Effects of High Sucrose or Starch-bran Diets on Glucose and Lipid Metabolism of Normal and Diabetic Rats

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ABSTRACT Normal and streptozotocin-diabetic rats were fed a low carbohydrate diet or one of two high carbohydrate diets (sucrose or starch-bran). High carbohydrate diets were associated with slightly higher plasma glucose levels in both control and diabetic rats. However, glucose tolerance tests were not altered by the diets in either the control or diabetic rats. Plasma and liver triglyceride values for control and diabetic rats fed the high sucrose diet were similar to those of rats fed the low carbohydrate diet. The starch-bran diet was associated with lower plasma and liver triglyceride values in both control and diabetic rats. Plasma triglyceride values of diabetic rats fed the high sucrose diet or the low carbohydrate diet were significantly higher than values of control rats fed the same diets. On the other hand, plasma triglyceride values of diabetic rats fed the starch-bran diet were similar to values for control rats fed the same diet. These studies suggest that a high carbohydrate diet containing starch and wheat bran is associated with a reduction in plasma triglyceride values in both normal and diabetic rats. Liver glycolytic enzyme activities were significantly lower in diabetic rats than in control rats in all three dietary groups. J. Nutr. 107: 584–595, 1977.

INDEXING KEY WORDS sucrose • starch-wheat bran • triglyceride • diabetes

High carbohydrate diets containing 75% to 85% of energy as carbohydrate are accompanied by a slight improvement of glucose metabolism of normal men (1, 2) and by a significant improvement in glucose metabolism of diabetic individuals (3–5). However, when high carbohydrate diets are fed to rats, some groups have reported improvement in glucose metabolism (6, 7) whereas other groups have reported deterioration of the glucose tolerance (8). High carbohydrate diets are accompanied by higher tissue activities for glycolytic enzymes in man (9) and in rats (10–12) than values obtained with low carbohydrate diets. Plasma insulin responses after oral glucose are lower in men fed high carbohydrate diets than responses when conventional diets are fed (1, 2). These observations (1, 2) have suggested that high carbohydrate diets are accompanied by an enhanced sensitivity of tissues to insulin. However, the mechanisms for alteration in the glucose metabolism when high carbohydrate diets are fed remains unclear.

Hypertriglyceridemia frequently has been reported when men or rats are fed high carbohydrate diets. When normal men or diabetic patients are fed formula diets containing sucrose, glucose or dextrin, increases in serum triglyceride values are observed with regularity (13–15). Similar observations have been made when rats are fed diets containing large amounts of

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sucrose or fructose (13, 16–19). Other studies have suggested that when diabetic patients are fed high carbohydrate diets composed of solid foods with generous amounts of starch and limited amounts of oligosaccharides, an increase in serum triacylglyceride values does not occur (3, 20–22). Recent studies (3) have suggested that the introduction of wheat bran into the diet protects diabetic patients from the development of hypertriglyceridemia when they are fed 75% carbohydrate diets. Thus certain high carbohydrate diets are accompanied by hypertriglyceridemia whereas other diets may not produce an increase in serum triglyceride values.

The primary objective of these experiments was to determine if high carbohydrate diets have different effects on the glucose metabolism of diabetic rats compared to the effects observed in normal rats. Whereas high carbohydrate diets have beneficial effects on the glucose metabolism of certain human diabetic patients (3–5), we observed no significant differences in the glucose metabolism of normal or diabetic rats fed high carbohydrate diets compared with normal or diabetic rats fed low carbohydrate diets. The secondary objective of these experiments was to determine if different types of high carbohydrate diets have differing effects on lipid metabolism of normal and diabetic rats. For this comparison, we selected two different high carbohydrate diets which we anticipated would be accompanied by differences in lipid metabolism. The high sucrose diet was selected because previous studies (13, 17–19) suggested that this diet would be accompanied by the greatest increases in serum triglyceride values. The high starch diet was selected because previous studies (3, 20–22) indicated that carbohydrates in this diet would be accompanied by the greatest increase in serum triglyceride values. The high sucrose diet was accompanied by hypertriglyceridemia whereas other diets may not produce an increase in serum triglyceride values.

