In the wealthy communities of the world, there has been a steady increase in the proportion of deaths due to coronary heart disease (C.H.D.) during this century. All authorities are agreed that there is no single factor causing atherosclerosis and C.H.D. - the list of known or suspected “risk factors” is a long one, and additional suspects continue to be added.

Considering the obvious complexity of the disease syndrome and the equally obvious difficulties of conducting long-term experiments on human beings, it can confidently (and regretfully) be forecast that it will be a very long time before the full picture emerges.

Nearly all authorities are agreed that, within this etiological complex, nutritional factors play a significant, and possibly crucial role. Although there are some dissentents, most are of the opinion that some foods of animal origin are important contributors.

The link between diet and atherosclerosis is the elevation of plasma lipids and lipoproteins. Epidemiological evidence shows clearly that in populations with consistently low levels of plasma cholesterol and fatty acids, atherosclerotic coronary heart disease does not occur as a major disease.

The dietary components of special concern are cholesterol and fat, and especially those fats with a high concentration of saturated fatty acids. In the present context, our interest lies in the relatively high cholesterol content of meats, dairy products and egg-yolk, and in the high degree of saturation of fats in ruminant tissues and milk.

It is timely, then, to examine the relation between ruminants as sources of food and the welfare of modern man.

Primitive man developed over a very long period (more than 2 million years) as a hunter and food gatherer. The social structures that we call civilized communities could never have arisen while man obtained his food in that way. This difficulty is well illustrated in the culture of the Australian aboriginals who colonized this country at least 25,000 years ago, at a time long before any known human beings had made the first recognizable moves toward agriculture, animal husbandry or settled communities. The aboriginal’s food technology probably advanced little, if at all, since arriving in Australia - indeed, it is improbable that development could have occurred in the absence of indigenous animal species suitable for domestication as sources of food and power. It would have been difficult for man to develop crop production prior to animal husbandry - however, once the techniques of crop production had been discovered, communities could emerge in areas where plant foods comprised their sole or main diet, perhaps supplemented with animal foods derived from hunting and fishing. It is not unreasonable to conclude that the difference between the aboriginal’s food technology of 1788 and that of our present society had its origin in the domestication of animals, and especially of ruminants.

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It seems that Mesolithic man domesticated the goat and sheep about 7000 B.C., before the commencement of agriculture, and that cattle were added during the next 2000 years as a post-agricultural acquisition (Zeuner 1963). The living "storehouse" of food enabled cultural progress at a rate depending on the proportion of the community needed for employment in food production. It is noteworthy that once the arts of domesticating animals had evolved, most of the food and draught animal species were, in fact, domesticated within a short period of time. It would be an interesting exercise for some biologist to explain why so few of the world's mammals and birds have been successfully domesticated.

The evolution of crop production by Neolithic man, originally designed for production of food seeds, would automatically have provided increasing scope for animal husbandry. The combination of crop and animal husbandry led to a swift progression so that, within a few thousand years, man moved from a stone-age culture to the highly developed civilizations that flourished and waned during the period of 4000 years B.C. It seems fair to say that the exploitation of ruminants has been woven into the tapestry of man's emergence from primitive hunter to urban dweller.

This agricultural revolution brought with it a new capacity of man to despoil his environment and its food-producing capacity. We cannot criticize primitive man for that; we are continuing to do the same today. Anyone with an inkling of agriculture and ecology would appreciate how difficult it must have been for the early farmers to understand how an environment could be changed to the detriment of food production. So, in spite of great developments in traditional crop and animal husbandry, food production remained a critical feature in human communities until recent times - the threat of famine was ever present, as it still is today for many peoples. Within the last two hundred years the technologies involved in food production, transport and storage have advanced so far that the spectre of famine has vanished for the present-day affluent nations.

This affluence has another very important consequence. For all practical purposes, most members of an affluent society can eat whatever they like - income available for food plays a relatively minor role in food selection. It is therefore of great interest that, within these societies there is an obvious tendency to consume a diet with a high content of fat and protein, and with these components substantially derived from foods of animal origin, and especially from ruminant animals.

