Diet, Serum Cholesterol and Coronary Artery Disease

By Menard M. Gertler, M.D., Stanley Marion Garn, Ph.D., and Paul Dudley White, M.D.

Diet, particularly the ingestion of cholesterol and fats, has been considered as causally related to the increasing incidence of coronary heart disease and atherosclerosis. This communication presents evidence which is not consonant with this viewpoint. The evidence presented here is based upon dietary and serum cholesterol studies of 97 males who had experienced coronary heart disease prior to the age of 40 and, for purposes of comparison, 146 healthy, nonhospitalized males. The evidence shows clearly that (a) there is virtually no correlation between ingested cholesterol and the level of cholesterol in the serum, and (b) there is virtually no difference, on the average, in the amount of cholesterol ingested by patients of the coronary disease group and the individuals of the control group. A discussion of the "dietary theory" of atherosclerosis is included in this presentation.

The prevalence and increasing incidence of coronary artery disease in the United States in the past few decades has been attributed partially to the allegedly high ingestion of cholesterol in the American diet. This contention is based primarily on the following series of observations. First, it is assumed that the relationship between serum cholesterol levels and coronary artery disease is a well-established causal relationship. Second, it is assumed that the serum cholesterol level depends largely on exogenous sources of cholesterol. Third, it is assumed that the coronary artery disease rate is actually higher in Americans than in certain other peoples such as Chinese and Okinawans.

In addition to these assumptions, certain clinical observations and experimental studies have been employed to support these tenets. The purported increase in incidence of atherosclerosis in diseases such as hypothyroidism, and xanthomatosis, and diabetes, in which the serum cholesterol is elevated, is accepted as evidence of an inescapable causal relationship between elevated serum cholesterol and atherosclerosis. Furthermore, the production of atherosclerosis in herbivorous animals such as rabbits, by feeding cholesterol in large quantities, and in carnivorous animals such as dogs, by the combination of a high cholesterol diet and thiouracil, is cited as further evidence that ingested cholesterol is the bête noire in atherosclerosis. Such evidence is suggestive that ingested cholesterol may be one factor in the production of atherosclerosis, but the evidence does not support the contention that ingested cholesterol is the only factor. In all the clinical or experimental examples named, there are additional factors such as hormonal imbalance or an increased (out of physiologic limits) supply of serum cholesterol from either endogenous or exogenous sources.

The best "support" for the idea that a high-cholesterol diet leads to coronary artery disease in human beings comes from the statements that coronary artery disease is rare in Chinese and other Mongoloids such as Okinawans because such individuals do not ingest cholesterol in great amounts. It is admitted by Public Health authorities that the mortality statistics in the United States could be improved and that individual death rates are not entirely accurate because they are based upon classified causes of death which are sometimes erroneous. Due to the technical lag, it is reasonable to suggest that the hospital records in China are not so well kept as they are in the United States; furthermore, local, territorial and national death records could not possibly be so well kept as in the United States because

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there is only one doctor to 50,000 Chinese in contrast to one doctor to 800 Americans. Even if the raw death rates were known as well in China as they are in the United States, extensive corrections of the statistical data would be required before interpreting them. In a country like China, where public health measures are either primitive or nonexistent, infantile mortality is higher than in the United States and the mean age of death would not be so high as in the American population. Such diseases as tuberculosis, vitamin deficiencies, macrocytic anemias, hookworm infestations, infectious diseases, liver disease, cholera and malaria are responsible for many deaths in the population at younger age levels. Thus, such findings may be interpreted as indicating that the low incidence of atherosclerosis exists in China because the death rate is higher in the younger age levels and fewer Chinese live to the age at which coronary artery disease manifests itself. These facts may be duplicated in the state of Massachusetts. It was found in Chinese in Massachusetts, that 52 died of tuberculosis during the year 1947, while only 7.5 Massachusetts inhabitants were expected to die from this disease; similarly in 1947, 33 Chinese died from diseases of the circulatory system while 66.7 were expected to die from these diseases. Thus, seven times the expected number died from an infectious disease while one-half as many died from cardiovascular diseases as were expected. The difference is significant by the chi square test. Thus, cardiovascular disease is spared the Chinese partially because infectious diseases take a higher toll. It is only when infectious and deficiency diseases are conquered that the bulk of any populace lives to the age when degenerative diseases predominate. Accordingly, even if the corrected coronary disease rate were the same in Chinese and Americans, the absolute frequency of this disorder in native Chinese would be far lower.

