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Diet and the Incidence of Heart Disease

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I. DIET AND THE INCIDENCE OF HEART DISEASE

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The facts to be discussed here concern three sets of items: 1) The first is the broad category of heart disease, or diseases, diagnosed by the clinician as angina pectoris, coronary heart disease, myocardial infarction, chronic myocarditis, and myocardial degeneration. In hospital and vital statistics it is rarely possible to differentiate these clearly so it is convenient to group them, for the present purpose, as "degenerative heart disease." Moreover, there is more than a suspicion that they all, in fact, share some common factors in basic etiology. 2) The second set of items concerns serum cholesterol and allied substances which are currently considered to be importantly related to the development of some, at least, of these conditions in man. The relationship is presumably through the atherosclerotic process but this assumption is not central for this argument. 3) Finally, there is the character of the habitual diet, particularly in regard to its content of cholesterol and of total fats.

From the facts to be presented here it will appear that the relationships between these three sets of items are of major public health importance. Perhaps they are also significant for the practice of clinical medicine but that is a somewhat different question.

For many decades the official as well as the general view of the subject of "public health" has been that it should be concentrated on a few major questions obviously requiring organized attention beyond the scope of the individual practice of medicine -- public sanitation, control of epidemics and infective diseases, record-keeping of mortality and community health status, and the correction of health hazards where many people congregate, as in schools and factories. These limited horizons are now being extended. There is general public insistence that efforts on a broad front should be made to prevent or decrease the incidence of all forms of illness and disability, not merely those that are infective or occupational in origin. But, obviously, increased knowledge, which can only come from research, is basic to such efforts. This means dependence on research workers and facilities whose contribution is to the common good and whose financial support must come, somehow, from the community.

On financial grounds, if for no other reason, public health activities, including research, cannot be expanded indefinitely. But the need for major public health attention is clear whenever two conditions exist. First, when there are large numbers of the population suffering disability and death from diseases against which private medical practice is making little headway. And, second, when there is any reason to hope that the incidence of these diseases may be altered by measures applicable to the general population, even if these measures are not yet known.

Degenerative heart disease fulfills these conditions. Great numbers of persons, and by no means merely the aged, are affected. And everyone must admit that the present practice and progress of diagnostic and therapeutic medicine is not solving the problem; the proof is only too clear in our vital statistics.

The mere existence of an undesirable state of affairs is not, in itself, enough reason to demand a major effort to correct it. There must also be some reason to believe that improvement is possible. In the past, a defeatist attitude about heart disease, particularly degenerative heart disease, has been a major hindrance to effort and even to careful consideration. But as will be shown, it is now abundantly clear that degenerative heart disease is not an inevitable consequence of aging, beginning in youth and progressing with the years, indifferent alike to medical efforts and the mode of life. Individual differences in the age of onset and the rate of progress of cardiovascular degeneration - or aging - might be ascribed to genetic

factors but this does not explain the differences between whole populations of the same or similar race and genetic make up.

Vital Statistics

Vital statistics are still far from perfect but there has been great improvement in recent years in the completeness and the accuracy of records of populations and deaths in many countries, particularly those of the Western World where genetic differences are at a minimum. Table 1 compares the total death rates, for given age and sex, of adults in 16 countries with the United States for the period 1947-1949. In all of these countries the enumeration of total population and total deaths is reliable. Together these countries, with a total population about 50 per cent greater than that of the United States, recapitulate the climate and, with the exception of the negroes, the racial background of our country.

With the exception of Portugal, and for women in South Africa and to a lesser extent women in a few other countries, it is clear that adults in these countries enjoy a considerably lower total mortality than in the United States. From age 20 to 40 years, the differences are smaller but the death rates at those ages are far lower

TABLE 1

Death rates, from all causes, in 16 countries with a total population of about 220 millions. All values are for the period 1947-1949 and are expressed as percentages of the rates in the United States in 1949.

Age	40-44		50-54		60-64	
Sex	M	F	M	F	M	F
Australia	75	91	87	96	94	94
Belgium	96	89	91	96	97	101
Canada	78	91	76	92	84	96
Denmark	59	83	63	88	70	100
England & Wales	68	78	76	83	93	88
France	96	100	91	91	93	91
Ireland	80	78	57	86	69	88
Italy	91	100	77	88	75	97
Netherlands	52	69	56	76	63	89
New Zealand	55	72	66	81	85	88
Norway	64	78	53	65	54	68
Portugal	139	125	99	96	99	103
Scotland	93	97	93	100	97	107
South Africa	93	108	102	115	94	104
Sweden	61	86	63	85	68	92
Switzerland	78	97	78	97	88	108
Mean	79.9	90.1	76.8	89.7	82.7	94.6

in all countries than at older ages. After age 70 the picture may be different but the statistics are less reliable and the present analysis is not concerned with mortality in the truly aged population. Roughly, these and other data show that over the age span of 20 to 70 years men in the United States have an excess mortality of about 20 per cent compared with the average of other comparable countries. For women our excess mortality is between 5 and 10 per cent.

These facts are surprising, perhaps. In the United States all official propaganda has long stressed our steady improvement in the so-called "expectation of life," but this is primarily a reflection of the infant death rate. In total expectation of life the United States is about midway in the countries listed in Table 1 which means that we are relatively healthy as infants and children but unhealthy as adults.

But, anyway, Portugal, a land of extreme poverty, has a worse record for adults. Closer analysis shows that Portugal's inferiority is largely the result of an appalling death rate from tuberculosis. Table 2 shows that our superiority over Portugal disappears when infective diseases and violence are removed as causes of death. Moreover, Table 2 shows a great excess of deaths in the United States from circulatory diseases and it is in this category of causes of death that we obviously have our biggest health problem.