This leads one to suspect that this dietary tendency derives from something quite fundamental in the human appetite, perhaps reaching back into the evolution of our primitive ancestors; if so, we can expect that it will be very difficult to convince affluent people that they should change their diet for health preservation.

It is a truism that the food chain for all animals starts with plants. It is known, too, that in all plant leaves, the fatty acids are highly unsaturated - indeed, the fats of nearly all plant products have this characteristic; a couple of notable exceptions are coconut oil and palm oil. This universal occurrence may have contributed to the loss during evolution of the capacity of animals to synthesize...
the fatty acids, linoleic and linolenic. As these acids are essential components of living cells, they have become indispensable components of the diet.

This is a highly specific loss of enzymic function. The animal has virtually no capacity to store energy as carbohydrate, and little to store protein - fat has become the buffer against energy deprivation. So we find that animal tissues can synthesize fatty acids from carbohydrates, protein, organic acids or dietary fats, and can store the fat in adipose tissue cells. The animal tissues can also produce mono-unsaturated fatty acids, notably oleic. It is curious that the animal can produce, for specific cellular functions, a whole array of highly unsaturated fatty acids from dietary linoleic and linolenic acids; but it cannot desaturate stearic or oleic to give these two essential-acids.

A second curious feature in fatty acid metabolism should be noted, viz., the amount of essential fatty acids required in the diet. For over a century it has been known that the fattening or lactating animal can synthesize very large amounts of fat even when the diet contains but little. Fat came to be thought of simply as a storage substance. During the latter decades of the nineteenth century and the early twentieth, careful experiments were undertaken to ascertain exactly what compounds were required in the animal's diet. It will be recalled that such experiments led, during the period 1908-1930, to the discovery of most of the vitamins, essential mineral elements, and essential amino acids.

The earliest experiments on so-called "fat-free" diets suggested that fats were not required nutrients. In considering this finding, it is well to remember that the chemistry of lipids was not well advanced - indeed, it is fair to say that lipid biochemistry laboured under a heavy analytical burden until the discovery of gas-liquid chromatography in 1954. The explanation of this experimental result can be found in four facts: (a) the extraction of fats by then currently acceptable methods was incomplete; (b) the small amount of residual fat contained lipids with essential fatty acids; (c) animal tissues tenaciously retain essential fatty acids when the diet provides an insufficiency; and (d) the need for essential fatty acids is least when the diet contains little fat. It is now known that a high fat intake requires a concomitant increase in the intake of polyunsaturated fatty acids. Current recommendations are for a ratio of about equal proportions of polyunsaturated, mono-unsaturated and saturated fatty acids in human diets.

The recognition that lipids are involved in the aetiology of atherosclerosis in man has come but lately and is not yet fully accepted. Indeed, it is only since 1935 that atherosclerosis has been looked upon as a metabolic disease; previously it was regarded simply as one of the unfortunate degenerations associated with growing old. Although experimental clues were available prior to 1913 from Russian research showing that atheromatous lesions could be induced in the aorta of rabbits by feeding cholesterol, this was considered irrelevant to the human disease. Leary (1935) started a new line of thought with his hypothesis that atherosclerosis arose from an inadequacy of cholesterol metabolism, that diet played a role in producing high blood cholesterol concentration, and that physical stresses on the arterial walls (especially in subjects with hypertension) were factors in the localization and progression of lesions.
Since the termination of the second World War, and particularly during the last two decades, there has been a rapidly growing interest in all aspects of coronary heart disease. There has been a tendency to look upon the disease as a newcomer to modern civilization; this view has, in fact, been strongly advanced by some writers. It is also commonly stated that the incidence is rising rapidly - one reads such striking phrases as "this terrifying modern epidemic". Both statements merit comment.

Leibowitz (1970) has undertaken a thorough examination of relevant historical documents and has reached the conclusion that the disease has probably always been with us, though recognition of the main clinical syndromes is of recent occurrence (e.g. of angina pectoris in the late eighteenth century, and of coronary thrombosis in the late nineteenth century).

