Serum Cholesterol in Man: Diet Fat
and Intrinsic Responsiveness

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Data obtained from a number of dietary experiments in man yield a formula by which
the responses in serum cholesterol to changes in diet can be predicted. In this paper
the reliability of this formula is tested and the effects of various types of fatty acids
on serum cholesterol are investigated.

The serum cholesterol concentration in
man is strongly influenced by both the
amount and kind of fat in the diet and it is
necessary to differentiate between at least 3
classes of fatty acids, saturated, monoenoic,
and polyenoic. Furthermore, even on a fixed
diet there are intrinsic differences between
individuals. Finally, intraindividual variabil-
ity is substantial as shown by spontaneous
day-to-day variations. All these factors comp-
licate the evaluation of the effects of the
diet and the prediction of serum cholesterol
responses to given dietary changes. How-
ever, the present paper will show that the
average cholesterol responses of groups of
men to changes of fat in the diet can be satis-
factorily predicted from knowledge of the
amounts of fat in the diet and that such pre-
diction can be made even for groups of men
who are relatively hypercholesteremic or
hypercholesteremic.

Preliminary Prediction from 41 Sets of Data

As a first approximation we proposed \(^1\) that
the serum total cholesterol concentration be
expressed as

\[
\text{Chol.} = k + bS + cM + dP
\]

(1)

where \(k\) refers to all factors, other than the
fats in the diet, that influence the cholesterol

level and \(S, M,\) and \(P\) are, respectively, the
percentages of the total calories in the diet
supplied by glycerides of saturated, mono-
enoic, and polyenoic fatty acids. Actually,
in the diets used, "monoenoic" is almost en-
tirely oleic acid, while "polyenoic" is almost
entirely linoleic acid. If everything else is
constant, then, a change in the diet in respect
to \(S, M,\) and \(P\) would produce a change in
the cholesterol level:

\[
\Delta \text{Chol.} = b\Delta S + c\Delta M + d\Delta P
\]

(2)

The values of the coefficients \(b, c,\) and \(d,\)
may be estimated from experimental data on
men who change their diets by the isocaloric
exchange of fats for the carbohydrates of
bread and jelly, or of one kind of fat for an-
other, the diet being otherwise constant. In
effect, for the solution of equation 2, we re-
quire a series of average serum cholesterol
values of the same men on diets differing in
respect to \(S, M,\) and \(P.\)

Accordingly, we carried out rigidly con-
trolled experiments on groups of 12 to 27
men who exhibited no disorder of cholesterol
metabolism. From the average values in 41
sets of comparisons, each involving one group
of men on each of 2 diets for periods of 2 to
4 weeks, we obtained \(^2\) the following values
for the coefficients in equation 2: \(b = 2.74,\)
\(c = 0, d = -1.31.\) Thus equation 2 becomes

\[
\Delta \text{Chol.} = 2.74 \Delta S - 1.31 \Delta P
\]

(2A)

where \(\Delta \text{Chol.}\) is measured in mg. per 100 ml.
of serum.

The fats involved in the comparisons that
yielded equation 2A, included butterfat, olive
oil, cottonseed oil, sunflower seed oil, safflower

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Chicago.
TABLE 1.—Design of Experiment N. 6 Men in Each Group

<table>
<thead>
<tr>
<th>Group</th>
<th>Dietary Period</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Days 1-28</td>
</tr>
<tr>
<td>W</td>
<td>H SO X X CO</td>
</tr>
<tr>
<td>Y</td>
<td>H CO X X SO</td>
</tr>
<tr>
<td>X</td>
<td>H X SO CO X</td>
</tr>
<tr>
<td>Z</td>
<td>H X CO SO X</td>
</tr>
</tbody>
</table>

H, house; SO, safflower oil; CO, corn oil.

Diet X maintained the constancy of calories and proteins but involved other variables not considered here.

oil, and the mixed fats of ordinary American diets at several levels of fat concentration. Comparisons involving corn oil in the diet were not included because of evidence that part of the effect of corn oil on the serum cholesterol concentration may be dependent on some factor other than the fatty acid in it.

Since equation 2A was published, we have carried out a critical experiment with 22 men to check the question of the effect of dietary monoene on the serum cholesterol level. It was found that the isocaloric substitution of 68 Gm. of oleic acid glyceride in the daily diet for carbohydrate, and vice versa, had no effect whatever. Since the only monoene contained in appreciable amount in all ordinary diets is oleic acid, it appears that the value of \( e = 0 \) in equation 2 is well demonstrated.

Equation 2A satisfactorily fitted the average cholesterol changes observed in the experiments that provided the data used in the derivation, with a coefficient of correlation of \( r = 0.978 \) between the 41 observed averages and those predicted. But it is important to examine equation 2 with additional experimental data, to consider the case of corn oil, and to evaluate the situation with men who differ intrinsically in their cholesterol levels on a given diet.

METHODS AND SUBJECTS

As in experiments previously reported, all experiments were conducted with strict 24 hour dietary control, and blood samples were drawn for analysis at least twice from each man in the last days of each dietary period. Serum total cholesterol was measured by the method previously described. The subjects were stabilized schizophrenic men who were physically healthy and were maintained on a regimen of constant activity. They were weighed, nude, in the basal state each week and the total calorie consumption was individually adjusted by adding or subtracting bread, jelly, and dry breakfast cereal from the standard measured ration to maintain constancy of body weight. Actual food intakes, minus any plate waste, were recorded for each man for each meal.

In all the experiments reported here the men were divided into matched subgroups and subsisted on the experimental diets in a cross over or switchback arrangement. This is illustrated by the design of experiment N, given in table 1.

Food actually eaten was recorded for each man and the nutrient contents, including fatty acids, were calculated from tables of average values for each food item. The experimental fats were analyzed independently in each case, and, in the case of the more recent experiments (since 1955) including the new experiment series N and P, the fats in samples of the whole diets, as eaten, were extracted and analyzed directly for fatty acid composition.

Series N Experiments

The design of the N series of experiments is given in table 1. Note that this comprises 2 independent sets of experiments on 2 different groups of 12 men each, each group being subdivided into 2 subgroups who made their safflower oil vs. corn oil dietary changes in reverse order so that any time trend independent of the diet was automati-
cally compensated for. This cross over arrangement was not feasible in the case of the H diet but the relative stability of the cholesterol values in repeated samplings indicates that no large time trend was present.

Table 2 shows the mean values for calories, proteins, and total fats in the diets and the body weights at the ends of the dietary periods in the N series. Table 3 gives the mean intakes of glycerides of saturated, monoene, and polyene fatty acids. Table 4 gives the serum cholesterol values (means of duplicates) for each of the 24 men on each of 2 occasions on each of the 3 types of diets.

