Diet and Sire Effects on Serum Cholesterol and Cholesterol Absorption in Infant Baboons (Papio cynocephalus)

GLEN E. MOTT, C. ALEX MCMAHAN, AND HENRY C. MCGILL, JR.

SUMMARY  One hundred three baboon infants, the progeny of an equal number of dams and seven sires, were breast fed or were fed prepared formulas containing 12, 29, or 61 mg/dl cholesterol for 14 weeks. We measured serum cholesterol and triglyceride concentrations at birth and at 3-week intervals, and cholesterol absorption at 12 weeks. Dietary cholesterol had a significant effect on serum cholesterol concentration at 12 weeks and on cholesterol absorption, but did not affect weight or serum triglyceride concentration. Sire had a significant effect on serum cholesterol concentration at 12 weeks and on cholesterol absorption, but did not affect weight or serum triglyceride concentration. Therefore, we have undertaken a long-term study of the influence of infant, juvenile, and adult diet and heredity on cholesterol metabolism and atherosclerosis in the baboon (Papio cynocephalus). The baboon is a large nonhuman primate with many physiological characteristics similar to those of man, including particularly its cholesterol metabolism and its serum cholesterol response to an atherogenic diet. We report here the results of the first phase of the experiment: the effects of diet, sire, and sex on serum lipid concentrations, cholesterol absorption, and growth in 103 baboon infants from birth to 12 weeks of age.

Methods

Breeding Colony and Infant Accession

One hundred-twenty female baboons were randomly assigned to six outdoor gang-caged groups with one male in each group. We determined the...
serum cholesterol concentrations of the breeders while being fed baboon chow manufactured by the Ralston Purina Co., and while being fed a high fat, high cholesterol diet. These data will be used to estimate genetic parameters when the offspring reach maturity. The means and standard deviations of the serum cholesterol concentrations of the females in each group were similar. The six males were selected for their breeding potential without consideration of their serum cholesterol concentrations. Low productivity required that three of the original sires be replaced and that a seventh sire and female group be added.

The breeders were fed baboon chow throughout mating, pregnancy, parturition, and nursing. Pregnant females were transferred to individual cages about 3 weeks before the estimated date of parturition. Between 8 and 20 hours after delivery, an attendant anesthetized the mother with ketamine (Ketaset; Bristol Laboratories) in order to weigh the infant, determine its sex, and draw a sample of venous blood. The measurements made at this time are referred to as "birth" values.

Each infant was assigned to one of three formulas or breast feeding. If assigned to breast feeding, the attendant returned the infant to the mother and placed both mother and infant in an outdoor gang cage with other nursing mothers. If assigned to one of the formulas, he placed the infant in an individual cage in the nursery. The infants were acquired over a period of about 2 years.

**Formula**

Ross Laboratories prepared the three experimental formulas in disposable 120-ml bottles. The manufacturer added sufficient cholesterol to portions of a single batch of oil mixture, before homogenizing the oil with other ingredients, to approximate final concentrations of 2, 30, and 60 mg/dl. Except for cholesterol content, the three formulas were identical to one another, and also were identical to the formula and the major components of breast milk as reported by Buss.4 We extracted and derivatized cholesterol and plant sterols from the formula and breast milk (Table 2) by the method of Miettinen et al. The silylated sterols were quantitated by gas liquid chromatography (GLC) on a column packed with 3% OV-17 on Gas Chrom Q (Applied Science Laboratories) with cholestane as internal standard. We analyzed fatty acids by GLC on a SP-2340 column as methyl esters produced by methylation with methanolic HCl.

**Nursery Rearing**

An attendant held each infant and fed it as much formula as it would take readily 5 times a day for the first 2 weeks, 4 times a day for the next 2 weeks, and 3 times a day for the next 10 weeks. He weighed each infant daily and estimated formula consumed at each feeding.

**Breast Feeding**

The mother of each baboon assigned to this group suckled its infant for 14 weeks after birth. Near the end of the breast-feeding period, infants often began to eat small quantities of baboon chow to a degree depending on dentition. At 12 weeks of age, mother and infant were placed in an individual cage for measurement of cholesterol absorption.