Let us also consider the Chinese diet. Is it generally low in ingested cholesterol? Is it generally adequate in vitamins and calorie intake? Snapper states that “the low caloric, protein, fat, mineral and vitamin intakes lead to a generally diminished resistance against disease. ...The low protein intake favors the development of hypoproteinemia.” Naturally, a person on such a diet could have a lower serum cholesterol, for not only is there a paucity of ingested cholesterol, but there is a paucity of those amino acids and fatty acids which are the precursors of endogenous cholesterol.

It is obvious that the relationship between diet and serum cholesterol is still a question of paramount importance. The question of the degree of correlation in man between the amount of ingested cholesterol and the level of serum cholesterol in two groups, one with good health and the other with coronary heart disease, is considered in this paper. This study deals not only with this degree of correlation between the level of serum cholesterol and ingested cholesterol in both groups, but also the weekly intake of individuals in both groups.

**Methods and Materials**

A total of 229 individuals were studied with regard to serum cholesterol and dietary cholesterol (dietary histories were not obtained in 17 individuals). One hundred thirty-nine of these individuals were healthy, active males employed by an eastern industrial plant; the occupational and racial background of this group has been reported in an earlier paper in this series. Ninety individuals (males) were obtained from the coronary disease group which consisted of individuals who had experienced myocardial infarction prior to the age of 40. The two groups were statistically comparable in mean age, as has been shown.

Dietary information was obtained from all of the individuals by the personal questionnaire method, stressing not only the obvious cholesterol-containing foods, but also those containing cholesterol in minute amounts and those containing the amino acid precursors of the cholesterol molecule. The dietary cards were computed to a common denominator such as weekly intake of the various foodstuffs and the amount of cholesterol, myristic acid, uric acid, fat, proteins, carbohydrates and amino acids such as alanine and leucine were computed from a standard table.

Correlations were made by the usual methods between the ingested cholesterol, myristic acid, uric acid, etc., and the level of serum cholesterol. Details will be published elsewhere.

The possible discrepancy between the questionnaire method of study and other methods of determining dietary composition has been considered. Errors in any questionnaire method are bound to be present, but there is evidence that, in this dietary study, (a) systematic errors (tending to make a
correlation lower or higher) were largely absent, and (b) the reported intakes were within reasonable approximations of actual values. Since the butter, egg, and milk content of the diet is one of the richest single sources of dietary cholesterol (65 per cent of the average dietary cholesterol in this study), and since these staples are easily remembered by the person interviewed, it is reasonable to suggest that correlations which may exist are not obscured by this technic.

Serum cholesterol determinations were made by the Chemistry Laboratory of the Massachusetts General Hospital as described in earlier papers.12

RESULTS

The amount of ingested cholesterol for both the control group and the coronary disease group was calculated in the manner described from the dietary histories. The final values and statistical calculations are included in table 1.

TABLE 1.—Calculated Cholesterol Intake: Grams Per Week in the Coronary Disease Group and the Control Group

<table>
<thead>
<tr>
<th></th>
<th>N</th>
<th>Range</th>
<th>S.E.</th>
<th>S.D.</th>
<th>C.V.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Coronary</td>
<td>90</td>
<td>.6-6.6</td>
<td>3.30 ± .15</td>
<td>1.41</td>
<td>42.73</td>
</tr>
<tr>
<td>Controls</td>
<td>129</td>
<td>.6-9.4</td>
<td>3.88 ± .12</td>
<td>1.46</td>
<td>37.63</td>
</tr>
</tbody>
</table>

X = Number
X = Mean
S.E. = Standard Error
S.D. = Standard Deviation
C.V. = Coefficient of Variation
* Three females are not considered and inadequate dietary histories were obtained in 7 males.
† Seven individuals were not available for dietary studies.
‡ Grams per week.

None in the control group was restricted in any way as to dietary intake. However, 9 members of the coronary disease group, either on physicians' advice or voluntary limiting, had placed themselves on a diet for the purpose of restricting fats and cholesterol.

As shown in table 1, the mean cholesterol intake of the coronary group was absolutely and significantly lower than the comparable intake of the control group (the critical ratio of the difference between the means equals 3.07); the difference is statistically highly significant on the .01 level of probability. However, despite a significantly lower cholesterol intake, the serum cholesterol level in the cor-
onary disease group was significantly higher. In percentages, the coronary group ingested 12 per cent less cholesterol, yet their serum lipids contained approximately 16 per cent more cholesterol.

