Dietary Starch Composition and Level of Energy Intake Alter Nutrient Oxidation in “Carbohydrate-Sensitive” Men

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ABSTRACT The effect of dietary starch type on components of 24-h energy expenditure (total, sleep, exercise) were examined in 13 hyperinsulinemic and nine control men, aged 28–58 y. Subjects consumed products containing 70% amylopectin or 70% amylose cornstarch for two 14-wk periods in a crossover design. A 10-wk period of starch replacement in the subjects’ self-selected diets was followed by a 4-wk controlled feeding period at 100% maintenance energy intake; diets during the last 4 d of the controlled feeding period provided excess energy, i.e., 125% of maintenance energy. Data for insulin, glucose, 24-h energy expenditure and its components, respiratory quotient and nutrient oxidation were analyzed by ANOVA for mixed models. Although insulin and glucose responses to a starch tolerance test remained greater for hyperinsulinemic than for control subjects, both were reduced with high amylose consumption (P < 0.04). No component of energy expenditure was significantly affected by dietary starch or subject type. However, excess energy intake did increase metabolic energy expenditure (P < 0.0001). Protein oxidation increased with excess energy intake when subjects consumed the high amylopectin starch but did not increase in response to excess energy consumption when the high amylose diet was consumed, suggesting increased protein retention. The magnitude of the response in carbohydrate and fat oxidation was blunted in hyperinsulinemic subjects consuming excess levels of the amylose diet. This may be due to an improvement in overall insulin response or to a change in available substrates for oxidation resulting from microbial fermentation. J. Nutr. 126: 2120-2129, 1996.

INDEXING KEY WORDS:
• energy expenditure
• hyperinsulinemic men
• amylose starch
• fuel oxidation
• overfeed

About 15–18% of the American population is believed to be “carbohydrate-sensitive.” Individuals identified as “carbohydrate sensitive,” by the criteria of Reiser et al. (1981), are characterized by hyperinsulinemia, normoglycemia and hyperlipidemia. Insulin sensitivity, low relative metabolic rate, and low fat oxidation rate are considered to be metabolic predictors to weight gain (Ravussin et al. 1993, Zurlo et al. 1990). Weight gain occurs when energy intake exceeds energy expenditure and results in higher resting metabolic rates (Tremblay 1992), decreasing insulin sensitivity (Ravussin and Swinburn 1992) and blunted glucose-induced thermogenesis (Golay 1993, Tremblay 1992). Golay (1993) has also suggested that the lowered glucose-induced thermogenesis may result in postprandial sparing of energy. Energy expenditure and nutrient oxidation have not been well characterized in “carbohydrate-sensitive” subjects fed either adequate or excess levels of energy.

Implementation of proposed dietary recommendations by the U.S. Department of Agriculture (1990) would decrease fat intake in the U.S. diet to no more than 30% of total energy and increase carbohydrates, particularly complex and high fiber carbohydrates, to 55–60% of total energy. Dietary starches occur as mixtures of amylose (AM) and amylopectin (AP). Insulin and glucose response curves have been shown to differ.

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2 To whom correspondence and reprint requests should be addressed.
3 Abbreviations used: AM, amylose starch; AP, amylopectin starch; BMI, body mass index; C, control subjects or group; EEexerc, energy expenditure during the exercise period; EEsleep, energy expenditure during the sleep period; H, hyperinsulinemic subjects or group; LBM, lean body mass; MEI, metabolizable energy intake; OGTT, oral glucose tolerance test; RQ, respiratory quotient; 24EE, 24-h energy expenditure.

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in relation to the amylose/amylopectin content of rice [Goddard et al. 1984]. Behall and Howe (1995) have demonstrated decreases in the postprandial glucose and insulin levels of human subjects following a high amylose starch meal compared with high amylopectin starch. Although insulin is not intrinsically a thermoregulatory hormone, its interactions with thermoregulatory hormones, viz. catecholamines, and its direct effects on protein, glucose and lipid metabolism could be expected to influence energy expenditure and nutrient oxidation. Therefore, diet-induced changes in insulin response could also affect energy expenditure and nutrient oxidation.

