A Soybean Peptide Isolate Diet Promotes Postprandial Carbohydrate Oxidation and Energy Expenditure in Type II Diabetic Mice

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ABSTRACT The aim of the present study was to determine the effects of dietary proteins on the oxidation of dietary carbohydrate and lipids in type II diabetic mice. KK-A'Y strain mice were provided free access to a high fat diet (30% of energy as fat) for an initial 4-wk period to induce diabetes. To reduce body weight gain, the mice were subsequently fed restrictive isocaloric and isonitrogenous diets (35% of energy as protein and 5% as fat) based on either casein or soy protein isolate hydrolysate (SPI-H) for 4 wk. To measure exogenous carbohydrate and lipid oxidation, the mice were fed a diet containing \(^{13}\text{C}\)-glucose or \(^{13}\text{C}\)-triolein while they were in a respiratory chamber for 72 h. Postprandial energy expenditure was higher in the SPI-H than in the casein group; this difference was due to an increase in postprandial exogenous and endogenous carbohydrate oxidation. There were no differences in 24-h energy expenditure between dietary groups. Oxidation of exogenous carbohydrate tended to be higher (\(P = 0.054\)) in the SPI-H group during the 24 h of measurement. Fecal excretion of \(^{13}\text{C}\)-glucose was lower but the excretion of lipid was higher in mice fed the SPI-H diet than in casein-fed mice. These results indicate that in type II diabetic mice, dietary SPI-H not only inhibits the absorption of dietary lipids and increases the absorption of dietary carbohydrates but also augments postprandial energy expenditure, which is accompanied by a postprandial increase in oxidation of dietary carbohydrates. J. Nutr. 133: 752–757, 2003.

KEY WORDS: KK-A' mice, energy restriction • diabetes • \(^{13}\text{C}\) • respiratory quotient

In obese people, fat-reduced diets are effective in inducing weight loss (1) and in preventing weight gain after weight loss (2). As a substitute for a high fat diet, a high protein diet has several advantages over a high carbohydrate diet because it promotes both satiety (3) and diet-induced thermogenesis (4). Except for the study by Mikkelsen et al. (5), there appears to be no detailed description of the effects of different dietary proteins on 24-h energy expenditure. Mikkelsen et al. (5) reported that the animal protein in pork meat produced higher 24-h energy expenditure than did the vegetable protein in soy, whereas soybean protein is generally considered an antiobesity dietary protein.

Soybean protein may be suitable as a protein source for preventing obesity and diabetes because long-term feeding of soy protein induces weight loss in obese (6–8) and in KK-A'Y mice (9,10), it has a hypocholesterolemic effect compared with animal proteins such as casein (11–13) and it markedly decreases hepatic mRNA concentration and activity of lipogenic enzymes (14–17).

To our knowledge, the effect of soybean protein on substrate oxidation has not been investigated. The present study was designed to investigate, by using indirect calorimetry, the effect of dietary protein on energy expenditure in genetically obese type II diabetic mice. We also fed mice diets containing \(^{13}\text{C}\)-labeled glucose or \(^{13}\text{C}\)-triolein to quantify the oxidation of dietary carbohydrate and lipid, and to measure the fecal excretion of these substances.

MATERIALS AND METHODS

Materials. \(\text{D-Glucose (U-}^{13}\text{C}_6, 99\%+\)) and triolein (1,1,1-\(^{13}\text{C}_3\), 99%) were purchased from Cambridge Isotope Laboratories (MA).

Animals and diets. Animals and feeding regimens in the present study were used as previously described by Aoyama et al. (9). Male KK-A' mice (\(n = 32\); 6 wk old) weighing 26–30 g were purchased from Nihon Clea (Tokyo, Japan). Mice were housed individually in plastic cages under a controlled atmosphere (temperature, 23°C ± 1; humidity, 55 ± 5%; light, 0700–1900 h). All mice were treated in accordance with the NIH Guide for the Care and Use of Laboratory Animals. Mice were fed a high fat diet, containing 30% of energy as fat, to induce obesity for 28 d before the start of experiment. Four extremely obese and another four lean mice were removed from the study, and the remaining 24 mice were randomly assigned to two groups matched by body weight. To reduce body weight gain over the next 4 wk (d 29–57), the mice were fed restrictively isocaloric and isonitrogenous diets (35% of energy as protein and 5% as fat) based on either high protein or casein diet.
casein or soy protein isolate hydrolysate (SPI-H). Foods were provided at 1700 h and left for 24 h every day. Food intake and body weights were recorded daily. Table 1 shows the composition of the experimental diets. Nitrogen content was determined by the Kjeldahl method (9). SPI-H was prepared by hydrolyzing soybean protein isolate (SPI) with protease from Bacillus subtilis (Hynute-D1; Fuji Oil, Osaka, Japan); the average peptide chain length was five to six. SPI-H is superior to SPI in solubility and taste, and is as effective as SPI in counteracting obesity (9).