MATERIALS AND METHODS

Animals. Male Sprague-Dawley rats (150–170 g) were housed in individual cages with a raised mesh floor. The rats were fed a commercial stock diet for at least 2 weeks before changing their diet. All diets and water were fed ad libitum. Diabetes was produced by the intravenous injection of streptozotocin after a 16 to 20 hour fast. At the time of streptozotocin injection, the weight of the injected rats matched that of the control rats. Diabetic rats in group 1 received 50 mg streptozotocin/kg body weight. Plasma glucose values and body weight were measured in these rats in the nonfasted state at 15 to 21 days after streptozotocin injection. Diabetic

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**TABLE 1**

<table>
<thead>
<tr>
<th>Composition of the diets</th>
<th>Low carbohydrate</th>
<th>High sucrose</th>
<th>Starch-bran</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carbohydrate, total</td>
<td>32</td>
<td>72</td>
<td>72</td>
</tr>
<tr>
<td>Sucrose</td>
<td>32</td>
<td>72</td>
<td>—</td>
</tr>
<tr>
<td>Starch</td>
<td>—</td>
<td>—</td>
<td>60</td>
</tr>
<tr>
<td>Bran¹</td>
<td>—</td>
<td>—</td>
<td>12</td>
</tr>
<tr>
<td>Protein, total</td>
<td>46</td>
<td>18</td>
<td>12</td>
</tr>
<tr>
<td>Casein</td>
<td>46</td>
<td>18</td>
<td>10</td>
</tr>
<tr>
<td>Bran¹</td>
<td>—</td>
<td>—</td>
<td>2</td>
</tr>
<tr>
<td>Vegetable oil</td>
<td>12</td>
<td>6</td>
<td>6</td>
</tr>
<tr>
<td>Dietary fiber, total</td>
<td>6</td>
<td>—</td>
<td>6</td>
</tr>
<tr>
<td>Cellulose</td>
<td>6</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Bran¹</td>
<td>—</td>
<td>—</td>
<td>6</td>
</tr>
<tr>
<td>Salt mixture²</td>
<td>4</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>Vitamin mixture³</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
</tbody>
</table>

¹Starch-bran diet contained 20% of wheat bran which had protein 10%, fat 3%, CHO 57% (values were from Watt, B. K. & Merrill, A. L., composition of Foods USDA Handbook No. 8, 1963) and dietary fiber 30% (unpublished observations). Therefore, this 20% wheat bran provided 12% carbohydrate, 2% protein and 6% dietary fiber. ²Salt mixture (ICN Nutritional Biochemicals, Cleveland, Ohio) contains: Sodium chloride, 13.99%; potassium biphosphate 38.91%; magnesium sulfate (anhydrous) 5.73%; calcium carbonate 38.14%; ferrous sulfate 2.70%; manganese sulfate 0.4%; potassium iodide 0.079% ; zinc sulfate 0.055%; cupric sulfate 0.045%. ³Vitamin mixture (ICN Nutritional Biochemicals, Cleveland, Ohio) contains (g/46 kg): Vitamin A concentrate (200,00 u/g), 4.5; Vitamin D concentrate (400,000 u/g), 0.25; α tocopherol, 5.0; ascorbic acid, 45.0; inositol, 5.0; choline chloride, 75.0; menadione, 2.25; p-aminobenzoic acid, 5.0; niacin, 4.5; riboflavin, 1.0; pyridoxine hydrochloride, 1.0; thiamin hydrochloride, 1.0; calcium pantothenate, 3.0; biotin, 0.02; folic acid, 0.09; vitamin B-12, 0.00135.

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1 Harlan Industries, Cumberland, Indiana.
2 Purina Laboratory Chow, Ralston Purina Co., St. Louis, Missouri.
rats in group 2 received 35 mg streptozotocin/kg body weight. Plasma glucose and body weight were measured in these rats after a 4 hour fast, 15 to 21 days after streptozotocin injection. This experiment used a randomized blocks design. Diabetic rats were divided into twelve blocks, three rats in each, by matching both plasma glucose level and body weight. Control rats were divided into nine blocks, again with three rats in each by matching body weight. Each of the three rats in a block of control or diabetic rats were randomly assigned to one of three experimental diets (table 1). One diet was a low carbohydrate diet and two diets were high carbohydrate diets, the high sucrose and the starch-bran diet. Preliminary studies of control rats indicated that the responses of glycolytic and pentose phosphate pathway enzymes in liver and jejunum to these experimental diets were maximal at 10 days and remained in the same range for up to 32 days. All rats in group 1 were given the diets on the same day and were killed 14 to 32 days (average of 25 days) later. Rats in group 2 were killed after being fed the diets for 21 days. The results for control rats in groups 1 and 2 were similar and were combined for analyses.

