Metabolic effects of digestible and partially indigestible cornstarch: a study in the absorptive and postabsorptive periods in healthy humans1,2

Lotfi Achour, Bernard Flourié, Françoise Briet, Claire Franchisseur, Francis Bornet, Martine Champ, Jean-Claude Rambaud, and Bernard Messing

ABSTRACT To compare the effects of digestible (pregelatinized) and partially indigestible (retrograded) cornstarches on some metabolic indexes, we studied eight healthy volunteers during two periods separated by 1 wk. In each period, fasting volunteers consumed at 0800 the test meal containing either the digestible or partially indigestible cornstarch; blood and breath were sampled in the absorptive period for 8 h. To study its late effects, the same test meal as that served at 0800 was given again at 2200, and blood and breath were sampled for 3 h in the postabsorptive period the next morning, i.e., 10 h after ingestion of the test meal. In the absorptive period, blood glucose and insulin were significantly higher after ingestion of digestible cornstarch than after partially indigestible cornstarch. In the postabsorptive period, concentrations of blood glucose, insulin, and fatty acids were not significantly different, whereas concentrations of blood acetate, breath hydrogen, methane, and 13CO2, and the respiratory quotient and satiety were significantly higher (P < 0.05) and concentrations of blood glycerol significantly lower (P < 0.05) after ingestion of partially indigestible cornstarch than after digestible cornstarch. We conclude that in healthy humans, digestion of partially indigestible cornstarch is slow in the small intestine and its colonic fermentation continues 10–13 h after its ingestion. Compared with pregelatinized cornstarch, the shift in starch digestion induced by retrogradation leads to a reduction in glycemic and insulminic responses in the absorptive period and in lipolysis in the postabsorptive period. Am J Clin Nutr 1997;66:1151–9.

KEY WORDS Digestible starch, partially indigestible starch, fermentation, metabolic responses, adults, insulin, glucose, colonic fermentation, short-chain fatty acids

INTRODUCTION

Until 1980, starch was thought to be completely degraded and absorbed in the small intestine by healthy humans (1). Since that time, several studies have shown that a small fraction of most dietary starches escapes α-amylase digestion in the small intestine and is subsequently fermented in the colon (2–5). It is now technologically possible to modify starch to slow down its digestion in the small intestine (6, 7). The digestion of technologically modified starch starts in the small intestine and continues in the colon, where its fermentation releases short-chain fatty acids (SCFAs) (acetate, propionate, and butyrate) and gases (hydrogen, carbon dioxide, and sometimes methane) (8). Diets containing resistant starch are useful models for studying the replacement of glucose absorption by SCFAs. Such replacement may alter liver and peripheral metabolism as well as hormonal response. For example, SCFAs may decrease hepatic cholesterol synthesis and improve carbohydrate tolerance (9, 10).

The aim of our study was to compare in healthy humans the metabolic responses to digestible and partially indigestible cornstarch, not only in the absorptive period but also in the postabsorptive period when fermentation takes place, i.e., to assess possible late effects.

SUBJECTS AND METHODS

Subjects
We studied eight healthy volunteers (two women and six men) with a mean age of 27 y (range: 18–35 y), weight of 64 kg (range: 47–90 kg), and body mass index (in kg/m2) of 22.4 (range: 19.6–26.9). None consumed a special diet, had a history of gastrointestinal or metabolic diseases, or had received recent treatment with antibiotics. Five subjects were methane producers as defined by breath methane concentrations > 2 ppm higher than in ambient air (11). All subjects gave written informed consent to the protocol, which was approved by the Lariboisière-Fernand-Widal-Saint-Lazare Hospital Ethics Committee.