TABLE 2

Death rates, per 1000, among men of three age groups in Portugal (1950) and in the United States (1949). Lines 1 and 2 give the rates from all causes of death except infective and parasitic diseases and violence. Lines 3 and 4 give the death rates from all diseases of the circulatory system (Category VII in the International Long List or items 24 and 25 in the Abridged List).

Ages	40-44	50-54	60-64
1. Portugal	4.2	9.0	23.3
2. U. S. A.	3.8	11.0	26.0
3. Portugal	0.9	1.8	5.2
4. U. S. A.	1.8	5.9	13.9

Comparison of the United States and Italy in regard to adult death rates is particularly illuminating as shown in Table 3. The recent vital statistics of Italy are very reliable and, at least for broad categories of causes of death, comparisons between the United States and Italy are valid. In the United States the death rate from violence is higher than in Italy but tuberculosis and other infective diseases are much

TABLE 3

Death rates in Italy (1948) as percentages of corresponding rates in the U.S.A. (1949) for men (M) and women (F) in three age groups for: 1) all causes, 2) all causes less infective and parasitic diseases and violence, 3) diseases of the circulatory system (Category VII in the International list), 4) degenerative heart disease (1938 Revised List nos. 93c, 93d, 94; 1948 Revised List nos. 420, 422).

	40-44		50-54		60-64	
	M	F	M	F	M	F
1. All causes	91	100	77	88	75	97
2. Excl. infective, violence	79	87	70	83	73	95
3. Circulatory system	35	79	31	59	28	67
4. Degenerative heart	20	56	23	48	25	56

more prominent in Italy. If both these categories of preventable causes of death are eliminated, the inferiority of the United States to Italy is much more marked. The reason for this inferiority is clearly because there is relatively a great excess of deaths from cardiovascular disease in the United States. And the biggest difference is in the "degenerative" types of cardiovascular disease - coronary disease, myocardial degeneration, etc.

It will be noted in Table 3, also, that there is a major difference between men and women when deaths from heart disease in Italy and the United States are compared. This is in conformity with the known facts of the situation in the United States where the death rate from degenerative heart disease before old age is 2 to 3 times as high among men as among women. That male excess is almost wholly absent in Italy.

One of the major results of considering these three tables, and many more data which could be cited, is the conviction that the present death rate from degenerative heart disease in the United States is not merely an inevitable consequence of aging. Hence two conclusions follow. First, much lower death rates should be attainable in the United States; and, second, research efforts should be directed towards discovering the characteristics of these foreign populations, and their mode of life, which may be related to their relative immunity from our own health problem with these diseases.

Characteristic Tendencies in Degenerative Heart Disease

What are the characteristics of persons who have, or are likely to have, degenerative heart disease before old age? A major part of the work of the Laboratory of Physiological Hygiene is devoted to the effort to provide some information on this question but a detailed answer is still in the distant future. However, some facts are clear.
1) It is a fact that, compared with healthy persons of the same age, patients with definite angina pectoris or who have survived a myocardial infarction tend to have blood serum characterized by high cholesterol and certain lipoprotein concentrations, a high cholesterol-phospholipid ratio, and a larger proportion of the total cholesterol in the beta lipoprotein fraction.
2) It is a fact that, on the average, persons afflicted with diabetes, myxedema and nephrosis tend to have high cholesterol and the other serum peculiarities mentioned above. Among these patients there is a high incidence of atherosclerosis and degenerative heart disease.
3) It is a fact that a major characteristic of the atherosclerotic artery is the presence of abnormal amounts of cholesterol in that artery. The atherosclerotic plaque consists of 40 to 70 per cent cholesterol. It is extremely probable that most or all of this cholesterol is derived from the blood. The cholesterol in the blood does not exist as a simple solution of free and ester cholesterol; it is carried in lipo-protein complexes.

All of this indicates that measurement of cholesterol and allied substances in the blood serum affords an indication of the tendency towards the development of atherosclerosis and degenerative heart disease. But two questions arise at once. How reliable is the indication derived from such measurements? And is one or the other of the various measurements much superior to the others for the recognition or prediction of degenerative heart disease in man?

In one sense the reliability is high. Whenever groups of individuals are compared, all of these measurements invariably seem to show statistically significant correlations between the measurements and the presence or absence of the tendency towards degenerative heart disease. Analysis of the data shows, however, that these correlations are not high enough to provide useful diagnostic or prognostic tools for dealing with individuals and that there is not much difference between the results from serum total cholesterol measurements or from ultracentrifugal analysis. Comparisons in this regard of total cholesterol and

the "S_f 10-20" fraction from the ultracentrifuge have been published (Keys, 1951). More recent data on the "giant molecule" concentration as a means of differentiating coronary patients from clinically healthy persons (Jones et al, 1951) do not change the picture very much. Table 4 shows, in summary form, the situation at present.

Table 4 shows that prediction from the ultracentrifuge is only about 20 per cent better than random guess work and that the results with the ultracentrifuge at Berkeley are almost identical to the results at Boston with serum total cholesterol. But both methods yield highly significant differentiations between groups. Hence there is every

TABLE 4

Discrimination between healthy men and patients with coronary disease (infarcts) by ultracentrifugal and total cholesterol measurements. "r"= biserial correlation coefficient; "Index" = index of forecasting efficiency; relative value (Rel. Val.) indicates the chance of assigning a correct diagnosis to a man with coronary disease in a population in which it is known that 10 per cent of the men have coronary disease. Data in lines 2 and 7 from Keys (1951) after Gertler et al. (1950) and Gofman et al. (1950); lines 3 - 6 from Jones et al. (1951).

