THE DIFFERENT EFFECTS OF ORAL SUCROSE AND GLUCOSE ON ALIMENTARY LIPAEMIA

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SUMMARY

1. A formula breakfast containing protein, carbohydrate and fat was given on two occasions to nine middle-aged male convalescent patients and to ten young men. The meals differed only in the type of carbohydrate given; sucrose or an isocalorific amount of glucose.

2. After the formula meal containing sucrose; (a) the alimentary lipaemia was cleared more slowly; (b) insulin response was smaller, and (c) there was a lower blood sugar curve than after the glucose meal.

3. The degree of lipaemia showed a significant positive correlation with insulin response which was, as expected, lower after sucrose than glucose.

While attempting to confirm a report (Schilling, Hashim & Leonardy, 1964) that serum triglycerides are not significantly elevated after the ingestion of a small mixed meal, it was noticed that the triglyceride concentrations seemed to depend on the type of carbohydrate given (Mann & Truswell, 1971). Albrink, Fitzgerald & Mann (1958) and Sullivan (1960) have shown that the lipaemia which occurs after a fatty meal is diminished by the addition of glucose to the meal. Krut & Barsky (1964) found that postprandial lipaemia in patients with ischaemic heart disease is decreased by intravenous infusion of glucose and insulin.

These considerations led us to examine the effects of glucose and sucrose and subsequent insulin release on alimentary lipaemia.

SUBJECTS AND METHODS

Nine middle-aged men (aged 30–58 years) and ten younger men (20–25 years) participated in the study. The older men were studied in the metabolism ward of Groote Schuur Hospital, and were all convalescent patients who had previously been admitted to hospital for non-metabolic illnesses. At the time of testing all had normal erythrocyte sedimentation rates and liver function tests, their weights were stable and they had all been on an ordinary hospital diet for at least 3 weeks before testing.

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least 2 weeks. The ten younger men were medical students and one of the authors, and were on diets of their own choice. They were not admitted to hospital. Informed consent was obtained from all subjects.

Two formula breakfasts (Table 1), providing the same total calories (565 kcal) and proportions of the proximate food constituents, were given to each of the volunteers. Protein was given in the form of powdered egg white and fat as sunflower seed oil. The meals differed only in their carbohydrate content: one contained glucose (meal G) and the other an isocalorific amount of sucrose (meal S). The dry constituents were mixed with 200 ml of warm water in a ‘Waring Blender’, approximately 0·5 h before the formula was ingested. The glucose and sucrose test meals were given in a randomized order at 8 a.m. on two different days, usually 2 days apart. As a ‘control’ experiment, five subjects in the metabolism ward were given 100 g of glucose and sucrose in turn without the other components of the test meal. All subjects fasted for 12 h before undergoing tests.

### Table 1. The formula breakfasts

<table>
<thead>
<tr>
<th>Meal G</th>
<th>Meal S</th>
<th>Composition (Watt &amp; Merrill, 1963)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Powdered egg white, 30 g</td>
<td>Powdered egg white, 30 g</td>
<td>Protein, 24 g</td>
</tr>
<tr>
<td>Sunflower seed oil, 25 g</td>
<td>Sunflower seed oil, 25 g</td>
<td>Fat, 25 g</td>
</tr>
<tr>
<td>Glucose, 60 g</td>
<td>Sucrose, 60 g</td>
<td>Carbohydrate, 60 g</td>
</tr>
</tbody>
</table>

A fasting blood sample was taken on each occasion at 7.40 a.m. This was repeated at 2 and 4 h after the ingestion of the formula in those subjects studied in the metabolism ward and at half-hourly intervals for 2·5 h in the ten younger men. The subjects were kept at rest during the experiment and did not smoke. Serum triglyceride was measured in the metabolism ward subjects in the first series of experiments. In the second series of experiments blood sugar and serum insulin were measured in addition to triglyceride.

Blood was spun down immediately and the serum frozen at −20° until the analyses were carried out. Serum triglyceride was measured by a modification (Young & Eastman, 1963) of the van Handel & Zilversmit (1957) method, with triolein (British Drug Houses Ltd, Poole, Dorset, U.K.) used as the standard. The samples taken from the ten younger subjects were also assayed for serum insulin by radioimmunoassay (Morgan & Lazarow, 1963). Pig insulin (Novo Research Institute, Copenhagen, Denmark) was used as standard and tracer. Antiserum was purchased from Wellcome Research Laboratories, Beckenham, Kent. Blood sugar was measured by the method of Hoffman (1937) adapted for the AutoAnalyzer.