Terminal plasma glucose values were obtained from tail vein blood 1 day before and the day of killing of nonfasted rats in group 1. In group 2, between 1 and 5 days before killing, the terminal plasma glucose value was obtained from rats fasted for 4 hours. On the day of killing, rats were anesthetized with ether, and blood was withdrawn by cardiac puncture. Blood was centrifuged for 30 minutes at 1,000 × g and 4°C; plasma was removed and stored in a freezer at −20°C until analysis.

Tissue preparations. After the rats were killed, the abdomen was opened and two portions of liver removed. Two segments of jejunum located from 12 to 25 cm and from 25 to 45 cm beyond the pylorus were removed, slit longitudinally, washed free of contents with distilled water at 4°C and blotted dry. Mucosa was obtained by scraping the intestine with a glass slide. One portion of liver and the distal portion of jejunal mucosa were homogenized in 30 volumes of a buffer containing 50 mM Tris, 100 mM KCl, 1 mM EDTA, 5 mM mercaptoethanol and 5 mM MgCl₂ at pH 7.4. The homogenates were centrifuged at 105,000 × g for 60 minutes in an ultracentrifuge. The 105,000 × g liver supernatant fraction was used for glucokinase (ATP: D-glucose-6-phosphotransferase, EC 2.7.1.2), glucose-6-phosphate (G-6-P) dehydrogenase (NADP oxidoreductase, EC 1.1.1.49) and malic enzyme (L(-)-malate:NADP oxidoreductase (decarboxylating), EC 1.1.1.40) assays. Hexokinase (ATP:D-hexose-6-phosphotransferase, EC 2.7.1.1) assays were performed on jejunal homogenates, and assays for hexokinase, C-6-P dehydrogenase and malic enzyme were obtained on supernatant fractions. The other portions of liver and jejunal mucosa were homogenized in 20 volumes of a buffer containing 20 mM TES (N-tris(hydroxymethyl)methyl-2-aminoethane sulfonic acid), 10 mM MgSO₄, 1 mM dithiotreitol and 100 mM sucrose at pH 7.4 and centrifuged in a similar fashion. The 105,000 × g supernatants were used for pyruvate kinase (ATP:pyruvate phosphotransferase, EC 2.7.1.40) assays. All tissue preparation and weighing were carried out at 4°C.

Enzyme assays. Enzymes were assayed at 25°C in a recording spectrophotometer. Hepatic glucokinase and jejunal hexokinase were assayed by the method of Vinuela et al. (23) using 0.1 M glucose for liver and 10 mM glucose for jejunal mucosa (12). Pyruvate kinase was measured by the method of Bucher and Pfleiderer (24), G-6-P dehydrogenase was measured by the method of Langdon (25) and the method of Ochoa (26) was used for malic enzyme assays. Protein concentrations in liver supernatants, jejunal supernatants and homogenates were measured by the method of Lowry et al. (27). Bovine serum albumin dissolved in the homogenizing buffer used for the original sample, was used as a standard. Enzyme activities were expressed as μmoles/minute/g tissue.

Glucose and glycogen measurements. Plasma glucose estimations were performed in duplicate by the glucose oxidase method of a Beckman Model L5-40, Fullerton, California.
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Liver glycogen was measured as described previously (28). Rabbit liver glycogen was used as a standard.

Total lipid and triglyceride measurements. The total lipid in liver was extracted by homogenizing the tissue with a 2:1 (v/v) chloroform-methanol mixture and washing the filtered extract with one-fifth volume of 0.9% NaCl (29). The resulting mixture separated into two phases. The lower chloroform layer contained the total lipid extract. The weight of total lipid in the chloroform layer was measured by the gravimetric method. A measured aliquot of the chloroform layer was used for triglyceride determinations. Liver triglyceride was estimated colorimetrically by the Dade method (7). Serum triglyceride was measured by the method of Levy (30) with triolein in isopropanol as the standard.