A diet containing a caloric excess may add weight to the individual in the form of fatty tissue. While such a diet may contain more cholesterol than the normal diet, it is unlikely because the excess calories usually are the result of excess carbohydrate intake which, to all intents and purposes, does not contain cholesterol. The coronary disease group ingested 52 Gm. of protein and 66 Gm. of fat per day in contrast to the control group, which ingested 61 Gm. of protein and 90 Gm. of fat per day. Carbohydrates were not assayed. Since the control group weighed 7 pounds more on the average than the coronary disease group, one wonders whether the total caloric intake was excessive in the coronary disease group as compared with the control group. Accordingly, reasons other than dietary alone must be considered to explain the higher levels of serum cholesterol in the coronary disease group, despite the smaller amount of ingested cholesterol.

It was of importance not only to determine the amount of ingested cholesterol in each group, but also to determine for both groups the relationship between the amount of ingested cholesterol and the amount of cholesterol in the serum. This was accomplished by determining the coefficients of correlation between exogenous cholesterol and serum cholesterol. A coefficient of correlation of ± 0.5 is considered moderate, ± 0.2 is considered low and correlations below ± 0.1 are usually not significant. The values of the coefficient of correlation are summarized in table 2.

In neither group is it possible to detect any significant correlation between the two variables; therefore, ingested cholesterol and serum cholesterol do not seem to be related. One question has been raised concerning the meaning of the insignificant but interesting negative correlation between cholesterol and serum cholesterol in the coronary artery disease group. This may be partially attributable to the fact that the diets which had been limited were in those individuals whose serum cholesterol was
highest. Accordingly, those patients who had higher levels of cholesterol in the serum were eating less cholesterol because of their doctors' advice; but it is clear that the advice did not affect the final level of the serum cholesterol.

It is important to know whether the product moment correlation as used might mask important associations at the ends of the scattergram. Accordingly, those individuals with low cholesterol levels (below the mean for normal people) and those with conspicuously high cholesterol levels in both groups have been removed and their dietary intake studied separately. As shown in the tables 3–6, there is no statistically significant difference between the amount of ingested cholesterol in each group for individuals with low or with high serum cholesterol. In other words, selecting on the basis of serum cholesterol, no difference in ingested cholesterol was found. It is clear, then that low serum cholesterol cannot be attributed to diet scarce in cholesterol in either group; nor can high serum cholesterol levels be attributed in diets replete with cholesterol.

In the individuals with lowest serum cholesterol, there is a significantly higher value of serum cholesterol in the coronary disease group

![Scattergrams](image)

**Fig. 1.** Scattergrams with regression lines showing the insignificant correlation between ingested cholesterol and the level of serum cholesterol in both the coronary disease group and the control group.

### Table 2.—Values of Coefficients of Correlation between Ingested Cholesterol and Serum Cholesterol in the Coronary Disease Group and the Control Group.

<table>
<thead>
<tr>
<th></th>
<th>Coefficient of Correlation</th>
<th>Standard error</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control Group</td>
<td>+0.05</td>
<td>±0.11</td>
<td>none</td>
</tr>
<tr>
<td>Coronary Disease Group</td>
<td>−0.09</td>
<td>±0.08</td>
<td>none</td>
</tr>
</tbody>
</table>

(critical ratio of the difference between the means equals 6.55), but there is no significant difference (critical ratio of the difference between the means equals 0.93) in the amount of ingested or exogenous cholesterol between the two groups.

While the serum cholesterol in the coronary disease group is significantly higher (critical
TABLE 3.—Ingested Cholesterol of 10 Individuals in Each Group with the Lowest Serum Cholesterol

<table>
<thead>
<tr>
<th>Coronary Disease Group</th>
<th>Control Group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serum Cholesterol</td>
<td>Ingested Cholesterol</td>
</tr>
<tr>
<td>167*</td>
<td>2.0†</td>
</tr>
<tr>
<td>171</td>
<td>2.2</td>
</tr>
<tr>
<td>194</td>
<td>5.1</td>
</tr>
<tr>
<td>194</td>
<td>1.7</td>
</tr>
<tr>
<td>200</td>
<td>2.1</td>
</tr>
<tr>
<td>202</td>
<td>5.3</td>
</tr>
<tr>
<td>208</td>
<td>1.3</td>
</tr>
<tr>
<td>208</td>
<td>3.6</td>
</tr>
<tr>
<td>208</td>
<td>4.6</td>
</tr>
<tr>
<td>208</td>
<td>4.9</td>
</tr>
</tbody>
</table>

* Mg. per cent.
† Grams per week.