The objectives of the current study were to determine energy expenditure in hyperinsulinemic men compared with control subjects, to determine the effect of excess energy intake on energy expenditure in this population and to examine the effect of changing the insulinogenic property of the diet (by varying the amylose/amylopectin starch ratio) on energy metabolism.

METHODS AND MATERIALS

Subjects for this study were recruited from the local area through advertising in newspapers, employee newsletters and fliers. Subjects taking drugs known to affect glucose, insulin, thyroid or lipid metabolism were excluded from the study. Of 34 male subjects interviewed, 29 volunteers were selected to participate in this study based on their response to a 2-h oral glucose tolerance test (OGTT; 1 g glucose/kg body wt). Two subjects were dropped for non-compliance, and three subjects withdrew for personal reasons. Twenty-four subjects completed the study.

Fourteen subjects were determined to be hyperinsulinemic (H) and normoglycemic [Reiser et al. 1981, Huttner 1991]. The remaining 10 subjects had normal insulin and glucose responses to a glucose load and served as the control (C) group. Serum insulin and glucose concentrations for the hyperinsulinemic and control subjects in response to the pre-study (screening) glucose tolerance test were shown in Table 1. Subjects ranged from 28 to 58 y of age, between 166 and 195 cm in height and weighed 54–114 kg. All medications taken by subjects during the study were recorded. The study protocol was approved by the Human Studies Committees of the U.S. Department of Agriculture and Georgetown University. Written informed consent was obtained from all volunteers prior to participation in the study.

The study consisted of two 14-wk periods in a crossover design. During the first 10 weeks of each period (starch replacement phase), subjects were given five research food products to replace their habitual starch sources in their self-selected diets. Subjects were provided with and instructed on the use of a starch/bread exchange list; subjects were told to consume only one starch source per day in addition to those provided. The research products were bread, muffins, cookies, cereal, and cheese puffs made with either 70% amylose (AM) or 70% amylopectin (AP) cornstarch as the primary carbohydrate. The quantities of research food products given to the subjects were calculated to provide 55% of carbohydrate energy as dietary starch. Estimates of total energy needs were initially based on metabolic body size plus an habitual activity factor, i.e., 420kJ/kg of body weight/75.7 kg (Stock and Rothwell 1982). Body weights were measured weekly throughout the study. Adjustments in energy intake were made if body weight varied by more than 2 kg. Research food products could be consumed at any time and in any order during the day. To encourage compliance, substitution of one product type for another was allowed. Subjects were instructed to return any uneaten products at the end of the week.

Subjects consumed a controlled diet prepared and weighed in the Beltsville Human Study Facility (BHSF) during the remaining 4 wk of each period. Subjects consumed breakfast and dinner at the BHSF on Monday through Friday; lunch and weekend or holiday meals were packaged for off-site consumption. The energy value of the control diet provided (7-d average) 15% of energy from protein, 34% from fat, and 51% from carbohydrate; 55% of the carbohydrate in the diet was from refined starches. The average daily cholesterol consumption was 410 mg. The ratio of polyunsaturated to saturated fat in the diet averaged 0.9. The starches and the analyses of the starch composition were obtained from American Maize-Product Company (Hammond, IN). The diets met the Recommended Dietary Allowance for all nutrients.

Due to experimental constraints in data collection during the controlled dietary phase, a group of four subjects were started on the study at the same time (two C and two H). Each starch (AM and AP) and subject type (C and H) was equally represented within each group, i.e., one C and one H were started on AM and the other C and H subjects were started on AP. There was a 2-wk break between diet periods.