Item	r	Index	Rel. Val.
1. No test or measurement	0	0	0.10
2. S_f 10-20, 31-50 yrs.	0.41	9	0.18
3. S_f 12-20, 41-50 yrs.	0.62	21	0.29
4. S_f 12-20, 51-60 yrs.	0.61	21	0.29
5. Cholesterol, 41-50 yrs.	0.46	12	0.21
6. Cholesterol, 51-60 yrs.	0.30	5	0.15
7. Cholesterol, 31-50 yrs.	0.61	21	0.29
8. Ideal test	1.00	100	1.00

reason to devote great effort to discovering what are the levels of serum cholesterol in different groups of men and what may affect or control that level. At present such researches may afford the best clues as to why population groups differ in regard to susceptibility to coronary disease and related conditions.

Serum Cholesterol Concentration

The serum cholesterol level is the resultant of several factors. All animals, including man, synthesize cholesterol, mainly in the liver, and eliminate it in the bile and by chemical degradation. If there is cholesterol in the food it may be absorbed and so enter the balance picture. But when exogenous cholesterol is supplied the synthesis in the liver is suppressed (Schoenheimer and Breusche, 1933; Gould, 1951; Frantz et al, 1952). Some animals, such as the rabbit and the chicken, have little or no dietary experience with cholesterol after weaning or hatching and have little capacity to destroy or otherwise eliminate it when they are fed large amounts. Others, like the rat, the dog and man, have much greater ability to handle cholesterol and are relatively unaffected by dietary administration.

The attempt to extrapolate to man the findings from cholesterol experiments with rabbits and chickens can lead to absurdities. The typical rabbit experi-

ment involves a diet containing 2 per cent added cholesterol, or some 5 mg. of cholesterol per Calorie. This means 15 grams of cholesterol in a human diet of 3000 Calories, an amount about 12 times higher than it is possible to attain in any habitual diet of ordinary human foods and 20 times what is considered a high-cholesterol diet for man (Keys, 1952). And it must be emphasized again that man is very different from the rabbit and the chicken in his metabolism of cholesterol.

In very many experiments in the Laboratory of Physiological Hygiene we have consistently found that ingestion of 10 gram of cholesterol in a meal suitable to promote absorption leads to only very trifling and transient increase of cholesterol in the blood serum. In 93 men studied the average maximum rise so produced was less than 4 mg. of cholesterol per 100 ml. of serum. But the results of continued daily administration of large amounts of cholesterol in the diet are perhaps even more significant.

Table 5 summarizes the results of experiments on man and on various animals. That man, and probably the monkey also, is very different from rabbits, chicks and even rats is evident. The dog, also, is very independent of the dietary cholesterol intake unless the thyroid gland is severely injured and

TABLE 5

Mean serum cholesterol responses to cholesterol feeding in different species. The dietary cholesterol values are rough approximations to enable them all to be expressed on the same scale of mg. of cholesterol per dietary Calorie. References (referring to line numbers) are: 1, 10, 11 - Cook and Thomson, 1951; 2, 3 - Kesten and Silbowitz, 1943; 4, 5 - Rodbard, Bolene and Katz, 1951; 6 - Stamler and Katz, 1950; 7, 8 - Horlick and Havel, 1948; 9 - Sperry and Stoyanoff, 1935; 12 - Greenberg and Rinehart, 1951; 13 - Moses, 1952; 14 - Steiner, 1949; 15, 16, 17 - Messinger, Porowsawska and Steele, 1950; 18 - Okey and Stewart, 1933.

	Species	Diet	Duration	Mg. per 100 ml.		%
		mg./Cal.	Weeks	Control	Final	Change
1.	Rabbit	5.0	8	149	1720	+1050
2.	"	0.6	16	(145)*	430*	+ 200
3.	"	1.5	5-8	141 *	959*	+ 580
4.	Chick	0.8	25	(100)	130	+ 30
5.	"	6.0	15	(100)	550	+ 450
6.	"	0.8	5-15	116	169	+ 46
7.	Rat	15.0	22	(59)*	113*	+ 108
8.	"	30.0	22	(54)*	129*	+ 139
9.	"	3.0	?	59	163	+ 176
10.	"	5.0	4	70	222	+ 217
11.	Guinea Pig	5.0	5	96	271	+ 182
12.	Monkey	3.0	29	175	213	+ 22
13.	Man	1.0	16	222	223	0
14.	"	1.5	6	-	-	"slight"
15.	"	1.3**	7	235	272	+ 16
16.	"	1.3**	6	253	287	+ 13
17.	"	12.0	9+	238	276	+ 16
18.	"	1.4	4	154	167	+ 8

* Whole blood.

** Cholesterol in dried egg yolk, powder fed. This powder also provided about 20 gm. of lecithin.

even then the dietary level to produce real hypercholesterolemia and atherosclerosis is colossal. It is also significant that man on an ordinary diet, with or without cholesterol in the food, has a higher serum cholesterol concentration than the other animals studied on their normal diets; these other animals include the cat, cow, dog, guinea pig, horse, mouse, rat, chicken and rabbit.

In the United States different persons have greatly different habitual dietary intakes of cholesterol. We have observed in dietary surveys individuals who rarely consume as much as 100 mg. of cholesterol daily while others average as much as 1000 mg. daily. Some years ago we reported that in such surveys of hundreds of men in the Twin Cities we could find no relationship between the habitual cholesterol intake and the concentration in the blood serum (Keys, 1949). Other investigators have had the same experience (Gertler, Garn and White, 1950; Wilkinson, Beecha and Reimer, 1950). Since then we have made three more such surveys with similar negative results.

Even more significant, perhaps, is the fact that men who made large changes in the cholesterol intake, without otherwise changing the diet significantly, did not show any effect in the serum cholesterol concentration. Some of the findings in these extensive researches are summarized in Table 6.

