Protective Effect of Riboflavin on Suppression of Growth Caused by Oxidized Oil

Yasuko Saito, Nobuko Ohishi, and Kunio Yagi

Institute of Biochemistry, Faculty of Medicine, University of Nagoya, Nagoya 466, Japan

(Received July 19, 1980)

Summary When oxidized corn oil (100 nmol in terms of malondialdehyde/day/rat) was administered to a riboflavin-deficient rat, the body weight gain was markedly suppressed. However, when 20 μg of riboflavin/day/rat was administered with the oxidized corn oil, reasonable growth and normal flavin levels in the liver, kidney and heart could be attained, though they were somewhat less than those of the animals fed on the normal diet containing non-oxidized corn oil. It was noted that the elevation of lipid peroxide level in blood plasma of animals administered with the oxidized oil was effectively prevented by riboflavin. These results indicate the protective effect of riboflavin on suppression of growth caused by the oxidized oil.

Key Words riboflavin, lipid peroxides
separately and mixed it with the diet just before administration. The present paper deals with the growth-suppression of rat by the oxidized oil and its protection by simultaneous administration of riboflavin.

MATERIALS AND METHODS

Male Wistar strain rats weighing about 50 g were used. Rats were divided into 4 groups according to the administration of riboflavin and of the oxidized corn oil: group A was administered with 20 μg riboflavin/rat/day, group B with riboflavin-deficient diet, group C with 100 nmol (in terms of malondialdehyde) lipid peroxide and 20 μg riboflavin/rat/day, and group D with 100 nmol lipid peroxide/rat/day and riboflavin-deficient diet. Rats were housed individually and bred for 4 weeks under controlled conditions. The experimental diet was prepared according to Forker and Morgan (7) except that fresh or oxidized corn oil was used in place of soybean oil. Vitamin B complex with or without riboflavin, and fat-soluble vitamins were compulsorily administered daily.

To obtain the oxidized corn oil containing lipid peroxides, commercially available corn oil was irradiated with a fluorescent lamp (F1 15SW, NEC, Tokyo) in the presence of resin-bound Rose-Bengal (8) as catalyzer, which was filtered off after irradiation. The amount of lipid peroxide was checked by the absorption of the conjugated diene at 233 nm, the peroxide value being obtained by iodometry and also by thiobarbituric acid value (9). The amount of lipid peroxides administered was expressed in terms of malondialdehyde measured by thiobarbituric acid reaction with tetramethoxypropane as external standard (9).

Vitamin-free casein was purchased from ICN NBC Laboratories, Inc., Cleveland, and other chemicals were obtained from Nakarai Chemical Co., Kyoto.

The levels of flavin in the liver, kidney and heart were measured by the lumiflavin fluorescence method according to Yagi (10). Lipid peroxide levels in blood plasma were assayed by the method of Yagi (11). After lipid peroxide was sedimented with protein, the reaction with thiobarbituric acid was made, and the resulting red pigment was measured by its fluorescence. Using tetramethoxypropane as external standard, the lipid peroxide level was expressed in terms of nmol of malondialdehyde per ml of blood.

RESULTS AND DISCUSSION

Figure 1 shows the growth curve. The rats of group A, control group, grew normally (curve A). The growth of the animals of group B, administered with riboflavin-deficient diet, almost ceased after a few days of breeding, as reported from many laboratories. When the oxidized corn oil containing lipid peroxides was administered to rats fed on riboflavin-deficient diet daily (100 nmol/day/rat), the body weight gain was markedly reduced (see curves B and D). After breeding for 22 days, the body weight gain was only 1/4 of that of riboflavin-deficient rats. The
Fig. 1. Body weight gain of rats. The mean values of body weight gain (n=5) were plotted. Administration of riboflavin: A and C, 20 μg/day/rat; B and D, riboflavin-deficient. Administration of lipid peroxide: A and B, fresh corn oil; C and D, oxidized corn oil (lipid peroxide content, 100 nmol in terms of malondialdehyde/day/rat).

Table 1. Flavin levels in various organs of rats.*

<table>
<thead>
<tr>
<th>Group</th>
<th>Total flavins (μg/g)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Liver</td>
</tr>
<tr>
<td>A</td>
<td>31.6 ± 2.26</td>
</tr>
<tr>
<td>B</td>
<td>15.0 ± 0.61</td>
</tr>
<tr>
<td>C</td>
<td>29.5 ± 1.33</td>
</tr>
<tr>
<td>D</td>
<td>12.5 ± 0.85</td>
</tr>
</tbody>
</table>

* The amounts of flavins are expressed in terms of riboflavin. Data are expressed as mean ± S.E. (n = 5).

features of rats in group D became those of severe ariboflavinosis. On the other hand, the daily administration of the oxidized corn oil with 20 μg of riboflavin for 22 days brought about fairly well body weight gain, even though the body weight gain at the mean time was less than that of the control (see curves A and C).

The levels of flavins in the liver, kidney and heart are shown in Table 1. The
level of flavins in each organ decreased with the administration of riboflavin-deficient diet, as usual. The administration of lipid peroxides tends to decrease the flavin levels, and the degree of decrease was more marked for the rats administered with riboflavin-deficient diet.

Checking the body weight gain and flavin levels in the body, it was found that they are in parallel. It seems, therefore, that the suppression of growth by the oxidized oil could be accounted for, at least partly, by the decrease in flavin levels.

As shown in Table 2, the lipid peroxide level in blood plasma was elevated after breeding with riboflavin-deficient diet for 22 days (compare group A with B). This elevation was strengthened by daily administration of the oxidized corn oil (group D). It should be noted that the lipid peroxide level of animals fed on the oxidized oil was maintained at the same level as that of control animals if riboflavin was administered simultaneously (compare group A with C).

Although somewhat different views were presented as to the absorption of lipid hydroperoxides through the small intestine (12–18), the present observation that the lipid peroxide level in blood plasma was increased by the administration of the oxidized oil should be taken into account in considering the chronic toxicity of the oxidized oil, which is actually contained in some processed foods. Since the presently adopted oxidized oil possesses a similar nature to that contained in processed foods, the obtained data can be safely applied to the practical cases. However, the problem of which component of the oxidized oil, viz., hydroperoxides, aldehydes or ketones, is responsible for the observed chronic toxicity should be examined by testing each of these components. The experiments in this line of study are under progress in our laboratory. Nevertheless, the effect of riboflavin in decreasing the level of lipid peroxides in blood plasma and in protecting the suppression of growth of animals administered with the oxidized oil should be noted from both nutritional and clinical points of view.

REFERENCES