Experimental design
Subjects participated in two 27-h test periods. For each period in both the morning (0800) and evening (1000) of the first day, subjects consumed the same mixed test meal (2643 kJ; 32%, 56%, and 12% from fat, carbohydrate, and protein, respectively) (Table 1). According to the period, this meal included either 50 g pregelatinized cornstarch (digestible cornstarch) or 50 g retrograded cornstarch (partially indigestible cornstarch).
Table 1

<table>
<thead>
<tr>
<th>Ingredients</th>
<th>Amount</th>
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</thead>
<tbody>
<tr>
<td>Hydrolyzed milk powder</td>
<td>30 g</td>
</tr>
<tr>
<td>Cheese</td>
<td>60 g</td>
</tr>
<tr>
<td>Sucrose</td>
<td>20 g</td>
</tr>
<tr>
<td>Tea or coffee</td>
<td>100 mL</td>
</tr>
<tr>
<td>Pregelatinized cornstarch or partially indigestible cornstarch</td>
<td>50 g</td>
</tr>
<tr>
<td>Water</td>
<td>200 mL</td>
</tr>
<tr>
<td>Energy(^2)</td>
<td>2643 kJ</td>
</tr>
</tbody>
</table>

\(^{1}\) The test meals provided 32% of energy as fat, 56% as carbohydrate, and 12% as protein.

\(^{2}\) The heat of combustion of starch is 17.5 kJ/g.

cornstarch) in addition to water, cheese, sucrose, hydrolyzed milk powder, tea, or coffee. The order in which the two periods were done was randomized, and there was a 1-wk interval between them. We used naturally \(^{13}\)C-enriched cornstarches. Pregelatinized cornstarch made from "normal" cornstarch was obtained from Cerestar (Vilvoorde, Belgium). Partially indigestible cornstarch (retrograded Hylon VII; Cerestar) was made from high-amyllose cornstarch (Hylon VII; National Starch, Bridgewater, NJ). The starch was first treated by extrusion cooking (10.5 kg dry starch in 13.5 L water, cooked at 170 °C and a screw speed of 250 rpm) and then cooled at 2 °C for 72 h. The product was manually broken into small pellets and dried at 60 °C in an air dryer for 2 h. This product was a reference material of the European Flair-Concerted Action (Euresta). Just before meal ingestion, 50 g of each starch and 20 g sucrose were mixed manually in 200 mL water. This resulted in a palatable porridge-like gel that was eaten with a spoon.

Subjects were asked to avoid alcohol consumption and exercise for 3 d before the experimental periods. On the day before each period, they consumed a low-residue dinner (300 g boiled rice, 100 g steak, and 1.5 g dietary fiber, with an energy content of 2412 kJ and 24%, 55%, and 21% of energy from fat, carbohydrate, and protein, respectively). On the morning of each period, a venous line was inserted in an antecubital vein for blood sampling. Fasting subjects were not allowed to smoke, and exercise was restricted during the study. After a 15-min rest, fasting blood and breath samples were taken, and basal respiratory exchanges were measured. At 0800, subjects consumed the test meal within 10 min. Blood and breath were sampled and respiratory exchanges were measured hourly for 8 h. This 8-h period was termed the absorptive period. At the end of measurements (1600), subjects consumed a low-residue standardized meal (100 g steak, 200 g boiled rice, three rusk, one orange, 4 g dietary fiber, and an energy content of 2575 kJ). The same test meal as that served at 0800 was given again at 2200. At 0800 on the next morning, ie, 10 h after the ingestion of the second test meal, blood and breath were sampled and respiratory exchanges were measured in the fasting subjects hourly for 3 h. This 3-h period (0800–1100 of day 2) was designated the postabsorptive period.

At sampling times, an end-expiratory breath sample for hydrogen and methane analysis was collected in a 50-mL syringe with a modified Haldane-Priestley tube and then subjects breathed for 3 min into a 50-L rubber bag. Three 10-mL aliquots were sampled from the bag for \(^{13}\)CO\(_2\) analysis. Respiratory exchange measurements were performed for 30 min under a ventilated hood connected to an open-circuit indirect calorimeter (MMC Beckman Horizon System, Beckman Instruments, San Diego). Subjective satiety ratings were obtained before and hourly during the absorptive and postabsorptive periods by using visual rating scales (from 0, extreme hunger, to 10, extreme satiety) according to the procedure of Hill et al (12). Urine was collected separately for five intervals: during the night before each test period (from 2000 to 0800) and during the test periods (from 0800 to 1600, from 1600 to 2200, from 2200 to 0800, and from 0800 to 1100). Urine volume was measured and aliquots were immediately frozen at -20 °C.