However, there is no doubt that the diet, apart from its cholesterol content, can have a profound effect on the serum cholesterol level. The rice-fruit diet is particularly effective in this regard (Kempner, 1948; Schwartz and Merlin, 1948; Chapman, Gibbons and Henschel, 1950). In many patients and experimental subjects we have consistently found the rice-fruit diet causes a decrease in serum cholesterol of 20 to 40 per cent in one month.

TABLE 6

Means and standard errors for serum total cholesterol, in mg. per 100 ml., in clinically healthy middle-aged men, matched as to age, with habitually different cholesterol intakes. The dietary intakes are indicated by the percentile ranks of the men in the total sample of 300 men of the upper economic bracket in the Twin Cities.

	First Year		Second Year*	
No. Men	Intake Percentile	Serum Conc.	Intake Percentile	Serum Conc.
35	90	244. 7 ± 5. 9	90	246. 0 ± 6. 7
28	10	240. 6 ± 8. 8	10	244. 0 ±10. 3
41	60	250. 1 ± 7. 1	25	250. 3 ± 6. 7
23	45	259. 7 ± 8. 3	70	252. 5 ±10. 2

* Corrected for increased age from data of Keys et al (1950)

For the past three years Dr. J. T. Anderson and I have been conducting completely controlled dietary experiments at the Hastings State Hospital. A major purpose of these experiments was to find what is the characteristic of the rice-fruit diet that causes the serum cholesterol concentration to fall. Briefly, it was abundantly shown that the rice, the fruit, the low salt, the low protein, the zero cholesterol content of the rice-fruit diet are unimportant in this regard. The controlling factor is the

total fat content of the diet. The major results of one experiment of six months on 21 men are summarized in Table 7. In these experiments there appeared to be no significant difference between the effects of animal fats and of vegetable fats.

TABLE 7

Mean changes in serum total cholesterol concentration, in mg. per 100 ml., in 21 men after subsistence for 4 weeks on controlled diets. (Detailed data to be published by A. Keys and J. T. Anderson).

Chol.	Fat, gm. daily			All
mg./day	15	72	114	Expts.
0	-64	-27	0	-46
600	-61	-21	0	-41
All Expts.	-63	-24	0	---

From these results, however, it was still not possible to be sure what would be the effect of very prolonged, even life-long, subsistence on different total fat intakes. Evidence on this point could not be secured in the United States because, in general, all habitual diets in this country are relatively high in total fats. The subjects we studied at the Hastings State Hospital, like other people in this country, were always on a high fat diet except during the experiments.

Studies on serum cholesterol in foreign countries required, of course, a firm base for comparison in this country. This was available in the data of Table 8 which have proved to be in good agreement with smaller series of men studied in other parts of the United States. It should be noted that the values in Table 8 should be increased by about 5 per cent for casual blood samples; the table refers to the basal state.

For the work abroad the apparatus, chemicals and cholesterol standards used in Minnesota were taken abroad and the method applied exactly as in Minneapolis. Further some samples and standards were sent by air to Minneapolis for check analysis. All the results, therefore, are strictly comparable in England, in Italy and in Spain.

TABLE 8

"Normal" values for the total cholesterol concentration, in mg. per 100 ml. of serum, for clinically healthy men in Minnesota. The data refer to 1492 men gainfully occupied in non-manual work in the metropolitan area of Minneapolis and St. Paul. Means and standard deviations (S.D.), and means expressed as percentages of the mean at age 25. These values refer to measurements using the Liebermann-Burchard reaction with the Bloor extract of bloods drawn in the basal state; for casual blood samples the values should be increased by about 5 per cent. With the Sperry-Schoenheimer method the values should be about 5 per cent lower. (Keys, et al, 1950).

Age	Mean S. D.	%	Age	Mean S. D.	%
20	173 ± 31	94	50	248 ± 45	135
25	184 ± 34	100	55	256 ± 46	139
30	195 ± 40	106	60	253 ± 34	137
35	200 ± 43	109	65	237 ± 34	129
40	219 ± 39	119	70	225 ± 42	122
45	236 ± 37	128	75	212 ± 37	115

Serum Cholesterol Values in Other Countries

In comparison with the United States the diet in Great Britain is interesting. The average fat consumption in Britain is high, though not so high as in the United States (about 11 per cent lower in Britain) and the pattern was not importantly changed during World War II and subsequent years. But the average cholesterol intake is much lower as indicated by the fact that the per capita consumption of eggs, meat and milk, as percentages of that in the United States, is, respectively, 52, 68, and 74 (from National Food Balance Data for 1949-50, supplied by the Nutrition Division, Food and Agriculture Organization of the United Nations).

In the spring of 1950 it was possible to study a carefully chosen sample of 48 clinically healthy men employed in Slough, a suburb of London, and these 40 to 55 year-old men, together with the sample of 44 healthy younger men studied in London a few months previously by Tanner (1951) are compared with the Minnesota men in Table 9.

TABLE 9

Serum total cholesterol values in clinically healthy men in the London area. Means, in mg. per 100 ml., adjusted to basal conditions, together with percentages of the means at age 25 in England and of the means for equal ages in Minnesota.

		% of Value of	
Age	Mean	Age 25	Minnesota
20	178	96	103
30	193	104	99
40	228	123	104
50	238	129	101

These Englishmen, then, conform closely to the Minnesota standards but this does not mean that the English and American populations are identical in these respects. The men studied in London are not a true sample because, for one thing, their fat intake was somewhat higher than the national average. A careful study made by the Ministry of Health of food actually eaten by the men at Slough gave an average of 35.4 per cent of calories from fats; the British national average is about 32%. For comparison, the retail average for Minneapolis in the winter of 1948 was 41.1 per cent of total calories as fats (data supplied from the U. S. Department of Agriculture by Dr. Esther F. Phipard), the intake value being perhaps 36 to 38 per cent.

