Comparison of Fat Intake of American Men and Women

Possible Relationship to Incidence of Clinical Coronary Artery Disease

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A detailed dietary survey of 46 women and their husbands belonging to an upper economic stratum was obtained. The caloric and percentage fat intakes of the sexes were almost identical. The data concerning the possible relationship of hormonal, dietary, and socioeconomic factors to the pathogenesis of coronary artery disease are reviewed.

RECENTLY a number of epidemiologic studies1-6 have been published concerning the possible causes of the relative immunity of various classes and groups of non-American men against overt coronary artery disease. Since a low-fat intake was thought to be a trait common to all of these groups, it was concluded that this dietary habit was chiefly responsible for the observed relative infrequency of clinical coronary artery disease in these individuals.

Impressive as is this relative immunity of the poverty-stricken Bantu,4,5 the similarly stricken Southern Chinese,7,8 the economically depressed Italian2 and Spaniard,6 and similar groups against overt coronary disease, the immunity of the young, premenopausal American woman of any class is perhaps even more striking. Thus, before the age of 40, if she is neither hypertensive nor diabetic, she exhibits more than 20 times more resistance to symptomatic coronary disease than does her American male counterpart.9-12

In view of this truly astounding immunity of the young American female, it has seemed rather strange to us that no attempts, as far as we know, have been made to determine her daily fat intake. Yet if the reputed greater intake of fat on the part of her male counterpart (as compared to that of the non-American men referred to above) is held responsible by some observers1-6 for his greater incidence of coronary artery disease, certainly her daily fat intake warrants scrutiny. Only in this manner can the possible relationship of fat intake to her immunity be assessed.

The present article describes the results obtained in such a survey in which the dietary intakes of 46 women and their husbands were recorded and evaluated. These results indicate that the fat intake of both sexes is identical and hence the relative immunity against coronary disease to be expected in these women is due to other than dietary causes.

METHODS

Selection of Subjects. Volunteers of an upper class urban and suburban feminine social organization were selected for the present studies. They were married, below the age of 50 years, free of overt cardiovascular disease, and adjudged intelligent and reliable enough for the purposes desired. These women were provided with a printed protocol providing space for the detailed daily recording for 14 days, during November 1955, of their own and their husband’s intake of all foods and beverages, both at regular mealtimes and otherwise. A standard but simple method of recording various solids and liquids was employed that seemed to make it simple for the subject to measure. In addition, information concerning the possible conscious restriction of caloric intake (i.e., dieting) was obtained.

Detailed instructions concerning the method of recording their food intake were given to these volunteers by the authors, in either group or personal interviews. During the latter, the general purpose of the research was explained to them so that their full cooperation in reporting food intakes honestly and accurately might be obtained. Such cooperation was more than satisfactory and we are as positive as one can be under the circumstances that the records chosen for the present study (46 of the 49 done) were meticulously assessed and recorded.

The dietary records thus compiled were submitted to 3 hospital dietitians who calculated from them, the total daily intake of calories, as well as the daily fractions of protein, carbohydrate, and fat.

RESULTS

Although the actual total caloric intake (1830) of the males of our group (table 1) was
slightly greater than that of the females (1577), if these values are correlated to the body surface area of each group, that of the average male (915/M.²), becomes somewhat less than that (986/M.²) of the average female.

The daily fat intake of the men also was not found to be greater than that of their wives. Thus the daily fat intake of the males was 775 calories or 387 per M.² of surface area and that of the females was 665 calories or 409 per M.² of surface area. As table 1 shows, a similarity exists in the fractions of carbohydrate and protein ingested by each sex.

It was of interest that 18 of the women (39 per cent) and 20 of the men (44 per cent) were in the habit of consciously restricting their diet. This tendency of the members of the upper class to watch their weight and consequent intake of food should be an important factor to be reckoned with in any attempt to assess the food intake of any class or group by utilization of governmental statistics.

These dietary values obtained by us are considerably lower than those assumed to represent the average food intake of the well-nourished American citizen. However, these latter values have never been determined by direct survey but have been derived from government statistics indicating only the amount of "food available for consumption." Such figures have at best a possible relevance for all classes and groups of the nation but it is extremely doubtful, in view of the present results, whether they are valid for a group of upper class, relatively sedentary Americans who rarely indulge in continuous heavy labor. Nevertheless, if there is to be a critical assessment of the dietary fat-atherosclerosis concept, the fat intake of this type of group may have more significance than a national survey embracing all groups because the male members of a group such as ours exhibit not an immunity but a proclivity to coronary artery disease.