Analytic methods

Exhaled hydrogen and methane were measured by gas chromatography (Microlyser model DP; Quintron, Milwaukee). Exhaled \(^{13}\)CO\(_2\) was measured by gas chromatography interfaced with an isotope ratio mass spectrometer (GC-IRMS) (13). The measured \(^{8}\)\(^{13}\)C was transformed to \(^{13}\)C atom percent (AP) with the following formula (14):

\[
AP = \frac{100 \times R(0.001 \times ^{13}C + 1)}{1 + R(0.001 \times ^{13}C + 1)}
\]

where R is the ratio of \(^{13}\)C to \(^{12}\)C of the international standard Pee Dee Belemnite (PDB) (\(R = 0.0112372\)) and \(^{13}\)C the value of the sample. The calculated AP was then transformed into AP excess (APE) with the following formula:

\[
APE = AP_s - AP_B
\]

where AP\(_s\) is the sample and AP\(_B\) is the basal value (before ingestion). Finally, the rate of \(^{13}\)CO\(_2\) excreted in breath was calculated by multiplying the APE by the rate of carbon dioxide production, and was expressed as a percentage of the ingested dose at each time point.

Plasma samples were analyzed for glucose by using the glucose hexokinase method, for insulin by radioimmunoassay (Oris, Gif sur Yvette, France), for glyceraldehyde 3-phosphate dehydrogenase (Boehringer Mannheim, Meylan, France), and for fatty acids by colorimetry (NEFA c; Unipath, Dardilly, France).

Plasma acetate was measured by using the automated head space gas chromatography method (15). Plasma samples (0.5 mL) were first acidified by addition of 0.05 mL H\(_2\)SO\(_4\); methanol (0.25 mL) was then added to esterify acetate. Samples were heated for 30 min at 55 °C in the gas chromatography oven. Esterified acetate was analyzed by using a 30-m capillary column (FFAP, Quadrex; Touzart & Maignon, Vitry sur Seine, France) and a flame-ionization detector. The analytic range was 0.02–0.5 mmol/L and the within-run CVs did not exceed 5%.

Urinary C-peptide was measured by radioimmunoassay (Oris), creatinine by colorimetry (Boehringer Mannheim), and urinary nitrogen by the Kjeldahl method.

Calculations and statistical analysis

Data are presented as changes from basal fasting concentrations in the fasting blood sample collected on the first day of each period. Areas under the curves (AUCs) for 8 h in the absorptive period were calculated as the difference between the integrated area of the response curve and the rectangular area
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Absorptive period

Postabsorptive period

Breath $^{13}$CO$_2$ (% of ingested dose)

Breath hydrogen (ppm)

Breath methane (ppm)

0800 1000 1200 1400 1600

Day 1

Time

Day 2

FIGURE 1. Changes in breath excretion of $^{13}$CO$_2$, hydrogen, and methane (in the five methane-producing subjects) after ingestion of digestible (dotted line) and partially indigestible (solid line) cornstarch meals. Data are expressed as differences from basal fasting values. Absorptive period (ANOVA): breath $^{13}$CO$_2$ (meal effect: $P < 0.01$; time effect: $P < 0.01$; interaction between meal and time: $P < 0.01$), breath hydrogen (interaction between meal and time: $P < 0.01$), and breath methane (interaction between meal and time: $P < 0.05$). Postabsorptive period (ANOVA): breath $^{13}$CO$_2$ (meal effect: $P < 0.01$; time effect: $P < 0.01$) and breath hydrogen (meal effect: $P < 0.01$; time effect: $P < 0.05$; interaction between meal and time: $P < 0.05$).

determined by the basal values. AUCs of glycemia and insulinemia were calculated only for the first 3 h in the absorptive period.