The important point about the data from England is that, with similar total fat intakes, the serum cholesterol values are also closely similar to those in Minnesota. But the cholesterol intake of these Englishmen was not much more than half that of the Minnesota men, owing to the strict rationing, since 1940, of eggs, butter and meats; margarine and vegetable oils are unrationed in Britain.

In Naples similar studies were carried out on 83 clinically healthy men covering the age range 20 to 56. In Naples the diet proved to be identical with the Italian national average for the percentage of total calories supplied by fats - 20 per cent - and therefore about half the U.S. (and Minnesota) level. Serum cholesterol data are summarized in Table 10.

TABLE 10

Serum total cholesterol values in clinically healthy men in Naples. Means, in mg. per 100 ml., adjusted to basal conditions, together with percentages of the means at age 25 and of the means for equal ages in Minnesota.

		% of Value of	
Age	Mean	Age 25	Minnesota
20	176	93	102
30	204	107	105
40	219	115	100
50	218	115	88

The picture is not different from that in the Englishmen through age 30 but thereafter the age trend does not continue as in England or the United States. From age 40 to age 50 in Minnesota there is a mean rise in serum cholesterol of 29 mg.; in Naples there is a fall of 1 mg. Statistical analysis, moreover, shows these differences to be highly significant.

In Madrid, Spain, similar studies were made on 55 poor men hibitually on a low-fat diet (about 22 per cent of calories from fats) and 57 prosperous professional men on a diet similar to that eaten by wealthy men in the United States. The poor men, moreover, were on a low-calorie diet, as indicated both by the dietary survey and by the body weights and measurements of skinfolds. The main findings on these two groups of clinically healthy men are indicated in Table 11.

The prosperous professional men in Madrid are not much different from the Minnesota standards, but they are in great contrast to the poor Madrilenos, especially after 30 years of age.

The Factor of Relative Obesity

TABLE 11

Serum cholesterol values in 2 classes of clinically healthy men in Madrid. Means, in mg. per 100 ml., adjusted to basal conditions, together with percentages of the means at age 25 and of the means for equal ages in Minnesota.

	Poor Men			Rich Men		
		% of Value of			% of Value of	
Age	Mean	Age 25	Minnesota	Mean	Age 25	Minnesota
20	166	91	95	193	98	111
30	199	109	102	201	102	103
40	209	114	95	230	117	105
50	197	105	79	251	127	101

The foregoing analysis emphasizes the role of the total dietary fats in determining the serum cholesterol concentration. The question may be asked, however, as to the role of total calorie balance, particularly in view of the very low serum values in the undernourished poor men in Madrid. However, it seems that relative obesity is not the major factor, short of real undernutrition, when the men in England, in Naples and in Minnesota are considered. The mean relative body weights, as percentages of the Medico-Actuarial standards for height and age used in the United States were as follows: Minnesota 103, Naples 105, England 93. In spite of the relative leanness of the Englishmen, their serum concentrations were not lower than the Minnesotans. And the neapolitan serum concentrations differ, after age 30, from the Minnesota values in spite of the fact that the Neapolitans were a trifle fatter than the Minnesotans.

These facts are in harmony with our findings in Minnesota where we have studied the correlation between serum cholesterol and relative obesity. There is a positive correlation but it is small in men of equal ages in the general population. However, chronic severe undernutrition is almost always associated with low serum cholesterol concentrations (Keys et al, 1950, Chap. 22; Horst, 1950). This may be in part because the fat content of the diet in such cases is always low. On the other hand, we have consistently found increased values in men when they were actively gaining weight from simple overeating. In simple fasting the usual tendency for the serum

cholesterol to rise may reflect the fact that the fasting man is primarily metabolizing fat; in a sense then he is on a high-fat diet (cf. Keys, et al, op. cit.).

The Diet and Mortality

The data summarized here suggest an important chain of relations between the total fat content of the diet (or the proportion of fat calories of the total metabolized), the cholesterol (and lipoprotein) concentration in the blood, the development of atherosclerosis, and the mortality from degenerative heart disease. The vital statistics data for Italy clearly fit this pattern.

Vital statistics data are not available for Spain but those from Portugal may serve to indicate what may be the case in Spain where the diet is similar. There seems to be no doubt that coronary disease and myocardial infarction are not common in Spain. This is agreed by the local doctors and is confirmed by a search through the hospitals. But it is also of interest that the leading fashionable physicians do not lack cases among their rich patients and tell of many relatives and colleagues who have died from occlusions. In general, rich people do not go to hospitals in Spain.

Besides Italy, however, there are several countries where there are good current data on the character of the national diet and the mortality rates, for given sex and age, from degenerative heart disease. The data for men from age 40 to age 70 in the United States, Canada, Australia, England and Wales, Italy and Japan show a very regular progression of death rate from this cause from Japan, with about 8 per cent of the dietary calories in the form of fats, to the United States with about 40 per cent of all calories from fats. The mortality rate differences are very large; for men aged 55 to 59, inclusive, for example, the U.S. rate is 4 times that of Italy and 10 times that of Japan.

There is other impressive evidence to this same effect. World War II brought with it dietary alterations in many areas and a reduction in dietary fat was prominent for several years in lands conquered by Germany. In Norway, the public health and vital statistics records were well maintained and it is clear that not long after the national dietary change began there was a marked decline in mortality from circulatory disease, particularly from arteriosclerotic heart disease (Strøm and Jensen, 1951). At first this change in mortality was attributed to a reduction in dietary cholesterol (Malmros, 1950), but detailed analysis indicates that a reduction in total dietary fats was the responsible factor (Pihl, 1952).