The critical ratio of the difference between the amount of ingested cholesterol equals 0.93.

TABLE 4.—Ingested Cholesterol of 10 Individuals in Each Group with the Highest Serum Cholesterol

<table>
<thead>
<tr>
<th>Coronary Disease Group</th>
<th>Control Group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serum Cholesterol</td>
<td>Ingested Cholesterol</td>
</tr>
<tr>
<td>376*</td>
<td>4.3†</td>
</tr>
<tr>
<td>379</td>
<td>5.2</td>
</tr>
<tr>
<td>382</td>
<td>5.7</td>
</tr>
<tr>
<td>388</td>
<td>6.2</td>
</tr>
<tr>
<td>390</td>
<td>2.8</td>
</tr>
<tr>
<td>400</td>
<td>4.5</td>
</tr>
<tr>
<td>410</td>
<td>1.7</td>
</tr>
<tr>
<td>480</td>
<td>—</td>
</tr>
<tr>
<td>490</td>
<td>3.0</td>
</tr>
<tr>
<td>490</td>
<td>—</td>
</tr>
</tbody>
</table>

* Grams per week.
† Mg. per cent.

ratio of the difference between the means equals 6.031), there is no significant demonstrable difference (critical ratio of the difference between the means equals 1.23) in the amount of ingested or exogenous cholesterol in the individuals with highest serum cholesterol.

In spite of statistically higher ingestion of cholesterol (the critical ratio of the difference between the means of ingested cholesterol equals 3.85) the serum cholesterol in the control group is significantly lower (the critical ratio between the values of serum cholesterol equals 3.02) than the serum cholesterol in the coronary group, as shown in table 5.

The cholesterol ingestion remained virtually the same for both groups, (the critical ratio of the difference between the means of ingested cholesterol equals 0.19) but the serum cholesterol was significantly higher (the critical ratio of the difference between the means of serum cholesterol equals 2.33) in the group with coronary artery disease, as shown in table 6.

Thus, (a) by use of the product moment correlation and (b) by combining the ten maximum and ten minimum independent variables
of serum cholesterol and ingested cholesterol in both groups and comparing the values, it is apparent that the exogenous cholesterol in the amounts observed in this study does not influence the level of serum cholesterol in either the control group or the coronary artery disease group.

Table 6.—Serum Cholesterol of 10 Individuals in Each Group with the Lowest Ingested Cholesterol

<table>
<thead>
<tr>
<th>Coronary Disease Group</th>
<th>Control Group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ingested Cholesterol</td>
<td>Serum Cholesterol</td>
</tr>
<tr>
<td>.6*</td>
<td>252†</td>
</tr>
<tr>
<td>1.0</td>
<td>316</td>
</tr>
<tr>
<td>1.3</td>
<td>208</td>
</tr>
<tr>
<td>1.3</td>
<td>284</td>
</tr>
<tr>
<td>1.4</td>
<td>286</td>
</tr>
<tr>
<td>1.5</td>
<td>287</td>
</tr>
<tr>
<td>1.5</td>
<td>347</td>
</tr>
<tr>
<td>1.6</td>
<td>290</td>
</tr>
<tr>
<td>1.6</td>
<td>218</td>
</tr>
<tr>
<td>N</td>
<td>10</td>
</tr>
<tr>
<td>R</td>
<td>.6–1.6</td>
</tr>
<tr>
<td>X</td>
<td>1.34</td>
</tr>
<tr>
<td>S.E.</td>
<td>.10</td>
</tr>
<tr>
<td>S.D.</td>
<td>.32</td>
</tr>
<tr>
<td>C.V.</td>
<td>23.88</td>
</tr>
</tbody>
</table>

* Grams per week.
† Mg. per cent.