Atherosclerosis, as the underlying pathological change, is a very slowly developing lesion, producing no untoward effects until interference with arterial blood flow occurs. In consequence, the incidence of coronary heart disease is strongly correlated with age.

It would seem that life-span (i.e. the maximum duration of life) has probably not changed during recorded history - the three score years and ten of biblical writings is just as relevant today; like all living creatures, we seem to be born with "in-built obsolescence". But as pointed out by Medawar (1957), probably none of us dies of the mere burden of years - we succumb to one or other of the classical causes: trauma, toxins, deficiencies, infections, neoplasms and degenerations.

When we look at life expectation, however, the picture is very different indeed. In the U.K. in 1700, the average life expectation was only 30 years, about the figure at the time of the great Greek and Roman civilizations. This value rose to 36 in 1750, 40 in 1850, 46 by the end of the nineteenth century, but then jumped to over 60 years by 1940. The early figures are approximations, but precise information is available from 1838 when the Registrar-General's records were commenced. In that year, the death rate from all causes in the U.K. was 21.7 per 1000 living persons; by 1926 the figure had fallen to less than half - 10.3 per 1000 living persons.

This change in age structure of the population can account for most of the apparent increase in heart disease. The reductions in death rate during the last two centuries were due initially to improved hygiene (in turn due, as wittily expressed by Pirie (1967), to technical skills in getting clean water into towns and getting dirty water out!) and later, in the nineteenth century, to the discovery of infective agents and methods for their control.

Angina pectoris was described as a clinical entity by Heberden in 1772 and was soon accepted and recognized by clinicians. However, clinical coronary occlusion was not described till 1876. It is definitely erroneous to conclude, as for example Michaels (1966) did, that the dramatic symptom complex of coronary thrombosis could not have been missed (and hence recorded) by the leading physicians. Rather, the evidence favours the view of Sprague (1966) who wrote, "Without much question, men have died of coronary narrowing and
occlusion for thousands of years" and "the prevalence of coronary atherosclerosis has probably remained much the same in Europe and the United States for at least several hundred years, relative to the population "at risk" as determined by the average age and freedom from other causes of death, especially tuberculosis and other infectious diseases."

Hammer's brilliant paper in 1878 recording the first authenticated clinical case of coronary occlusion provides the illuminating comment that he had "never heard of such a diagnosis". Yet there is clear evidence that at this time the pathologists already had a sound knowledge of the major changes in the heart associated with constriction or occlusion of the coronary arteries. The association of clinical and pathological findings was slow in coming. Herrick's major clinical contribution on the disease in 1912, was reported by its author to have "fallen like a dud" on his contemporaries. As late as 1925, the celebrated cardiologist Mackenzie wrote an entire textbook on diseases of the heart with but one oblique mention of coronary thrombosis; in contrast, in 1922, Paul White and Myers unequivocally include myocardial infarction in their classification of cardiac diagnosis.

If further evidence of slow recognition is required, it could be mentioned that the word "coronary" does not appear until 1931 in the U.K. Registrar-General's records of causes of death.

It is also commonly stated that during the last few decades there has been a notable tendency for younger persons to be afflicted. The available data do not seem to support this notion. For example, in New Zealand, the rate of increase in deaths from coronary heart disease and myocardial infarction over the period 1950-1969 is substantially the same in all age groups from 35 to 65 years and over. This uniform trend suggests an improved diagnosis rather than a real increase in incidence. Further, deaths per 100,000 in the various age groups in 1966 were 1.4 in the range 0-34 years, 69 in 35-44, 295 in 45-54, 858 in 55-64, 1939 in 65-74, and 3723 in the group of 75 years and older. The data for Australia, U.S.A. and Canada are closely similar. Taking deaths from all heart diseases, the average ages at death are surprisingly high - for example, in the U.K., in 1910 the average age at death was 59 years, and this rose steadily: 1921, 62 years; 1931, 68 years; 1938, 69; and 73 years in 1959.