**DISCUSSION**

If these dietary results can be assumed to have national pertinency, then it is clear that the American, upper class, premenopausal female ingests as much or more fat than her male counterpart. In view of her known relative immunity to clinical coronary disease, these results suggest either that femaleness protects against the otherwise supposed atherogenic properties of a high-fat intake or else that a high-fat intake itself is not specifically or uniformly atherogenic. It therefore may be of profit to review some available evidence concerning both of these possibilities.

First, although there is no experimental evidence available from animal studies that femaleness as such plays the slightest part in certain species in preventing atherosclerosis resulting from ingestion of diets high in fat and cholesterol, there is evidence that administration of pharmacologic doses of estrogen protects the coronary vasculature of the chick against the atherogenic properties of a high cholesterol-high fat diet. However the production of hypopotassemia and nonreactivity to desoxycorticosterone acetate in these chicks and possibly of associated hypotension makes it questionable whether such data explain the striking immunity to clinical coronary disease of the American woman. Nor can estrogen entirely be credited for the relatively low β-lipoprotein content found in the premenopausal American female simply because
lowering of the $\beta$-lipoprotein content of men suffering from coronary artery disease can be accomplished by administration of pharmacologic doses of estrogen. The possible fallibility of such reasoning is emphasized by the fact that the rural Guatemalan female (but not her urban compatriot) displays a high $\beta$-lipoprotein spectrum and this despite her undoubted abundance of female sex hormone. Before it therefore can be stated with assurance that either the feminality or estrogen content of the American woman is responsible for her low plasma $\beta$-lipoprotein content, the blood of females of other groups and races both in and out of Western society must be analyzed.

Another reason presented as evidence that estrogen accounts for the relative immunity of the American woman against this disease is the supposed increase in its incidence after the menopause. However, examination of available statistics suggests that similar to the male, there appears to be no sudden upsurge in the severity of coronary atherosclerosis in the American woman in the immediate postmenopausal years but a gradual and smoothly ascending increase from the fourth to the seventh decade. At all ages, however, the degree apparently remains less than that in the male.

Secondly, protection against clinical coronary disease does not appear to be a sex-linked phenomenon in all races. Thus the American Negro woman completely lacks this protection and, according to Keys, the incidence of degenerative heart disease in Italy is about the same in both sexes.

Finally, it seems difficult to envision the striking immunity to this disease as due to the sex of the young American woman, if only because it dwindles considerably when she becomes hypertensive and disappears entirely when she becomes diabetic. Certainly a supposed protection of the potency of 20 to 1 afforded the American female before the age of 40 might be expected to retain some influence in the face of these diseases.

These observations, when viewed as a whole, give little or no support to the belief that the American female may have a sex-linked protection against the atherogenic properties of a high-fat diet. This conclusion forces us to examine the second possibility, namely that a high-fat intake, of and by itself, is not specifically responsible either for the greater incidence of clinical coronary disease in the American male as compared to the American female or to men of other groups ingesting lower amounts of fat, or for their supposed higher level of serum cholesterol and $\beta$-lipoproteins.

Several authors have presented data suggesting that the male members of certain groups of various countries and races ingesting low amounts of fat (10 to 20 per cent of the total caloric intake) also show a markedly lower incidence of deaths due to coronary artery disease and its complications (i.e., thrombosis and myocardial infarction) as well as lower levels of serum cholesterol and $\beta$-lipoproteins. It also has been pointed out that ingestion of excess fat when given with cholesterol accelerates processes of hypercholesteremia and atherosclerosis in the chick, rat, rabbit, and monkey. These epidemiologic and experimental observations have been emphasized recently by some investigators as support for their belief in the role of dietary fat in the pathogenesis of coronary artery disease.

Critical analysis however of the above data and of other existing data not taken into consideration or emphasized by these dietary proponents, leads the impartial observer to believe that the differences found in the incidence of clinical coronary disease in various groups and races cannot be ascribed with certainty only to differences in the increment of fat ingested. This conclusion was reached for the following reasons.

First, much dietary data now used to incriminate a high-fat intake in the pathogenesis of coronary artery disease were not obtained by detailed, systematically conducted food diaries but were interpolated from governmental statistics. As Mann, Munoz, and Scrimshaw have pointed out, the usual dependence upon such data may lead to considerable error. Indeed our own survey and that of Davis and Scoular indicate that among supposedly well-nourished persons, the actual dietary intake was only one half that...
which could be deduced from statistics of the U. S. Department of Agriculture on "foods available for consumption."

