Increased Excretion of Fecal Fat in Hyperlipidemic Patients

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NAGASAKI, A. and MARUHAMA, Y. Increased Excretion of Fecal Fat in Hyperlipidemic Patients. Tohoku J. exp. Med., 1980, 131 (1), 99-100 — Total fat balance was studied in 13 healthy controls and 14 patients with primary hyperlipidemia by estimating dietary and fecal fat. Although no difference in fat intake was noted between hyperlipidemics and controls, fecal fat excretion in hyperlipidemics increased significantly as compared with that in controls. The increase in fat excretion was greater in patients with type IIa hyperlipidemia (hypercholesterolemia) than in those with types IIb and IV hyperlipidemia (endogenous hyperlipidemia). hyperlipidemia; fecal fat; fat balance study

The lipoprotein metabolism in circulating blood has been studied intensively in recent years. As a result, it has been shown that both genetic and nutritional factors are important in the pathogenesis of hyperlipidemia. However, little is known about the behavior of lipid in the alimentary tract in hyperlipidemia. In the present study, therefore, dietary fat intake and fecal fat excretion were estimated in hyperlipidemics in order to see the total fat balance in alimentary tract in the hyperlipidemic stage. 13 healthy controls (8 males and 5 females) and a total of 14 patients with primary hyperlipidemia (6 males and 8 females) were examined for dietary intake during the sequential 3 days prior to sampling of serum and feces according to the quantitative diet record method as described previously (Maruhama et al. 1977). The daily fat intake was calculated in each subject by an experienced dietitian according to the Japanese food composition table (Ministry of Science and Technology 1969). Serum triglyceride (Fletcher 1968), total cholesterol (Zlatkis and Zak 1969) and lipoproteins (Noble 1968) were determined after an overnight fast, and the total feces excreted during the day was collected and homogenized in a vinyl bag, a portion of the sample being used for analysis of total fat (Van de Kamer 1949). Hyperlipidemia was classified by lipoprotein phenotypes according to the WHO criteria (WHO Committee 1970). Since the mechanisms of type IIb and type IV hyperlipidemia are known to be very near as endogenous hyperlipidemia, the patients with these types of hyperlipidemia were combined.

As shown in Table 1, no significant difference in age was noted between hyperlipidemics and controls, although the mean age of type IIa patients was slightly higher than that of controls. Relative body weight was significantly greater in patients with either type IIb or type IV hyperlipidemia than in controls. Serum lipid profiles were compatible with each type of hyperlipidemia, showing a marked elevation of total cholesterol in type IIa patients and an increase in triglyceride with and without elevated cholesterol in types IIb and IV patients. Daily fat intake was about 40 g on an average both in hyperlipidemics and controls and no difference in fat intake was found between the groups. Fecal fat was significantly increased in hyperlipidemics as compared with controls. The fat excretion in type IIa patients was found to be most increased, being twice as high as that in controls. Miettinen et al. (1967) reported a low excretion of fecal bile acids in type IIa patients, and Kottke (1963) confirmed the decreased bile acids in duodenal contents in type IIa patients but not in type IIb or IV patients. These authors suggested that the decreased bile

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99
### Table 1. Clinical and laboratory data of the subjects

<table>
<thead>
<tr>
<th>Group</th>
<th>Age (years)</th>
<th>RBW (%)</th>
<th>Serum lipids</th>
<th>Dietary fat (g/day)</th>
<th>Fecal fat (g/day)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control (n=13)</td>
<td>44±13</td>
<td>96±5</td>
<td>114±14</td>
<td>170±7</td>
<td>41±2</td>
</tr>
<tr>
<td>Hyperlipidemia</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Type IIa (n=7)</td>
<td>54±12</td>
<td>112±7</td>
<td>89±12</td>
<td>331±35*</td>
<td>37±3</td>
</tr>
<tr>
<td>Types IIb, IV (n=7)</td>
<td>48±10</td>
<td>123±6*</td>
<td>325±78*</td>
<td>250±17*</td>
<td>40±2</td>
</tr>
<tr>
<td>Total (n=14)</td>
<td>51±11</td>
<td>117±5*</td>
<td>207±50</td>
<td>291±22*</td>
<td>38±2</td>
</tr>
</tbody>
</table>

RBW, (relative body weight) = actual body weight/standard body weight × 100. Standard body weight = (height in cm − 100) × 0.9 kg; TG, triglyceride; TC, total cholesterol. Grouping of hyperlipidemia was based on WHO phenotyping (see text). Figures express mean±s.e. * p<0.01, † p<0.05 against control.

Acid excretion is the result of deranged cholesterol catabolism in the liver in type IIa patients. The present results, coupled with the observations of the above authors, may suggest a decreased rate of fat absorption in type IIa patients, as the bile acids are known to be important in the facilitation of fat absorption (Davenport 1977). However, this assumption seems unlikely since the type IIa patients could well tolerate fatty meal and showed no symptoms of chronic fat malabsorption. Furthermore, when the individual data of fecal fat were plotted against the amounts of dietary fat taken, there was no significant positive correlation between them in type IIa patients (r=-0.55), as in controls (r=-0.09), nor in types IIb and IV patients (r=0.25). Thus, the fecal fat seems to increase in type IIa patients independently of the amount of intake and there seems to be no actual disturbance in fat absorption in these patients. It is likely that the observed increase in fecal fat in hyperlipidemics reflects an increase in the excretion of endogenous fat into the alimentary tract. However, why more fat is excreted in type IIa patients than in types IIb and IV patients remains obscure.

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**References**