**Experimental design and computations of substrate oxidation.**

The aim of the study was to compare the effects on 24-h nutrient oxidation of two diets (the SPI-H and casein diets) used in conjunction with energy restriction in type 2 diabetic mice. Each group of mice was housed in the metabolic chambers for 72 h, from 1700 h on d 53 to 1700 h on d 56. Diets [77.4 kJ/(mouse · d)] were provided at 1700 h and left for 24 h every day. During the first 24 h of measurement (d 53–54), mice were given the diet containing unlabeled carbohydrate and lipid, which was the same in composition and amount as the diet fed during the 24 d of energy restriction. For the second 24 h of measurement (d 54–55), half of the mice in each group were fed a diet containing 13C-triolate (3 g/kg diet) instead of sucrose to measure the oxidation of exogenous carbohydrate. To measure oxidation of exogenous lipid, the remaining mice in each group were fed a diet containing 13C-triolate (3 g/kg diet) instead of soybean oil. During the final 24 h of measurement (d 55–56), all mice were fed the unlabeled diets.

The oxidation of total glucose, lipid, exogenous glucose and exogenous lipid was computed from oxygen consumption (V02), carbon dioxide production (VCO2) and the ratio of breath 14CO2/13CO2. Gas analysis was performed using an open-circuit metabolic gas analysis system connected directly to a mass spectrometer (Model RL-600; ArcoSystem, Chiba, Japan). The gas analysis system is described in detail elsewhere (18,19). Briefly, each metabolic chamber had a 125.4 cm2 floor and was 6.5 cm in height. Room air was pumped through the chambers at a rate of 0.5 L/min. Expired air was dried in a cotton thin column and then directed to an O2/CO2 analyzer, which used mass spectrometry.

From the volume of CO2 production per unit time (L/min; VCO2) and V02, total glucose and lipid oxidation were calculated using the stoichiometric equations of Frayn (20) as follows:

$$\text{Exogenous carbohydrate or lipid} = \frac{\text{VCO}_2 \times (\text{Rexp} - \text{Rref})/(\text{Rexo} - \text{Rref})}{k}$$

where VCO2 (not corrected for protein oxidation) is in L/min, Rexp is the observed isotopic composition of expired CO2 before the mice were fed the diet containing 13C, Rexo is the isotopic composition of exogenous diet containing 13C and k (0.7426 L/g) is the volume of CO2 provided by the complete oxidation of glucose (21). Endogenous oxidation of carbohydrate or lipids was calculated by subtracting exogenous from total carbohydrate oxidation as assessed by indirect calorimetry.

**Fecal excretion of triglycerides and analysis of 13C in feces.**

Feces were collected from metabolic chambers every day and were analyzed for lipids as described previously (22,23). Briefly, fecal lipids were extracted and separated, and a known weight of powdered feces was extracted in ice-cold chloroform and methanol (2:1, v/v) for 19 h at 4°C. After centrifugation at 900 × g for 10 min, the supernatant was collected and dried at 75°C. The residue was then saponified by heating at 75°C for 45 min in 5% KOH in 95% ethanol. Triacylglycerol content was then determined by measuring free fatty acids by a commercial kit (NEFA C, Wako, Osaka, Japan). Absorption of 13C-glucose was calculated from the concentration of 13C in feces, which was analyzed by NipponSanso (Kawasaki, Japan).

**Statistical analyses.**

Data are presented as means ± SEM. All statistical analyses were performed using StatView version 4.5 (SAS Institute, Cary, NC). Comparisons between two dietary groups were evaluated by Student’s t test. Differences were considered significant at P < 0.05.