Analysis of 19 Additional Sets of Comparisons

The basic data of 10 sets of experiments involving corn oil or fish oil have been published elsewhere, but were not used in the derivation of equation 2A. Full data on 3 sets of new experimental comparisons (the P series) have also been published. Table 5 summarizes these data, as well as the 6 sets of comparisons from the N series, in a form suitable for application to equation 2.

We have shown that a small part of the effect on serum cholesterol of corn oil is apparently attributable to the larger amount of nonsaponifiable matter in this oil as compared with the other fats tested. When fed at the level of 100 Gm. daily in a diet balanced at about 3,200 calories, as in the experiments involving corn oil in table 5, an average depression of about 6 mg. cholesterol per 100 ml. serum is attributable to the excess nonsaponifiable matter (phytosterol) in the corn oil. Accordingly, table 5 allows for this fact in the column of predicted "Δ Chol., corrected."

It is clear that equation 2A provides satisfactory prediction of the average cholesterol changes observed in these 19 sets of experiments, and that this is improved by allowing for the nonsaponifiable matter in corn oil. The standard error of estimate was calculated as $\text{SEE} = (\Sigma \Delta^2/N)^{1/2}$ where $\Delta$ is the discrepancy between observed and predicted changes in cholesterol concentration in the serum and $N$ is the number of pairs (19). The result with equation 2A is $\text{SEE} = \pm 7.1$ mg. per 100 ml. With use of the prediction corrected for the nonsaponifiable matter in corn oil (when applicable), the value is $\text{SEE} = \pm 5.1$.

A completely independent solution of equation 2 with the method of least squares was obtained by using the observed raw data as given in table 5 and again using these data corrected by 6 mg. per 100 ml. in the case of the corn oil experiments. In both cases the value of the coefficient $c$ in equation 2 was found to be statistically not different from zero. The results of the least squares solutions of the regression equation are given in table 6 for the 19 sets of observed data of table 5, for these data corrected by 6 mg. in the cases where corn oil was involved, and also for the 15 sets of data when the comparisons with the H diets were omitted (because a switchback design was not employed to eliminate all possibility of artefact by time trend). The latter solution with 15 sets of data avoids any possibility of error from uncompensated time trends in the comparisons involving the H diets.

The coefficients obtained in the 4 solutions are statistically not different from each other or from those in the previous solution (eq. 2A) from the data in 41 different sets of experiments. A test of the homogeneity of variances indicated that the data in table 5 may be considered to be homogeneous with those in the previously reported 41 sets (table VII), so it was considered justifiable to pool the entire material of 60 sets of data. The method of least squares was used to obtain another solution for equation 2A, with use of the raw data of the 60 sets. This yielded

$$\Delta \text{Chol.} = 2.73 \Delta S - 1.30 \Delta P \quad (2B)$$

When the corrected $\Delta$ Chol. values for the experiments involving corn oil were used, the 60 sets of data yielded the solution

$$\Delta \text{Chol.} = 2.68 \Delta S - 1.23 \Delta P \quad (2C)$$

The standard errors of estimate for equations 2B and 2C were calculated from the differences between observed and predicted $\Delta$ Chol. and it was found that $\text{SEE} = \pm 5.79$ for equation 2B and $\text{SEE} = \pm 4.88$ for equation 2C. The standard errors of the 2 coefficients in equation 2B are $SE_a = \pm 0.086$, $SE_b = \pm 0.100$, and in equation 2C, $SE_a = \pm 0.084$, $SE_d = \pm 0.073$.

Intrinsic Differences Among Men

The data in table 4 illustrate the well known fact that even on exactly the same diet and when other conditions in the mode of life are standard-ized, there are consistent differences in serum cholesterol level among individual men. Subjects

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**Table 3.—Experiment N. Mean Intakes of Glycerides of Saturated, Monoene, and Polyene Fatty Acids**

<table>
<thead>
<tr>
<th>Group</th>
<th>Saturated</th>
<th>Monoene</th>
<th>Polyene</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Gm./day</td>
<td>% Cal.</td>
<td>Gm./day</td>
</tr>
<tr>
<td>WY</td>
<td>65.9</td>
<td>18.6</td>
<td>61.9</td>
</tr>
<tr>
<td>WY SO</td>
<td>29.3</td>
<td>8.2</td>
<td>26.4</td>
</tr>
<tr>
<td>WY CO</td>
<td>33.6</td>
<td>9.5</td>
<td>42.4</td>
</tr>
<tr>
<td>XZ</td>
<td>64.6</td>
<td>18.5</td>
<td>61.1</td>
</tr>
<tr>
<td>XZ SO</td>
<td>28.8</td>
<td>8.2</td>
<td>25.7</td>
</tr>
<tr>
<td>XZ CO</td>
<td>32.8</td>
<td>9.3</td>
<td>41.5</td>
</tr>
</tbody>
</table>

H, house; SO, safflower oil; CO, corn oil.
TABLE 4.—Serum Total Cholesterol Values, Mg. per 100 Ml., for the 24 Men in the X Series of Experiments

<table>
<thead>
<tr>
<th>Subject</th>
<th>Diet H</th>
<th>Diet SO</th>
<th>Diet CO</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Day 26</td>
<td>Day 30</td>
<td>Day 19</td>
</tr>
<tr>
<td>W1</td>
<td>232</td>
<td>249</td>
<td>142</td>
</tr>
<tr>
<td>W2</td>
<td>207</td>
<td>222</td>
<td>150</td>
</tr>
<tr>
<td>W3</td>
<td>176</td>
<td>184</td>
<td>134</td>
</tr>
<tr>
<td>W4</td>
<td>220</td>
<td>231</td>
<td>172</td>
</tr>
<tr>
<td>W5</td>
<td>270</td>
<td>276</td>
<td>179</td>
</tr>
<tr>
<td>W6</td>
<td>227</td>
<td>224</td>
<td>192</td>
</tr>
<tr>
<td>Y1</td>
<td>204</td>
<td>202</td>
<td>180</td>
</tr>
<tr>
<td>Y2</td>
<td>219</td>
<td>230</td>
<td>178</td>
</tr>
<tr>
<td>Y3</td>
<td>235</td>
<td>228</td>
<td>170</td>
</tr>
<tr>
<td>Y4</td>
<td>184</td>
<td>178</td>
<td>154</td>
</tr>
<tr>
<td>Y5</td>
<td>170</td>
<td>164</td>
<td>137</td>
</tr>
<tr>
<td>Y6</td>
<td>188</td>
<td>176</td>
<td>142</td>
</tr>
</tbody>
</table>

Mean 211.00 213.66 160.83 161.66 161.00 156.91
Grand mean 212.33 ± 6.23 161.25 ± 3.86 158.95 ± 4.28

X1 174 179 129 132 153 144
X2 182 166 138 142 149 134
X3 256 241 196 200 199 204
X4 214 206 148 132 149 144
X5 208 196 160 136 161 162
X6 213 210 175 170 178 172
Z1 196 189 128 126 150 152
Z2 179 160 144 142 142 137
Z3 149 137 120 119 122 121
Z4 288 304 227 226 230 236
Z5 235 234 188 183 195 190
Z6 168 172 133 134 140 142

Mean 205.16 199.50 157.16 156.83 163.25 161.50
Grand mean 202.33 ± 8.44 157.00 ± 7.68 162.37 ± 6.46

The number of days of subsistence on each type of diet is indicated at the heads of the columns.