During the third week of each controlled diet phase, fasting (12 h) subjects were given a starch meal of bread as a tolerance test (1 g carbohydrate/kg body wt). The bread for the test was taken from that day's allotment rather than as an addition to energy intake. The bread was mixed with an appropriate amount of water and blended into a slurry, which the subjects consumed through a straw. Blood was drawn prior to and at 0.5, 1, 2 and 3 h after the meal, centrifuged at 100 times g for 20 min at 4°C and stored at -80°C. Plasma glucose was analyzed by the hexokinase method [Trace America, Miami, FL] on an automated spectrophotometric system (Centrifichem, Baker Instruments Corp., Allentown, PA). Insulin concentrations in plasma were quantified by radioimmunoaasay using commercially available kits [ICN Biomedicals, Costa Mesa, CA].
Control sera were analyzed with every insulin and glucose assay. Areas under the curve for glucose and insulin were calculated as described by Wolever and Jenkins (1986).

In the week following the starch tolerance test, 24-h energy expenditure (24EE) was measured. The subjects were then overfed (125% of maintenance energy) for the next 3 d. On the fourth day of overfeeding, 24EE was again measured.

Twenty-four hour EE was determined by indirect calorimetry using the Beltsville room calorimeter. A complete description of the Beltsville room calorimeter was previously reported (Seale et al. 1990). Subjects entered the calorimeter at 0800 and remained for 23.5 h. Initial and final gas compositions of the air in the chamber were recorded. Data on gas composition and air flow were collected for the entire time the subject was in the chamber (Rumpler et al. 1990). While in the calorimeter, subjects followed an activity protocol similar to that reported by Rumpler et al. (1990). Scheduled activities included 7.5 h of sleep, 4 h of desk work, 30 min of exercise, and 1.5 h of meal consumption; Table 2 shows the 117MJ (2800 kcal) level of the menu used on calorimeter days. The calorimeter day meals provided 16% of total energy from protein, 34% from fat and 50% from carbohydrate and provided 507 mg/d of cholesterol. The remaining time was spent watching television, listening to radio, reading, or desk work. Subjects fasted overnight (12 h) in the calorimeter. The 23.5-h measured EE was corrected for differences in initial and final gas composition of the chamber and for excretion of urinary nitrogen, and then extrapolated to 24-h EE. Sleep EE (EEsleep) was measured during the hours of 0030–0530. The rate of EEsleep is an index of basal metabolic rate and was also extrapolated to 24 h. The EE during exercise (EEexercise) represents the subjects’ EE during one 30-min period on a bicycle ergometer minus the sleep rate of energy expenditure. Measurement of EEexercise was therefore not confounded by variation in basal metabolic rate as estimated by EEsleep.

The subjects were allowed to set their own exercise pace and tension on the bike. The exercise period was included in the activity protocols to provide subjects with some physical activity while in the chamber. The EEexercise values provide only an estimate of the level of activity exhibited by the subject only while in the chamber and are not an accurate assessment of the energy expenditure for a certain amount of exercise.

Energy expenditure was calculated using the Weir equation [Weir 1949]. Respiratory quotient (RQ) was calculated as the total 24-h production of carbon dioxide divided by the total 24-h consumption of oxygen. Rates of use of substrates (carbohydrate, protein and fat) were calculated using the equations described by Consolazio et al. (1963). These factors are derived from the daily production of carbon dioxide, consumption of oxygen and excretion of urinary nitrogen (AOAC 1980; A/SN Kjel-Foss automatic 16210 analyzer, Foss Electric, Hillerod, Denmark). The formulas of Weir (1949) and Consolazio et al. (1963) differ slightly in the underlying assumptions related to the oxygen consumption and carbon dioxide production related to substrate oxidation; however, results vary by <2%.

Before subjects entered the calorimeter, body weights of fasted subjects without clothing were determined with a Type E 1200 Balance (August Sauter, Ebingen, Germany). Heights were measured using a stadiometer (Perspective Enterprises, Kalamazoo, MI). Lean body mass (LBM) was determined from measurement of total body water by bioelectric impedance analysis (model BIA-101, RJL Systems, Detroit, MI) using the equations for males developed by Kushner and Schoeller (1986).