**RESULTS**

**Energy intake and body weight.**

Energy intake of mice consuming the high fat diet was 126 ± 0.1 kJ/d. During the

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**TABLE 1**

**Composition of experimental diets**

<table>
<thead>
<tr>
<th>Ingredients</th>
<th>High-fat</th>
<th>Casein</th>
<th>SPI-H</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>g/kg</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Commercial feed2</td>
<td>600</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Casein3</td>
<td>391</td>
<td></td>
<td>404</td>
</tr>
<tr>
<td>SPI-H4</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Corn starch3</td>
<td>362</td>
<td></td>
<td>349</td>
</tr>
<tr>
<td>Sucrose3</td>
<td>100</td>
<td></td>
<td>100</td>
</tr>
<tr>
<td>Shortening5</td>
<td>250</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Condensed milk6</td>
<td>150</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Soybean oil7</td>
<td>50</td>
<td></td>
<td>50</td>
</tr>
<tr>
<td>Cellulose powder3</td>
<td>50</td>
<td></td>
<td>50</td>
</tr>
<tr>
<td>Mineral mixture8</td>
<td>35</td>
<td></td>
<td>35</td>
</tr>
<tr>
<td>Vitamin mixture8</td>
<td>10</td>
<td></td>
<td>10</td>
</tr>
<tr>
<td>Choline bitartrate7</td>
<td>2</td>
<td></td>
<td>2</td>
</tr>
</tbody>
</table>

1 SPI-H, soybean protein hydrolysate.
2 Powdered, CRF-1 Oriental Yeast Co., Tokyo, Japan.
3 Oriental Yeast Co., Tokyo, Japan.
4 Hinute-D1, Fuji Oil Co., Osaka, Japan. Free amino acid content, 0.8%.
5 Panpas Deluxe, Fuji Oil Co., Osaka, Japan.
6 Morinaga Milk Co., Tokyo, Japan.
7 Wako Pure Chemical Industries, Ltd., Osaka, Japan.
8 AIN-76 mixtures, Oriental Yeast Co., Tokyo, Japan.

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**TABLE 2**

**SPI-H diet isoflavones**

<table>
<thead>
<tr>
<th>Isoflavone</th>
<th>mg/100 g protein</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total daidzin2</td>
<td>75.8</td>
</tr>
<tr>
<td>Total genistin3</td>
<td>144.1</td>
</tr>
<tr>
<td>Total glycitin4</td>
<td>7.2</td>
</tr>
<tr>
<td>Total isoflavones</td>
<td>227</td>
</tr>
<tr>
<td>Conjugated5</td>
<td></td>
</tr>
<tr>
<td>Malonyl6</td>
<td>29.9</td>
</tr>
<tr>
<td>Acetyl7</td>
<td>50.8</td>
</tr>
<tr>
<td>Aglycones8</td>
<td>9.7</td>
</tr>
</tbody>
</table>

1 SPI-H, soybean protein hydrolysate.
2 Powdered, CRF-1 Oriental Yeast Co., Tokyo, Japan.
3 Oriental Yeast Co., Tokyo, Japan.
4 Hinute-D1, Fuji Oil Co., Osaka, Japan.
5 Panpas Deluxe, Fuji Oil Co., Osaka, Japan.
6 Morinaga Milk Co., Tokyo, Japan.
7 Wako Pure Chemical Industries, Ltd., Osaka, Japan.
8 AIN-76 mixtures, Oriental Yeast Co., Tokyo, Japan.

Glucose oxidation = 4.55 VCO2 – 3.21 V02, and

Lipid oxidation = 1.67 V02 – 1.67 VCO2

The isotopic composition was expressed as the 13C/12C ratio in the sample. The oxidation rate of exogenous carbohydrate and lipid, in g/min, was computed as follows (21):