W5 and Z4, for example, are consistently relatively hypercholesteremic while subjects Y4 and Z6 are consistently low in cholesterol level. But do such men differ in responsiveness to a dietary change?

This question can be examined by comparing the magnitudes of the serum cholesterol responses, to a given dietary change, of experimental subjects classified according to their cholesterol values on a given control diet. This has been done by subdividing the 24 men in the X series into 2 groups of 12 men each. The "higher" group averaged 235.3 mg. per 100 ml. on the H diet and exhibited an average fall of 58.7 on the SO diet; the "lower" group had corresponding averages of 179.4 and 37.8 mg. per 100 ml. These results are given in table 7 together with a similar analysis of the data published by Starko on 154 patients on a control diet and again after subsistence on a rice-fruit diet. Both sets of data show an important relationship between responsiveness and the intrinsic (control) level of cholesterol.

Theoretically, to some extent such a difference in "responsiveness" could be more apparent than real. With a single measurement of serum cholesterol on the H diet, both random intrindividual variability and measurement error would combine to group overestimates in the "higher" group and underestimates in the "lower" group. Then a second measurement on the SO diet, with both sources of variation again acting at random, would tend to show larger Δ Chol. in the "higher" than in the "lower" group, even if there were, in fact, no real relationship between intrinsic level and Δ Chol. But this possibility can be eliminated by a more elaborate analysis of the data in table 4.

Comparison of the serum cholesterol values on the first and second occasions of blood drawing on a constant diet will reveal the magnitude of any such artefactitious trend. This comparison may be made by solving, with the method of least squares, the regression equation, Y = a + bX, where X is the value on the first occasion, Y is that predicted for the second occasion, a and b are the intercept and slope of the regression line, respectively. Table 8 shows that on each of the 3 diets the slope of the regression line relating the first to the second blood samples is not significantly different from unity and therefore there is no significant artefactitious trend.

Table 8 also shows the results of this regression analysis applied to the prediction of the individual cholesterol values on the SO and CO diets from the cholesterol values of the same individuals on the H diet. In all cases the slope, b, is different from unity with high significance.

The regression constants in table 8 pertain only to the data of table 4. It is more convenient to make the analysis in percentage terms which have general application:

\[ \Delta \% = a + bX\% \]  

where X\% is the cholesterol value of an individual expressed as a percentage of the mean of a group or population on the same given diet, and Δ\% is the predicted cholesterol change of that individual in response to a particular diet change, again expressed as a percentage of the mean change of the group undergoing the same dietary alteration. In these terms, for the data of table 4, we have the following solution for equation 4: H to SO, a = -73, b = 1.73; H to CO, a = -64, b = 1.64. Note that Δ\% = 100 when X\% = 100 and a = 100 (1-b).

Table 9 summarizes 3 more sets of original data suitable for analysis in the above terms. Four blood samples were drawn, on different days, from
SERUM CHOLESTEROL IN MAN

TABLE 5.—Mean Differences between % of Calories Provided by Glycerides of Saturated (ΔS), Monoene (ΔM), and Polyene (ΔP) Fatty Acids, Together with Mean (and S.E.) Serum Total Cholesterol Differences (Δ Chol.) Observed and Predicted from Equation 2.1

<table>
<thead>
<tr>
<th></th>
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<th></th>
<th></th>
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</thead>
<tbody>
<tr>
<td>13</td>
<td>HWX</td>
<td>Cottonseed-Corn Oil</td>
<td>3.4</td>
<td>-2.2</td>
<td>-1.5</td>
<td>19.3±4.2</td>
<td>11.3</td>
<td>17.3</td>
</tr>
<tr>
<td>14</td>
<td>JWX</td>
<td>Butterfat-Corn Oil</td>
<td>11.7</td>
<td>1.0</td>
<td>-13.5</td>
<td>45.1±3.6</td>
<td>49.7</td>
<td>55.7</td>
</tr>
<tr>
<td>14</td>
<td>JWX</td>
<td>Olive-Corn Oil</td>
<td>0.3</td>
<td>12.1</td>
<td>-12.6</td>
<td>27.2±3.6</td>
<td>17.3</td>
<td>23.3</td>
</tr>
<tr>
<td>14</td>
<td>JWX</td>
<td>Sunflower-Corn Oil</td>
<td>-0.2</td>
<td>1.9</td>
<td>1.5</td>
<td>10.5±2.8</td>
<td>1.5</td>
<td>7.5</td>
</tr>
<tr>
<td>12</td>
<td>JYZ</td>
<td>Cottonseed-Sardine Oil</td>
<td>0.6</td>
<td>0.1</td>
<td>-0.7</td>
<td>3.9±5.2</td>
<td>2.5</td>
<td>2.5</td>
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<tr>
<td>12</td>
<td>JYZ</td>
<td>Olive-Sardine Oil</td>
<td>-3.3</td>
<td>16.4</td>
<td>-13.0</td>
<td>10.1±4.6</td>
<td>8.0</td>
<td>8.0</td>
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<td>Sardine-Corn Oil</td>
<td>3.6</td>
<td>-2.7</td>
<td>-0.7</td>
<td>20.1±3.8</td>
<td>10.8</td>
<td>16.8</td>
</tr>
<tr>
<td>12</td>
<td>JYZ</td>
<td>Olive-Corn oil</td>
<td>0.3</td>
<td>13.7</td>
<td>-13.7</td>
<td>30.5±5.6</td>
<td>18.7</td>
<td>24.7</td>
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<td>JYZ</td>
<td>Butterfat-Corn Oil</td>
<td>13.2</td>
<td>1.4</td>
<td>-14.8</td>
<td>59.5±5.7</td>
<td>55.6</td>
<td>61.6</td>
</tr>
<tr>
<td>12</td>
<td>JYZ</td>
<td>Butterfat-Sardine Oil</td>
<td>9.6</td>
<td>4.1</td>
<td>-14.1</td>
<td>39.4±5.6</td>
<td>44.8</td>
<td>44.8</td>
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<tr>
<td>12</td>
<td>NWY</td>
<td>House-Safflower</td>
<td>10.4</td>
<td>10.1</td>
<td>-19.8</td>
<td>51.1±7.1</td>
<td>54.4</td>
<td>54.4</td>
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<tr>
<td>12</td>
<td>NWY</td>
<td>House-Corn Oil</td>
<td>9.1</td>
<td>5.5</td>
<td>-14.4</td>
<td>53.4±6.2</td>
<td>43.8</td>
<td>49.8</td>
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<tr>
<td>12</td>
<td>NWY</td>
<td>Safflower-Corn Oil</td>
<td>-1.3</td>
<td>4.6</td>
<td>5.4</td>
<td>2.3±2.7</td>
<td>-10.6</td>
<td>-4.6</td>
</tr>
<tr>
<td>12</td>
<td>NXZ</td>
<td>House-Safflower</td>
<td>10.3</td>
<td>10.2</td>
<td>-19.8</td>
<td>45.3±4.2</td>
<td>54.1</td>
<td>54.1</td>
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<tr>
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<td>NXZ</td>
<td>House-Corn Oil</td>
<td>9.2</td>
<td>5.7</td>
<td>-14.3</td>
<td>40.0±3.8</td>
<td>43.9</td>
<td>49.9</td>
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<tr>
<td>12</td>
<td>NXZ</td>
<td>Safflower-Corn Oil</td>
<td>-1.1</td>
<td>4.5</td>
<td>5.5</td>
<td>-5.4±2.4</td>
<td>-10.2</td>
<td>-4.2</td>
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<tr>
<td>12</td>
<td>P</td>
<td>Olive Oil-Low Fat</td>
<td>4.1</td>
<td>20.1</td>
<td>2.6</td>
<td>12.5±4.4</td>
<td>14.4</td>
<td>14.4</td>
</tr>
<tr>
<td>12</td>
<td>P</td>
<td>Safflower Oleo.-Low Fat</td>
<td>4.2</td>
<td>2.0</td>
<td>2.6</td>
<td>12.5±4.5</td>
<td>14.9</td>
<td>14.9</td>
</tr>
<tr>
<td>12</td>
<td>P</td>
<td>Safflower Oleo.-Olive Oil</td>
<td>0.1</td>
<td>-18.1</td>
<td>0</td>
<td>0±3.1</td>
<td>0.3</td>
<td>0.3</td>
</tr>
</tbody>
</table>