Insulin and glucose values were evaluated by repeated measures analysis, using the general linear model procedure of SAS (PCSAS, version 6.1, SAS Institute, Cary, NC). The model included sources of variation for among subjects, within subjects in different periods, and within subjects and periods for each insulin or glucose curve. The interactions of hour × diet,
TABLE 2

Typical study menu: 11.7 MJ calorimeter day

<table>
<thead>
<tr>
<th>Meal/Food Item</th>
<th>Weight</th>
<th>Meal/Food Item</th>
<th>Weight</th>
</tr>
</thead>
<tbody>
<tr>
<td>Breakfast</td>
<td></td>
<td>Lunch</td>
<td></td>
</tr>
<tr>
<td>Corn flakes</td>
<td>36</td>
<td>Chicken breast, roasted</td>
<td>45</td>
</tr>
<tr>
<td>Bran muffin</td>
<td>100</td>
<td>Imitation mayonnaise</td>
<td>15</td>
</tr>
<tr>
<td>Margarine, corn, regular</td>
<td>25</td>
<td>Lettuce, iceberg</td>
<td>25</td>
</tr>
<tr>
<td>Eggs, scrambled</td>
<td>49</td>
<td>Tomato</td>
<td>60</td>
</tr>
<tr>
<td>Jelly, regular</td>
<td>14</td>
<td>Bread</td>
<td>94</td>
</tr>
<tr>
<td>Milk, whole</td>
<td>246</td>
<td>Apple</td>
<td>189</td>
</tr>
<tr>
<td>Apple juice</td>
<td>120</td>
<td>Snack</td>
<td></td>
</tr>
<tr>
<td>Dinner</td>
<td></td>
<td>Cheese puffs</td>
<td>75</td>
</tr>
<tr>
<td>Pork, broiled</td>
<td>90</td>
<td>Cashews, oil-roasted</td>
<td>15</td>
</tr>
<tr>
<td>Beef gravy, canned</td>
<td>30</td>
<td>Peaches, packed in juice</td>
<td>140</td>
</tr>
<tr>
<td>Mushrooms, canned</td>
<td>20</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stuffing</td>
<td>154</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Broccoli, frozen</td>
<td>140</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lettuce</td>
<td>60</td>
<td></td>
<td></td>
</tr>
<tr>
<td>French dressing, regular</td>
<td>20</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Angel food cake</td>
<td>65</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Orange</td>
<td>166</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Millet, whole</td>
<td>246</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

1 Contained either high amylose or amylopectin starch; 1 of the carbohydrate content was derived from the experimental starch products.

RESULTS

Calorimeter data were obtained for 22 of the 24 subjects. Table 3 shows the physical characteristics of the 22 subjects who completed all aspects of the study. Although hyperinsulinemic subjects were heavier (P = 0.11) and had greater LBM (P = 0.19), percentage of body fat (P = 0.22) and BMI (P = 0.07), these components of body composition were not significantly different from those of the control group. One hyperinsulinemic subject and one control subject could be considered obese by BMI standards (BMI >30 kg/m²); four hyperinsulinemic and two control subjects would be considered obese if the criterion was percentage of body fat >25%.

Figure 1 shows the effect of a starch load on the serum insulin and glucose response of 10 control and 14 hyperinsulinemic subjects. Regardless of diet (P < 0.03), serum insulin response was significantly greater in hyperinsulinemic than in control subjects, whether expressed as a response curve or as area under the curve. Long-term consumption of amylose significantly reduced the insulin response of all subjects when compared with amylopectin starch (P < 0.02 for the response curve, P < 0.003 for area under the curve). The glucose response showed a pattern similar to that of insulin. Consumption of high amylose starch decreased glucose response significantly (P < 0.04) and was similar for both subject types. These data, as well as the effect of diet and subject type on serum cholesterol, triglyceride and lipoprotein levels, have been previously reported (Behall and Howe 1995).