Exogenous carbohydrate or lipid

$$= \frac{\text{VCO}_2 \times (\text{Rexp} - \text{Rref})/(\text{Rexo} - \text{Rref})}{k}$$

where VCO2 (not corrected for protein oxidation) is in L/min, Rexp is the observed isotopic composition of expired CO2 before the mice were fed the diet containing 13C, Rexo is the isotopic composition of exogenous diet containing 13C and k (0.7426 L/g) is the volume of CO2 provided by the complete oxidation of glucose (21). Endogenous oxidation of carbohydrate or lipids was calculated by subtracting exogenous from total carbohydrate oxidation as assessed by indirect calorimetry.
A 4-wk period of food restriction, intake was ~80% of that during the ad libitum consumption period and did not differ in mice fed the SPI-H and casein diets (103 ± 2.4 and 105 ± 5.3 kJ/d). During the 4 wk of consuming the high fat diet, mice gained from 28.7 ± 0.1 to 46.9 ± 0.4 g. During the 4 wk of food restriction, body weight in the SPI-H and casein-fed mice decreased to 40.4 ± 0.49 and 40.6 ± 0.4 g, respectively.

There were few changes in body weight during the 72 h of respiratory measurement, and no significant differences in body weight between dietary groups during all experimental periods. Kidney weights were significantly lower in the SPI-H group compared with the casein group (502 ± 34 and 466 ± 35 mg, P < 0.05). The weights of other tissues such as the epididymal, inguinal and perirenal fat pads, brown adipose tissue, gastrocnemius and quadriceps skeletal muscle, liver, heart and spleen did not differ (data not shown).

Changes in energy expenditure after SPI-H feeding. Postprandial energy expenditure, measured after every meal at 1700 h, was 1–8% higher in mice fed the SPI-H diet than in those fed the casein diet. This augmentation of energy expenditure was equivalent to ~25% of resting metabolic rate (RMR). The difference in postprandial energy expenditure between the SPI-H and casein groups was ~8–12 J/min, and the lowest energy expenditure fluctuated between 31 and 38 J/min from 1000 to 1700 h (Fig. 1).

Contribution of carbohydrate oxidation to energy expenditure. Most of the increase in postprandial energy expenditure was due to the increase in postprandial carbohydrate oxidation, which was 8% higher in mice fed the SPI-H diet than in those fed the casein diet; the difference between the SPI-H and casein groups was ~13 J/min (Fig. 2). Although postprandial lipid oxidation was lower in mice fed SPI-H than in casein-fed mice (Fig. 3), the difference in postprandial oxidation of lipid was negligible compared with the increase in postprandial carbohydrate oxidation in both dietary groups.

Oxidation of dietary carbohydrate. Postprandial oxidation of dietary carbohydrate was 30% higher in mice fed the SPI-H diet than in those fed the casein diet. Dietary 13C-glucose was expired into 13CO2 within 30 min of consumption of the diet containing 13C-glucose at 1700 h (d 54); a gradual decrease in expired 13CO2 was observed in both groups after 2000 h. After ingestion of meals containing unlabeled carbohydrate, oxidation of 13C-unlabeled carbohydrate decreased and oxidation of unlabeled carbohydrate increased (d 55). The second increase in oxidation of dietary 13C-glucose was observed 18 h after the mice were fed the 13C-unlabeled diet. The second increase in oxidation of dietary carbohydrate was slightly and significantly higher in the mice fed the SPI-H diet than in those fed the casein diet (P < 0.05, Fig. 4).

Oxidation of dietary lipids. Exogenous lipid oxidation in the mice fed the SPI-H diet was calculated by measuring expired 13CO2 from dietary 13C-trioleate. When mice were fed diets containing 13C-trioleate at 1700 h on d 54, and then exogenous lipid oxidation was measured between 0600 and 1600 h on the next day (d 55), exogenous lipid oxidation was significantly lower in mice fed SPI-H than in casein-fed mice. The increases in oxidation of 13C-trioleate were observed in both groups 5–22 h after consumption of diets containing 13C-trioleate. For this period, expired 13CO2 from dietary 13C-trioleate was considerably lower in mice fed the SPI-H diet than in those fed the casein diet (Fig. 5, P < 0.01).

FIGURE 1  Energy expenditure during 72 h in KK-Ay mice fed casein or soybean protein isolate hydrolysate (SPI-H) diets. Mice were provided free access to a high fat diet for the first 28 d and then restrictively fed a high protein diet for 28 d before measurement of energy expenditure (d 53–56). Food was provided at 1700 h (arrow) and left for 24 h every day. Black square shows the dark period. Values are means ± SEM, n = 9. *Different from the casein group, P < 0.05.