Energy expenditure and substrate oxidation in the postprandial and postabsorptive periods were calculated according to standard equations (16–18) from the oxygen consumed ($\text{VO}_2$, in L/min), carbon dioxide produced ($\text{VCO}_2$, in L/min) and urinary nitrogen excreted (N, in g/min) as follows:

Energy expenditure = $15.91 \, \text{VCO}_2 + 5.21 \, \text{VCO}_2$

$- 4.65 \, N \quad (3)$

Carbohydrate oxidation = $4.57 \, \text{VCO}_2 - 3.23 \, \text{VO}_2$

$- 2.6 \, N \quad (4)$

Fat oxidation = $1.67(\text{VO}_2 - \text{VCO}_2) - 1.92 \, N \quad (5)$

In the postabsorptive state, carbohydrate oxidation was corrected as follows because it was assumed that glycogen was used as the carbohydrate energy source:

Carbohydrate oxidation = $4.12 \, \text{VCO}_2 - 2.91 \, \text{VO}_2$

$- 2.33 \, N \quad (6)$

Diet-induced thermogenesis was calculated over 8 h by subtracting resting energy expenditure from postprandial energy expenditure.

Results are expressed as means ± SEMs. AUCs in the absorptive period and fasting values on days 1 and 2 were compared by one-way within-subjects analysis of variance (ANOVA). Responses to the two test meals were also compared by two-way within-subjects ANOVA with meal and time as factors (19). When $F$ values were significant ($P < 0.05$) comparisons were made by using the Neuman-Keuls test.
RESULTS

Absorptive period

The excretion of $^{13}$CO$_2$ in breath increased more rapidly after the ingestion of digestible cornstarch than after partially indigestible cornstarch (Figure 1). The percentage of the ingested dose that was excreted over the 5 h after ingestion of digestible cornstarch was significantly higher than after ingestion of partially indigestible cornstarch (35 ± 3% compared with 28 ± 2%, meal effect and AUC, $P < 0.01$, Figure 1 and Table 2). The mean excretion of hydrogen and methane in breath did not show any increase after the ingestion of either starch (Figure 1). At the end of the absorptive period (8 h), the breath-hydrogen concentration was > 5 ppm over basal values in only one subject.

Blood glucose and insulin concentrations were significantly higher (meal effect and AUC, $P < 0.05$ and $P < 0.01$, respectively) after ingestion of digestible cornstarch than after partially indigestible cornstarch (Figure 2 and Table 2). Blood glycerol concentrations decreased after ingestion of digestible cornstarch and then rose linearly, whereas there was no marked variation after ingestion of partially indigestible cornstarch (Figure 3). Blood glycerol concentrations were significantly different after the two test meals (meal effect and AUC, $P < 0.05$; Figure 3 and Table 2). Blood fatty acids decreased after the two test meals and then increased up to values higher than basal values from 3 to 4 h after meals (Figure 3). Blood fatty acid curves were not different after the digestible and partially indigestible cornstarch meals. Blood acetate did not change within the first 3 h after the ingestion of starches. From the fourth hour, acetate increased in blood similarly after the digestible and partially indigestible cornstarch meals (Figure 3). After both meals, satiety sensations showed an early rise followed by a late decrease (Figure 4). The 8-h AUCs of satiety sensation were not significantly different between both meals (Table 2). However, by ANOVA satiety estimated for 5 h after the ingestion of the digestible cornstarch was significantly higher than after the partially indigestible cornstarch (meal-time interaction, $P < 0.05$; Figure 4).

A rise in energy expenditure was noted after ingestion of both meals. Energy expenditure was maximal 60 min after meal ingestion (5.0 ± 0.2 and 5.1 ± 0.2 kJ/min for digestible and partially indigestible cornstarch, respectively) (Figure 5). Diet-induced thermogenesis calculated over 8 h was 287 ± 31 and 233 ± 37 kJ after the digestible and partially indigestible cornstarch meals, respectively. These values were not significantly different.