Secondly, it is claimed that a low-fat intake possibly protects against coronary artery disease by lowering plasma cholesterol. However, it is to be doubted quite seriously whether only a low-fat intake is per se responsible for the lower plasma cholesterol observed in relatively immune groups and races. This is so, not only because plasma cholesterol is not consistently lowered by a low-fat intake, but it can even be increased by augmenting the protein or carbohydrate fractions of the diet. Indeed, plasma cholesterol can even rise if physical activity is curtailed. Furthermore, the "low fat intake-low plasma cholesterol" concept fails to explain the low plasma cholesterol of the Navajo who eats as much animal fat as the average American white man. Indeed, when the plasma cholesterol of United States citizens ingesting 40 per cent of the diet as fat is compared with that of Italian males ingesting only half as much fat, it is actually extremely doubtful if a statistically significant (not to mention a physiologically significant) difference can be shown to exist. These facts appear to us to negate the possible over-all inevitability of "low fat intake-low plasma cholesterol" relationship. Actually the basic mechanism responsible for the plasma cholesterol level remains unknown.

Thirdly, it has been proposed that a high-fat intake per se leads to a higher concentration of plasma \( \beta \)-lipoprotein, but this appears untenable in view of recent evidence that complete dissociation may exist not only between intake of fat and the level of \( \beta \)-lipoproteins, but also between serum levels of cholesterol and \( \beta \)-lipoproteins. Actually it would appear that the serum \( \beta \)-lipoprotein content, similar to the cholesterol level, may be determined by factors other than just fat intake.

Fourthly, with possible exception of the Bantu, epidemiologists have not stressed the relative infrequency of hypertension that also has been observed in their low-fat-eating groups found relatively immune to heart disease. In view of the well-known association between hypertension and augmentation of coronary disease, their failure to consider this second peculiarity of these immune persons makes their final conclusions open to doubt.

Fifthly, almost all of the relatively immune groups, classes, and races studied, besides ingesting less fat, also indulge in considerably more physical exertion than American groups with whom they were compared. This difference, although seemingly discarded by some observers, nevertheless may have considerable influence upon the incidence of coronary disease in the groups compared.

Sixthly, mortality statistics and not data obtained by pathologic study frequently have been used to determine the incidence or prevalence of coronary artery disease in epidemiologic studies. However, deaths due to myocardial infarction cannot be employed necessarily as an index of the respective incidence of coronary artery disease in various groups. Thus the abrupt decline in cardiac deaths in Norway occurred in a matter of a few months, a phenomenon that cannot be ascribed to any unique resolution of coronary atherosclerosis itself. Moreover, there was a similar decline in deaths due to other types of heart disease and also just as marked a decline in postoperative deaths following pelvic surgery in females.

Actually coronary atherosclerosis may be almost ubiquitous in men of Western society, but myocardial infarction is a sudden affliction of a minority. This latter concept also is borne out by the recent study of Morris who noted a 7-fold increase in the incidence of deaths due to coronary disease in English persons from 1907 to 1949 although an actual decrease occurred in the incidence of advanced coronary arteriopathosis in the same population. This dissociation of morbidity and mortality is also found to a lesser extent in the Bantu and the Melanesians.

These latter findings suggest that if a high-fat intake does increase the death rate due to coronary heart disease, it may do so, not by increasing the degree of atherosclerosis as such, but by increasing the incidence of thrombotic complications by inducing changes in blood
coagulation,77-79 clumping of red blood cells,73-75 and possible capillary obstruction by chylomicra,74-78 which, when superimposed upon the basic coronary disease, increase its lethality.

Seventhly, as we have previously pointed out,79 the fat intake of several races9, 56-62, 79, 80 is comparable to that of the middle or upper class American, yet their incidence of coronary artery disease is almost nonexistent. Nor can these exceptions be dismissed as due to hereditary influences. The high incidence of coronary artery disease in the American Jew has been so explained by some observers,81 but the immunity of the non-Westernized Yemenite Jew99 makes it necessary to adopt extreme caution before an hereditary protection is promulgated to explain glaring deviations from an arbitrarily erected concept.

Finally, it should be pointed out that in most epidemiologic studies, little attention has been paid to the really striking fact that immune groups invariably have belonged to either the lower socioeconomic class of Western society or to groups not belonging to this society. Conversely stated, only middle and upper class Western man has this disease in abundance.

