Note
Effect of Dietary Sodium Phytate on the Hepatic and Serum Levels of Lipids and on the Hepatic Activities of NADPH-generating Enzymes in Rats Fed on Sucrose

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The effect was investigated of dietary 0.5% sodium phytate on the hepatic and serum lipid status in rats fed on sucrose for 29 or 30 days. The increase in hepatic weight, levels of hepatic total lipids, triglyceride, cholesterol, serum triglyceride and phospholipid, and the activities of hepatic NADPH-generating enzymes due to sucrose feeding were generally depressed by dietary sodium phytate.

Phytic acid (myo-inositol hexaphosphoric acid) is an abundant plant constituent, constituting 1–5% by weight of edible legumes, cereals, oil seeds, pollens, and nuts. Until recently, most published reports on phytic acid have focused on concern over possible decreased mineral bioavailability. However, little attention has been paid to other nutritional effects of phytate. Our studies have recently demonstrated that increases in the hepatic and serum levels of lipids and in the hepatic activities of lipogenic enzymes such as glucose-6-phosphate dehydrogenase (G6PD, EC 1.1.1.49) and malic enzymes (ME, EC 1.1.1.40) that had been caused by sucrose feeding were clearly depressed by dietary myo-inositol. To investigate whether dietary phytate would act similarly to dietary myo-inositol, the effect of dietary sodium phytate on the hepatic and serum levels of lipids and on the hepatic activities of NADPH-generating enzymes in rats fed on sucrose was examined in the present study.

Male rats of the Wistar strain (Hiroshima Laboratory Animal Center, Hiroshima) weighing 76 g were divided into three groups (six animals per group). The animal room was maintained at 24 ± 1°C and illuminated for 12 h from 8.00 a.m. Diets and tap water were supplied ad libitum for 29 or 30 days. The basal diet was composed of 20% casein, 65% carbohydrate (x-corn starch or sucrose), 5% corn oil, 5% cellulose powder, 3.5% mineral mixture (AIN-76), 1% vitamin mixture (AIN-76), 0.3% dl-methionine and 0.2% choline bitartrate. The dietary addition of 0.5% sodium phytate was made at the expense of sucrose. Sodium phytate was purchased from Naaiatal Tesque, Kyto (purity ≥ 95%). At the end of the experimental period, the diets were removed from the cages at 8:00 a.m. and the rats were anesthetized with diethyl ether and killed (1:00 p.m.–3:00 p.m.). Half of the rats were killed on day 29 and the other half were killed on day 30. Blood was collected by heart puncture in a syringe and serum samples were isolated by centrifugation. The liver was homogenized in 9 volumes of 0.14 M KCl (pH 7.0). The homogenate was centrifuged at 105,000 × g for 60 min, and the resulting supernatant was stored at −30°C until needed for assays of G6PD, ME, and 6-phosphogluconate dehydrogenase (6PGD, EC 1.1.1.44). Enzyme activity is expressed as the amount of NADPH formed. Protein was measured by the method of Lowry et al., using bovine serum albumin as a standard. Liver lipids were extracted by the method of Folch et al., and liver triglyceride was determined by the method of Danno et al. Liver cholesterol, phospholipid and total lipids, and serum lipids were determined as described previously. The statistical significance of differences between the values of data was analyzed by ANOVA and Duncan’s multiple-range test.

As shown in Table, growth and food intake were not affected by the dietary manipulation. Consistent with previous reports, the sucrose-fed rats showed significant increases in hepatic weight, in the levels of hepatic total lipids, triglyceride, cholesterol, serum triglyceride and phospholipid, and in the hepatic activities of G6PD, ME, and 6PGD (Table). Of special interest was the finding that the rises in hepatic weight, hepatic levels of total lipids and triglyceride, and hepatic activities of these NADPH-generating enzymes due to sucrose feeding was significantly depressed by dietary sodium phytate (Table). A similar depressing effect of phytate was also observed in the hepatic levels of cholesterol and in the serum levels of triglyceride and phospholipid in rats fed on sucrose (Table). On the other hand, liver phospholipid and serum cholesterol were not apparently affected by the dietary carbohydrate source and phytate (Table). These results suggest that dietary phytate can protect sucrose-fed rats from hyperlipidemia.

Table
Effect of Dietary Sodium Phytate on the Hepatic and Serum Levels of Lipids and on the Hepatic Activities of NADPH-generating Enzymes in Rats Fed on Sucrose

<table>
<thead>
<tr>
<th>Groups</th>
<th>Corn starch (g/day, on days 26–28)</th>
<th>Sucrose (g/day, on days 26–28)</th>
<th>Sucrose + phytate (g/day, on days 26–28)</th>
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<tr>
<td>Liver weight</td>
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<td>(% of body wt.)</td>
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<td>Liver total lipids (mg/g of tissue)</td>
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<tr>
<td>Liver triglyceride (mmol/g of tissue)</td>
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<td>Liver cholesterol (mmol/g of tissue)</td>
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<tr>
<td>Liver phospholipid (mmol/g of tissue)</td>
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<tr>
<td>Liver G6PD (μmol of protein)</td>
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<td>Liver ME (μmol of protein)</td>
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<tr>
<td>Liver 6PGD (μmol of protein)</td>
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<tr>
<td>Serum triglyceride (mmol/l)</td>
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<td>Serum cholesterol (mmol/l)</td>
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<td>Serum phospholipid (mmol/l)</td>
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</tbody>
</table>

1 Initial body weight was an average of 76 g (62–87 g), and the feeding period was 29 or 30 days.
2 Mean ± SE (N=6). Means not followed by the same letter are significantly different (p<0.05).
3 1 μU enzyme = 1 nmol of NADPH formed per minute at 25°C.
animals against an accumulation of hepatic lipids.

Jariwalla et al.\textsuperscript{13,14} have recently shown that the addition of 9% potassium phytate to a diet for 6 weeks lowered the serum cholesterol and triglyceride levels in rats. However, the high level of phytate (9%) used in their study is not practical for human nutrition. The present study suggests that a much lower level of dietary phytate (0.5%) may affect hepatic lipids and the activities of lipogenic enzymes such as NADPH-generating enzymes rather than the serum levels of lipids. By virtue of binding to proteins, phytic acid has been found to inhibit such enzymes as polyphenol oxidase, alcohol dehydrogenase and trypsin.\textsuperscript{21} Thus, further studies are in progress on the effect of dietary phytate on intestinal sucrose activity in rats fed on sucrose to investigate the underlying mechanism for this phenomenon. The nutritional status of such minerals as copper, zinc, and magnesium affects lipid metabolism,\textsuperscript{14,15} and it is also necessary to evaluate the effect of dietary phytate on these minerals under the present experimental conditions.

It is known that some plant proteins such as those from soy protein contain endogenous phytate.\textsuperscript{5,16} Hunter\textsuperscript{16} has mentioned that the endogenous phytate content of a soy protein diet is roughly equivalent to the 0.5% level in the diet if the soy protein is supplied at around 20–24% by weight of the diet, although the dietary level may vary with the purity of the soy protein. In addition, these plant proteins generally lower the hepatic and serum levels of lipids and of hepatic lipogenic enzymes including NADPH-generating enzymes, when compared with animal proteins.\textsuperscript{17–19} These facts together with the results of the present study imply that the depressing effect of plant proteins on hepatic and serum lipids and on hepatic lipogenesis might be in part related to the endogenous phytate in plant proteins, especially when the purity of these plant proteins is not very high.

References