Responses of Serum Lipoproteins to Dietary Cholesterol and Type of Fat in the Baboon

Henry C. McGill, Jr., C. Alex McMahan, Arthur W. Kruski, Jim L. Kelley, and Glen E. Mott

We examined the effects of dietary cholesterol (<0.01 and 1.7 mg/Kcal) and type of fat (saturated, coconut oil; polyunsaturated, corn oil) on very low density plus low density lipoprotein (VLDL + LDL) and high density lipoprotein (HDL) cholesterol in 24 young baboons (12 male, 12 female) (Papio sp.) in a crossover design experiment. The oils contributed 40% of calories. Total serum cholesterol concentration on the low cholesterol-polyunsaturated fat diet averaged 120 mg/dl; on the high cholesterol-saturated fat diet, 245 mg/dl; and on the other two cholesterol-fat diet combinations, about 200 mg/dl. There was a significant interaction between cholesterol and type of fat in their effects on VLDL + LDL cholesterol, but not in their effects on HDL cholesterol. Dietary cholesterol elevated VLDL + LDL cholesterol more when fed with polyunsaturated fat than with saturated fat. Dietary cholesterol elevated VLDL + LDL cholesterol when dietary cholesterol was low, but not when dietary cholesterol was high. Saturated fat consistently elevated HDL cholesterol more than did dietary cholesterol. The response of apolipoprotein B concentrations to dietary components was similar to that of VLDL + LDL cholesterol. These results indicate that dietary cholesterol and type of fat have different effects on the distribution of cholesterol among the major serum lipoproteins of the baboon.

(Arteriosclerosis 5:337-344, September/October 1981)
Table 1. Nutrient Composition and Ingredients of Diets

<table>
<thead>
<tr>
<th>Nutrients</th>
<th>Chow*</th>
<th>Low cholesterol-polyunsaturated fat</th>
<th>Low cholesterol-saturated fat</th>
<th>High cholesterol-polyunsaturated fat</th>
<th>High cholesterol-saturated fat</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carbohydrate (% Kcal)</td>
<td>62</td>
<td>40</td>
<td>40</td>
<td>39</td>
<td>39</td>
</tr>
<tr>
<td>Protein (% Kcal)</td>
<td>28</td>
<td>20</td>
<td>20</td>
<td>21</td>
<td>21</td>
</tr>
<tr>
<td>Fat (% Kcal)</td>
<td>10</td>
<td>40</td>
<td>40</td>
<td>40</td>
<td>40</td>
</tr>
<tr>
<td>Energy (Kcal/100 g)</td>
<td>329</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td>1.7</td>
<td>1.7</td>
</tr>
<tr>
<td>Cholesterol (mg/Kcal)</td>
<td>0.03</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Ingredients (% dry wt)

<table>
<thead>
<tr>
<th>Ingredient</th>
<th>Chow mixture†</th>
<th>Chow</th>
<th>LCPF</th>
<th>LCSF</th>
<th>HCPF</th>
<th>HCSF</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chow mixture</td>
<td></td>
<td>81.8</td>
<td>81.8</td>
<td>79.1</td>
<td>79.1</td>
<td></td>
</tr>
<tr>
<td>Egg yolk, dried</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>4.8</td>
<td>4.8</td>
<td></td>
</tr>
<tr>
<td>Coconut oil</td>
<td>0</td>
<td>17.0</td>
<td>0</td>
<td>14.3</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Corn oil</td>
<td>0</td>
<td>17.0</td>
<td>0</td>
<td>14.3</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>NaCl</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Retinyl acetate</td>
<td>0.001</td>
<td>1.1</td>
<td>1.1</td>
<td>1.1</td>
<td>1.1</td>
<td></td>
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<tr>
<td>Ascorbic acid</td>
<td>0.01</td>
<td>0.01</td>
<td>0.01</td>
<td>0.01</td>
<td>0.01</td>
<td></td>
</tr>
<tr>
<td>Cholesterol</td>
<td>0.01</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0.6</td>
<td></td>
</tr>
</tbody>
</table>

* Baboon Chow manufactured by Ralston Purina Company, St. Louis, Missouri.
† Purina Monkey Meal 25-5045-6, Special Mix with no added fat, dehydrated alfalfa, NaCl, ascorbic acid, or retinyl acetate.