The last column gives the predicted values when allowance is made for an average of 6 mg. % cholesterol depression for non-saponifiable matter in corn oil when this oil comprises about 28% of total calories.

Each man in the third and fourth weeks of subsistence on fixed control diets (not identical in the 3 experiments). Two to 4 more blood samples were drawn, again on different days, after several weeks of subsistence on another diet, variously low in total fat or involving substitution of a vegetable oil for saturated fats, in the several experiments. Calory equilibrium was maintained throughout each blood sample was analyzed, in duplicate, for serum cholesterol. Table 9 gives the means of these duplicates for each individual, expressed as percentages of group means in the control period and as percentages of the group mean changes in the experimental period to facilitate analysis in the form of equation 4.

In all 3 experiments it was found that the cholesterol response to the dietary change was linearly related to the intrinsic level of the individuals as indicated by their values on the control diet. The slopes of the least-squares regression lines, in equation 4, were b = 1.92, b = 1.93, and b = 1.59 in experiments V, VII, and IX, respectively.

The applicability of equation 4 to other groups of persons and to other situations producing serum cholesterol changes may be checked with data in the literature. Individual "before" and "after" serum total cholesterol values are provided by Hatch et al.8 on 24 hypertensive men changed to a rice-fruit diet, by Best et al.9 on 14 men before and during daily saponifiable matter, and beta lipoprotein cholesterol values by Farquhar and Sokolow10 on 15 patients before and after substitution of safflower oil for a fixed part of the saturated fats in the diet. The findings from these data are summarized in table 10, which also gives the comparable figures from the original data of the present paper.

The estimates of the slope, b (± S. E.), are given in table 10. None of these differs significantly from each other. The largest difference, that between the series of Hatch et al. (b = 2.80) and experiment IX (b = 1.59), has a standard error of ±0.79, which gives a t value of 1.53. Since for a probability of 0.1 the required t value is 1.7, the difference is insignificant.

A good general estimate for the slope, b, would be the weighted mean of all 8 estimates in table 10, with the number of persons in each series used as the weights. This weighted mean is b = 1.91. Accordingly, we have

$$\bar{b} = 1.91 \times \% - 1$$ (4A)

It may be noted that the individual data of Starke7 are not available for a least-squares solu-
TABLE 6.—Solutions of Equation 2, \( \triangle \) Chol. =
\[ b \Delta S + d \Delta P, \]
with the Data in Table 5

<table>
<thead>
<tr>
<th>Data</th>
<th>Sets of data</th>
<th>b</th>
<th>d</th>
</tr>
</thead>
<tbody>
<tr>
<td>Raw</td>
<td>19</td>
<td>2.65</td>
<td>-1.33</td>
</tr>
<tr>
<td>Corrected</td>
<td>15</td>
<td>2.39</td>
<td>-1.27</td>
</tr>
<tr>
<td>Raw</td>
<td>15</td>
<td>2.67</td>
<td>-1.43</td>
</tr>
<tr>
<td>Corrected</td>
<td>15</td>
<td>2.41</td>
<td>-1.31</td>
</tr>
</tbody>
</table>

'Raw' uses the observed data without changes; 'Corrected' uses the same data but allows 6 mg. of cholesterol per 100 ml. for the effect of extra non saponifiable matter in corn oil. The solutions with only 15 sets of data were obtained by omitting comparisons involving the H diets in the N series of experiments.

The solution of equation 4, but the means of the 2 groups allow a solution by simultaneous equations. The result is

\[ \bar{b} \% = 1.99 \times X\% - 99 \]  

(4B)

which is in good agreement with equation 4A.

**General Prediction of Response to Dietary Change**

Equation 2C (and 2B) was derived from data on men with a narrow range of group average intrinsic cholesterol values. On a diet of the current American type, with S about 20 and P about 3 (per cent of total calories), these group averages were in the range of 215 to 235 with a grand average of about 225. Equation 4A shows the relative difference in response to diet of men who differ intrinsically in cholesterol level. Hence, from the information from equations 2C and 4A, it is now possible to formulate a general prediction of cholesterol responses to change in dietary fat.