Carbon dioxide production, oxygen consumption, and respiratory quotient were not significantly different after the digestible and partially indigestible cornstarch meals (Table 3 and Figure 5). The rate of carbohydrate oxidation reached a maximum value of $\sim$300 mg/min 1 h after ingestion of the meals and then decreased slowly to a basal value for the rest of the absorptive period. The rate of fat oxidation followed an inverse pattern. Neither carbohydrate nor fat oxidation were significantly different between the two starches (Figure 5). The net postprandial carbohydrate oxidation rate was 28.4 ± 9.7 and 35.9 ± 10.8 g/8 h after the digestible and partially indigestible cornstarch meals, respectively (Table 3). The net fat oxidation rate was $-5.3 ± 3.7$ and $-8.4 ± 3.7$ g/8 h after the digestible and partially indigestible cornstarch meals, respectively.

Urinary excretion of nitrogen, creatinine, and C-peptide were not different in the absorptive period after the ingestion of the digestible and partially indigestible cornstarch (data not shown).

Postabsorptive period

The excretion of $^{13}$CO$_2$ in breath was significantly higher (meal effect, $P < 0.01$) after ingestion on the previous evening of the partially indigestible cornstarch than after digestible cornstarch (Figure 1). Similarly, hydrogen and methane (in the five methane producers) excreted in breath were higher when the evening meal contained partially indigestible cornstarch than digestible cornstarch (Figure 1). In all subjects, breath-hydrogen concentrations 10–13 h after ingestion of partially indigestible cornstarch were $> 5$ ppm over the basal values measured the previous morning.

In each period the fasting concentrations of glucose, insulin, and fatty acids measured in the postabsorptive period on day 2 were not significantly different from the fasting values measured on day 1. The concentrations of blood glucose and insulin (Figure 2) and fatty acids (Figure 3) were not different after the ingestion of the digestible and partially indigestible cornstarch on the previous evening. Glycerol concentrations were significantly higher (meal effect, $P < 0.05$) after ingestion on the previous evening of the digestible cornstarch than after the partially indigestible cornstarch (Figure 3). When the previous meal contained partially indigestible cornstarch, satiety (Figure 4) and fasting blood acetate concentrations (Figure 3) were higher (meal effect, $P < 0.05$) than after the digestible cornstarch meal.

In the postabsorptive period, the respiratory quotient was significantly higher (meal effect, $P < 0.05$) after ingestion of partially indigestible cornstarch (Figure 5), and this was accounted for by an increase in carbon dioxide production.

Net postabsorptive carbohydrate oxidation rate was $-1.1 ± 3.1$ and $5.9 ± 3.0$ g/3 h and net fat oxidation rate was $-1.9 ± 1.1$ and $1.1 ± 0.9$ g/3 h after the ingestion of the digestible and

### Table 2

<table>
<thead>
<tr>
<th></th>
<th>Digestible cornstarch</th>
<th>Partially indigestible cornstarch</th>
</tr>
</thead>
<tbody>
<tr>
<td>Breath $^{13}$CO$_2$ (% of ingested dose)</td>
<td>35.3 ± 3.0</td>
<td>28.2 ± 1.8$^2$</td>
</tr>
<tr>
<td>Breath hydrogen (ppm · min/L)</td>
<td>3.0 ± 1.0</td>
<td>7.0 ± 2.5</td>
</tr>
<tr>
<td>Breath methane (ppm · min/L)</td>
<td>13.0 ± 2.6</td>
<td>11.0 ± 1.8</td>
</tr>
<tr>
<td>Blood glucose (mmol · min/L)</td>
<td>4.0 ± 0.8</td>
<td>2.3 ± 0.8$^4$</td>
</tr>
<tr>
<td>Blood insulin (μmol · min/L)</td>
<td>463 ± 55</td>
<td>277 ± 28$^2$</td>
</tr>
<tr>
<td>Blood fatty acids (μmol · min/L)</td>
<td>126 ± 433</td>
<td>7 ± 455</td>
</tr>
<tr>
<td>Blood glycerol (μmol · min/L)</td>
<td>$-207 ± 62$</td>
<td>38 ± 84$^4$</td>
</tr>
<tr>
<td>Blood acetate (μmol · min/L)</td>
<td>294 ± 102</td>
<td>298 ± 125</td>
</tr>
<tr>
<td>Satiety sensation</td>
<td>7.3 ± 6.8</td>
<td>$-3.2 ± 4.3$</td>
</tr>
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</table>

$^1$SEM.