Statistical analysis. Data were analyzed by using the method of randomized block design (31). When statistical significance was found, Duncan's Multiple Range Test (31) was applied to determine which mean values were significantly different.

RESULTS

Plasma glucose values. Terminal plasma glucose values in control rats fed high sucrose or starch-bran were slightly but not significantly higher than values for the rats fed the low carbohydrate diet. Initial plasma glucose values were similar for all three groups of diabetic rats before the test diets were initiated (table 2). Diabetic rats in group 1 fed high sucrose or starch-bran diets had significantly higher terminal plasma glucose values than the rats fed the low carbohydrate diet. Terminal plasma glucose values for all diabetic rats in group 2 fed the high sucrose diet (441 ± 180 mg/100 ml, mean ± sd) or the starch-bran diet (483 ± 149) were slightly but not significantly higher than values for the low carbohydrate group (411 ± 142). There were no significant differences between the plasma glucose values of rats fed the high-sucrose diet or the starch-bran diet. These observations are similar to those of Baker et al. (32).

Glucose tolerance tests were performed on streptozotocin-injected rats with plasma glucose values less than 300 mg/100 ml; 2 g glucose/kg body weight (33) was injected intraperitoneally after a 4-hour fast. Glucose tolerance tests were also performed on four control rats from each diet group.

TABLE 2

Plasma glucose in control and diabetic rats

| Diet              | Control Initial | Control Terminal | Group 1 Initial | Mild | Moderate | Severe
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>mg/100 ml</td>
<td>mg/100 ml</td>
<td>mg/100 ml</td>
<td>mg/100 ml</td>
<td>mg/100 ml</td>
<td>mg/100 ml</td>
</tr>
<tr>
<td>Low carbohydrate</td>
<td>162 (9)±12</td>
<td>444 (12)±43</td>
<td>514 (12)±43</td>
<td>375 (12)±120</td>
<td>187 (3)±14</td>
<td>450 (5)±44</td>
</tr>
<tr>
<td>High sucrose</td>
<td>175 (9)±22</td>
<td>446 (12)±69</td>
<td>679 (11)±43</td>
<td>375 (12)±123</td>
<td>218 (4)±36</td>
<td>569 (7)±36</td>
</tr>
<tr>
<td>Starch-bran</td>
<td>180 (9)±34</td>
<td>444 (12)±64</td>
<td>645 (12)±71</td>
<td>371 (12)±113</td>
<td>246 (2)±78</td>
<td>580 (6)±64</td>
</tr>
</tbody>
</table>

1 Mild diabetes with mean plasma glucose levels less than 300 mg/100 ml. 2 Moderate diabetes with mean plasma glucose level between 300 to 500 mg/100 ml. 3 Severe diabetes with mean plasma glucose level above 500 mg/100 ml. 4 Mean ± sd with the number of rats in parenthesis. Means in the same vertical column with different superscript letters differ significantly (P < 0.05).
TABLE 3
Body weight and weight changes in control and diabetic rats

<table>
<thead>
<tr>
<th>Diets</th>
<th>Controls</th>
<th>Group 1</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low carbohydrate</td>
<td>494 ±60 (+109)</td>
<td>338 ±35 (+43)</td>
<td>477 ±45 (+64)</td>
<td>298 ±62 (+14)</td>
<td>336 ±56 (+18)</td>
</tr>
<tr>
<td>High sucrose</td>
<td>486 ±40 (+100)</td>
<td>320 ±38 (+19)</td>
<td>485 ±94 (+63)</td>
<td>—</td>
<td>328 ±50 (+12)</td>
</tr>
<tr>
<td>Starch-bran</td>
<td>483 ±76 (+100)</td>
<td>303 ±38 (+19)</td>
<td>512 ±76 (+55)</td>
<td>382 ±19 (+19)</td>
<td>398 ±74 (+18)</td>
</tr>
</tbody>
</table>