Let \( Z_1, Z_2, \) and \( Z_3 \) refer to the average serum cholesterol values of a reference group of men on diets characterized by \( S_1, P_1; S_2, P_2; \) and \( S_3, P_3, \) respectively, the reference group being so defined that they will have \( Z_1 = 225 \) when \( S_1 = 20 \) and \( P_1 = 3. \) In other words, equation 2C will apply strictly to this reference group, and for such a reference group a change from a diet with \( S_1 = 20, P_1 = 3 \) to a second diet represented by \( S_2, P_2, \) should result in an average serum cholesterol change, as predicted by equation 2C, of \( \Delta \) Chol. = \( Z_2 - 225 = 2.68 (S_2 - 20) - 1.23 (P_2 - 3), \)

For another group of men, let \( G_1, G_2, \) and \( G_3 \) refer to their average cholesterol values on the above diets \( (S_1, P_1; S_2, P_2; S_3, P_3), \) where \( G_1 \neq 225 \) when \( S_1 = 20 \) and \( P_1 = 3. \) These men, in other words, are intrinsically different from the reference group to whom equation 2C applies without correction.

Now consider the situation where \( G_2 \) has been measured on a diet with \( S_2 \) and \( P_2 \) and it is desired to predict the value of \( G_3 \) when the diet is changed to \( S_3 \) and \( P_3. \) In order to correspond with the form of equation 4A, it is necessary to put the cholesterol values into percentage terms: \( X\% = 100 \frac{G_2}{Z_2} \) and change from diet 2 to diet 3, \( \Delta \% = 100 \left( \frac{G_3 - G_2}{Z_3 - Z_2} \right). \)

These expressions may be put into equation 4A:

\[ \Delta \% = 100 \left( \frac{G_3 - G_2}{Z_3 - Z_2} \right) = 1.91 \left( \frac{100 G_2}{Z_2} \right) - 91 \]  

(4C)

which may be rearranged:

\[ G_3 = 0.91 (Z_2 - G_2 - Z_3) + 1.91 G_2 \frac{Z_3}{Z_2} \]  

(4D)

The values of \( Z_2 \) and \( Z_3 \) necessary for the solution of equation 4D are found by the application of equation 2C, using the reference value of \( Z_1 = 225 \) when \( S = 20 \) and \( P = 3: \)

\[ Z_2 - 225 = 2.68 (S_2 - 20) - 1.23 (P_2 - 3) \]  

(2D)

whence

\[ Z_2 = 175 + 2.68 S_2 - 1.23 P_2 \]

\[ Z_3 = 175 + 2.68 S_3 - 1.23 P_3 \]  

(2E)

As an example of the calculation, take the case where \( G_2 = 300 \) when \( S_2 = 25, P_2 = 4, \) and it is desired to predict \( G_3 \) when \( S_3 = 10, P_3 = 3. \) In this case we have

\[ Z_2 = 175 + 2.68 (25) - 1.23 (4) = 237.1 \]

\[ Z_3 = 175 + 2.68 (10) - 1.23 (10) = 195.6 \]

\[ G_3 = 0.91 (237.1 - 300.0 - 195.6) + 1.91 (300) (195.6) / (237.1) = 235.8 \]

**Test of Predictions with Published Data**

There are surprisingly few data in the literature suitable for test of equations 2E and 4D (or 2C and 4A). Malmonos and Wiggand have reported data from 4 dietary experiments on "healthy volunteers" and these are summarized, together with predictions, in table 11. In the first 2 of those
experiments the dietary periods were long enough (4 weeks each) to assure approximation to a cholesterol plateau, but in the other experiments the periods are too short (1 to 2½ weeks) to be sure on this score.

The predictions in table 11 make no allowance for the cholesterol-lowering effect of the nonsoap-lipid fraction, and the values observed for the control diet would be about 6 to 8 mg. of cholesterol per 100 ml. A reasonable estimate of this effect would be about 6 to 8 mg. of cholesterol per 100 ml. If this is included, the predicted values (line 11 of table 11) would be approximately 60, 55, 50, and 45. While the prediction may tend to exceed the observed values, especially in experiment 1, it appears that the same general relationships hold for the volunteers in Land, Sweden, as for the Hastings subjects in Minnesota.

The data published by Farquhar and Sokolow do not include fully adequate details for the "control" diet eaten by their patients but an approximate test is possible. Fats provided an average of 41 per cent of the calories in the control diet and 95 per cent of these fats were from animal sources or were hardened vegetable fats. The experiment consisted of unsupplanting 30 per cent of the fat calories in the control diet by safflower oil. On the presumption that the control diet conformed to the usual American food pattern, a rough estimate is that the average composition of the fats in the control diet was about 50 per cent saturated and from 6 to 8 per cent polyunsaturated. The safflower oil used to supplant these fats in the amount of 30.8 per cent of the total calories contained 6 per cent saturated and 73 per cent polyunsaturated fatty acids. Accordingly, if the patients of Farquhar and Sokolow were like our

![Graph](http://circ.ahajournals.org/)

**Fig. 1 Top.** Observed and predicted individual serum cholesterol values following dietary change by the men studied by Ahrens and others. The circles with crosses correspond to data from patients 13 and 15 whose cholesterol values were unusually resistant to dietary effects.

**Fig. 2 Bottom.** Observed and predicted individual serum cholesterol values for the men in experiment N.

The own subjects, the change from the control to the safflower oil diet would produce an average serum total cholesterol change of ∆ Chol. = 0.308 (2.68 (6 - 50) + 1.23 (73 - 7)) = - 59 mg. %.

Farquhar and Sokolow reported an average change of - 52 ± 5.8 mg. of beta lipoprotein cholesterol per 100 ml. with no change in the alpha fraction. Hence we have observed ∆ Chol. = - 52, predicted ∆ Chol. = - 59.

### Table 8

<table>
<thead>
<tr>
<th>Diets compared</th>
<th>N</th>
<th>a</th>
<th>b</th>
</tr>
</thead>
<tbody>
<tr>
<td>H₂ vs. H₁</td>
<td>24</td>
<td>25.2</td>
<td>1.11±0.06</td>
</tr>
<tr>
<td>SO₂ vs. SO₁</td>
<td>24</td>
<td>9.7</td>
<td>0.04±0.06</td>
</tr>
<tr>
<td>CO₂ vs. CO₁</td>
<td>24</td>
<td>5.9</td>
<td>1.02±0.05</td>
</tr>
<tr>
<td>Hₙ, CO₂ vs. H₁, SO₂, CO₁</td>
<td>24</td>
<td>5.3</td>
<td>1.02±0.03</td>
</tr>
<tr>
<td>SO₁ vs. H₁</td>
<td>24</td>
<td>20.3</td>
<td>0.67±0.09</td>
</tr>
<tr>
<td>SO₁ vs. H₂</td>
<td>24</td>
<td>68.5</td>
<td>0.44±0.08</td>
</tr>
<tr>
<td>SO₂, SO₁ vs. H₁, H₂</td>
<td>48</td>
<td>35.2</td>
<td>0.60±0.06</td>
</tr>
<tr>
<td>CO₁ vs. H₁</td>
<td>24</td>
<td>37.9</td>
<td>0.60±0.08</td>
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<tr>
<td>CO₁ vs. H₂</td>
<td>24</td>
<td>58.4</td>
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<tr>
<td>CO₁, CO₂ vs. H₁, H₂</td>
<td>48</td>
<td>29.7</td>
<td>0.63±0.05</td>
</tr>
<tr>
<td>SO₂, SO₁, CO₁, CO₂ vs. H₁, H₂</td>
<td>96</td>
<td>32.4</td>
<td>0.61±0.04</td>
</tr>
</tbody>
</table>