Discussion

The argument and evidence assembled here make a consistent picture which holds promise of a preventive hygiene but many details are lacking. The mechanism of the action of the diet on the blood cholesterol concentration has not really been examined. We would like to know, apart from dietary peculiarities, why hypercholesterolemia tends to run in families. Moreover, other factors besides the blood concentration are important in the development of arteriosclerotic heart disease. Why are men and women so different, until well beyond the female reproductive age range, in susceptibility?

Recognition of all those limitations should not interfere with the analysis of the facts now at hand. There is obviously a fruitful field for epidemiological research as yet scarcely touched. And it is not too soon to begin the application, by educational means, of epidemiological findings.

The present high level of fat in the American diet did not always prevail and this fact may not be unrelated to the indication that coronary disease is increasing in this country. In the past 40 years the contribution of fats to the total metabolism in the United States has risen by more than 25 per cent; in the past 20 years the rise has been almost 13 per cent. In the face of all the evidence, is this situation desirable? One may seriously ask whether the current nutritional and dietetic teaching in this

country is as completely on the right track as some may suppose. Complete indifference to the amount of the fat in the diet is the attitude currently expressed by both technical and popular books and articles on diet.

From the statistics of the U.S. Department of Agriculture it is clear that the biggest contributor to the fats in the American diet is fats and oils as such, excluding butter, which comprise 46.5 per cent of the total. Meats, poultry and fish combined make a poor second at 22.1 per cent. Dairy products (milk, cream, ice cream, cheese), excluding butter, contribute 15 per cent, and butter makes up 3 per cent of the total. Any attempt to reduce the total fat intake must begin, then, with cooking fats and oils.

Acknowledgment

It is a pleasure to acknowledge my great obligation for help in getting subjects and for laboratory space and facilities to Drs. H. M. Sinclair, H. E. Magee, E. R. Bransby, Austin Eagger, D. Engel and Madeline Keech in England, to Professors Gino Bergami and Ferruccio di-Lorenzo and Drs. Flaminio Fidanza, Vicenzo Scardi and A. del Vecchio in Naples, Italy, and to Professors Carlos Jimenez Diaz, Francisco Grande and Drs. Francisco Vivanco, Minon, Palacios, Merchante, Segovia and Pariennes in Spain. Dr. J. T. Anderson provided able collaboration in Minneapolis. My wife, Mrs. Margaret H. Keys, performed many of the blood analyses in England, Italy and Spain.

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II. MEDICAL SCHOOL NEWS

Coming Events

March 2-4	Continuation Course in Clinical Dietetics
March 26	Special Lecture; "Trace Elements in Biochemistry and Medicine"; Dr. Burt L. Vallee; Peter Bent Brigham Hospital, Boston, Massachusetts; Owre Amphitheater; 4:00 p.m.
April 6-11	Continuation Course in Proctology for General Physicians
April 16-18	Continuation Course in Gynecology for Specialists

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Continuation Course

The University of Minnesota announces a continuation course in Clinical Dietetics which will be held at the Center for Continuation Study on March 2 to 4, 1953. The course in intended primarily for dietitians who are actively engaged in the practice of clinical dietetics. During the two-and-a-half day session, recent advances in the field of clinical dietetics will be considered. In addition, a part of the course will deal with some problems in administrative dietetics. The program will be under the direction of Miss Gertrude I. Thomas, Director of Nutrition and Professor of Dietetics, and the remainder of the faculty will include full-time and clinical members of the faculty of the University of Minnesota.

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Faculty News

Dr. Gaylord W. Anderson, Mayo Professor and Director, School of Public Health, is heading a Public Health Teaching Mission which has just gone to Cairo, Egypt. This teaching mission, which is being conducted under the joint sponsorship of the World Health Organization and of the Egyptian government, will undertake unstruction at a postgraduate level for approximately 40-50 trainees. Most of these trainees will be from Egypt. However, a few are expected from other Middle Eastern countries. It is expected that problems of public health administration, of epidemiology, and of sanitary engineering will be discussed by the mission members. In addition to Dr. Anderson, the mission includes Dr. Waldo Treuting, Professor of Public Health Education, Tulane University, and Mr. Clarence I. Sterling, Jr., Chief Sanitary Engineer, Massachusetts Department of Public Health. Dr. Anderson will return to Minneapolis on approximately March 15.

Doctors H. S. Diehl, Howard L. Horns, James S. McCartney, and Robert B. Howard attended the meeting of the Forty-Ninth Annual Congress on Medical Education and Licensure on February 8-10 at Chicago.

On February 14 Dr. F. W. Hoffbauer, Associate Professor, Department of Medicine, spoke on the subject, "Experimental Dietary Hepatic Necrosis: Absence of Cirrhosis following Recurrent Attacks" at the Conference on Nutritional Factors and Liver Diseases held in New York City.

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III. UNIVERSITY OF MINNESOTA MEDICAL SCHOOL
WEEKLY CALENDAR OF EVENTS

Physicians Welcome

February 23 - 28, 1953

Monday, February 23 (HOLIDAY)

Tuesday, February 24

Medical School and University Hospitals

9:00 - 9:50 Roentgenology-Pediatric Conference; L. G. Rigler, I. McQuarrie and Staff; Eustis Amphitheater, U. H.

9:00 - 12:00 Cardiovascular Rounds; Station 30, U. H.

12:30 - 1:20 Pathology Conference; Autopsies; J. R. Dawson and Staff; 102 I. A.

12:30 - 1:30 Physiology 114D -- Current Literature Seminar; 129 Millard Hall.