Diets

We prepared four diets which provided two levels of cholesterol and two types of fat in all combinations (Table 1). Analyses of the oils by gas liquid chromatography showed that the corn oil (Mazola, Best Foods Company, Atlanta, Georgia) contained 12.6% saturated, 24.9% monounsaturated, and 62.4% polyunsaturated fatty acids. Coconut oil (white coconut oil, Lou Ana Foods, Opelousas, Louisiana) contained 90.9% saturated, 6.7% monounsaturated, and 1.4% polyunsaturated fatty acids. The mixed diets were pelleted and stored in a freezer; each animal received 500 g daily, an amount that in our experience has proved more than adequate to maintain growth.

Table 2. Diet Sequences Assigned to Each of Four Groups of Animals in Each of Five 6-Week Time Periods

<table>
<thead>
<tr>
<th>Period</th>
<th>Diet sequence</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Chow Chow Chow Chow</td>
</tr>
<tr>
<td>1</td>
<td>LCPF HCSF LCSF HCPF</td>
</tr>
<tr>
<td>2</td>
<td>LCSF HCPF HCSF HCPF</td>
</tr>
<tr>
<td>3</td>
<td>HCPF LCSF CPDF HCSF</td>
</tr>
<tr>
<td>4</td>
<td>HCSF LCPF HCPF LCSF</td>
</tr>
<tr>
<td>5</td>
<td>LCSF HCSF LCPF HCPF</td>
</tr>
</tbody>
</table>

Diet code: LC = low cholesterol; HC = high cholesterol; PF = polyunsaturated fat (corn oil); SF = saturated fat (coconut oil).

Experimental Design

Each of 24 animals received four diets in a crossover design (Table 2). Each assigned diet was given for 6 weeks. We assigned the animals randomly within sex to one of the four diet sequences so that each diet sequence contained three males and three females.

Blood Collection

We collected venous blood in vacutainers from each baboon after an overnight fast and under ketamine anesthesia (Vetalar, Parke, Davis and Company, Detroit, Michigan) after 4, 5, and 6 weeks on each diet.

Lipoprotein-Cholesterol Analyses

High density lipoprotein (HDL) cholesterol was measured in the supernatant solution after precipitation of the very low density plus low density lipoproteins (VLDL + LDL) by the dextran sulfate CaCl₂ procedure. VLDL + LDL cholesterol was calculated as the difference between whole serum cholesterol and HDL cholesterol. Cholesterol was measured by an enzymatic method using the ABA 100 Bichromatic Analyzer (Abbott Laboratories, South Pasadena, California). Analyses were performed in blind duplicate. The coefficients of variation between duplicates were 2.1% for VLDL + LDL cholesterol and 0.8% for HDL cholesterol.

The precipitation procedure was validated by comparison with the separation of lipoprotein density classes by preparative ultracentrifugation. In a separate experiment using 59 female...
baboons fed an atherogenic diet, the VLDL + LDL cholesterol values obtained by the dextran sulfate CaCl₂ precipitation method were slightly lower than the VLDL + LDL cholesterol value obtained by preparative ultracentrifugation. The means were 82.7 and 90.9 mg/dl, with standard deviations of 39.4 and 44.6 for the precipitation method and the ultracentrifugal method respectively. The correlation coefficient between the two methods was 0.955. The percentage of the VLDL + LDL cholesterol contributed by the VLDL + LDL cholesterol measured in samples isolated in the ultracentrifuge.

We also compared the dextran sulfate CaCl₂ method with the heparin MnCl₂ precipitation method in a group of 20 baboons. The mean VLDL + LDL cholesterol was 94.5 mg/dl for the heparin MnCl₂ method and 96.6 mg/dl for the dextran sulfate CaCl₂ method with standard deviations of 27.9 and 26.0 respectively. The correlation coefficient between these two methods was 0.991.

**Apolipoprotein B Measurement**

We measured apolipoprotein B (apo B) by electroimmunoassay using antisera to baboon apo B. This antisera was specific for baboon apo B as determined by electroimmunoassay, immunodiffusion, and immunoelectrophoresis. LDL (1.019–1.063 g/ml), isolated by preparative ultracentrifugation from a pool of serum from chow-fed animals, was used as the primary standard for the apo B assay. This LDL preparation was analyzed for apoprotein content by immunodiffusion using antisera to human apolipoproteins and was found to be free of albumin, apo A-I, and apo E. These observations were confirmed by SDS-polyacrylamide gel electrophoresis. Low molecular weight polypeptides accounted for less than 1% of the total as shown by densitometry of the polyacrylamide gels.