Table 4 shows the MEI, energy expenditure, and 24-
Table 3
Physical characteristics of subjects

<table>
<thead>
<tr>
<th>Subject type</th>
<th>Age</th>
<th>Height</th>
<th>Weight</th>
<th>BMI&lt;sup&gt;1&lt;/sup&gt;</th>
<th>LBM&lt;sup&gt;2&lt;/sup&gt;</th>
<th>Body fat&lt;sup&gt;2&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>y</td>
<td>cm</td>
<td>kg</td>
<td>kg/m²</td>
<td>kg</td>
<td>%</td>
</tr>
<tr>
<td>Hyperinsulinemic</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>[n = 13]</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean ± SEM</td>
<td>40</td>
<td>177</td>
<td>83</td>
<td>26.4 ± 0.99</td>
<td>64 ± 2.1</td>
<td>21.6 ± 1.88</td>
</tr>
<tr>
<td>Control</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>[n = 9]</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean ± SEM</td>
<td>36</td>
<td>178</td>
<td>74</td>
<td>23.5 ± 1.18</td>
<td>60 ± 1.2</td>
<td>18 ± 2.37</td>
</tr>
<tr>
<td>Range</td>
<td>29–49</td>
<td>170–184</td>
<td>63–88</td>
<td>19.7–30.3</td>
<td>55–65</td>
<td>6.8–27.8</td>
</tr>
</tbody>
</table>

<sup>1</sup> Body mass index = weight/height²

<sup>2</sup> Lean body mass (LBM) determined from measurements of total body water by bioelectric impedance analysis using equations developed by Kushner and Schoeller (1986); % body fat = 100 − %LBM.

<sup>3</sup> No statistically significant differences were obtained for any variable (ANOVA).

h RQ for all subjects. The MEI was similar for all subjects, regardless of subject type or diet, and reflected the energy level of the diet. Metabolizable energy intake as a percentage of total energy intake was as follows [mean ± SEM]: 90 ± 0.3 and 88 ± 0.2 for C and H subjects consuming amylose, respectively, and 91 ± 0.2 and 90 ± 0.2 for C and H subjects fed amylopectin, respectively. There were no significant interactions of subject type, diet, and energy level on energy expenditure measurements [13 H and 9 C]. Twenty-four hour EE was not affected by diet or subject type, even after adjustment for body weight or LBM. Variance in 24EE was significantly reduced (P < 0.0001) by 75% or 67% following adjustment of the data for body weight or LBM, respectively.

The EE<sub>sleep</sub> was unaffected by diet or subject type. However, consumption of excess energy resulted in a significant increase [P < 0.0001] in EE<sub>sleep</sub>, regardless of subject type or diet. The EE<sub>sleep</sub> was significantly associated with body weight [P < 0.0001], which accounted for 73% of the variance among subjects. Lean body mass was also a significant contributor to the variance in EE<sub>sleep</sub>, but that relationship was affected by diet [P < 0.011]. The slope of the linear relationship between EE<sub>sleep</sub> and LBM was 94 kJ/kg LBM when the diet contained the high amylose starch and 130 kJ/kg LBM when subjects consumed the high amylopectin diet.

The EE<sub>exercise</sub> was unaffected by diet, energy level or subject type (Table 4), nor was body weight or BMI a significant contributor to the variance in EE<sub>exercise</sub>. However, the variance in EE<sub>exercise</sub> was significantly affected by the interaction of LBM and subject type. The slope of the linear relationship between EE<sub>exercise</sub> and LBM was −6.41 kJ/kg LBM for hyperinsulinemic subjects and 53.5 kJ/kg LBM for control subjects.

Respiratory quotient was unaffected by subject type or diet (Table 4). The RQ did increase [P < 0.02] with excess energy intake; RQ [mean ± SEM] was 0.836 ± 0.007 at maintenance energy level and 0.854 ± 0.007 at 25% energy excess (data combined across subjects and diet).