4:00 - 5:00 Pediatric Rounds on Wards; I. McQuarrie and Staff; U. H.

4:30 - 5:30 Clinical-Medical-Pathological Conference; Todd Amphitheater, U. H.

4:30 - ECG Reading Conference; James C. Dahl, et al; Staff Room, Heart Hospital.

5:00 - 6:00 X-ray Conference; Presentation of Cases from Ancker Hospital; Drs. Aurelius, Peterson, and Azad; Eustis Amphitheater, U. H.

Ancker Hospital

8:30 - 9:30 Medical-Roentgenology Conference; Auditorium.

1:00 - 2:30 X-ray - Surgery Conference; Auditorium.

Minneapolis General Hospital

9:30 - 10:30 Obstetrics and Gynecology Staff Rounds; William P. Sadler and Staff; 301 Harrington Hall.

10:00 - Pediatric Rounds; Spencer F. Brown; Stations I and J.

10:00 - Cardiac Rounds; Paul F. Dwan; Sta. I, Classroom.

10:30 - 12:00 Medicine Rounds; Thomas Lowry and Staff; Station F.

12:30 - Grand Rounds; Fractures; Sta. A; Willard White, et al.

12:30 - Neuroroentgenology Conference; O. Lipschultz, J. C. Michael and Staff.

12:30 - EKG Conference; Boyd Thomes and Staff; 302 Harrington Hall.

1:00 - Tumor Clinic; Drs. Eder, Cal, and Lipschultz.

1:00 - Neurology Grand Rounds; J. C. Michael and Staff.

Veterans Administration Hospital

7:30 - Anesthesiology Conference; Conference Room, Bldg. I.

8:30 - Infectious Disease Rounds; Dr. Hall.

8:45 - Surgery Journal Club; Conference Room, Bldg. I.

9:00 - Liver Rounds; Drs. Nesbitt and MacDonald.

9:30 - Surgery-Pathology Conference; Conference Room, Bldg. I.

Tuesday, February 24 (Cont.)

Veterans Administration Hospital (Cont.)

10:30 - Surgery Tumor Conference; L. J. Hay, J. Jorgens; Conference Room, Bldg. I.

1:00 - Review of Pathology, Pulmonary Tuberculosis; Conference Room, Bldg. I.

1:30 - Combined Medical-Surgical Chest Conference; Conference Room, Bldg. I.

2:00 - 2:50 Dermatology and Syphilology Conference; H. E. Michelson and Staff; Bldg. III.

3:30 - 4:20 Clinical Pathological Conference; Conference Room, Bldg. I.

Wednesday, February 25

Medical School and University Hospitals

8:00 - 9:00 Roentgenology-Surgical-Pathological Conference; Paul Lober and L. G. Rigler; Todd Amphitheater, U. H.

11:00 - 12:00 Pathology-Medicine-Surgery Conference; Medicine Case; O. H. Wangensteen, C. J. Watson and Staff; Todd Amphitheater, U. H.

12:30 - 1:30 Radioisotope Seminar; Report of 30th Class at Oak Ridge Institute of Nuclear Studies; Joseph Ryan; 12 Owre Hall.

1:30 - 3:00 Physiology 114B -- Circulatory and Renal System Problems Seminar; Dr. M. B. Visscher, et al; 214 Millard Hall.

4:00 - 5:30 Physiology 114C -- Permeability and Metabolism Seminar; Nathan Lifson; 214 Millard Hall.

4:30 - ECG Reading Conference; James C. Dahl, et al; Staff Room, Heart Hospital.

5:00 - 5:50 Urology-Pathological Conference; C. D. Creevy and Staff; Eustis Amphitheater, U. H.

8:00 - 10:00 Dermatological-Pathology Conference; Review of Histopathology Section; R. Goltz; Todd Amphitheater, U. H.

Ancker Hospital

8:30 - 9:30 Clinico-Pathological Conference; Auditorium.

2:00 - 4:00 Medical Ward Rounds;

3:30 - 4:30 Journal Club; Surgery Office.

Minneapolis General Hospital

8:30 - 9:30 Grand Rounds; William P. Sadler and Staff; Sta. C.

9:30 - Pediatric Rounds; Max Seham; Stations I and J.

10:30 - 12:00 Medicine Rounds; Thomas Lowry and Staff; Station D.

11:00 - Pediatric Seminar; Arnold Anderson; Classroom, Station I.

11:00 - Pediatric Rounds; Erling S. Platou; Station K.

12:15 - Pediatrics Staff Meeting; Classroom, Station I.

1:30 - Visiting Pediatric Staff Case Presentation; Station I, Classroom.

Veterans Administration Hospital

8:30 - 10:00 Orthopedic X-ray Conference; E. T. Evans and Staff; Conference Room; Bldg. I.

Wednesday, February 25 (Cont.)

Veterans Administration Hospital (Cont.)

8:30 - 12:00 Neurology Rehabilitation and Case Conference; A. B. Baker.

2:00 - 4:00 Infectious Disease Rounds; Main Conference Room, Bldg. I.

4:00 - 5:00 Infectious Disease Conference; Wesley W. Spink; Conference Room, Bldg.I.

4:00 - Combined Medical-Surgical Conference; Conference Room, Bldg. I.

7:00 p.m. Lectures in Basic Science of Orthopedics; Conference Room, Bldg. I.

Thursday, February 26

Medical School and University Hospitals

8:00 - 9:00 Vascular Rounds; Davitt Felder and Staff Members from the Departments of Medicine, Surgery, Physical Medicine, and Dermatology; Heart Hospital Amphitheater.

9:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; E-221, U. H.

11:00 - 12:00 Cancer Clinic; K. Stenstrom and A. Kremen; Todd Amphitheater, U. H.