Equivalence between standard and whole serum was determined by assaying whole serum at dilutions within the range of the standard curve. The relationship between rocket height and apo B concentration was linear over a range of 50 to 500 ng. Between and within assay coefficients of variation were 2.0% and 2.6% respectively.

**Statistical Methods**

For the statistical analyses, the three serum and lipoprotein cholesterol measurements made after 4, 5, and 6 weeks on each diet, and the single apo B measurement made after 6 weeks on each diet, were transformed with a natural logarithm transformation to normalize the data and stabilize the variances. The means of the three measurements of serum and lipoprotein cholesterol on each diet were used in the statistical analysis.

The linear model (see pages 135–138) included terms for the overall mean, sequence, sex, sequence by sex interaction, animal within sequence and sex, time period, time period by sex interaction, diet, diet by sex interaction, and carryover.

The data were analyzed by analysis of variance. The effects of time period, time period by sex, diet, diet by sex, and carryover were tested against a within animal error term. The overall mean, sequence, sex, and sequence by sex were tested against the animal within sequence and sex mean square. The estimated means (appropriate linear combination of regression coefficients) and confidence intervals were based upon robust Huber M-estimates (c = 1.2) of the parameters. The robust estimator gives less weight to observations far from the values predicted under the model and thus is less sensitive than the classical methods to outliers in the data. The choice of c gives the number of standard deviations to be used as a transition in the weighting procedure.

The factorial effects (see pages 153–154) were the main effect of dietary cholesterol (C), main effect of fat (F), and the cholesterol by fat interaction (CF). These are given in terms of the group means as

\[ C = \frac{1}{2}(HCSF - LCSF + HCPF - LCPF) \]

\[ F = \frac{1}{2}(HCSF - HCPF + LCSF - LCPF) \]

\[ CF = \frac{1}{2}(HCSF - HCPF - LCSF + LCPF) \]

The simple effects were the effect of cholesterol in the presence of (@) polyunsaturated fat, effect of cholesterol in the presence of saturated fat, effect of saturated fat in the presence of low cholesterol, and effect of saturated fat in the presence of high cholesterol. These are given as

\[ C@PF = (HCPF) - (LCPF) \]

\[ C@SF = (HCSF) - (LCSF) \]

\[ F@LC = (LCSF) - (LCPF) \]

\[ F@HC = (HCSF) - (HCPF). \]

The main effect of dietary cholesterol is the average of the simple effect of C @ PF and C @ SF and thus represents the effect of dietary cholesterol averaged over type of fat. Similarly, the main effect of fat is the average of F @ LC and F @ HC and represents the effect of fat averaged over the levels of cholesterol. The interaction is the difference between C @ SF and C @ PF (also the difference between F @ HC and F @ LC). If the interaction is zero, the effect of cholesterol is the same regardless of the type of fat. If the interaction is positive, dietary cholesterol has a larger effect in the presence of saturated fat than in the presence of polyunsaturated fat.
Table 3. Multiplicative Carryover Effects for Total, VLDL + LDL, and HDL Cholesterol, and Apo B Concentrations In Serum

<table>
<thead>
<tr>
<th>Diet Fat</th>
<th>Total cholesterol</th>
<th>VLDL + LDL cholesterol</th>
<th>HDL cholesterol</th>
<th>Apo B</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>95% confidence limits</td>
<td>95% confidence limits</td>
<td>95% confidence limits</td>
<td>95% confidence limits</td>
</tr>
<tr>
<td>Low Polyunsaturated</td>
<td>1.06 1.06-1.10</td>
<td>1.08 1.00-1.17</td>
<td>1.07 1.02-1.11</td>
<td>1.04 0.94-1.14</td>
</tr>
<tr>
<td>Low Saturated</td>
<td>1.03 1.00-1.07</td>
<td>1.06 0.98-1.15</td>
<td>0.98 0.94-1.02</td>
<td>1.12 1.02-1.23</td>
</tr>
<tr>
<td>High Polyunsaturated</td>
<td>0.96 0.93-1.00</td>
<td>0.97 0.90-1.05</td>
<td>0.96 0.92-1.00</td>
<td>0.88 0.80-0.97</td>
</tr>
<tr>
<td>High Saturated</td>
<td>0.95 0.91-0.98</td>
<td>0.89 0.82-0.96</td>
<td>1.00 0.94-1.04</td>
<td>0.97 0.89-1.07</td>
</tr>
</tbody>
</table>

Effects are statistically significant ($p < 0.05$) if 95% confidence limits do not include 1.00.