Protein oxidation (Fig. 2A) was significantly affected by the interaction of diet and energy level [P < 0.03]. Protein oxidation increased 25% [P < 0.01] in hyperinsulinemic subjects and 14% in control subjects when total dietary intake was increased by 25% and the dietary starch was amylopectin. Protein oxidation was greater when diets consumed at maintenance level contained amylose rather than amylopectin [P = 0.056]. However, protein oxidation did not increase with overfeeding when amylose was the carbohydrate source. Carbohydrate oxidation (Fig. 2B) was significantly increased [P < 0.005] with excess energy intake, whereas fat oxidation was reduced [P < 0.03; Fig. 2C]. Fat oxidation decreased 16–18% for all subjects overfed the high amylopectin diet and for control subjects overfed the amylose diet. However, fat oxidation decreased only 6% when hyperinsulinemic subjects were overfed the high amylose diet. Substrate oxidation did not differ with subject type per se.

**DISCUSSION**

Starch products high in amylose content have been reported to decrease postprandial insulin and glucose responses to a meal (Goddard et al. 1984), van Amelsvoort and Weststrate (1992). All subjects in this study had significantly reduced insulin responses following a high amylose starch meal, when compared with an amylopectin load. However, the significantly lower [P < 0.04] blood glucose area following the high amylose starch meal may indicate that the lowered insulin response was due to slower and less complete digestion of the amylose starch. Van Amelsvoort and Weststrate (1992) fed 24 normal, non-obese, male subjects starch
meals containing either no amylose or an amylose:amylopectin ratio of 0.82. These investigators (van Amelsvoort and Weststrate 1992) also observed a reduced insulin response to high amylose consumption, but noted a slightly higher level of blood glucose during the latter parts of the tolerance test. The higher blood glucose levels reported by van Amelsvoort and Weststrate (1992), compared with those in the present study, may have been due to several reasons: the time frame for the tolerance test used to calculate the area under the curve was longer (6 h); the mixed meals used in the test contained other food products that could have contributed to the tolerance results; the amylose:amylopectin ratio of the test meal was lower (45:55) than in the current study (70:30), which may have resulted in greater starch digestion and absorption. In fact, these
investigators reported that the expected increase in
breath hydrogen following the high amylose:amylopectin meal was not observed (van Amelsvoort and Weststrate 1992). Data for the current study show that
breath hydrogen expired was significantly higher after
consuming the amylose meal (1 g carbohydrate/kg body
wt) than after an amylopectin load (P < 0.001) and that
amylose starch was indeed digested more slowly than amylopectin and therefore available for bacterial digestion.

Both 24EE and EE_{sleep}, an indicator of basal metabolic rate, were similar for all subjects. Long-term consumption of high amylose starch had no effect on either total energy expenditure or basal energy expenditure, per se. Although postprandial insulin response was decreased with high amylose consumption, there was no measurable impact on energy expenditure.

Because energy expenditure is affected by factors such as body size, sex, age and physical activity (Weinsier et al. 1992), energy expenditure data are frequently adjusted by body composition. Energy expenditure was slightly higher in the hyperinsulinemic subjects than in the controls, reflecting their slightly larger size, but was slightly lower (H < C) after adjustment for body weight or LBM. Weinsier et al. (1992) reported that energy expenditure adjusted for weight decreases with increased body fat. Furthermore, energy expenditure was somewhat lower (nonsignificant) for hyperinsulinemic subjects during the exercise period when compared with the control subjects; this may also be a reflection of their larger body size. Heavier people tend to be less active than lighter people (Tryon et al. 1992).

Total energy expenditure was increased slightly (1–3%) when subjects consumed 125% of maintenance requirements for 4 d. Dauncey (1980) reported increases in 24EE (8%) and basal energy expenditure (12%) within 1 d when subjects consumed 65% excess energy as a mixed diet of fixed composition (16% from protein, 34% from carbohydrate and 50% from fat). Excess energy consumption did significantly increase EE_{sleep} (P < 0.0001) for all groups in the present study from 8% to 15%, which confirms the findings of Dauncey (1980). No apparent relationship to subject type or diet was observed.