H, house; SO, safflower oil; CO, corn oil. Subscripts indicate the occasions. N, number of comparisons.
TABLE 9.—Individual Serum Total Cholesterol Values, $X$, and the Changes, $Y$, in Response to Diet Changes, Expressed as Percentages of the Mean Values for the Group

<table>
<thead>
<tr>
<th>Subject</th>
<th>Experiment V</th>
<th></th>
<th>Subject</th>
<th>Experiment VII</th>
<th></th>
<th>Subject</th>
<th>Experiment IX</th>
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<tr>
<td></td>
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<td></td>
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<td>$Y$</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>J34</td>
<td>92</td>
</tr>
</tbody>
</table>

Mean (mg. %) (238.1) (34.9) Mean (mg. %) (217.3) (38.9) Mean (mg. %) (218.4) (33.2)

Four blood samples were analyzed from each man on each of two diets in each experiment. The absolute grand means, in mg. per 100 ml., are given in parentheses at the end of each column.

However, the agreement between observation and prediction is not quite this good. On the average, the patients of Farquhar and Sokolow seem to be intrinsically somewhat hypercholesteremic, so the predicted $\Delta$ Chol. should allow for this fact. Accordingly, the best prediction for $\Delta$ Chol. would be somewhat greater than -59, more like -70 mg. per 100 ml. Alternatively, it could be suggested that their patients did not precisely exchange 30.8 per cent of control diet fat calories for safflower oil. If it is presumed that they faithfully ingested the safflower oil daily in an amount calculated to represent about 31 per cent of their calorie requirements, it is questionable whether the corresponding reduction in their control diet was achieved entirely at the expense of control diet fat. For example, if they were no longer allowed to have butter, did they actually continue to eat the same amount of bread as before? An average reduction of only a slice or two of bread daily during the safflower oil period would account for the discrepancy between observation and prediction.

Ahrens et al. provided adequate dietary data for their experiments with formula feeding but their material is peculiar in several regards: 1.
SERUM CHOLESTEROL IN MAN

Table 10.—Estimates of the Intercept, a, and the Slope, b, in Equation 4: \( \Delta \% = a + bX\% \), where \( X\% \) is the Individual Serum Cholesterol Value as \% of the Group Mean on a Reference Diet, and \( \Delta \% \) is the Predicted Change of the Same Person as \% of the Group Mean Change

<table>
<thead>
<tr>
<th>Series</th>
<th>N</th>
<th>Ref. Chol. (mg. %) Mean ± S.D.</th>
<th>( \Delta ) Chol. (mg. %) Mean ± S.D.</th>
<th>a</th>
<th>b ± SEa</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hatch et al.14</td>
<td>24</td>
<td>222.1 ± 34.0</td>
<td>34.6 ± 34.8</td>
<td>-180</td>
<td>2.80 ± 0.74</td>
</tr>
<tr>
<td>Best et al.15</td>
<td>14</td>
<td>312.1 ± 188.9</td>
<td>53.0 ± 55.4</td>
<td>-110</td>
<td>2.10 ± 0.22</td>
</tr>
<tr>
<td>Farquhar and Sokolow16</td>
<td>15</td>
<td>232.7 ± 41.7</td>
<td>48.7 ± 22.5</td>
<td>-92</td>
<td>1.69 ± 0.56</td>
</tr>
<tr>
<td>N series, H to SO</td>
<td>24</td>
<td>207.3 ± 25.4</td>
<td>48.2 ± 19.9</td>
<td>-73</td>
<td>1.75 ± 0.25</td>
</tr>
<tr>
<td>N series, H to CO</td>
<td>24</td>
<td>207.3 ± 25.4</td>
<td>46.7 ± 19.2</td>
<td>-64</td>
<td>1.64 ± 0.24</td>
</tr>
<tr>
<td>Expt. V</td>
<td>18</td>
<td>238.1 ± 44.9</td>
<td>34.9 ± 15.6</td>
<td>-92</td>
<td>1.92 ± 0.39</td>
</tr>
<tr>
<td>Expt. VII</td>
<td>24</td>
<td>217.3 ± 34.4</td>
<td>38.9 ± 18.2</td>
<td>-93</td>
<td>1.93 ± 0.58</td>
</tr>
<tr>
<td>Expt. IX</td>
<td>26</td>
<td>218.4 ± 35.5</td>
<td>53.2 ± 21.4</td>
<td>-59</td>
<td>1.59 ± 0.28</td>
</tr>
<tr>
<td>Weighted mean</td>
<td>169</td>
<td>226.7</td>
<td>44.5</td>
<td>-91</td>
<td>1.91</td>
</tr>
</tbody>
</table>

Group means (and standard deviations) are in absolute terms (mg. %).
N, number of persons.

They used no ordinary diets but only liquid formulas. 2. Individual patients but studied repeatedly but it is not possible to analyze group averages so as to reduce the factor of individual variation. 3. Most of their patients were metabolically abnormal, as indicated by hypercholesteremia, xanthomatisosis, etc. Two patients (nos. 13 and 15), who suffered from arteriosclerotic heart disease and myocardial infarction as well as xanthomatisosis and hypercholesteremia, were remarkable in being almost completely uninfluenced by the diet.

However, it is of interest to consider the predictions for the results of changes in the fats in the formula diet. Figure 1 includes all of the data on the men studied by Ahrns et al. with the exception of comparisons involving coconut oil. The latter were omitted because of our suspicion that the short-chain fatty acids in coconut oil have a rather different quantitative effect from the fatty acids in most common food fats. The mean chain length in coconut oil is only 12.4 compared with 15.0 for butter and 16 to 17.5 for most other food fats and oils. The predicted values in figure 1 were obtained by the application of equations 4D and 2E. The coefficient of correlation between predicted and observed values is \( r = 0.94 \). If the 3 values from patient no. 15 are omitted, the coefficient of correlation, between the observed cholesterol change in response to diet change and that predicted, is \( r = 0.955 \). Their patient no. 1, who had the most extreme hypercholesteremia, makes a large contribution to the correlation coefficient, but even when he is omitted the correlation between observed and predicted \( \Delta \) Chol. is \( r = 0.86 \).