**Experimental Design**

The observations on infants up to 12 weeks of age were part of a longer term experiment in which the diet after weaning also was manipulated. Infant diet, adult diet, and sex were arranged as a factorial experiment.4 Four infant diets, four adult diets, and two sexes made 32 diet-sex combinations. We planned to assign 96 infants to the experiment, three to each diet-sex combination. We also planned to balance for sires within each infant diet (across adult diets) and adult diet (across infant diets) for both sexes, to assign a male and a female infant of one sire to each diet combination, and to avoid more than one offspring of a sire in each diet-sex group. We established rules to assign infants at birth to diet combinations by a restricted random process. Since sires were not equally productive, we did not achieve complete balance for sires. Each dam contributed only one infant to the experiment.

**Table 1 Diet Composition**

<table>
<thead>
<tr>
<th>Component</th>
<th>Source in formula</th>
<th>U/liter</th>
<th>Baboon breast milk¹</th>
</tr>
</thead>
<tbody>
<tr>
<td>Protein</td>
<td>Nonfat milk solids</td>
<td></td>
<td>16</td>
</tr>
<tr>
<td>Fat</td>
<td>Oils (40% soya, 40% coconut, 20% corn)</td>
<td>37</td>
<td>50</td>
</tr>
<tr>
<td>Carbohydrate</td>
<td>Lactose</td>
<td>g</td>
<td>70</td>
</tr>
<tr>
<td>Minerals</td>
<td>Mineral mix</td>
<td>g</td>
<td>1.43</td>
</tr>
<tr>
<td>Vitamin A</td>
<td>USP units</td>
<td>2800</td>
<td>ND</td>
</tr>
<tr>
<td>Vitamin C</td>
<td>mg</td>
<td>87</td>
<td>ND</td>
</tr>
<tr>
<td>Vitamin B₃</td>
<td>mg</td>
<td>0.96</td>
<td>ND</td>
</tr>
</tbody>
</table>

ND = not determined.
The laboratory maintained quality control for both the cholesterol and triglyceride procedures by analysis of pooled serum from the Lipid Standardization Program of the Center for Disease Control (CDC), Atlanta, Georgia. Our analyses of the CDC serum samples met our criteria for acceptable limits of the mean and standard deviation.

**Cholesterol Absorption**

Cholesterol absorption was measured during the 13th week by the methods of Borgstrom 14 and Sodhi et al. 15 with \( \beta \)-sitosterol[4,14C] and cholesterol-[1,2,3H]. We purified the isotopes (Amersham-Searle and New England Nuclear) by thin layer chromatography before use. Approximately 3 \( \mu \)Ci each of tracer amounts of radiolabeled cholesterol and \( \beta \)-sitosterol dissolved in 100 \( \mu \)l of acetone and blue food coloring as a fecal flow marker were added to dry milk powder in a gelatin capsule or to 25 ml of the appropriate milk formula and administered to the unanesthetized animal followed by the regular meal. The food coloring dissolved in the formula was readily observed in the stools, and stool samples containing the marker were highly radioactive. Stools were collected for 6 days and pooled except for a small sample containing fecal flow marker, which was analyzed separately. In preliminary experiments, daily stool collection for 8 days showed that radioactivity from \( \beta \)-sitosterol[4-14C] did not decrease to less than 1% of the administered dose until 5 to 6 days. The 6-day collection period maximized the recovery of the isotope and minimized the collection of radioactive cholesterol which had been absorbed and reexcreted. Duplicate samples of the 6-day pool and the stool sample containing the marker were homogenized, saponified, extracted, and the ratio of \( ^3H/^14C \) in the extracts was measured by liquid scintillation spectrometry. We calculated cholesterol absorption by the Borgstrom method from the total recovery of isotopic sterols in the 6-day stool collection plus the small stool sample containing the fecal flow marker. Cholesterol absorption by the Sodhi method was calculated from the \( ^3H/^14C \) recovered only from the small stool sample containing the marker. The amount of cholesterol absorbed (mg/day) was calculated from the average cholesterol intake during the week of the absorption measurement and the percent absorption. Mean recovery of \( \beta \)-sitosterol-[14C] was 71% over the 6-day period.
Cholesterol absorption measurements were complete on 15 to 17 animals in each diet cell for a total of 65 animals by the Sodhi method and 61 by the Borgstrom method. Of these, 47 were measured by both methods.