Results

Carryover Effects

Carryover effects were small (table 3). The largest positive carryover effect was due to the low cholesterol, saturated fat diet while the most extreme negative effect was due to the high cholesterol, polyunsaturated fat diet, both on apo B concentration. We estimate that the mean on subsequent diets would be increased or decreased by 12% respectively. Although carryover effects were small, estimated means and diet effects in subsequent analyses were adjusted for these effects.

Serum and Lipoprotein Cholesterol Concentrations

Both lipoprotein cholesterol levels and the apo B levels on the low cholesterol, polyunsaturated fat diet were similar to the corresponding levels on the chow diet (table 4, figure 1). As anticipated, the low cholesterol-polyunsaturated fat diet produced the lowest (120 mg/dl) and the high cholesterol-saturated fat diet produced the highest (245 mg/dl) mean serum cholesterol concentrations. The increment in total serum cholesterol was made up of about 60 mg/dl each of VLDL + LDL and HDL cholesterol. The low cholesterol-saturated fat and high cholesterol-polyunsaturated fat diets produced nearly identical total serum cholesterol levels (198 and 193 mg/dl), but different distributions of cholesterol between VLDL + LDL and HDL: on the high cholesterol-polyunsaturated fat diet, VLDL + LDL cholesterol was higher and HDL cholesterol was lower than on the low cholesterol-saturated fat diet (VLDL + LDL cholesterol, 95 vs 77 mg/dl; HDL cholesterol, 90 vs 114 mg/dl).
The factorial effects of dietary components are shown in the first three lines of table 5. The main effects of dietary cholesterol and saturated fat on total serum cholesterol were similar (1.41 and 1.44). That is, the effect of dietary cholesterol averaged over the two types of fat was to increase total serum cholesterol by 41%. In contrast, the main effect of dietary cholesterol on VLDL + LDL cholesterol (1.76) was greater than that of saturated fat (1.38); but the main effect of saturated fat on HDL cholesterol (1.48) was greater than that of dietary cholesterol (1.20). The interpretation of these main effects must be qualified, however, because there were significant interactions between the two dietary components in their effects on both total serum cholesterol and VLDL + LDL cholesterol.

The negative interaction of dietary cholesterol and saturated fat on total serum cholesterol (0.87) was due entirely to the interaction effect on VLDL + LDL cholesterol (0.74), since there was no interaction on HDL cholesterol (1.0).

The simple effects of the two dietary components are shown in the last four lines of table 5. Dietary cholesterol in the presence of polyunsaturated fat increased total serum cholesterol more (1.62) than it did in the presence of saturated fat (1.24). This difference was due predominantly to its greater effect on VLDL + LDL cholesterol with polyunsaturated fat (2.38) than with saturated fat (1.31). In contrast, dietary cholesterol elevated HDL cholesterol only slightly with either fat (1.19 and 1.20).

Saturated fat raised total serum cholesterol more with a low cholesterol intake (1.65) than with a high cholesterol intake (1.26). Saturated fat with a low dietary cholesterol intake raised VLDL + LDL cholesterol (1.86), but had little effect on VLDL + LDL cholesterol (1.03) when cholesterol intake was high. It raised HDL cholesterol to the same degree with (1.49) or without (1.48) dietary cholesterol.

There were no significant differences between the sexes in serum or lipoprotein cholesterol responses to dietary components nor any interaction of sex with the dietary components.

### Apo B Concentrations

Apo B concentrations (table 4) responded to diets as did VLDL + LDL cholesterol. The ratio of VLDL + LDL cholesterol to apo B was higher (1.82 and 1.50) with the two high cholesterol

### Table 5. Factorial and Simple Multiplicative Effects of Dietary Components on Total, VLDL + LDL, and HDL Cholesterol, and Apo B Concentrations in Serum (mg/dl)

