.8[4] |
| Aftertaste (0: none, 10: much) | 5.4 ± 1.1 | 7.0 ± 0.7 |
| Texture (0: firm, 10: loose) | 7.7 ± 0.8 | 3.4 ± 0.8[4] |
| Palatability (0: good, 10: bad) | 7.4 ± 0.6 | 8.2 ± 0.6 |

[1] $\bar{x}$ ± SEM. R, raw potato starch meal; S, pregelatinized potato starch meal.
[2–4] Significantly different from R (unpaired $t$ test, $df = 9$): [2]$P = 0.007$, [3]$P = 0.02$, [4]$P = 0.0004$.

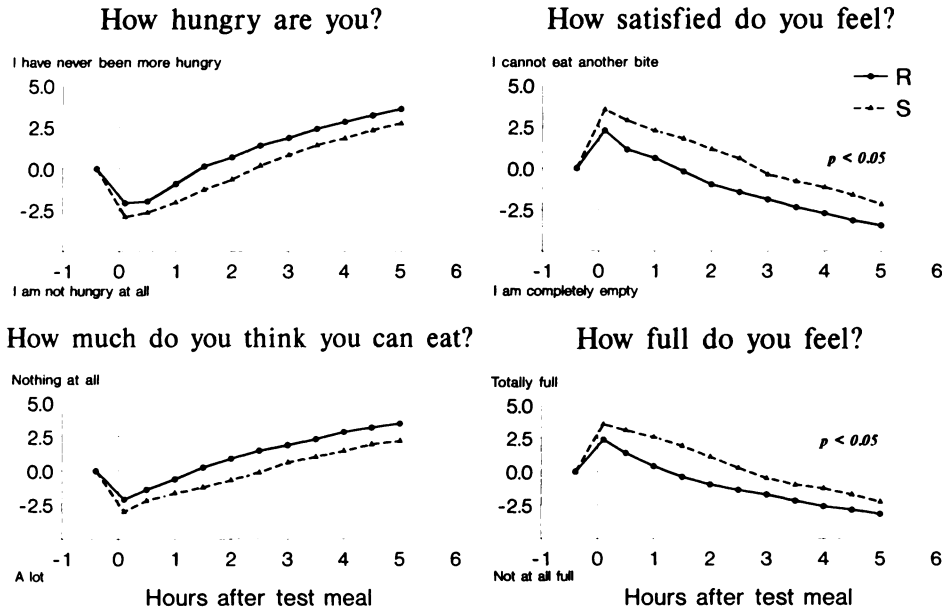


FIG 4. Subjective satiety scores after a raw potato starch meal (54% resistant starch [RS]) (R) and a pregelatinized potato starch meal (0% RS) (S) in 10 healthy, normal-weight, male subjects. All data are mean (±SEM), expressed as differences from fasting values. ANOVA: all (time effect: $F_{[11,198]}$, $P < 0.0001$); satiety (top right); diet effect: $F_{[1,18]} = 4.94$, $P = 0.04$; interaction between diet and time: $F_{[11,198]} = 1.95$, $P = 0.03$). Fullness (right bottom); interaction between diet and time: $F_{[11,198]} = 2.30$, $P = 0.012$).

some caution, and need to be confirmed before any further conclusions can be made.

Because the volume of the test meals and the time spent on consuming the meals on the 2 test days were similar, these factors cannot have influenced the satiating power of the two meals. However, the gelatinization of the S meal and the fact that it was a solid meal may have resulted in a reduction in gastric emptying rate (36) and in increased feelings of satiety and fullness after this meal compared with the liquid R meal. Also, the different ratings of taste, visual appeal, and texture may have influenced satiety and fullness ratings. In fact, positive correlations were found between aftertaste and satiety and fullness. This partly confirms the previous study by Hill et al (23) in which increased hunger was found with increased preference (palatability) of a meal. However, in their study there was no effect on fullness ratings of the meal preference (23).

The effect of RS on satiety shown in the present study may be considered a negative effect of RS with regard to appetite regulation and perhaps also weight control. Replacement of digestible starch with nondigestible starch in the diet may pose a risk of increasing the overall fat-to-carbohydrate ratio in the diet. Because a high-fat, low-carbohydrate diet has been shown to increase energy intake and body weight under ad libitum conditions, whereas a low-fat, high-carbohydrate diet has been shown to decrease spontaneous food intake and result in unexpected weight reductions (37–39), a replacement of digestible starch with RS may be undesirable in long-term weight regulation. Whether RS in a mixed meal has the same effect on appetite as in the present study remains, however, to be elucidated. Moreover, it is not possible to predict the effect on 24-h appetite sensations from the present 5-h results. The RS not digested in the small intestine will at a later stage, ie, 7–12 h after ingestion, be digested and fermented by bacteria in the large intestine, resulting in the production and uptake of short-chain fatty acids (5, 6, 40). Whether this may contribute to increasing satiety remains to be elucidated.

In conclusion, the intake of RS resulted in significantly lower postprandial plasma glucose, lactate, insulin, GIP, and GLP-1 responses and in a reduction of the satiating power compared with digestible starch. The changes in plasma glucose were not correlated with satiety or fullness, but correlations were found between GIP and satiety scores. Differences in texture and palatability of the test meals may also have influenced the subjective satiety ratings. Further studies are needed to clarify the effect of RS in a mixed meal, both acutely and long term.

We gratefully thank Lene Mellem, Kjas Densen, the laboratory technicians Bente Knap, Inge Timmermann, John Lind, Bente Mathiasen, and Karen Klausen; the dietitians Claudia Trentani and Caterina Mamini; and Inger-Lise Grønfeldt and her kitchen staff for expert technical assistance as well as Grethe Thorbek for consultive assistance.

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