The degree of lipaemia at each sampling was measured as the increase in triglyceride above the fasting value. The total lipaemic response, glycaemic stimulus and insulin response to the meal were calculated in mg-h or μunit-h by a modification of the method described for total insulin response by Perley & Kipnis (1965). The following formula was devised:

Lipaemic response (mg-h), glycaemic stimulus (mg-h) or insulin response (μunit-h)

\[
= 0.5 \left[ \frac{t^0 + t^{30}}{2} + \frac{t^{30} + t^{60}}{2} + \frac{t^{60} + t^{90}}{2} + \frac{t^{50} + t^{120}}{2} + \frac{t^{120} + t^{150}}{2} \right]
\]
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Where \( t \) = triglyceride, glucose or insulin increase above fasting concentration at the particular sampling interval, \( t^0 \) therefore would always = 0.

The significance of the difference between two responses was determined by Student's \( t \) test. The interrelationships between responses were determined by calculating the correlation coefficient, \( r \) (Snedecor, 1950).

RESULTS

Middle-aged subjects

The triglyceride results are shown in Fig. 1. The degree of lipaemia was significantly higher after meal S than after meal G at 2 and 4 h. Glucose and insulin concentrations were not measured in these subjects.

To test the effect of sucrose and glucose eaten without fat, five patients were given 100 g of sucrose and glucose on different mornings without the other components of the test meal. After sucrose alone mean serum triglyceride concentrations were 107 mg/100 ml fasting, 99 mg/100 ml at 2 h and 102 mg/100 ml at 4 h. The corresponding values after glucose alone were 97, 96 and 98 mg/100 ml.

Younger subjects

(i) Serum triglyceride. There were no significant differences in the degree of lipaemia for 1·5 h after meals G and S had been ingested (Fig. 2). At 2 and 2·5 h, however, values were significantly lower after meal G than after meal S, and tended to decrease towards fasting values. The total lipaemic response to the meal (Table 2) was significantly greater after meal S than after meal G (\( P<0·01 \)). Subjects 4 and 7 showed negligible lipaemic responses but the remaining eight all showed similar patterns.

(ii) Blood sugar. Concentrations of sugar in the blood were significantly higher at 0·5 and 1 h
after meal G than after meal S (Fig. 2), the glycaemic response (Table 2) being greater in all ten cases ($P < 0.005$).

(iii) Serum insulin. Serum insulin concentrations were significantly higher at 1 and 1.5 h after meal G than after meal S (Fig. 2) and the total insulin response (Table 2) was greater in all ten cases ($P < 0.001$).

(iv) Interrelationships between the triglyceride, blood sugar and insulin responses. There was a close inverse relationship between the insulin response and the total lipaemic response to the meal. The correlation coefficient, $r$, was $-0.66$, which was significant at the 1% level. There was also an inverse correlation between the glycaemic stimulus and the lipaemic response, but the correlation coefficient was $-0.43$ and not statistically significant.

The insulin response seemed to be related to the glycaemic stimulus and there was no significant difference between ratios of insulin response to glycaemic stimulus after either glucose or sucrose (see Table 3).
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Table 2. Total lipaemic response, glycaemic stimulus and insulin response after the glucose and sucrose test meals

<table>
<thead>
<tr>
<th>Subject No.</th>
<th>Lipaemic response (mg-h)</th>
<th>Glycaemic stimulus (mg-h)</th>
<th>Insulin response (μunit-h)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Glucose</td>
<td>Sucrose</td>
<td>Glucose</td>
</tr>
<tr>
<td>1</td>
<td>33.00</td>
<td>42.25</td>
<td>42.50</td>
</tr>
<tr>
<td>2</td>
<td>58.50</td>
<td>69.50</td>
<td>23.00</td>
</tr>
<tr>
<td>3</td>
<td>9.50</td>
<td>18.00</td>
<td>46.50</td>
</tr>
<tr>
<td>4</td>
<td>No lipaemia</td>
<td>32.50</td>
<td>26.50</td>
</tr>
<tr>
<td>5</td>
<td>25.00</td>
<td>43.25</td>
<td>31.00</td>
</tr>
<tr>
<td>6</td>
<td>4.50</td>
<td>35.75</td>
<td>47.50</td>
</tr>
<tr>
<td>7</td>
<td>No lipaemia</td>
<td>75.00</td>
<td>44.75</td>
</tr>
<tr>
<td>8</td>
<td>42.25</td>
<td>76.75</td>
<td>76.00</td>
</tr>
<tr>
<td>9</td>
<td>21.25</td>
<td>28.25</td>
<td>35.00</td>
</tr>
<tr>
<td>10</td>
<td>40.50</td>
<td>47.25</td>
<td>13.50</td>
</tr>
<tr>
<td>Mean</td>
<td>23.45</td>
<td>36.10</td>
<td>42.25</td>
</tr>
</tbody>
</table>