1. Nine rats per dietary group.  
2. 11 or 12 rats per dietary group.  
3. Mild diabetic rats included two to four rats per dietary group. Moderate diabetic rats included two to five rats per dietary group. Severe diabetic rats included four to seven rats per dietary group.  
4. Mean body weight (Mean ± SD) at the end of experiment with mean weight changes during experimental period in parenthesis. Mean weight changes in the same vertical column with different superscript letters differ significantly (P < 0.05).

etary group. After 30 minutes, control rats had plasma glucose values of 420 ± 76 mg/100 ml, 432 ± 97, 350 ± 107 and diabetic rats had values of 537 ± 221, 535 ± 87 and 534 ± 70 for the low carbohydrate, the high sucrose and the starch-bran diets, respectively. After 90 minutes, control rats had plasma glucose values of 238 ± 40 mg/100 ml, 215 ± 30, and 216 ± 24 and diabetic rats had values of 462 ± 197, 399 ± 98 and 427 ± 231 for the low carbohydrate, the high sucrose and the starch-bran diet, respectively. These mean plasma glucose values for diabetic rats were significantly higher than values for control rats. Glucose tolerance tests were also performed on moderately and severely diabetic rats (four rats fed each diet). Plasma glucose values were 780 ± 115, 785 ± 257 and 816 ± 135 mg/100 ml at 30 minutes and 640 ± 96, 741 ± 102, 588 ± 94 mg/100 ml at 90 minutes for the low carbohydrate, the high sucrose and the starch-bran diets, respectively; these differences were not significantly different. The plasma glucose values obtained after a glucose load did not differ significantly between the three dietary groups for either control or mildly diabetic rats. Thus, glucose tolerance was neither exacerbated nor improved in these control or diabetic rats fed either high carbohydrate diet when compared with values for the low carbohydrate diet.

**Body weight and weight changes.** At the time of streptozotocin injection, mean body weight changes in the same vertical column with different superscript letters differ significantly (P < 0.05).

TABLE 4
Liver glycogen in control and diabetic rats

<table>
<thead>
<tr>
<th>Diets</th>
<th>Controls</th>
<th>Group 1</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low carbohydrate</td>
<td>56.1 ±20.3³</td>
<td>26.2 ±11.4**³</td>
<td>37.6 ±18.3³</td>
<td>23.9 ±13.7**³</td>
<td>31.4 ± 7.6**³</td>
</tr>
<tr>
<td>High sucrose</td>
<td>82.7 ±16.9³</td>
<td>39.5 ±12.2**³</td>
<td>86.0 ±18.8³</td>
<td>—</td>
<td>53.6 ±15.1**³</td>
</tr>
<tr>
<td>Starch-bran</td>
<td>76.0 ±9.7³</td>
<td>26.4 ±10.7**³</td>
<td>69.9 ± 7.2³</td>
<td>60.5 ± 9.0**³</td>
<td>22.4 ±10.8**³</td>
</tr>
</tbody>
</table>

1. Mean ± SD with the number of rats in parenthesis.  
2. Means in the same vertical column with different superscript letters differ significantly (P < 0.05).  
3. Significantly different from control rats (P < 0.01).
weights were similar in control and diabetic rats. As would be expected, body weights of control rats were greater than those of moderately or severely diabetic rats at the time of killing (table 3).

Control rats had a similar weight gain with each of the three experimental diets. Diabetic rats in group 1 fed the high sucrose or starch-bran diets gained significantly less weight than rats fed the low carbohydrate diet. The weight gains for diabetic rats fed the high sucrose and starch-bran group were similar.

Liver glycogen. The liver glycogen content for control rats fed both high carbohydrate diets (high sucrose and starch-bran) was significantly higher compared to the low carbohydrate group (table 4). These observations are in agreement with previous studies (6, 7, 34). A significant increase in liver glycogen was also noticed in mildly diabetic rats fed high carbohydrate diets; those rats fed the high sucrose diet had a similar liver glycogen content compared to the rats fed the starch-bran diet.

As anticipated, the liver glycogen content of moderately and severely diabetic rats was significantly lower than the value for control rats fed the corresponding diet. However, the glycogen content of mildly diabetic rats was similar to that of control rats. Studies from other groups (35) have indicated that early diabetes is accompanied by a higher concentration of liver glycogen.