For comparison with figure 1, we have used equations 2E and 4D to predict the individual cholesterol responses of our subjects in changing from the H to the SO diet in experiment N. The result is given in figure 2. The correlation between observed and predicted cholesterol values of the individual men on the SO diet is \( r = 0.85 \). It should be observed, of course, that intrapersonal variability in serum cholesterol, plus the error of the method of measurement, means that any single estimate of cholesterol change is subject to an appreciable error. Accordingly, in a sample of subjects who exhibited only a limited range of intrinsic differences, it cannot be expected that an extremely high correlation will be found between observed and predicted values.

**Discussion**

The variations in total serum cholesterol concentration considered here are the result of variations in the cholesterol in the beta lipoprotein fraction as separated by paper electrophoresis or by cold ethanol fractionation. The alpha lipoprotein cholesterol is not altered by changes in the fat in the diet and, for that matter, no dietary manipulation is known to affect the level of the alpha fraction. Farquhar and Sokolow16 found the average serum concentration of alpha lipoprotein cholesterol to be the same in their subjects on their control diet, on their safflower oil diet, and during the periods of sitosterol administration. Similar constancy of the alpha fraction was found in all of our experiments with different amounts and kinds of fats in the diet, as well as in our comparisons of populations habitually subsisting on different diets.13, 14
The present analysis is concerned entirely with adults. We have conducted no experiments with women or children. There is no information in the literature which proves that cholesterol responses to the diet are identical in the 2 sexes but if they are not, they are similar, at least, to judge from the experimental data of Groen et al.,15 Ahrens et al.,12 and Malmros and Wigand.11 Surveys of both sexes in populations subsisting habitually on different diets also indicate that men and women are much alike in their cholesterol response to the diet.18

The surveys of Scrimshaw, Balsam and Arroyave16 suggest that the general cholesterol response of children to fats in the diet is similar to that of adults. It is clear that children17 are like adult men in being uninfluenced by large variations in dietary cholesterol.18 However, though we suspect that the relative effects on the serum cholesterol of different types of fatty acids in the diet are the same in children and in adults, the available data do not make it possible to decide whether the absolute effect is the same. It cannot be stated, for example, that intrinsic serum cholesterol level in a child bears the same relationship to serum cholesterol responsiveness as found among adults.

The present analysis is concerned with the isocaloric exchange of fats for other fats or for carbohydrates in the diet. What may happen when a fat supplement is added to the diet is conjectural in the absence of other information. If the body weight remains constant in spite of such supplementation, this means that either the calories spent for physical activity have been increased or that the basic diet has been reduced in an amount to correspond with the calories in the supplement. The latter would seem to be more likely in most cases and the question then is as to the particular kinds of foods reduced in the basic diet.

We presume that, in general, the ingestion of a fat supplement will tend to decrease the appetite for fat and that, in effect, the prescription of a fat supplement such as corn or safflower oil may automatically bring about some degree of substitution for other fat in the basic diet. The "other fats" would be, of course, those most easily altered in the diet of the patient by his spontaneous choice, i.e., the saturated fats of butter and of meats. Unless allowance were made for the possibility of such spontaneous substitution, it would be
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erroneous to credit a cholesterol response solely to the effect of the fat supplement.

We have discussed this point in connection with our experiments with supplements of different kinds of fats for medical students who were instructed to continue with their ordinary diet while taking the supplements.\(^{19}\)

That some substitutions did occur was shown by the fact that the students did not gain enough weight to correspond with the extra calories in the supplements.

The recent report of Perkins, Wright, and Gatje\(^ {20}\) is of interest in this connection. Their 24 male medical students received, alternately, an emulsion providing 48 Gm. safflower oil daily or a carbohydrate “placebo” providing 450 calories. The safflower oil preparation, which also contained a mixture of 5 emulsifying (surface active) agents, supplied 450 calories. During the period on the supplement there was an average weight gain of 2 Kg, but it is not clear whether this refers only to the 6 to 7 weeks on the safflower oil supplement or to the entire 13 weeks on the oil or placebo supplements. In any case, the weight gain is less than would correspond to the extra calories provided in the supplement, an excess which adds up to about 21,000 calories during the oil period and 11,000 during the placebo. In terms of adipose tissue these calories correspond to about 3 Kg. for the oil period and close to 5 Kg. for the entire 13 weeks. We conclude therefore that the basic diet must have been reduced or that these young men increased their physical activity by about 200 calories daily.

Perkins et al. stated that the average basic diet (estimated by questionnaire) before the start of the study provided 2,350 calories daily. The usual composition of safflower oil being assumed, with about 12 per cent saturated and 78 per cent polyunsaturated fatty acids, this means that the 48 Gm. oil supplement provided a daily average of about 5.8 Gm. saturated and 37.4 Gm. polyunsaturated fatty-acid glycerides. The basic diet, of the usual American type, provided 42 per cent of total calories from fats, so it may be estimated as being characterized as \(S_1 = 21\) and \(P_1 = 3\) (per cent of calories from glycerides of saturated and poly-unsaturated fatty acids). During the oil supplementation, the basic diet was not actually decreased, the diet would have been \(S_2 = 19.5\) and \(P_2 = 14.5\).

From equation 2C the average expected cholesterol change would be then \(\Delta \text{Chol.} = 2.68 (19.5 - 21.0) - 1.23 (14.5 - 3.0) = -18.1\) mg. per 100 ml. However, these young men are, compared with our reference, intrinsically hypocholesteremic, with an average of 191.5 mg. cholesterol per 100 ml. on the control diet, while the reference men would have an average of 229.5 on that diet. Correcting for this fact (by applying equation 4D), the predicted result of the oil supplement on the cholesterol level of these young men is an average fall of 15.1 mg. per 100 ml. Actually, in both safflower oil and placebo periods the serum cholesterol steadily rose, and after 13 weeks the average was an increase of 22 mg. for the 12 men ending with the placebo and of 29 mg. for the 12 who had the oil emulsion during the final 6 weeks.

Consideration of the experiment of Perkins, Wright, and Gatje leads to the conclusion that only a small effect of the safflower oil emulsion could have been expected and that other factors, probably including changes in the basic diets, were operative during the period of the experiment. The latter conclusion is also in accord with the expectation that freely selected diets will necessarily change from late summer (the control period) to early winter.

**Summary**

The effects on serum cholesterol concentration of different percentages of total calories supplied by glycerides of saturated (S), monoene (M) and polyunsaturated (P) fatty acids were analyzed in terms of the prediction equation

\[\text{Chol}_2 - \text{Chol}_1 = b(S_2 - S_1) + c(M_2 - M_1) + d(P_2 - P_1)\]

where Chol. is in mg. per cent and the subscripts refer to first and second diets.