**Statistical Methods**

The responses were analyzed in separate univariate analyses. The linear model included the main effects of diet, sex, and sire and all interactions. We assumed all effects to be fixed. Parameters of the linear model were estimated by least squares.

Lack of homogeneity of variance of the serum cholesterol concentrations due to the smaller variance of the low cholesterol formula group did not appear to affect the results seriously. Also, skewness in either the serum cholesterol or serum triglyceride concentrations did not substantially affect the results. When a logarithmic transformation was used, the hypothesis tests yielded the same results.

Specific null hypotheses tested for each response were the following: (1) \( H_0 \): no difference between breast feeding and formula feeding; (2) \( H_0 \): no linear trend associated with formula cholesterol concentration; (3) \( H_0 \): no quadratic trend associated with formula cholesterol concentration; (4) \( H_0 \): no difference between sexes; (5) \( H_0 \): no difference between sires. In addition, all interactions, that is, diet by sex, diet by sire, sex by sire, and diet by sex by sire, were tested.

Null hypotheses were tested by an F test. The interactions in all cases were not statistically significant. The model was reduced to include only the diet and sire effects, except in the case of body weight, where sex also was included. Final estimates were then obtained. Contrasts between the individual diet group means were tested using Tukey's multiple comparison test.

For serum lipids, the mean of two blind duplicate values was used in the statistical analyses.

**Results**

**Formula Consumption and Growth**

The amount of formula consumed by the three formula groups during the 13th week did not differ by formula cholesterol concentration or sire. Although males weighed more at 12 weeks than did females (1.64 kg vs. 1.44 kg; \( P < 0.01 \)), formula consumption between sexes was not significantly different. There were no statistically significant effects of diet or sire on body weight at 12 weeks.

**Serum Triglyceride Concentrations**

The overall mean serum triglyceride concentration at birth was 36.7 mg/dl (SD = 32.5), and at 12 weeks, 52.7 mg/dl (SD = 37.4). Individual values at birth ranged from 10 mg/dl to 263 mg/dl and at 12 weeks from 14 mg/dl to 270 mg/dl. By 3 weeks of age, the overall mean was 56.2 mg/dl and remained essentially the same thereafter. There were no statistically significant differences by diet or sex. The sire effect at 12 weeks approached statistical significance (\( P = 0.10 \)).

**Age, Sex, and Diet Effects on Serum Cholesterol Concentrations**

The mean total serum cholesterol concentration at birth was 75 mg/dl (SD = 29.0), and ranged from 31 mg/dl to 178 mg/dl. Means of the two sexes were similar throughout the 12 weeks. Figure 1 shows the serum cholesterol concentrations in each of the four diet groups with increasing age. The groups began to diverge immediately after birth and all continued to increase until 12 weeks of age.

In the low formula group, which received essentially no dietary cholesterol, serum cholesterol increased from a mean of 75 mg/dl at birth to a mean of 94 mg/dl at 12 weeks. At 12 weeks, the serum cholesterol concentrations of individual animals receiving the low formula ranged from 63 mg/dl to 143 mg/dl. These differences presumably were due to factors other than dietary cholesterol, since the average cholesterol intake was less than 5 mg/day. Serum cholesterol concentrations of the breast-fed and medium formula groups were similar throughout. The relationship between formula cholesterol and serum cholesterol concentrations at 12 weeks of age (Fig. 2) showed statistically significant linear (\( P < 0.01 \)) and quadratic (\( P < 0.05 \)) trends. As dietary cholesterol increased, the resulting increase...
FIGURE 2  Mean serum cholesterol concentrations and standard errors of three formula groups at 12 weeks of age.

in serum cholesterol concentration was less. At 12 weeks (Table 3), the low formula group was significantly lower than the other three groups. The high formula group was higher than the medium formula and breast-fed groups, but the difference was not statistically significant.

Sire Effect on Serum Cholesterol Concentrations at Birth and 12 Weeks

The sire effect at birth was statistically significant ($P = 0.05$) (Table 4). The sire effect on serum cholesterol concentration at 12 weeks shown in Table 4 and Figure 3 also was statistically significant ($P < 0.01$). However, as can be seen in Table 4, the sire effect at birth did not predict the effect at 12 weeks. Table 5 shows that the rank order of the sire groups within each diet group was similar at 12 weeks. Although Table 5 seems to indicate a diet by sire interaction (that is, a greater sire effect on the high formula than on the medium or low formulas), this effect was not statistically significant.

