SHORT COMMUNICATION

Effect of Peas (Pisum sativum) in the Treatment of Experimental non-insulin-dependent Diabetes

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A study was made on the mechanism by which the consumption of peas affects beneficially the glycaemia in a rat model of non-insulin-dependent diabetes. With a standard diet, the diabetic rats showed elevated glycaemia throughout the study, varying between 8.3 and 10.0 mmol/L. With the administration of a diet of peas, the glycaemia in the diabetic rats declined significantly from the second day, and stayed at levels near normal for the rest of the study. A significant inhibitory activity of the pancreatic amylase enzyme was detected in the raw pea extract. A possible mechanism of the hypoglycaemic effect of a diet of peas might therefore reside partially in its inhibitory effect on carbohydrate digestion. © 1997 by John Wiley & Sons, Ltd.

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day 10 to approximately 80%, and to around 85% on day 20, being maintained even 6 days after ending the diet period. The standard-diet-fed normal rats presented no significant variations in glycaemia. In the standard-diet-fed diabetic rats, the levels of glycaemia stayed at around 8.33–10.0 mmol/L, with small fluctuations throughout the study. In the pea-fed diabetic rats, there was a significant ($p<0.05$) fall in the levels of glycaemia at 2–3 days of treatment, then a maintenance of the levels close to the controls (4.4 mmol/L) for the rest of the study, the descent in glycaemia values relative to the untreated rats being approximately 4.4–5.0 mmol/L. In the pea-fed diabetic rats, a reduction of 50% in the levels of glycaemia was observed on day 10, and then remained at these levels for the remainder of the treatment period and even for 6 days posttreatment, although a slight rising tendency was observed. After the end of the diet period, the glycaemia levels stayed in this range even though the rats had gone over to a standard feed diet. In the standard-diet-fed diabetic rats, there was a slight, non-significant, decline in the percentage reduction of the glycaemia which was maintained throughout the study.

Table 1. Weight increment (g/day), fluid intake (mL/day), and food intake (g/day) in normal and diabetic rats given a standard diet and a pea-based diet for 21 days.

<table>
<thead>
<tr>
<th></th>
<th>Normal</th>
<th>Diabetic</th>
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<tbody>
<tr>
<td>Weight increase (g/day)</td>
<td>0.95±0.00</td>
<td>0.70±0.09</td>
</tr>
<tr>
<td>Peas</td>
<td>1.39±0.13a</td>
<td>0.67±0.14b</td>
</tr>
<tr>
<td>Water intake (mL/day)</td>
<td>34.7±1.80</td>
<td>72.3±1.5b</td>
</tr>
<tr>
<td>Peas</td>
<td>29.9±0.7a</td>
<td>32.6±1.4a</td>
</tr>
<tr>
<td>Food intake (g/day)</td>
<td>23.1±0.7</td>
<td>28.9±0.8b</td>
</tr>
<tr>
<td>Peas</td>
<td>25.6±0.4a</td>
<td>23.8±0.8a</td>
</tr>
</tbody>
</table>

$^a p<0.05$ vs standard, $^b p<0.01$ vs standard, $^c p<0.05$ vs normal, $^d p<0.01$ vs normal ($n=6$ for each group).

DISCUSSION

Recent years have seen a recognition of the importance of legumes in the diabetic patient’s diet (Southgate, 1990; Jenkins et al., 1981; Hansen et al., 1992; Karlstrom et al., 1987). Pulses may exert their action via other mechanisms which might be of more interest than the fibre itself in delaying intestinal glucose absorption. Several studies have shown amylase inhibitors purified from white beans and administered to diabetic patients to exert beneficial effects (Layer et al., 1985; Layer et al., 1989; Boivin et al., 1988).

Today, its inhibitory potential has been totally characterized and studied, as well as its effect on digestion, and its activity on the pancreatic exocrine function (Koike et al., 1995). Our study found that the glycaemia of diabetic rats was normalized by giving them an exclusive diet of lyophilized peas. This improvement was not due to a reduction in body weight. The weight gain was similar in diabetic rats treated with the pea diet and those with a conventional diet, but there was nevertheless a clear reduction to 50% of the initial value of the diabetic rats’ glycaemia. Furthermore, there was a reduction in the intake of food and water in diabetic rats, as a consequence of the improvement in the pea-fed diabetic state. In the non-diabetic rats, the weight gain was greater in those given the pea diet than in those receiving a conventional diet, but here too we observed a 20% reduction in glycaemic levels relative to the beginning of the study. As was noted in Materials and Methods, there were no significant differences in either the energy content of the composition of the two diets that could clearly suggest that therein lay the cause of the effect observed with the pea diet. Even the total fibre content (g/100 g) was similar (standard diet 5 and pea diet 5). It is also known that in the peas, only 2.3 g of the total fibre is soluble fibre (Terán, 1994), contained mainly in the cotyledon (Hansen et al., 1992), and that it is only soluble fibre which reduces postprandial glycaemia (Elsenhans and Caspary, 1988; Johnson et al., 1981; Jenkins et al., 1981; Anderson and Okanj, 1991). This, together with the demonstration of the presence of inhibitory activity against pancreatic amylase in
the raw pea extract, allows our results to be taken as compatible with the presence in the peas of natural inhibitors of intestinal carbohydrate digestion.

REFERENCES