<table>
<thead>
<tr>
<th>Dietary components</th>
<th>Total cholesterol</th>
<th>VLDL + LDL cholesterol</th>
<th>HDL cholesterol</th>
<th>Apo B</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Effect 95% confidence limits</td>
<td>Effect 95% confidence limits</td>
<td>Effect 95% confidence limits</td>
<td>Effect 95% confidence limits</td>
</tr>
<tr>
<td>Cholesterol</td>
<td>1.41 1.36–1.47</td>
<td>1.76 1.62–1.93</td>
<td>1.20 1.15–1.25</td>
<td>1.20 1.08–1.33</td>
</tr>
<tr>
<td>Fat</td>
<td>1.44 1.39–1.50</td>
<td>1.38 1.27–1.56</td>
<td>1.48 1.43–1.54</td>
<td>1.39 1.25–1.54</td>
</tr>
<tr>
<td>Cholesterol × Fat</td>
<td>0.87 0.84–0.91</td>
<td>0.74 0.68–0.81</td>
<td>1.00 0.96–1.04</td>
<td>0.91 0.83–1.01</td>
</tr>
<tr>
<td>Cholesterol @ Polyunsaturated</td>
<td>1.62 1.53–1.71</td>
<td>2.38 2.11–2.68</td>
<td>1.19 1.13–1.27</td>
<td>1.31 1.14–1.52</td>
</tr>
<tr>
<td>Cholesterol @ Saturated fat</td>
<td>1.24 1.17–1.31</td>
<td>1.31 1.16–1.48</td>
<td>1.20 1.13–1.27</td>
<td>1.09 0.95–1.27</td>
</tr>
<tr>
<td>Fat @ Low cholesterol</td>
<td>1.65 1.56–1.74</td>
<td>1.86 1.66–2.09</td>
<td>1.48 1.40–1.57</td>
<td>1.52 1.32–1.75</td>
</tr>
<tr>
<td>Fat @ High cholesterol</td>
<td>1.26 1.19–1.33</td>
<td>1.03 0.91–1.15</td>
<td>1.49 1.40–1.57</td>
<td>1.27 1.10–1.46</td>
</tr>
</tbody>
</table>

Effect is statistically significant (p < 0.05) if 95% confidence intervals do not include 1.00.
Table 6. Apo B Concentrations in Serum (mg/dl) by Sex and Type of Fat

<table>
<thead>
<tr>
<th>Sex</th>
<th>Fat</th>
<th>Mean</th>
<th>95% confidence limits</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>Polyunsaturated</td>
<td>50</td>
<td>43-58</td>
</tr>
<tr>
<td></td>
<td>Saturated</td>
<td>62</td>
<td>53-71</td>
</tr>
<tr>
<td>Female</td>
<td>Polyunsaturated</td>
<td>43</td>
<td>37-50</td>
</tr>
<tr>
<td></td>
<td>Saturated</td>
<td>67</td>
<td>58-77</td>
</tr>
</tbody>
</table>

Diets enriched in cholesterol and saturated fat usually lower HDL levels in nonhuman primates.9-13 However, Srinivasan et al.14 found increased α-lipoprotein cholesterol in spider, green, patas, and squirrel monkeys, but not in rhesus monkeys or chimpanzees, with a diet containing 1.84 mg cholesterol/Kcal and 10% of calories from butter. The consistent elevation of HDL cholesterol by both dietary cholesterol and saturated fat in our baboons may represent a species difference.

Comparison with Dietary Effects in Other Nonhuman Primates

Most other commonly used nonhuman primate species respond to diets enriched in cholesterol and saturated fat with a much greater increase in VLDL + LDL cholesterol than does the baboon.9 Both rhesus and patas monkeys developed LDL cholesterol levels of 600 mg/dl or greater on a diet containing 1 mg/Kcal of cholesterol, and the increase was associated with dietary cholesterol rather than with the accompanying saturated fat.10,11 Thus, although the magnitude of change was not as great, the direction of change of VLDL + LDL cholesterol associated with dietary cholesterol in baboons was similar to that in other species. However, baboons responded to saturated fat with a greater change in VLDL + LDL cholesterol than is seen in other species.

Individual Variability In Response to Fat

In the presence of a high cholesterol intake, saturated fat decreased VLDL + LDL cholesterol in nine animals and increased it in 15; and saturated fat decreased apo B in five animals and increased it in 19. In contrast, saturated fat increased HDL cholesterol concentrations in all animals with both high and low dietary cholesterol intakes.