$^2$Significantly different from digestible cornstarch: $^2P < 0.01$.

$^3$Significantly different from partially indigestible cornstarch: $^4P < 0.05$.

$^5$In the five methane-producing subjects.
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FIGURE 2. Changes in blood concentrations of glucose and insulin after ingestion of digestible (dotted line) and partially indigestible (solid line) cornstarch meals. Data are expressed as differences from basal fasting concentrations. Absorptive period (ANOVA): blood glucose (meal effect: \( P < 0.05 \); time effect: \( P < 0.01 \); interaction between meal and time: \( P < 0.01 \)) and blood insulin (meal effect: \( P < 0.01 \); time effect: \( P < 0.05 \); interaction between meal and time: \( P < 0.01 \)). Postabsorptive period (ANOVA): there were no significant differences for blood glucose and insulin for meal effect, time effect, and interaction between meal and time.

The use of a pregelatinized cornstarch, which is easily digested in the human small intestine (20), and a partially indigestible cornstarch. We have shown by the intubation technique that \( 51 \pm 2\% (x \pm SEM) \) of this partially indigestible cornstarch is not digested in the small intestine of healthy individuals, but is almost completely fermented in the colon because only small amounts of starch are excreted in stools (21). The slow digestion of partially indigestible cornstarch in the small intestine and its subsequent colonic fermentation were also noted in the present study. Indeed, compared with the ingestion of pregelatinized cornstarch, the increase in \( ^{13} \text{CO}_2 \) in breath was slower and the peak of \( ^{13} \text{CO}_2 \) excretion occurred later when partially indigestible cornstarch was ingested. Moreover, in the postabsorptive period 10–13 h after the ingestion of partially indigestible cornstarch, the amounts of \( ^{13} \text{CO}_2 \) exhaled in breath were higher than after the ingestion of pregelatinized cornstarch on the previous evening. The absence of an increase in breath hydrogen and methane after ingestion of pregelatinized cornstarch confirmed that this starch was completely digested in the small intestine. In contrast, breath-hydrogen excretion increased after ingestion of partially indigestible cornstarch from the eighth hour in one subject and later in the remaining subjects. This prolonged orocecal transit time agrees with previous determinations performed after ingestion of resistant starches (22–24).

In the present study, we aimed to assess the metabolic consequences of the shift in starch digestion. In addition to the changes expected in the absorptive period, and because fermentation of starch is a delayed and slow process, we wanted to determine the effects of partially indigestible cornstarch ingested the previous evening on metabolic indexes measured in the postabsorptive state the next morning.

In the absorptive period, ingestion of the meal containing partially indigestible cornstarch resulted in significantly lower glycemic and insulinemic responses than after ingestion of the same meal containing digestible cornstarch. This agrees with previous studies that showed lower glycemic and insulinemic responses to ingestion of resistant starch (25–28). Increasing the amount of partially indigestible cornstarch with a resultant decrease in the amount of absorbed carbohydrate also led to a reduction in the satiating power of the meal for 5 h in the absorptive period, as shown previously after ingestion of raw potato starch (29).
Blood fatty acid concentrations decreased after ingestion of both starches. From the fourth hour after ingestion of the meals, blood fatty acids increased up to values higher than the initial fasting concentrations. Blood glycerol followed the same pattern after ingestion of the digestible cornstarch. The rapid fall in the concentrations of fatty acids and glycerol after the ingestion of digestible cornstarch meal could be related to the inhibitory effect of insulin on lipolysis in the adipose tissue (30). After ingestion of partially indigestible cornstarch, blood glycerol remained roughly constant. A different response close to the significant level (P = 0.06) for blood glycerol was also noted by Raben et al (29) in healthy subjects after the ingestion of pregelatinized and raw potato starch. This could be explained by the reduced hepatic synthesis of triacylglycerol related to the lower insulin response to the partially indigestible cornstarch meal (31).