Respiratory quotient, an index of carbohydrate to fat oxidation, was unaffected by subject type or diet, but did rise in response to excess energy intake (P < 0.06). This rise reflected an overall increase in carbohydrate oxidation (P < 0.005) with overfeeding. Fat oxidation, which is inversely correlated to carbohydrate oxidation (van Amelsvoort and Weststrate 1992), was reduced during the periods of excess energy intake (P < 0.03).

The overall function of many endocrine and enzymatic regulators is to adjust carbohydrate oxidation to carbohydrate intake (Flatt 1991) and to maintain carbohydrate balance, not fat balance (Zurlo et al. 1990). Fat intake does not exert much influence on fat oxidation in the short term (Flatt 1991). Maintenance of fat balance differs greatly from that of carbohydrate balance (Schutz et al. 1989). In a study of 11 lean and 10 obese subjects, Thomas et al. (1992) examined the effect of feeding high carbohydrate or high fat diets on 24EE and resting metabolic rate. Obese subjects oxidized more carbohydrate and less fat than their lean counterparts.

| Table 4 |
|------------------|------------------|------------------|------------------|
| **Effect of dietary starch and energy level on metabolizable energy intake and components of 24-h energy expenditure in hyperinsulinemic and control men** | **Amylose diet** | **Amylopectin diet** |
| **Control subjects** | **Hyperinsulinemic** | **Control subjects** | **Hyperinsulinemic** |
| **M, E/d** | **M, E** | **M, E** | **M, E** |
| MEL, M/d | 11.61 ± 0.69 | 14.29 ± 0.69 | 11.39 ± 0.58 | 14.3 ± 0.57 |
| 24EE, M/d | 10.31 ± 0.49 | 10.46 ± 0.49 | 10.54 ± 0.48 | 10.77 ± 0.40 |
| EE_{sleep}, ** | 6.93 ± 0.33 | 7.39 ± 0.33 | 7.16 ± 0.27 | 8.01 ± 0.27 |
| M, E/d | 640 ± 129 | 719 ± 129 | 540 ± 113 | 466 ± 109 |
| RQ* | 0.841 ± 0.012 | 0.861 ± 0.012 | 0.835 ± 0.011 | 0.842 ± 0.010 |

1 Values are least squares means ± SEM for 13 hyperinsulinemic and 9 control men; 1 kcal = 4.184 kJ.
2 Abbreviations used: M, maintenance energy requirement; E, 25% excess energy intake in relation to maintenance level; MEL, metabolizable energy intake; 24EE, total 24-h energy expenditure; RQ, respiratory quotient = CO2/O2; EE_{sleep}, the rate of energy expenditure during sleep (1230–1730) extrapolated to 24 h as an index of basal energy expenditure; EE_{exercise}, the energy expenditure during a 30-min exercise period on a bicycle ergometer; LBM, lean body mass determined from measurement of total body water by bioelectric impedance analysis using equations developed by Kushner and Schoeller (1986).
3 Significant effect of energy level (ANOVA): *P < 0.02; "P < 0.0001.
DIETARY STARCH, HIGH FOOD INTAKE AND NUTRIENT OXIDATION

Figure 2 Effect of diet and energy intake on protein oxidation (A), carbohydrate oxidation (B) and fat oxidation (C) in 13 hyperinsulinemic and 9 control men. Values are least squares means and standard errors. H = hyperinsulinemic subjects, C = control subjects. Carbohydrate oxidation (B) was significantly increased during overfeeding ($P < 0.005$); fat oxidation (B) decreased ($P < 0.03$) during overfeeding. Protein oxidation was affected by the interaction of diet and energy level ($P < 0.003$); protein oxidation increased ($P < 0.009$) with overfeeding when the amylopectin diet was consumed, but was unaffected when amylose was the carbohydrate source.