Severely diabetic rats fed the high sucrose diet had a significantly higher liver glycogen concentration compared to rats fed the low carbohydrate or starch-bran diet.

Liver lipid and plasma triglyceride. The total liver lipid concentrations of control and diabetic rats fed low carbohydrate diets were significantly higher than values for rats fed high sucrose and starch-bran diets (table 5). This may be related to the dietary fat since the low carbohydrate diet contained twice as much fat as the high carbohydrate diets. No significant differences were observed in total liver lipid concentration between those rats fed high sucrose or starch-bran diets.

Liver triglyceride values in both control and diabetic rats were also significantly
higher in rats fed the low carbohydrate diet than values from starch-bran fed rats. Furthermore, in each of these groups, liver triglyceride was significantly higher in the high sucrose group than in the starch-bran group. This could not be related to fat intake since the fat content of these two high carbohydrate diets was identical.

Plasma triglyceride values followed a similar pattern to that observed for liver triglyceride concentrations; values were significantly greater in both groups (control and diabetics) fed the long carbohydrate diet than in the high sucrose group. This could not be related to fat intake since the fat content of these two high carbohydrate diets was identical.

Liver enzyme activities. In control rats, the high sucrose diet was accompanied by a significant increase in the activities of all four liver enzymes (Table 6). When the starch-bran diet was fed, only glucokinase and pyruvate kinase activities were significantly higher than the values for the low carbohydrate group. Consequently, G-6-P dehydrogenase and malic enzyme activities were significantly lower when the starch-bran diet was fed than when the high sucrose diet was fed.

Diabetic rats fed the high sucrose diet had significantly higher pyruvate kinase and malic enzyme activities than the rats fed low carbohydrate or starch-bran diet; the activities of glucokinase and G-6-P dehydrogenase were similar among those rats fed the three experimental diets. As noted previously (38, 39), activities for these four hepatic enzymes were significantly lower in severely diabetic rats fed each diet than in control rats fed the same diet ($P < 0.01$).

Jejunal enzyme activities. Hexokinase activities in the homogenate for control rats fed both high carbohydrate diets were significantly higher than values for rats fed low carbohydrate diets ($P < 0.05$), but
hexokinase activities in the supernatant were similar for the high and low carbohydrate groups (table 7). These data indicate that hexokinase activity in the particulate fraction was increased in control rats fed high carbohydrate diets. Our previous study (12) suggested that a high carbohydrate diet (75% glucose) produced proportionally greater increases in particle-bound hexokinase activity than in soluble hexokinase. Pyruvate kinase activity for control rats fed the high sucrose diet was significantly higher than the values for rats fed the low carbohydrate and starch-bran diets \( (P < 0.05) \). Jejunal G-6-P dehydrogenase and malic enzyme activities were similar for control rats in the three dietary groups.

In diabetic rats, hexokinase activity in homogenate from both high carbohydrate groups was significantly higher than the values for the low carbohydrate group \( (P < 0.05) \). Hexokinase activity of the supernatant fraction in the starch-bran group was also significantly higher than values for the low carbohydrate group \( (P < 0.05) \); the activities for the high sucrose and low carbohydrate diet were similar. Hexokinase activity in the supernatant fraction was higher in diabetic rats fed starch-bran than in control rats fed the same diet; homogenate activity was not increased. Consequently, the percentage of hexokinase activity in supernatant fraction of diabetic rats, \( 52 \pm 13\% \), was significantly higher than in control rats \( 32 \pm 8\% \) \( (P < 0.05) \). These observations are consistent with our previous studies (40, 41) demonstrating that diabetes is accompanied by a significant reduction in mitochondria-bound hexokinase activity and by an increase in the percentage of soluble hexokinase activity in jejunal mucosa.

**DISCUSSION**

The present study indicates that these high carbohydrate diets (high sucrose and starch-bran) did not significantly alter the glucose tolerance of control rats. This contrasts with previous studies which have shown either improvement (6) or deterioration of the glucose tolerance (8, 42) when normal rats were fed high carbohydrate diets. These differences may be due to the genetic or strain differences between the rats studied since some strains of rats have an inherited tendency toward glucose intolerance (43, 44). Likewise, we were unable to demonstrate that high carbohydrate diets altered the glucose tolerance of diabetic rats.