FIGURE 2  Total carbohydrate oxidation during postprandial 24 h (on d 54) in KK-Ay mice fed casein or soybean protein isolate hydrolysate (SPI-H) diets. Mice were provided free access to a high fat diet for the initial 28 d and then restrictively fed high protein diets for 28 d before measurement of energy expenditure (d 53–56). Food was provided at 1700 h (arrow) and left for 24 h every day. Black square shows the dark period. Values are means ± SEM, n = 9. *Different from the casein group, P < 0.05.
Energetic contribution of substrate. Substrate oxidation data are summarized in Figure 6. Energy expenditure by mice fed the SPI-H diet in the postprandial period was significantly higher than by those fed the casein diet (P < 0.05). The increase in postprandial energy expenditure in SPI-H mice was due mainly to the increase in exogenous carbohydrate oxidation. The groups did not differ in exogenous lipid oxidation during the postprandial period. During the 24 h after feeding, there were no significant differences between groups in energy expenditure, although total carbohydrate oxidation was significantly higher and total lipid oxidation tended to be lower (P = 0.07) in the SPI-H than in the casein group. Similarly, exogenous carbohydrate oxidation tended to higher (P = 0.054) and exogenous lipid oxidation was significantly lower in the SPI-H than in the casein group.

Absorption of substrates. The absorption of lipid and carbohydrates was estimated from the content of lipid and 13C-glucose in feces during the 72 h in the respiratory chamber. Fecal volume was higher in mice fed SPI-H than in those fed the casein diet. The amount of 13C in feces in mice fed the SPI-H diet was 71% of that in casein-fed mice. In contrast, the amount of lipids in feces of mice fed the SPI-H diet was 153% of that in mice fed the casein diet (Table 3).

DISCUSSION

In a previous study by Aoyama et al. (9), body fat content was significantly lower in genetically obese diabetic KK-AV mice fed an energy-restricted SPI-H diet compared with mice fed a casein diet. Aoyama et al. (9) also reported that SPI and

**FIGURE 4** Exogenous carbohydrate oxidation during 48 h (d 54–55) in KK-AV mice fed casein or soybean protein isolate hydrolysate (SPI-H) diets. For the initial 28 d, mice were provided free access to a high fat diet, and then restrictively fed high protein diets for a further 28 d before measurement of energy expenditure (d 53–56). Foods were provided at 1700 h and left for 24 h every day. Values are means ± SEM, n = 6. *Different from the casein group, P < 0.05.

**FIGURE 5** Exogenous lipid oxidation during 24 h (d 55) in KK-AV mice fed casein or soybean protein isolate hydrolysate (SPI-H) diets. Mice were fed a high protein diet containing 13C-triolein (3 g/kg diet) on d 54 (white arrow). Values are means ± SEM, n = 3. *Different from the casein group, P < 0.05.

**FIGURE 6** Summary of total substrate utilization during postprandial 6 h (1700–2300 h; A) and 24 h (1700–1700 h; B) in KK-AV mice fed casein or soybean protein isolate hydrolysate (SPI-H) diets. Endogenous carbohydrate oxidation (diagonal hatched bars); exogenous carbohydrate oxidation (solid bars); exogenous lipid oxidation (open bars); exogenous lipid oxidation (vertical hatched bars). Values are means, n = 3–6. *Different from the casein group, P < 0.05; †P < 0.05 in total energy expenditure between mice fed SPI-H and the casein-fed groups.

| TABLE 3 | Fecal 13C enrichment, triglyceride and water in mice 24 h after being fed a diet containing 13C-glucose (d54–55) |  |
| --- | --- | --- | --- |
| Casein | SPI-H |  |
| d 54 |  |
| 13C, atom% | 2.62 ± 0.15 | 1.88 ± 0.13* |
| Weight, g | 0.49 ± 0.05 | 0.63 ± 0.05* |
| Triglyceride, % | 0.29 ± 0.02 | 0.43 ± 0.06* |
| Water, % | 44.2 ± 4.4 | 45.5 ± 0.99 |
| d 55 |  |
| 13C, atom% | 1.2 ± 0.02 | 1.18 ± 0.01 |
| Water, % | 0.33 ± 0.04 | 0.48 ± 0.03 |