[Thomas et al. 1992]. These investigators (Thomas et al. 1992) suggested that the greater insulin sensitivity of lean subjects may be the key to adjusting nutrient oxidation to nutrient intake. Thomas and co-workers (1992) proposed that reduced insulin sensitivity would require more circulating insulin to maintain a high level of carbohydrate oxidation and that increased insulin circulation may inhibit or limit lipolysis and subsequently fat oxidation. Although the postprandial insulin response of subjects while in an overfed state was not determined in this study, the magnitude of change in carbohydrate and fat oxidation in response to excess energy intake was blunted in hyperinsulinemic subjects consuming the amylose diet. This could be due to a change in overall insulin response.

The breath hydrogen data from this study (Behall and Howe 1996) indicate that amylose starch was digested less completely than amylopectin and subjected to bacterial fermentation, particularly in hyperinsulinemic subjects. The short-chain fatty acids resulting from fermentation would then be available for absorption and could provide substrates for oxidation. The difference in carbohydrate and fat oxidation observed in hyperinsulinemic subjects could be due to the relative production of these short-chain fatty acids. For example, increased acetate levels would favor fat storage due to its antilipolytic and lipogenic effects, whereas propionate is gluconeogenic in nature. Furthermore, hyperinsulinemic subjects may metabolize these short-chain fatty acids differently than do the control subjects. Impaired acetate metabolism has been demonstrated in persons with noninsulin-dependent diabetes (Wolever 1995). Moreover, it has been hypothesized that acetate overproduction in diabetes is due to enhanced fat oxidation (Wolever 1995).

As expected, protein oxidation increased with increased food consumption, but only when amylopectin was the principal carbohydrate. If one assumes that insulin response would be directly related to the size of a starch load, then an increase in protein retention might result. Insulin has been shown to affect protein turnover by reducing the rate of protein degradation, resulting in net protein retention (McHardy et al. 1991, Melville et al. 1989). Indeed, control subjects oxidized only 14% more protein when presented with a 25% increase in intake. However, the hyperinsulinemic subjects seem to have oxidized all the excess intake. Perhaps insulin had reached a maximum response and no further increase in insulin occurred with added challenge, or perhaps these results simply reflect the insulin insensitivity of hyperinsulinemic subjects.

Only part of the slight increase in protein oxidation observed (10 g/d) for both control and hyperinsulinemic subjects fed diets high in amylose (as compared with amylopectin at maintenance levels) can be explained by the protein content of the diets. Protein intake was 96 g/d when subjects consumed the amylopectin diet and 100 g/d when they consumed the amylose diet. Insulin was stimulated to a lesser extent following intake of starch products containing amylose rather than amylopectin, which could also contribute to lower protein retention. Increasing food intake to 125% of maintenance energy did not result in increased protein ox-
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diation when subjects were consuming the amylose diets. If one assumes a corresponding increase in insulin, then a reduced rate of protein degradation could occur with a resulting decrease in protein oxidation. Normalization of insulin sensitivity with high amylose intake might also contribute to protein retention. A substantial increase in lower gut fermentation, as indicated by increased breath hydrogen expiration (Behall and Howe 1996), could also lead to loss of nitrogen as microbial protein in feces.

In summary, energy expenditure in “carbohydrate-sensitive” men was similar to that of control subjects and was not affected by the type of starch in the diet. However, foods high in amylose were not as insulinogetic as foods containing amylopectin. When subjects were consuming excess energy, amylose starch did significantly affect nutrient oxidation. Protein oxidation did not increase in response to excess energy consumption when the high amylose diet was consumed, which suggests an increase in protein retention. Although reduced postprandial insulin response to amylose did not significantly affect energy expenditure, it may have been a factor in moderating protein turnover. Furthermore, the blunted response of carbohydrate and fat oxidation in hyperinsulinemic subjects to excess levels of the amylose diet may reflect a normalization of insulin response due to incomplete starch digestion, or a change in available substrates for oxidation resulting from microbial fermentation, i.e., absorption of short-chain fatty acids.

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LITERATURE CITED