12:30 - Physiological Chemistry Seminar; Factors Influencing Adrenal Cholesterol; Robert Bahn; 214 Millard Hall.

1:30 - 4:00 Cardiology X-ray Conference; Heart Hospital Theatre.

4:00 - 5:00 Physiology-Surgery Conference; Todd Amphitheater, U. H.

4:30 - 5:20 Ophthalmology Ward Rounds; Erling W. Hansen and Staff; E-534, U. H.

4:30 - ECG Reading Conference; James C. Dahl, et al; Staff Room, Heart Hospital.

5:00 - 6:00 Radiology Seminar; Thoracic Surgery Conference; Thomas Kinsella; Eustis Amphitheater, U. H.

7:30 - 9:30 Pediatric Cardiology Conference and Journal Club; Review of Current Literature 1st hour and Review of Patients 2nd hour; 206 Temporary West Hospital.

Ancker Hospital

4:00 - Medical-Pathological Conference; Auditorium.

Minneapolis General Hospital

9:30 - Neurology Rounds; Heinz Bruhl; Station I.

10:00 - Pediatric Rounds; Spencer F. Brown; Station K.

10:00 - Psychiatry Grand Rounds; J. C. Michael and Staff; Sta. H.

1:00 - Fracture - X-ray Conference; Dr. Zierold; Classroom.

1:00 - House Staff Conference; Station I.

2:00 - 4:00 Infectious Disease Rounds; Classroom.

4:00 - 5:00 Infectious Disease Conference; Wesley W. Spink; Classroom.

Veterans Administration Hospital

8:00 - Surgery Ward Rounds; Lyle Hay and Staff; Ward 11.

8:00 - Surgery Grand Rounds; Conference Room, Bldg. I.

11:00 - Surgery-Roentgen Conference; J. Jorgens; Conference Room, Bldg. I.

Friday, February 27

Medical School and University Hospitals

8:00 - 10:00 Neurology Grand Rounds; A. B. Baker and Staff; Station 50, U. H.

9:00 - 9:50 Medicine Grand Rounds; C. J. Watson and Staff; Todd Amphitheater, U. H.

10:30 - 11:50 Medicine Rounds; C. J. Watson and Staff; Todd Amphitheater, U. H.

10:30 - 11:50 Otolaryngology Case Studies; L. R. Boies and Staff; Out-Patient Department, U. H.

11:45 - 12:50 University of Minnesota Hospitals Staff Meeting; ACTH and Cortisone in Certain Allergic Disorders; J. S. Blumenthal; Powell Hall Amphitheater.

1:00 - 2:50 Neurosurgery-Roentgenology Conference; W. T. Peyton, Harold O. Peterson and Staff; Todd Amphitheater, U. H.

3:00 - 4:00 Neuropathological Conference; F. Tichy; Todd Amphitheater, U. H.

4:00 - 5:00 Physiology 124 -- Seminar in Neurophysiology; Ernst Gelhorn; 113 Owre Hall.

4:30 - ECG Reading Conference; James C. Dahl, et al; Staff Room, Heart Hospital.

5:00 - Urology Seminar and X-ray Conference; Eustis Amphitheater, U. H.

Ancker Hospital

1:00 - 3:00 Pathology-Surgery Conference; Auditorium.

Minneapolis General Hospital

9:30 - Pediatric Rounds; Wallace Lueck; Station J.

10:30 - Pediatric Surgery Conference; Oswald Wyatt; Tague Chisholm; Station I. Classroom.

12:00 - Surgery-Pathology Conference; Dr. Zierold, Dr. Coe; Classroom.

1:00 - 3:00 Clinical Medical Conference; Thomas Lowry; Classroom, Station M.

1:15 - X-ray Conference; Oscar Lipschultz; Classroom, Main Bldg.

2:00 - Pediatric Rounds; Robert Ulstrom; Stations I and J.

Veterans Administration Hospital

10:30 - 11:20 Medicine Grand Rounds; Conference Room, Bldg. I.

1:00 - Chest Follow-Up Conference; E. T. Bell; Conference Room, Bldg. I.

Saturday, February 28

Medical School and University Hospitals

7:45 - 8:50 Orthopedic X-ray Conference; W. H. Cole and Staff; M-109, U. H.

9:00 - 10:00 Infertility Conference; Louis L. Friedman, David I. Seibel, and Obstetrics Staff; Station 54

9:00 - 10:30 Pediatric Grand Rounds; I. McQuarrie and Staff; Eustis Amphitheater.

9:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; Heart Hospital Amphitheater.

9:15 - 10:00 Surgery-Roentgenology Conference; L. G. Rigler, J. Friedman, Owen H. Wangensteen and Staff; Todd Amphitheater, U. H.

10:00 - 11:30 Surgery Conference; Todd Amphitheater, U. H.

Saturday, February 28

Medical School and University Hospitals (Cont.)

10:00 - 12:50 Obstetrics and Gynecology Grand Rounds; J. L. McKelvey and Staff; Station 44, U. H.

11:30 - Anatomy Seminar; Histopathology of Certain Experimental Renal Lesions; Dennis J. Kane; 226 Institute of Anatomy.

Ancker Hospital

8:30 - 9:30 Surgery Conference; Auditorium.

Minneapolis General Hospital

11:00 - 12:00 Medical - X-ray Conference; O. Lipschultz, Thomas Lowry, and Staff; Main Classroom.

Veterans Administration Hospital

8:00 - Proctology Rounds; W. C. Bernstein and Staff; Bldg. III.

8:30 - 11:15 Hematology Rounds; Drs. Hagen, Goldish, and Aufderheide.
11:15 - 12:00 Morphology . . . . . Dr. Aufderheide.