Cholesterol Absorption

Table 6 shows the overall means and standard errors of percent absorption of dietary cholesterol by diet groups as determined by both the Sodhi and Borgstrom methods. Both percent absorption and amount absorbed as determined by both methods show statistically significant ($P < 0.01$) linear trends associated with formula cholesterol concentration. The absolute amount absorbed increased

<table>
<thead>
<tr>
<th>Sire</th>
<th>Sire's serum cholesterol concentration (mg/dl)</th>
<th>No. of progeny</th>
<th>Sire effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>A982</td>
<td>116</td>
<td>12</td>
<td>-28 (9.6)</td>
</tr>
<tr>
<td>A776</td>
<td>115</td>
<td>18</td>
<td>-24 (8.2)</td>
</tr>
<tr>
<td>X84</td>
<td>118</td>
<td>3</td>
<td>-15 (15)</td>
</tr>
<tr>
<td>A947</td>
<td>139</td>
<td>18</td>
<td>-7.6 (6.8)</td>
</tr>
<tr>
<td>A943</td>
<td>135</td>
<td>21</td>
<td>7.5 (8.2)</td>
</tr>
<tr>
<td>A966</td>
<td>98</td>
<td>12</td>
<td>9.7 (7.7)</td>
</tr>
<tr>
<td>A772</td>
<td>196</td>
<td>19</td>
<td>20 (9.6)</td>
</tr>
</tbody>
</table>

* Sire effect is expressed as deviation from the overall mean serum cholesterol concentration of all baboons at birth or 12 weeks of age.
† Determined during 3-month feeding of monkey chow plus 20% saturated fat (1 mg cholesterol/kcal). Mean of three monthly analyses.
‡ Standard errors are in parentheses.

FIGURE 3  Estimated sire effect on 12-week serum cholesterol concentration of progeny of each sire. Sire effect is expressed as deviation from the overall mean serum cholesterol concentration. Vertical lines extending from bars are standard errors.

TABLE 3  Serum Cholesterol Concentration in Infant Baboons by Diet at 12 Weeks of Age

<table>
<thead>
<tr>
<th>Diet</th>
<th>No. of animals</th>
<th>Serum cholesterol (mg/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Breast</td>
<td>25</td>
<td>126 (7.6)*</td>
</tr>
<tr>
<td>Formula 1.15</td>
<td>25</td>
<td>91 (7.6)</td>
</tr>
<tr>
<td>Formula 28.8</td>
<td>27</td>
<td>136 (7.6)</td>
</tr>
<tr>
<td>Formula 61.1</td>
<td>26</td>
<td>152 (7.0)</td>
</tr>
</tbody>
</table>

* Standard errors are in parentheses.

TABLE 4  Sire's Observed Serum Cholesterol Concentration (mg/dl) and Estimated Sire Effect* on Serum Cholesterol Concentration of Infant Baboons at Birth and 12 Weeks of Age

<table>
<thead>
<tr>
<th>Sire</th>
<th>Sire's serum cholesterol concentration (mg/dl)</th>
<th>No. of progeny</th>
<th>Sire effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>A982</td>
<td>116</td>
<td>12</td>
<td>-28 (9.6)</td>
</tr>
<tr>
<td>A776</td>
<td>115</td>
<td>18</td>
<td>-24 (8.2)</td>
</tr>
<tr>
<td>X84</td>
<td>118</td>
<td>3</td>
<td>-15 (15)</td>
</tr>
<tr>
<td>A947</td>
<td>139</td>
<td>18</td>
<td>-7.6 (6.8)</td>
</tr>
<tr>
<td>A943</td>
<td>135</td>
<td>21</td>
<td>7.5 (8.2)</td>
</tr>
<tr>
<td>A966</td>
<td>98</td>
<td>12</td>
<td>9.7 (7.7)</td>
</tr>
<tr>
<td>A772</td>
<td>196</td>
<td>19</td>
<td>20 (9.6)</td>
</tr>
</tbody>
</table>

