THE EFFECT OF AN EXCLUSIVE MEAT DIET LASTING ONE YEAR ON THE CARBOHYDRATE TOLERANCE OF TWO NORMAL MEN.*

BY EDWARD TOLSTOI.

(From the Russell Sage Institute of Pathology in Affiliation with the Second Medical (Cornell) Division and Pathological Department of Bellevue Hospital, New York.)

(Received for publication, June 20, 1929.)

During the past year exhaustive studies were made of the metabolism of two normal men whose diet consisted for 1 year exclusively of lean and fat meat. This was essentially a high fat, low carbohydrate diet. The tolerance of these men to carbohydrate was tested by the glucose meal. As far as can be ascertained, no experiments have been reported where subjects lived on a similar diet for 1 year. This communication presents such experiments.

EXPERIMENTAL.

The subjects were healthy adult men whose diet consisted solely of lean and fat meat. This was eaten either cooked or raw. The protein of the diet averaged 120 gm. with a caloric intake of 2600 to 3000 calories. Other data and general plan of the experiment will be published by McClellan and Du Bois (1). Two glucose tolerance tests were carried out on each of the two subjects, K. A. and V. S.; one test immediately after the meat-fat diet was discontinued, the other 2 or more weeks after the general diet had been resumed. Some 8 or 9 hours after the first glucose tolerance test one of the men (K. A.) developed a pneumonia from which he recovered and reported in good condition for his second test 4 weeks later. Subject V. S. was in good health and showed no abnormalities either before or after any of the tests. Each man

* This work was in part supported by a grant to the Russell Sage Institute of Pathology by the Institute of American Meat Packers.
Carbohydrate Tolerance

received 100 gm. of glucose in 230 cc. of water. About 20 cc. of orange juice were added for flavoring. This solution was given 10 to 12 hours after the last meal. Blood samples were obtained from a vein at the elbow before the test meal, and \( \frac{1}{2} \), 1, 2, and 3 hours after it. The blood was discharged into a flask containing about 2 mg. of potassium oxalate for each cc. of blood. A portion was at once precipitated and the sugar determined by the method of Folin and Wu (2). The urine of each subject was examined for reducing substances whenever samples were obtained during the course of the test.

The results obtained by these procedures are presented in Table I and Charts 1 and 2. Both show that a marked rise in the blood sugar occurred after the test meal following the previous high fat, low carbohydrate diet. The curve of K. A. not only in-

### Table I.

**Reducing Bodies.**

<table>
<thead>
<tr>
<th>Subject</th>
<th>Time</th>
<th>Blood</th>
<th>Urine</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>K. A.</td>
<td>Fasting</td>
<td>110</td>
<td>0</td>
<td>After diet of lean and fat meat for 1 yr.</td>
</tr>
<tr>
<td></td>
<td>( \frac{1}{2} )</td>
<td>235</td>
<td>++++</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>235</td>
<td>++++</td>
<td></td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>250</td>
<td>++++</td>
<td></td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>200</td>
<td>++++</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Fasting</td>
<td>87</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td></td>
<td>( \frac{1}{2} )</td>
<td>166</td>
<td>0</td>
<td>After general diet for 4 wks.</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>153</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>97</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>84</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>V. S.</td>
<td>Fasting</td>
<td>105</td>
<td>0</td>
<td>After diet of lean and fat meat for 1 yr.</td>
</tr>
<tr>
<td></td>
<td>( \frac{1}{2} )</td>
<td>200</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>210</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>182</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>117</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Fasting</td>
<td>105</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td></td>
<td>( \frac{1}{2} )</td>
<td>143</td>
<td>0</td>
<td>After general diet for 2 wks.</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>122</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>116</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>98</td>
<td>0</td>
<td></td>
</tr>
</tbody>
</table>
creased in height, but the hyperglycemia was prolonged also. He presented a glycosuria. V. S. had only an increase in the height of the curve. His urine was sugar-free. After a mixed diet for the periods mentioned above, both men reacted normally to the glucose tolerance tests, the height and the duration of the blood sugar curve being well within normal limits, and the urine sugar-free.

**CHART 1.** Blood sugar curves after glucose tolerance tests. Solid line indicates results after a diet of lean and fat meat for 1 year, dotted line after a general diet.

DISCUSSION.

The above results are not new. They were emphasized by Odin (3), Malmros (4), Stenstrom (5), Staub (6), Kageura (7), Greenwald, Gross, and Samet (8) and others. It was believed that during a period of a low carbohydrate, high fat diet, the need for insulin was diminished, with a resulting decrease in its production. Then, upon administration of a large quantity of carbohydrate to a subject subsisting on such a diet, the carbohydrate
mechanism is heavily taxed. The production of insulin cannot keep up with the demand, the result being a hyperglycemia and often a glycosuria as well. The extensive and excellent work of Malmros (4) also supports the above view. He worked with normal human beings who had glucose tolerance tests after general diets as well as after high fat, low protein, and low carbohydrate mixtures. The duration of such diets varied from 1 to 23 days.