Discussion

Comparison with Dietary Effects In Other Nonhuman Primates

Although diet effects on apo B concentration were similar to the effects on VLDL + LDL cholesterol concentration, differences were not as great for apo B as for VLDL + LDL cholesterol. The effect of saturated fat on apo B concentration in the baboon was similar to the effect of saturated fat on apo LDL in humans.22

Relationship of Apo B to VLDL + LDL Cholesterol

In general, the effects of dietary saturated fat on total serum cholesterol15 have been greater and more consistent in humans than those of dietary cholesterol.16 Among the few experiments designed to test for interactions between dietary cholesterol and saturated fat, the results have been inconsistent — some have found a positive interaction;17 others, no interaction.18,19 None has reported a negative interaction.

A few recent experiments have examined the effects of dietary cholesterol and type of fat on lipoprotein cholesterol concentrations. Increased cholesterol intake without a change in type of fat raised only LDL cholesterol.20 Reducing both cholesterol intake and saturation of fat lowered LDL cholesterol in both hyperlipidemic and normolipidemic men and women, and lowered HDL cholesterol in both hyperlipidemic and normolipidemic men, but did not change HDL cholesterol significantly in women.21 Substituting polyunsaturated for saturated fat and maintaining cholesterol intake constant (400 mg/day) lowered both LDL cholesterol and HDL cholesterol in normolipidemic men.22 Changing from a low cholesterol, polyunsaturated fat diet to a high cholesterol, saturated fat diet raised both LDL and HDL cholesterol in normolipidemic young adults.23 A number of other reports concerning the effects of cholesterol and type of fat on HDL cholesterol were reviewed recently.24

These results suggest that, in humans, dietary cholesterol predominantly raises LDL cholesterol, and saturated fat raises both LDL and HDL cholesterol. This pattern of response is similar to the pattern we have found in baboons.
The ratio of VLDL + LDL cholesterol to serum apo B concentrations was lowest in sera of animals fed the low cholesterol polyunsaturated diet and was increased when saturated fat or cholesterol were added to the diet.

Nazir et al.26 reported a ratio of LDL cholesterol to apo LDL that ranged from 0.66 to 1.72 (mean = 1.24) for normal human subjects. Hypercholesterolemic subjects showed an increased ratio of LDL cholesterol to apo LDL. Bautovitch et al.26 found a ratio of LDL cholesterol to LDL apo B of 1.31 for normal human subjects and an increased ratio for hypercholesterolemic subjects. Albers et al.27 reported a ratio of LDL cholesterol to LDL apo B of 1.7 for normal human subjects and an increase in the ratio for hypercholesterolemic subjects. The variation in the ratios of LDL cholesterol to LDL apo B (1.2 vs 1.7) in normal human subjects could be due to differences in analytical methods or differences in diet. Our results show that the type of dietary fat and cholesterol content of the diet affect the VLDL + LDL cholesterol to apo B ratio. Our ratio of VLDL + LDL cholesterol to apo B in animals fed either type of fat plus cholesterol is similar to that observed in hypercholesterolemic human subjects.26-27

**Contrast Between Coconut Oil and Corn Oil**

This experiment used coconut oil as representative of saturated fat, and corn oil as representative of polyunsaturated fat. These do differ greatly in degree of saturation of fatty acids, and in other characteristics as well. For example, coconut oil contains a larger proportion of short chain fatty acids than does corn oil. Consequently, some of the contrasting effects observed may be due to differences other than saturation alone. It will be important to compare other fats and oils with regard to their effects on the various lipoprotein species.

**Significance**

In the baboon, different combinations of dietary cholesterol and type of fat can produce identical levels of total serum cholesterol with varying proportions of VLDL + LDL and HDL cholesterol. If, as now seems likely, the balance between VLDL + LDL and HDL is an important determinant of atherosclerosis in humans3 and in baboons, atherogenicity of a diet may depend on how it affects the distribution of cholesterol among the lipoproteins as well as how it affects the total serum cholesterol concentration. Interactions between dietary cholesterol and type of fat, differences in their effects on VLDL + LDL and HDL cholesterol concentrations, and variations among individuals in responses to each dietary component (particularly to saturated fat) may have contributed to some of the inconsistencies among results of studies of diet and atherosclerotic disease.

**Acknowledgments**

We thank K.D. Carey for caring for the baboons used in this experiment, Jamie Wene for designing and preparing the diets, and Yolan Marinez for coordinating the project.

**References**

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Index Terms: baboon (Papio sp.) • lipoprotein • dietary cholesterol • saturated fat • polyunsaturated fat
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