The glucose tolerance tests were similar when control rats were fed the high sucrose diet or the starch-bran diet. This finding is in contrast with the results of Cohen and Teitelbaum (8, 45) who reported that glucose tolerance was impaired in sucrose-fed rats compared with the starch-fed rats.

**TABLE 7**

Jejunal mucosal enzyme activities in control and diabetic rats

<table>
<thead>
<tr>
<th></th>
<th>Total hexokinase</th>
<th>Hexokinase</th>
<th>Pyruvate kinase</th>
<th>G-6-P dehydrogenase</th>
<th>Malic enzyme</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control rats ( n = 9 )</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low carbohydrate</td>
<td>( 5.51 \pm 1.03^{*} )</td>
<td>( 1.34 \pm 0.21 )</td>
<td>( 127.79 \pm 34.09^{*} )</td>
<td>( 5.54 \pm 1.86 )</td>
<td>( 2.66 \pm 0.67 )</td>
</tr>
<tr>
<td>High sucrose</td>
<td>( 8.72 \pm 1.12^{*} )</td>
<td>( 1.24 \pm 0.23 )</td>
<td>( 233.98 \pm 58.83^{*} )</td>
<td>( 6.00 \pm 1.75 )</td>
<td>( 2.70 \pm 0.69 )</td>
</tr>
<tr>
<td>Starch-bran</td>
<td>( 9.31 \pm 1.72^{*} )</td>
<td>( 1.25 \pm 0.18 )</td>
<td>( 110.14 \pm 21.69^{*} )</td>
<td>( 4.87 \pm 1.86 )</td>
<td>( 3.15 \pm 0.75 )</td>
</tr>
</tbody>
</table>

|                |                  |            |                  |                      |              |
| Diabetic rats \( n = 16 \) |                  |            |                  |                      |              |
| Low carbohydrate | \( 7.11 \pm 1.99^{**} \) | \( 1.31 \pm 0.66^{*} \) | \( 160.08 \pm 39.86^{*} \) | \( 6.35 \pm 2.46 \) | \( 3.39 \pm 0.84^{*} \) |
| High sucrose    | \( 10.77 \pm 1.57^{**} \) | \( 1.59 \pm 0.50^{*} \) | \( 203.96 \pm 55.91^{**} \) | \( 7.73 \pm 2.39 \) | \( 4.69 \pm 1.65^{*} \) |
| Starch-bran     | \( 9.84 \pm 2.06^{**} \) | \( 2.08 \pm 0.48^{*} \) | \( 119.22 \pm 33.34^{*} \) | \( 6.90 \pm 2.80 \) | \( 4.13 \pm 1.50^{*} \) |

\* Total hexokinase were measured in jejunal homogenates. \* These four enzymes were measured in 105,000 \( \times g \) supernatant fractions. \* Mean \( \pm S E \) means within a column with different superscript letters differ significantly \( (P < 0.05) \). \* Values of group 1 and group 2 diabetic rats were statistically similar and have been pooled. \* Significantly different from control rats \( (P < 0.05) \).
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These differences could be related to the observations that different strains have different responses to sucrose (46). In diabetic rats, glucose tolerance also was not altered when responses of rats fed the high sucrose diet were compared with responses of rats fed the starch-bran diet.

Triglyceride values in liver and plasma of control rats fed the high carbohydrate diet containing sucrose were similar to values for rats fed the low carbohydrate diet despite the fact that they had significantly higher glycolytic and lipogenic enzyme activities. These data differ from previous observations that high carbohydrate diets increase the liver (36, 47, 48) and plasma triglyceride values (7, 34, 45) in normal rats. The incorporation of [6-14C]glucose, [6-14C]fructose or 14C acetate into hepatic fatty acids are higher in rats fed high carbohydrate diets than in animals fed a stock diet (16, 47). Waddell and Fallon (48) demonstrated that high carbohydrate diets increased the capacity for triglyceride formation from α-glycerol-3-phosphate in rat liver homogenates and also increased the incorporation of [1,3-14C]glycerol into hepatic triglyceride in the intact animal. The activities of the enzymes supporting lipogenesis, G-6-P dehydrogenase, NADP-malate dehydrogenase and acetyl CoA carboxylase, are also higher in animals fed high carbohydrate diets (17, 47). Even in diabetic rats, however, our studies demonstrate that the values of liver and plasma triglyceride for rats fed the high sucrose diet are not different from the values for rats fed the low carbohydrate diet.