The coefficients in this equation obtained by the method of least squares from the data of
19 sets of dietary comparisons, with 12 to 22 men in each set, were not different with statistical significance from those obtained in a previous analysis of 41 sets of similar data. The final result from the combined analysis of all 60 sets of comparisons was \( b = 2.68 \pm 0.08, c = 0, d = -1.23 \pm 0.07 \).

These coefficients apply to the average \( \Delta \text{Chol.} \) of reference men in calory equilibrium who have an average of about 225 mg. cholesterol per 100 ml. of serum on a diet in which the values of \( S \) and \( P \), as percentages of total calories, are about 20 and 3, respectively.

Men who are intrinsically hypercholesteremic exhibit greater cholesterol response to dietary changes, while hypocholesteremic men are less responsive than reference men. In general, if \( X\% \) is the cholesterol value of an individual, expressed as percentage of the average of a group of men on the same diet, and \( \Delta% \) is that individual's cholesterol response in changing to another diet, also expressed as percentage of the group average response to the same dietary change, we have

\[
\Delta% = 1.91 X\% - 91.
\]

Analyses of 9 sets of data (5 original and 4 from the literature) yielded coefficients not significantly different from those in the above equation.

Published data from controlled dietary experiments in Sweden and in New York show serum cholesterol changes resulting from given dietary changes that are highly correlated with the results predicted from calculations based on the equations and coefficients given in the foregoing.

It is concluded that in man (a) oleic acid glyceride in the diet is substantially the equivalent, in serum cholesterol effect, of equal calories from carbohydrate, (b) the glycerides of saturated fatty acids in ordinary food fats raise the serum cholesterol level, those of polyunsaturated fatty acids (mainly linoleic acid) lower it, and in this respect it requires slightly more than 2 Gm. of linoleic acid to counter the effect of 1 Gm. of a saturated fatty acid such as stearic or palmitic acid, (c) intrinsic relative hypercholesteremia is generally associated with relative hyper-responsiveness to dietary fat changes, and (d) the above relationships allow reasonably accurate predictions of group average responses in serum cholesterol level to given changes in dietary fats.

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**Summario in Interlingua**

Le effectos produce in le concentration de cholesterol del sero per supplere le calorias total in le forma de varie proportiones de glyceridos de acidos grassse saturate (S), monoenic (M), e poly-nonsaturate (P) esseva analysate super le base del equation de predicition:

\[
\text{Chol}_2 - \text{Chol}_1 = b(S_2 - S_1) + c(M_2 - M_1) + d(P_2 - P_1)
\]

in que Chol representa cholesterol in mg pro cento e in que le cifras abassate se refere a un prime e secunde dieta.

Le coefficientes obtenite per iste equation secundo le metodo de quadratos minime ab le datos de 19 experimentos de comparation dietari, con inter 12 e 22 homines participante in cata un del experimentos, non differeva a grados de signification statistic ab le coefficientes correspondentie que esseva obtenite previemente in 41 experimentos de natura similair. Le resultato final ab le analyse combine de omme le 60 comparasiones esseva \( b = 2.68 \pm 0.08, c = 0, d = -1.23 \pm 0.07 \).
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Iste coeficientes es applicabile al valor medie de $\Delta$ Chol de subjectos de referentia in equilibrio caloric qui ha un valor medie de circa 225 mg de cholesterol per 100 ml de sero a un dieta in que le valores de S e P, como procentage del calorias total, es circa 20 e 3, respectivemente.

Homes qui es intrinsecamente hypercholesterolemic exhibi plus pronunciate respon-sas cholesterol a alterationes dietari, durante que homes hypocholesterolemic es minus responsive que homes de referentia. In general, si $X\%$ es le valor de cholesterol in un certe individuo, exprimite como procentage del valor medie de un grupo de homes con le mesme dieta, e si $\Delta\%$ es le responsa cholesterolic del mesme individuo como efecto del transition a un altre dieta, etiam exprimite como procentage del valor medie del responsas de un grupo al mesme alteration dietari, nos obtenete:

$$\Delta\% = 1.91 \times X\% - 91.$$ 

Le analyse de 9 series de datos, 5 original e 4 ab le litteratura, resultava in coefficientes non significativamentemente differente ab illos in le supra-presentate equation.

Datos in le litteratura, ab controlate experimentos dietari effectuate in Sveda e in New York, monstra alterationes del cholesterol del sero in consequentia de specific alterationes dietari que es correlationate a alte grados con le resultatos predicite per calculationes super le base del equationes e coefficientes supra-presentate.

Es concludite que in humanos (a) glycerido de acido oleic in le dieta es essentialmente le equivalente, quanto al efecto super le cholesterol del sero, de carbohadrato con le mesme valor caloric, (b) le glyceridos de saturate acidos grasse in ordinari grassias alimentari augmenta le nivello de cholesterol in le sero, illos de poly-nonsaturate acidos grasse (principalmente acido linoleic) reduce lo, e in iste respecto levemente plus que 2 g de acido linoleic es requirite pro contrariar le efecto de 1 g de saturate acido grasse como per exemplo acido stearic o palmitic, (c) intrinsec hypercholesterolemia relative es generalmente associate con hyper-responsivitate relative a alterationes de grassia dietari, e (d) le relationes hie discutite permitte satis accurate predictiones del responsas medie del nivello de cholesterol del sero in gruppus de individuos como efecto de specific alterationes del grassia dietari.

References


Medical Eponyms

By Robert W. Buck, M.D.

Aschoff Bodies. These structures were first described by Ludwig Aschoff (1866-1936), then Professor of Pathology at Freiburg, in an article entitled “The Problem of Myocarditis” (“Zur Myocarditisfrage”) which may be found in the Verhandlungen der Deutschen Pathologischen Gesellschaft, Achte Tagung, Jahrgang 1904, Heft 2, Jena, 1905, pp. 46-53.

“We succeeded in finding peculiar nodules which seemed to be specific for rheumatic myocarditis. These nodules were clearly marked, it is true, in only two cases of recurrent endocarditis, but corresponded in their location exactly to the cellular proliferation found in other cases. They usually lay in the neighborhood of small or medium sized blood vessels and often showed the most intimate relation to the adventitia of these vessels, or there was found simultaneous involvement of all layers of the blood vessels, such as has been described in arteritis nodosa. The nodules are extremely small, at most submiliary in size, and arise through the collection of notably larger elements with one or more abnormally large, slightly notched, or polymorphic nuclei. The aggregation of cells often occurs in the form of a fan or rosette. The periphery is formed by the large nuclei; the center by the confluent protoplasm of the cells which often seems to stain weakly, or otherwise appears to be a necrotic mass. The fan-shaped foci recall, when superficially observed, the tiny necrotic areas of gout with the peripheral cell mantle as they are frequently found in gouty kidneys.”
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