![Subject V.S. Chart](chart.png)

**Chart 2.** Blood sugar curves after glucose tolerance tests. Solid line indicates results after a diet of lean and fat meat for 1 year, dotted line after a general diet.

In every case a lowering of the tolerance to carbohydrate was noted irrespective of the duration of the preceding diet. Greenwald, Gross, and Samet (8) were of the same opinion. They believed that a diet consisting chiefly of protein does not lower the tolerance for glucose as much as a diet consisting principally of fat. This inference might be used to explain the difference between the results of Heinbecker (9) and the ones presented in this paper.
Heinbecker studied the tolerance of Eskimos to carbohydrate. His subjects, by necessity, lived on a practically exclusive meat diet for years, before their carbohydrate tolerance tests were made. In spite of the fact that their diets were low in carbohydrate, the results of the tests indicated that they assimilated carbohydrate well. The blood sugar curves were within the normal range and the urine remained free of sugar. Is it possible that Heinbecker's subjects derived sufficient carbohydrate-forming substance from the protein in their diet to keep the insulin producing mechanism sufficiently stimulated to handle large quantities of carbohydrate? His Eskimos consumed about 280 gm. of protein, 135 gm. of fat, and 54 gm. of carbohydrate of which more than half is obtained from the glycogen of the meat. This seems a likely explanation.

If then a low carbohydrate, low protein, and high fat diet lowers the tolerance to carbohydrate in normal human beings, is it reasonable to assume that a high carbohydrate diet will raise the tolerance?

This too has been demonstrated. John (11) performed glucose tolerance tests on two normal subjects to whom he gave 100 gm. of glucose on 5 successive days. And although the peak of the blood sugar curve was 260 mg. on the 1st day, the maximum was only 90 mg. on the 5th day. Traugott's (12) experiments concur with this work. He gave an initial dose of 20 gm. of glucose, obtaining an increase in the height of the blood sugar curve. When, an hour later, after the first 20 gm. additional amounts of glucose were given in various quantities, from 20 to 100 gm., no hyperglycemia was noted. Apparently the initial stimulus of 20 gm. evoked sufficient amounts of sugar-metabolizing hormone, to take care of the additional quantities of glucose given later.

Such experiments and the ones presented above lead to the belief that in normal human beings, the quantity of insulin produced is dependent upon the amount of carbohydrate ingested irrespective of whether this foodstuff is administered as such or derived from the protein fraction of the diet.

1 These figures are based on the analytical data of Krogh and Krogh (10), and are acceptable as Heinbecker points out that the statements regarding the Greenland Polar Eskimo, the peoples studied by the Kroghs apply to Baffin Island Eskimos, the group studied by him.
Carbohydrate Tolerance

The question of the effect of ketosis on the glucose tolerance has not been discussed as that phase of the subject has been presented fully and clearly by Malmros (4).

SUMMARY AND CONCLUSIONS.

Two normal men were given glucose tolerance tests after both had lived for 1 year on lean and fat meat exclusively, (protein 120 gm., 2600 to 3000 calories) and later after a general diet. Following the meat diet there was a diminution of the tolerance to glucose as demonstrated by the blood sugar curve of both men and a glycosuria of one man. After 2 to 4 weeks of a general diet the blood sugar curve presented no abnormalities and the urine was sugar-free. The explanation of this phenomenon is that the normal carbohydrate mechanism needs daily stimulation for good function. Should that stimulus be lacking as is the case in low carbohydrate, high fat diets, and in prolonged fasting, it is temporarily incapable of handling large quantities of carbohydrate. In normal human beings this mechanism recovers fully after a general diet. These data are of practical importance in pointing out a probable fallacy in the interpretation of the glucose tolerance test, when the factors here discussed are not considered.

BIBLIOGRAPHY.

THE EFFECT OF AN EXCLUSIVE MEAT DIET LASTING ONE YEAR ON THE CARBOHYDRATE TOLERANCE OF TWO NORMAL MEN
Edward Tolstoi


Access the most updated version of this article at http://www.jbc.org/content/83/3/747.citation

Alerts:
• When this article is cited
• When a correction for this article is posted

Click here to choose from all of JBC's e-mail alerts

This article cites 0 references, 0 of which can be accessed free at http://www.jbc.org/content/83/3/747.citation.full.html#ref-list-1