* Sire effect is expressed as deviation from the overall mean serum cholesterol concentration of all baboons at birth or 12 weeks of age.
† Determined during 3-month feeding of monkey chow plus 20% saturated fat (1 mg cholesterol/kcal). Mean of three monthly analyses.
‡ Standard errors are in parentheses.
TABLE 5  Mean Total Serum Cholesterol Concentration (mg/dl) of Infant Baboons at 12 Weeks of Age by Diet and Sire

<table>
<thead>
<tr>
<th>Sire</th>
<th>Diet</th>
<th>A982 (mg/dl)</th>
<th>A776 (mg/dl)</th>
<th>X84 (mg/dl)</th>
<th>A947 (mg/dl)</th>
<th>A943 (mg/dl)</th>
<th>A856 (mg/dl)</th>
<th>A772 (mg/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Breast</td>
<td>104.5 (2)*</td>
<td>112.0 (3)</td>
<td>120.9 (5)</td>
<td>136.8 (4)</td>
<td>151.4 (4)</td>
<td>153.4 (7)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Formula 1.15</td>
<td>81.3 (2)</td>
<td>87.8 (6)</td>
<td>98.5 (5)</td>
<td>87.3 (6)</td>
<td>97.7 (2)</td>
<td>113.9 (4)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Formula 61.1</td>
<td>105.7 (3)</td>
<td>106.6 (5)</td>
<td>157.3 (5)</td>
<td>147.9 (5)</td>
<td>136.4 (4)</td>
<td>176.0 (4)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* Numbers of baboons in parentheses. Estimated standard error for each mean is 34.9/\sqrt{n}.

with increasing cholesterol concentration of the formula, but the percent absorbed decreased. Percent and amount of cholesterol absorbed are associated with average serum cholesterol concentration of formula groups, but when the relationships were examined for individual baboons within formula groups, there were no statistically significant associations. Therefore, the variability of serum cholesterol within diet groups could not be explained by differences in cholesterol absorption.

Sire effects on the absorption values were not consistent between the two methods. The sire effect on percent absorption by the Sodhi method was statistically significant \( P = 0.05 \) using all four diet groups, and not statistically significant for the Borgstrom method, using only the three formula groups. Sire effects on amount of cholesterol absorbed by either the Sodhi of Borgstrom method were not statistically significant. Thus, it appears that variations in absorption as determined by each method do not explain the variability in serum cholesterol concentration observed among the different sire groups.

The Sodhi and Borgstrom methods are indistinguishable when applied to the high cholesterol formula group. However, in the medium and low cholesterol formulas, percent cholesterol absorbed measured by the Sodhi method was significantly higher than by the Borgstrom method.

Discussion

Genetic Control of Serum Cholesterol Concentration

This experiment shows that the serum cholesterol concentration in young baboons is partially under genetic control and that the control probably is not accomplished by regulating absorption of dietary cholesterol. A genetic component in the control of serum cholesterol concentration has been demonstrated in several primates, including humans, squirrel monkeys, and rhesus monkeys. To these may be added the baboon, at least in infancy.

Attempts to characterize the metabolic processes responsible for high and low cholesterolemic response to diet in animals have yielded conflicting results. Lofland et al. concluded that the genetic control of serum cholesterol in the squirrel monkey was principally by regulation of bile acid secretion, although Jones et al. also observed an association with cholesterol absorption. Eggen concluded that differences in cholesterol absorption could partially explain high and low cholesterolemic response in rhesus monkeys. The difference between sire effects at birth and at 12 weeks suggests that the biochemical mechanism of genetic control of serum cholesterol concentration may vary with age. Investigations of both environmental and genetic factors controlling cholesterol metabolism, therefore, should take into account the age of the individual.

The failure of the sire effect at birth to predict the 12-week sire effect is consistent with reports that cord blood cholesterol concentration does not detect familial hypercholesterolemia in humans. The 12-week values are more likely to correspond to adult values, since observations on humans suggest that children maintain similar rank order by serum cholesterol levels over several years.

Diet Effects on Serum Cholesterol

The effects of feeding a low cholesterol formula or breast feeding on serum cholesterol concentration are similar to those reported for human infants.