As we have previously emphasized,79 the American executive differs from these immune groups in more than just dietary respects. One of the outstanding differences is his exposure to, indeed his immersion in, the rapidly increasing profound emotional stresses and tensions of this society. A succinct description of this emotional stress was given by de Tocqueville as early as 183692 and it was allied with coronary artery disease by Osler in 189993 and by Stewart in 1950.94 Indeed, the physiologic and pathologic effects of the application of this stress have begun to be recorded.95-99 Nevertheless, its possible importance has been brought up in epidemiologic studies only to be summarily dismissed.3, 91

Despite the negation of emotional stress by its neglect, if its absence in a group is used as a standard of comparison, as good or perhaps a better correlation can be obtained with it and the decreased incidence of coronary artery disease1, 5, 14, 19 as can be obtained with a low-fat intake. Thus, absence of socioeconomic stress in the placid Navajo92 and Eskimo65-92 correlates with their rarity of coronary artery disease, whereas no such correlation could be found if their fat intake were employed as a standard. Again as Brock and Bronte-Stewart1, 8 pointed out in their studies in Cape Town, if job responsibility were used as an index, as good a correlation could be found between it and the increased incidence of coronary artery disease as was found with the fat intake.

In the same regard, it should be mentioned that, both in the study of Gertler and White9 and that of Yater and co-workers,96 although no difference was found between the dietary habits of their young coronary patients and those of the controls, considerable difference was found in the type of civil positions held by the 2 groups. The young coronary patient almost always had occupied a position demanding responsibility and frequently associated with emotional stress.

In view of these observations, it seems almost incredible that a factor so rampant in the American community has received so little considered objective attention. How can one compare the incidence of a disease (whose etiology is obscure) in the American male with that in the American housewife, the male eunuch, the Chinese coolie, or the Bantu without giving as much weight to this factor of "socioeconomic stress" as to any other factor? Undoubtedly the measurement of the dietary fat intake of a group of persons is infinitely easier than a similar evaluation of their socioeconomic stresses. Such difficulty, however, should not lead us to assume that the latter may not be an important factor. The forces of nature unfortunately are not yet applied according to the ease with which we can discover and understand them, much less measure them.

****SUMMARY****

A detailed dietary history was obtained of 46 women and their husbands, belonging to an upper economic stratum. No significant difference was found in the caloric or percentage fat intake of each sex, when corrected for differences in surface area.
A critical review of various data relating to the relative immunity of the American woman to clinical coronary artery disease suggests that this immunity cannot be due entirely to some endocrine-induced protection against the supposed atherogenic properties of a high-fat dietary.

A similar review was made of the data relating the high intake of fat to the increased incidence of clinical coronary artery disease as observed in the American male. It was concluded that such a relationship is not only unproved but probably untenable.

The possible atherogenic potential of the socioeconomic stress peculiar to and characteristic of the middle and upper class male of Western society is discussed.

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SUMMARIO IN INTERLINGUA

Esseva obtenite un detaliate historia dietari de 46 feminas e lor sposos pertinente a un strato economic superior. Nulle significative differentias inter le sexos esseva constatare in le ingestion calorie o procentual de grassia post compensation del valores pro differentias in le areas de superficie corporee.

Un revista critic del varie datos concernente le immunitate relative de feminas american contra clinic morbo de arteria coronari pare indicar que iste immunitate non pote esser le effecto de alium protection de origine endocrine contra le proprietates supponitmente atherogene de dietas ric in grassia.

Un simile revista esseva facite pro le datos concernente le stipulate relation inter alte valores del ingestion de grassia e le augmentate incidentia de clinic morbo de arteria coronari observate in le masculo american. Le conclusion esseva que iste relation es non solmente non demonstrate sed probablemente etiam non tenibile.

Es discutite le possibile potential atherogene del stresses socio-economic que es un charactaristica particular del masculo del classes medie e superior in le societate occidental.

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Clinical and pathologic findings were correlated in 32 patients who developed clinical evidence of ischemic heart disease within 3 months of death. Of 53 coronary occlusions, only 7 were not followed by myocardial infarctions. The occlusions occurred in vessels supplying muscle previously infarcted. Of 77 infarcts, 52 were recent. Twenty-eight patients had ischemic pain longer than 30 minutes, 2 angina pectoris, 2 aggravation of dyspnea, and 19 had no apparent symptoms. Painless myocardial infarction usually occurs in patients who do not have coronary occlusion. The authors believe that pain may be due to the intensity of the ischemia (greatest when a vessel is occluded) and infarction due to the duration of ischemia. There is no evidence that coronary occlusion without infarction causes symptoms.

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