Table 3. The ratios insulin response/glycaemic stimulus after the glucose and sucrose formulae

<table>
<thead>
<tr>
<th>Subject</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
<th>Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glucose</td>
<td>2.52</td>
<td>2.78</td>
<td>2.42</td>
<td>1.32</td>
<td>1.81</td>
<td>1.91</td>
<td>0.81</td>
<td>1.05</td>
<td>2.43</td>
<td>2.52</td>
<td>1.96</td>
</tr>
<tr>
<td>Sucrose</td>
<td>2.86</td>
<td>2.62</td>
<td>2.72</td>
<td>1.58</td>
<td>1.91</td>
<td>2.23</td>
<td>0.56</td>
<td>1.04</td>
<td>2.20</td>
<td>2.48</td>
<td>2.02</td>
</tr>
</tbody>
</table>

DISCUSSION

This study has shown that after a formula meal similar in composition to a normal breakfast, the lipaemic response is greater when the carbohydrate component of the formula is sucrose rather than glucose.

We have considered three possible mechanisms for this.

1. Glucose might impair fat absorption (or sucrose might enhance it).
2. Fructose, derived from the sucrose in meal S might stimulate hepatic (Zakim & Herman, 1969) or intestinal (Nikkilä & Pelkonen, 1966) triglyceride synthesis and so add an endogenous component to the alimentary lipaemia.
3. Alimentary lipaemia might be cleared more rapidly in the presence of glucose.

There is no physiological basis for the first possibility which in addition would seem to be ruled out by our finding that the differences between meal S and meal G were only significant in the descending portion of the triglyceride curve (Fig. 2).

Endogenous triglyceride synthesis does not seem to affect human serum triglycerides under the conditions of this experiment. We found no significant change in serum triglycerides after 100 g of glucose or sucrose without fat and neither did Swan, Davidson & Albrink (1966). It would therefore seem most likely that the smaller degree of lapaemia after the meal containing glucose resulted from enhanced chylomicron clearance. Adipose tissue lipoprotein lipase,
which plays the major role in clearing chylomicron triglycerides after a fatty meal, is potentiated by insulin (Korn, 1955). Thus, in rats made diabetic with alloxan the enzyme activity is low and is restored to normal by insulin treatment (Kessler, 1963). Diabetic animals clear ingested or infused glycerides better when treated with insulin (Hirsch & Perl, 1966).

Glucoce has been shown to be a more potent stimulator of insulin than any other monosaccharide both in vitro (Grodsky, Batts, Bennett, Vcella, McWilliams & Smith, 1963; Mayhew, Wright & Ashmore, 1969) and in man, in whom oral fructose is much less effective (Swan et al., 1966; Aitken & Dunnigan, 1969). It would therefore be expected that glucose would stimulate a greater insulin response than an isocalorific amount of sucrose, which is digested to glucose and fructose. This was suggested by the work of Swan et al. (1966), who showed higher peak insulin concentrations after 100 g of oral glucose than after sucrose (both taken without other food). The present investigation shows a significantly greater insulin response to glucose when individual concentrations at 1 and 1·5 h or total insulin response are considered. The protein and fat in the formula meals presumably contributed to the insulin secretion as well but these two constituents were kept constant in the two meals and are weaker stimulators of insulin secretion than carbohydrate (Mayhew et al., 1969).

Infusion of insulin to hyperglyceridaemic patients results in decreased plasma glyceride concentrations as compared with control infusions (Schlierf & Kinsell, 1965). In this particular study protamine, a known inhibitor of lipoprotein lipase (Bragdon & Havel, 1954), prevented the insulin-induced decrease of glyceride without preventing the decrease in blood sugar and plasma free fatty acids which also resulted from the insulin infusion. This suggests that insulin lowers glyceride concentrations through increased removal of glyceride by peripheral tissue, rather than by decreased endogenous synthesis as a result of decreased fat mobilization from adipose tissue.

We therefore suggest that in our experiments the smaller degree of lipaemia seen when glucose was included in the mixed meal resulted from more rapid clearing of chylomicrons by lipoprotein lipase activity, which was enhanced by the larger insulin secretion after glucose than after sucrose.

Patients with ischaemic heart disease on average have a greater degree of lipaemia after a fat-containing meal than controls. There is also a direct relation between postprandial lipaemia and fasting serum triglyceride concentrations (Denborough, 1963). However, the relevance of our study to the pathogenesis of coronary heart disease must await more information on the epidemiology of serum triglycerides.

ACKNOWLEDGMENTS

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