TABLE 6  Cholesterol Absorption in Infant Baboons by Diet

<table>
<thead>
<tr>
<th>Diet</th>
<th>Sodhi method</th>
<th></th>
<th>Borgstrom method</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>%</td>
<td>mg/day</td>
<td>%</td>
<td>mg/day</td>
</tr>
<tr>
<td>Breast</td>
<td>52 (3.7)*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Formula 1.15</td>
<td>63 (3.4)</td>
<td>4.7 (5.9)</td>
<td>51 (3.1)</td>
<td>2.1 (5.1)</td>
</tr>
<tr>
<td>Formula 28.8</td>
<td>45 (3.5)</td>
<td>42 (6.8)</td>
<td>36 (3.2)</td>
<td>33 (5.2)</td>
</tr>
<tr>
<td>Formula 61.1</td>
<td>32 (3.5)</td>
<td>80 (5.9)</td>
<td>32 (3.4)</td>
<td>77 (5.6)</td>
</tr>
</tbody>
</table>

* Standard errors are in parentheses.
† Could not be estimated for breast-fed baboons.
Fomon and Bartels found mean serum cholesterol concentrations in breast-fed human infants of 172 mg/dl; in infants fed cow's milk, 156 mg/dl; and in infants fed a formula containing corn oil, about 128 mg/dl. Other investigators observed similar serum cholesterol concentrations in infants on comparable infant diets, except that those on cow's milk were more nearly like breast-fed infants. Potter and Nestel showed that increasing the polyunsaturated fatty acid content of breast milk by altering maternal diet does not change the cholesterol concentration of the milk, but reduces the nursing infant's serum cholesterol concentration. In the only comparable animal study, Pickering et al. found that a milk fat formula produced much higher serum cholesterol concentrations in rhesus monkeys at 12 weeks (281 mg/dl) than did a vegetable oil formula (150 mg/dl).

In the observations on both man and monkeys, investigators usually attributed the lower serum cholesterol values associated with vegetable oil formulas to their higher proportion of polyunsaturated fatty acids rather than to their lower cholesterol content. Since we did not test different fatty acid combinations in the formulas in the present experiment, we have no evidence bearing directly on the response of the infant baboon to type of fat. In spite of some differences in fat content and fatty acid composition (Tables 1 and 2), the differences between the responses to the breast and medium formula diets in this experiment were not statistically significant. This experiment demonstrates clearly that formula cholesterol concentration affects serum cholesterol when type of fat is held constant.

**Cholesterol Absorption**

The differences between the Borgstrom and Sodhi absorption methods shown in Table 4 may result from radioactive cholesterol which was absorbed, secreted in the bile, and collected in the stools 3-5 days after excretion of the flow marker. This phenomenon would decrease the percent absorption calculated by the Borgstrom method but not the Sodhi method as previously suggested by Sodhi et al. However, to explain the inverse relationship among the diet groups between cholesterol intake and the differences between the two methods, there must be a difference in the fractional turnover rate of the rapidly exchanging cholesterol pool. Cholesterol (and also β-sitosterol) also may be taken up by the mucosa and slowly lost when the mucosal epithelial cells are sloughed. Cholesterol in the mucosa also may be partially exchanged with cholesterol in the lumen. The latter possibilities would explain the large differences between the two absorption methods in the low cholesterol formula group in which the cholesterol-specific radioactivity entering the mucosa is probably many times greater than the cholesterol of the high cholesterol formula group.

Several experiments in humans had found no decrease in percent cholesterol absorption as cholesterol intake was increased, even up to as much as 2 g/day. We observed a decrease in percent absorption of cholesterol as cholesterol intake was increased in these infant baboons. This result is consistent with results obtained by Sodhi et al. in rats and by Borgstrom in adult humans.

**Diet Effects on Growth (Weight)**

The relative merits of breast feeding vs. formula feeding for human infants have been debated widely. The present experiment showed no differences in weight gain up to 12 weeks of age between breast-fed and formula-fed infants. Furthermore, variation in the cholesterol content of the formula did not affect weight up to 12 weeks of age. Longer term effects of infant diet on juvenile and adult characteristics will be determined as these animals are followed into adulthood.

**Acknowledgments**

We would like to express our appreciation to Cynthia Gaudot and Michael Rogers for the analyses of serum lipids, to Evelyn Jackson for supervising the cholesterol absorption measurements, and to Jose Silva and Ned Zamora for care of the infant baboons. We are grateful to Dr. Herman S. Wigodskey for his helpful comments throughout the experiment and in preparation of the manuscript.

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14. Borgstrom B: Quantification of cholesterol absorption in...
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