**Discussion**

The coefficients of correlation in this study between ingested cholesterol and serum cholesterol for the coronary disease group and the control group are $-0.09 \pm 0.08$ and $+0.05 \pm 0.11$ respectively. These values suggest a startling independence of ingested cholesterol and serum cholesterol in the two groups studied. The present study does not preclude the remote possibility that a significant association may exist in some individuals, but indicates clearly that it is not prevalent on a group basis of either normal individuals or individuals with coronary artery disease. In previous papers in this series and in other citations from the literature, it is evident that the level of cholesterol in the serum is influenced by age, morphological, idiosyncratic or genetic, hormonal and perhaps, dietary factors. The dietary factor contributes very little to the level of cholesterol in the serum if the daily ingestion of cholesterol is within the normal range of the individual. The difference in the levels of cholesterol in the sera of both groups cannot be attributed to age differences for the average age of both groups is only two years different. The difference in morphologic makeup of the two groups accounts for only part of the difference in the level of serum cholesterol. The coronary disease group is predominantly mesomorphic at the expense of ectomorphy. However, the mean serum cholesterol for the matched control group was 17 mg. per cent greater than that of the control group, but 45 mg. per cent less than the mean serum cholesterol of the coronary disease group. The importance of this difference is emphasized when it is recalled that the matched control group is of the same ethnic origin and body build as the coronary disease group.

The most important hormone influencing serum cholesterol appears to be thyroid. The presence of hypothyroidism and hyperthyroidism produces hypocholesterolemia or hypercholesterolemia respectively. While the basal metabolic rate is on the average, minus 13 in the coronary artery group, studies with radioactive iodine did not reveal any degree of hypothyroidism. Thus, to all intents and purposes, the elevation of the serum cholesterol in the coronary group is not due to a depressed thyroid function. Furthermore, the degree of hypercholesterolemia and the value of the cholesterol:lecithin ratio do not favor this etiology.

If dietary, age, morphologic and hormonal factors are ruled out, how then is it possible to account for the elevated serum cholesterol in coronary artery disease? Individuals with coronary artery disease do not appear predominantly in families with hypercholesterolemia in spite of the fact that their serum cholesterol is elevated. Familial coronary artery disease and familial hypercholesterolemia may co-exist but there is no evidence to demonstrate a causal relationship. Thus, the only remaining explanation for the elevation of serum cholesterol in the coronary artery disease group appears to
be the endogenous one, assuming that utilization remains constant.

Studies which employed isotope and deuterium labelled substances, such as alanine, leucine, sodium acetate and myristic acid, indicate that these substances are incorporated into the steroid nucleus of cholesterol.\textsuperscript{24,25} About 45 per cent of the carbon atoms in cholesterol come from the acetate radical.\textsuperscript{26} In mice there is a positive cholesterol balance even when the animals are on a virtually noncholesterol diet, indicating that cholesterol is being synthesized within the organism.\textsuperscript{27} Furthermore, if cholesterol were derived only from the exogenous or the ingested form, then how could we explain its existence in the most diverse phyla of the vertebrates and in most invertebrates except sponges and mollusks?\textsuperscript{28} Considering all these factors, one is lead to conclude as have others, that cholesterol is synthesized by endogenous means in the herbivora and obtained by endogenous means and (when available) exogenous means in the carnivora. The final level of serum cholesterol is the result of the interdependency of utilization, synthesis and availability of the substance for metabolic needs.\textsuperscript{29}

Thus far, it has been shown that the level of serum cholesterol is independent of dietary cholesterol within normal limits of ingestion and that the serum cholesterol is mainly dependent upon the balance between synthesis and utilization. Accordingly, if dietary cholesterol contributes little if anything to the value of serum cholesterol, of what value is a diet restricting cholesterol in the treatment of coronary artery disease, especially those cases of coronary artery disease in which the value of serum cholesterol is below that of the mean value of serum cholesterol in the normal group? In experiments conducted in this laboratory, no remarkable or significant variation was found in the serum cholesterol following the ingestion of eight eggs in the form of scrambled eggs, two 8 oz. glasses of milk and one oz. of cream with one oz. of butter; (cholesterol intake equals 5.0 Gm.). These results are confirmed by Steiner who fed 10 Gm. of cholesterol crystal to a human subject without any significant rise in the level of serum cholesterol.\textsuperscript{30}

Similar results were observed by other authors.\textsuperscript{31,32} Steiner\textsuperscript{33} and Okey\textsuperscript{34} have purposefully fed individuals diets which were inordinately high in cholesterol as compared with the normal diet. Doubtless the serum cholesterol would be elevated temporarily if such high amounts of cholesterol (5 Gm. daily) were fed over prolonged periods of time. But, does this prove that the incidence of atherosclerosis or coronary artery disease was higher in these patients or that an average ingestion of cholesterol over prolonged periods will augment the level of serum cholesterol? Finally, does this prove that elevated serum cholesterol is causally related to atherosclerosis? The ingestion of abnormally high amounts of cholesterol occurs infrequently in human diets and the facts as presented are acceptable and applicable only to those individuals who live on diets replete with eggs, milk, calves' brains and oysters! Furthermore, there is evidence that indicates that such individuals may have a low level of serum cholesterol.