The time course of acetate in blood during the absorptive period was similar after the ingestion of both test meals. Blood acetate did not change for the first 3 h. It then rose and was still increased at the end of the absorptive period. Exogenous acetate is produced during the colonic fermentation of dietary residues whereas endogenous acetate is produced from the hepatic or peripheral metabolism of glucose and fatty acids (32, 33). In our study, hydrogen did not increase within 8 h after ingestion of both meals. This argues against the fermentative origin of blood acetate. Furthermore, the concomitant increase in acetate and fatty acids in blood from the fourth hour after the ingestion of both meals suggests that acetate originated from the oxidation of fatty acids. This was also indicated by respiratory gas exchanges, which showed that lipids were the main fuel oxidized from the fourth hour after both meals.

In the postabsorptive period, fasting blood concentrations of glucose, insulin, and fatty acids were at the same concentration as on the previous morning. They were not significantly different whether the digestible or partially indigestible cornstarch was ingested on the previous evening. Similarly, fasting glycerol and acetate concentrations after ingestion of digestible cornstarch were close to those measured on the previous morning. In contrast, after ingestion of partially indigestible cornstarch, blood glycerol was significantly lower and acetate significantly higher than after ingestion of digestible cornstarch. It is likely that additional acetate in blood originates from colonic

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**FIGURE 3.** Changes in blood concentrations of fatty acids, glycerol, and acetate after ingestion of digestible (dotted line) and partially indigestible (solid line) cornstarch meals. Data are expressed as differences from basal fasting concentrations. Absorptive period (ANOVA): all (time effect: P < 0.01), and blood glycerol (meal effect: P < 0.05; interaction between meal and time: P < 0.05). Postabsorptive period (ANOVA): blood glycerol (meal effect: P < 0.01), and acetate (meal effect: P < 0.05).
fermentation of partially indigestible cornstarch as supported by the concomitant increase in hydrogen and methane in breath. Acetate has no effect on glucose metabolism (34, 35). However, it may influence lipid metabolism. Indeed, acetate replaces fat as an oxidative fuel and has antilipolytic effects (34, 36, 37). Acetate inhibits glycerol release in rat adipocytes in culture (38) and reduces blood glycerol in humans (39). This could explain why fasting blood glycerol concentrations were lower after ingestion of partially indigestible cornstarch. Our results for fatty acids do not agree with those of Morand et al (40), who found in rats that resistant starch decreased blood fatty acid concentrations in the postabsorptive period compared with digestible starch. In addition, acetate (given orally, intravenously, or intrarectally) is known to reduce the normal rise in plasma fatty acid concentration related to the fasting state (39, 41–43). In our study, there was a trend toward reduced blood fatty acids (P = 0.10) after ingestion of partially indigestible cornstarch on the previous evening, and we cannot exclude the possibility that a significant effect would have been obtained with a larger number of volunteers.

In addition to acetate, butyrate and propionate are produced during the fermentation of starch. Butyrate is oxidized in the colonic mucosa, but propionate reaches the liver where it may affect glucose and lipid metabolism. In humans, propionate is associated with an improvement in carbohydrate tolerance and an increase in serum triacylglycerol (9, 10). This last effect could also account for the reduced amount of glycerol found in the postabsorptive period after the ingestion of partially indigestible cornstarch on the previous evening.

In the postabsorptive period, subjective scores of satiety were significantly higher after partially indigestible cornstarch than after digestible cornstarch. This delayed change in satiety is a priori not mediated through different palatability of ingested starches, rate of gastric fullness and emptying, plasma glucose, or hepatic glycogen stores. Whether the fermentation products of partially indigestible cornstarch may play a role in increasing satiety remains to be elucidated.