1 Values are means ± SEM, n = 6. * Different from mice fed casein, P < 0.05.
2 Feces were collected over 72 h in a metabolic chamber 24 h after either the casein or SPI-H diet.
SPI-H diets decreased the apparent digestibility of energy and fat in Sprague-Dawley rats fed an energy-restricted diet for 4 wk. It was concluded that the antiobesity effects of SPI were caused by a decrease in net energy intake, although these authors did not discuss the effect of SPI on energy expenditure. Respiratory gas analysis is the most valid method for assessing whole-animal energy expenditure. We optimized the respiratory gas analysis system for use with small animals by direct linkage with mass spectrometry (18,19), a method that can differentiate $^{13}$CO$_2$ from $^{12}$CO$_2$. In the current study, we use respiratory gas analysis to measure energy expenditure in KK-A$^+$ mice fed SPI-H or casein diets for 4 wk after energy restriction. We also fed mice diet containing $^{13}$C-labeled sucrose or triolein to quantify the oxidation of dietary carbohydrate and lipid.

Compared with the casein diet, postprandial energy expenditure increased by 6% after 4 wk of consuming the SPI-H diet (Fig. 1). Dietary-induced thermogenesis plays an important role in the balance between daily energy intake and expenditure. Saito et al. (24) reported that soy peptide increases the activity of uncoupling protein-1 in brown adipose tissue of rats. In the present study, increased energy expenditure by mice fed the SPI-H diet was observed during the postprandial but not during the preprandial period (Fig. 1).

In diabetic patients, there is a significant inverse correlation between respiratory quotient (RQ) and blood glucose levels (25). RQ reflects the amount of energy derived from carbohydrate as opposed to fat metabolism. In the study by Nakaya et al. (25), diabetic patients treated with insulin had lower blood glucose levels and significantly higher RQ values than untreated diabetic patients. Panigagua et al. (26) reported that 0.4 mg Cerivastatin therapy improved first-phase insulin secretion and increased insulin-mediated glucose uptake, glucose oxidation and RQ in the early stage of obese type 2 diabetes (26).

In the present study, the postprandial increase in energy expenditure in mice fed the SPI-H diet was accompanied by a postprandial increase in carbohydrate oxidation (Figs. 2 and 4) and RQ. In a previous study, plasma insulin concentration was significantly higher after administration of a glucose solution in obese rats fed an SPI-H diet containing corn oil compared with those fed a casein diet (14). We speculate that in mice fed the SPI-H diet in our study, the increase in postprandial carbohydrate oxidation was due to higher insulin and lower glucose concentrations in the blood during the postprandial period. Further studies are required to elucidate the mechanism(s) responsible for the higher postprandial carbohydrate oxidation in mice fed SPI-H.

It is possible that the higher postprandial carbohydrate oxidation in mice fed SPI-H is related to inhibition of lipid absorption and increased carbohydrate absorption. Inhibition of lipid absorption is consistent with the observation that dietary SPI-H may reduce lipid absorption in genetically obese type II diabetic mice (9). In the present study, significantly higher glucose digestibility was found in mice fed SPI-H compared with those fed the casein diet when we fed the mice a diet containing $^{13}$C-labeled glucose and then assessed $^{13}$C concentrations in their feces. KK-A$^+$ mice develop hyperglycemia, hyperinsulinemia, glucose intolerance and obesity by 8 wk of age. The principal cause of diabetes in these mice is insulin resistance. Body composition analysis shows that both fat and lean tissue mass increase compared with nonobese mice; in the obese mice, fat accounts for 30–35% of total body weight. Food restriction normalizes levels of blood glucose, blood insulin and body fat, and improves glucose tolerance (27). Although we did not investigate the effect of the SPI-H diet on diabetic nephropathy, kidney weights were significantly lower in mice fed SPI-H (see Results). Williams et al. (28–31) reported previously that rats fed soy diets demonstrated improved survival, and less proteinuria, renal hypertrophy and renal histological damage. In conclusion, the present study of food-restricted mice indicates that there is a greater increment in postprandial energy expenditure and promotion of carbohydrate oxidation in mice fed the SPI-H diet compared with mice fed the casein diet.

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LITERATURE CITED