It cannot be denied that the level of serum cholesterol decreases on a low cholesterol diet. However, in the study of 6 individuals who were followed for at least 9 to 18 months on a low-cholesterol diet, the serum cholesterol dropped 25 to 30 per cent within a period of three weeks and remained at that level for approximately two months. Following this stationary level, the serum cholesterol began to rise slowly and within six to nine months reached its predietary level. This is probably explained by the gradual replacement of the dietary cholesterol by the endogenous synthetic cholesterol mechanisms which lay dormant during the normal period of cholesterol ingestion, because there was no need for this mechanism to function.

The purported low incidence of coronary artery disease or atherosclerosis in Okinawans is held to be causally related to their diets which are supposed to contain little or no cholesterol. Assuming that the Okinawan diet is deficient in cholesterol, (the diet cited was a wartime diet) there is considerable evidence to indicate that it is replete with proteins and carbohydrates.\textsuperscript{35} There is also evidence to show that humans on a high protein diet maintain an elevated level of serum cholesterol which dis-
appears when the diet is returned to normal. It is not possible, (no actual values are given) therefore, that the level of serum cholesterol in Okinawans would not be lower than in Americans? Furthermore, there is no evidence which indicates that (a) the dietary cholesterol is deposited in the atherosclerotic plaque and (b) the level of serum cholesterol is causally related to atherosclerosis. Finally, is atherosclerosis necessarily coronary artery atherosclerosis?

The statement that there is a low incidence of atherosclerosis in Okinawa is open to considerable question and interpretation. In reviewing Steiner’s material on autopsies performed on 150 Okinawans (51 males and 99 females) from 1 year to 100 years of age, it was evident that 7 (6 over 66 and one 91 years of age) individuals had visible aortic atherosclerosis. Thus, 6 males out of 51 examined, or 12 per cent, showed atherosclerosis. Does this incidence of atherosclerosis differ from autopsy material in any American hospital on males of a similar age group? The reports from Okinawan physicians state that (1) infectious diseases such as tuberculosis and pneumonia take a high death toll among these peoples and (2) that degenerative cardiovascular disease is rare. These two statements cannot be taken as separate facts, for, as has been shown with the discussion on the Chinese, where there is an increased loss from infectious diseases at an early age, there is bound to be a paucity of degenerative diseases in the older decades.

A defense of the dietary causation of atherosclerosis is maintained by those who quote the evidence that (a) atherosclerosis was low in Germany immediately following World War I (b) atherosclerosis is more common in obese, overnourished persons than in lean and undernourished (c) lower incidence in chronic alcoholics than in nonalcoholics. The common denominator in all these three factors appears to be a low fat intake due either to restriction or expediency. Moreton has claimed that following a fatty meal there exists a state of hyperchylomiconemia and the larger particles are absorbed by the intima and built up into an atherosclerotic plaque. There are several objections to this theory which are summarized best by Leary. It should be stressed at this point that a high fat diet is not synonymous with a high cholesterol diet and the two terms should not be used loosely or be confused.

From the evidence submitted it is believed that not only is a low cholesterol diet of questionable value in the treatment of coronary artery disease, but there is complete independency of the level of serum cholesterol and the amount of cholesterol ingested within the normal dietary variations.

SUMMARY AND CONCLUSIONS

1. Dietary histories with emphasis on cholesterol and purines were obtained from 139 healthy males and 90 males who had experienced myocardial infarction prior to the age of 40 years.

2. The average amount of cholesterol ingested per week in the healthy and in the coronary disease group was 3.88±1.46 Gm. and 3.30±1.41 Gm. respectively.

3. The coefficients of correlation between serum cholesterol and ingested cholesterol were +0.05±.11 and −0.09±.08 for the control group and coronary disease group respectively. This indicates that for the two groups in question and the amounts of cholesterol in consideration, there is no definite relation between the amount of cholesterol ingested and the level of cholesterol in the serum.

4. The question is raised as to the wisdom of removing cholesterol from the diet of individuals with coronary artery disease. From the evidence gathered from this study and other dietary studies in this laboratory, it is believed that there is no advantage to be gained from imposing a low cholesterol diet on patients with coronary artery disease.

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