In the postabsorptive state, the respiratory quotient was higher after partially indigestible than after digestible cornstarch. The rise in respiratory quotient would indicate an increase in carbohydrate oxidation, which is quite unexpected and nonphysiologic in the postabsorptive state. In addition, oxygen consumption measured after ingestion of partially indigestible cornstarch on the previous evening was similar to that measured after ingestion of digestible cornstarch, and the changes in respiratory quotient after partially indigestible cornstarch were linked only to increased carbon dioxide production. The excess carbon dioxide may arise from the oxidation in the body of absorbed SCFAs and from carbon dioxide produced by bacteria in the colon (bacterial carbon dioxide). Ritz et al (35) as well as our group (44) found a significant increase in the respiratory quotient and carbon dioxide production during the fermentation of lactulose. In the later study the excretion of carbon dioxide in breath was strongly correlated with that of hydrogen, allowing us to correct the carbon dioxide production when the concomitant hydrogen concentration in breath was known. By using lactulose data from that study in the present study, bacterial carbon dioxide may be estimated from breath-hydrogen concentrations, and carbon dioxide production corrected by accounting for bacterial carbon dioxide. As a result, the respiratory quotient determined in the postabsorptive period after ingestion of partially indigestible cornstarch was no longer significantly different from the corresponding values measured after digestible cornstarch. Thus, the changes in the respiratory quotient and substrate oxidation observed in our study after ingestion of partially indigestible cornstarch are, at least in part, artificial and linked to the colonic fermentation of starch.

In conclusion, the substitution of a highly digestible cornstarch by a partially indigestible cornstarch leads to reduced glycemia, reduced insulinemia, reduced early satiety, and presumably reduced triacylglycerol synthesis in the absorptive period. To avoid uncontrolled factors, experiments were per-
formed in the postabsorptive period under conditions of sustained fasting. Under these conditions, acetate produced in the colon after ingestion of partially indigestible cornstarch re-

TABLE 3
Areas under the curve of oxygen consumption, carbon dioxide production, respiratory quotient, and substrate oxidation after ingestion of the digestible and partially indigestible cornstarch meals in the absorptive period†

<table>
<thead>
<tr>
<th></th>
<th>Digestible cornstarch</th>
<th>Partially indigestible cornstarch</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oxygen consumption (L • 8 h)</td>
<td>13.50 ± 1.71</td>
<td>13.22 ± 3.58</td>
</tr>
<tr>
<td>Carbon dioxide production (L • 8 h)</td>
<td>15.45 ± 2.07</td>
<td>17.08 ± 3.10</td>
</tr>
<tr>
<td>Respiratory quotient</td>
<td>0.28 ± 0.15</td>
<td>0.47 ± 0.17</td>
</tr>
<tr>
<td>Carbohydrate oxidation (g • 8 h)</td>
<td>28.39 ± 9.67</td>
<td>35.94 ± 10.84</td>
</tr>
<tr>
<td>Fat oxidation (g • 8 h)</td>
<td>−5.28 ± 3.66</td>
<td>−8.42 ± 3.69</td>
</tr>
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</table>

†± SEM.

places fatty acid oxidation, lipolysis is reduced, and satiety is increased. It may be expected that the ingestion of a breakfast during concomitant fermentation may alter some metabolic measures. Further studies will help to better define the postabsorptive metabolic consequences of resistant starch ingestion on the amount of food ingested at breakfast and on nutrient oxidation and metabolic responses to breakfast.

Overall, the acute effect of partially indigestible cornstarch on the glycemic and insulminemic responses shown in the present study in the absorptive period may be considered a positive effect with regard to glucose metabolism, whereas its effect on fat synthesis may appear to be counterbalanced by its antilipolytic action in the postabsorptive period. Likewise, with regard to weight control, the reduced lipolysis induced by partially indigestible cornstarch in the postabsorptive period may be counterbalanced by increased satiety. Further studies are needed to clarify the consequences of resistant starch consumption in the long term.
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REFERENCES