Many investigators have reported that high sucrose diets result in higher liver and plasma triglyceride values (17–19, 49, 50) and are associated with higher G-6-P dehydrogenase and malic enzyme activities (17, 51) than the values observed with starch diets. These observations are confirmed by the present study demonstrating that sucrose feeding was accompanied by elevated liver and plasma triglyceride values. The remarkable rises in the activities of liver enzymes for NADPH production and especially the more than fivefold rise in the activity of acetyl-CoA carboxylase, which is directly involved in fatty acid synthesis, emphasize the acquired capacity for enhanced lipogenesis when rats are fed sucrose (17). Bruckdorfer et al. (52) pointed out that endogenous hepatic lipogenesis or fatty acid synthetase activity may be the major determinant of triglyceride concentrations during sucrose feeding. These metabolic differences between sucrose and starch may be related to either the fructose moiety in sucrose (13, 16, 52) or to the disaccharide configuration of sucrose (51).

In this study, the higher fiber content of starch-bran diet (Table 1) may be responsible for the lower liver and plasma triglyceride values in both control and diabetic rats than values observed when these rats were fed the high sucrose diet. Dietary fiber has been demonstrated to exert a hypocholesterolemic effect in rats (53, 54), but the influence of dietary fiber on plasma triglyceride values in rats has not been carefully evaluated. Our data suggest that this high carbohydrate diet containing starch and wheat bran may have beneficial effect in lowering the plasma triglyceride values of diabetic rats.

The effects of diet on carbohydrate and lipid metabolism in rats were similar in some instances but differed in other instances to that of men. High carbohydrate diets are associated with improved glucose tolerance in normal subjects (1, 2), in mildly diabetic patients (3, 55) and in moderately to severely diabetic patients (3–5). Despite the higher hepatic glycolytic enzymes for control rats fed the high carbohydrate diets, we did not see an improvement in glucose tolerance in these rats. The lack of improvement in glucose metabolism of streptozotocin treated rats with moderate to severe diabetes fed high carbohydrate diets is consistent with the observations that glucose metabolism in untreated moderately to severely diabetic patients fed high carbohydrate diets is not improved (3, 20, 21). Since we were unable to produce mildly diabetic rats, streptozotocin treated rats may not be a good model for studying the influence of high carbohydrate diets.

Elevations in plasma triglyceride concentrations can be induced in men by the feeding of diets high in carbohydrate (13, 56–
plasma triglyceride values occurs (13-15). This phenomenon was not observed in the rats in this study which were fed the starch-bran diet. Significant increases in serum triglyceride have been found in normal subjects eating diets in which monosaccharides or disaccharides are substituted for starch (59-62). Similar findings were observed in control rats in this study. When diabetic patients receive a liquid high carbohydrate diet containing no solid foods, a significant increase in fasting plasma triglyceride values occurs (13-15). However, fasting plasma triglyceride values are not increased when diabetic subjects are fed a sugar-limited or low oligosaccharide, high carbohydrate diet (3, 20-22). Likewise, this study demonstrated that diabetic rats fed the high sucrose diet had a higher plasma triglyceride value than rats fed the starch-bran diet.

Some investigators have reported that serum triglyceride can be lowered by the inclusion of high levels of dietary fiber in the diet in normal subjects (63, 64) while others fail to confirm these findings (65, 66). A recent study (3) demonstrated that a high carbohydrate diet containing generous amounts of dietary fiber including wheat bran was accompanied by a reduction in the serum triglyceride value in diabetic patients. In our present study, we observed that the starch-bran diet (containing 6 g fiber/100 g diet) tended to prevent hypertriglyceridemia in diabetic rats. The effect of dietary fiber on plasma lipid remains ill-defined and controversial (67-69). Further studies are necessary to determine the effect of varying amounts and type of dietary fiber on plasma lipid levels.

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LITERATURE CITED