The statistical data prompted Campbell (1963) to calculate the expected incidence of deaths from heart disease, with 1876 as the starting point, taking into consideration the declining mortality from infectious diseases. To do this, he assumed (a) that most of the lives saved were young persons, with a mean age of 20 years, and (b) that one quarter of those saved subsequently died from heart disease at the ages when other persons die from this disease (on the average about 40 years later). The calculated death rates from all diseases of the heart showed close correspondence with observed values from 1878 to 1956; the rates would be expected to continue rising until they stabilize about 1990!

Campbell makes the striking observation that "it is not necessary to search for any changed conditions in the environment of the last 40 years as possible causes of the increased death rate from heart disease. It could result entirely from the greatly reduced death rate from all causes in the period 1880-1920." He also questions the validity of the increased proportion of coronary heart disease in
the death rate from all heart diseases, and ascribes this to a shift of diagnosis from other groups to coronary heart disease. He states "the deaths from other diseases of the myocardium diminished enough to account for much of the increase in 1940-1949 and for all of the increase in deaths from coronary disease in 1950-59." He adds: "The cause of coronary heart disease should therefore be searched for mainly on general pathological and biochemical principles, and not specially on any changes in the environment, though these may be of some lesser importance."

All this evidence leads to a tentative conclusion that coronary heart disease is not a "modern disease" and that the basic aetiological factors have been operating in the European and North American communities for a long time - at least a century, and perhaps for several centuries. Much futile argument has ensued from a failure to recognize this historical situation, and from a search for correlations between environmental factors and the rising incidence of deaths from heart disease.

Within the very complex pathological changes involved in coronary heart disease, we are concerned here only with the initial changes that subsequently lead to frank disease. The initial change is hyperlipidaemia or, more precisely, hyperlipoproteinaemia (HLP). It is now recognized that there are at least five forms of primary HLP, which differ in genetic predisposition, in the dietary factors involved, in the blood lipid changes, and in the risk of developing C.H.D.. The dietary factors are intakes of total energy (especially in relation to obesity), total fat, carbohydrate, cholesterol, and ratio of saturated to unsaturated fatty acids in the total fat. Only the last-named factor is of concern in this discussion.

It has been noted above that in most affluent communities there is a strong tendency toward a diet with high fat content and for a large proportion of this fat to be derived from ruminant meats and dairy products. The high degree of saturation of ruminant fats, consequent on the ruminal hydrogenation of fatty acids, is a well-recognized condition, and it is this feature that has led medical authorities to recommend the reduction or elimination of these foods from the diet of persons considered to be at high risk of coronary heart disease.

The epidemiological evidence relating dietary lipids to the aetiology of atheroma receives strong support from experimental work on several species of animals. It is, however, only fair to note that this dietary hypothesis has been challenged by some workers who have incriminated sucrose, lack of fibrous food, the hardness of water supplies,, psychological characteristics, or immune reactions to milk proteins. The difficulties in resolving these conflicting hypotheses are very great. Some of the reasons are:-

(1) Atheromatous lesions in the arteries develop extremely slowly (with some rare exceptions). One must think in terms of years, and even decades.

(2) It is virtually impossible to design long-term experiments on human beings that would be free from criticism. In addition, ethical and financial problems become truly formidable.
From a knowledge of species-specificity in comparative pathology, it can be said that no amount of experimental work on laboratory animals can establish beyond doubt the nature of pathogenesis in man. By one means or another, information on man himself must be obtained.

With these reservations in mind, one can only accept the present overwhelming body of opinion from medical investigators - namely, that the above-quoted dietary factors, operating on a genetic predisposition, lead to the formation of atheromatous lesions, which form the basis for subsequent atherosclerosis, and thus to the various clinical syndromes induced by obstruction of blood flow - in the present context, especially coronary heart disease.

If dietary modifications are to be effective in preventing or reducing the formation of atheromatous lesions, or in aiding the regression of existing lesions, it is evident that the diet must be continuously used over very long periods of time - some authorities would recommend a full lifetime. In turn, it follows that the diet should, if at all possible, be compounded from foodstuffs that are particularly favoured. In view of the long tradition for the use of ruminant meats and dairy products by peoples of Caucasian origin, there is the strongest incentive to find means for modification of these foods to render them medically acceptable to persons considered to be at risk of developing atherosclerosis.

This objective has, in considerable measure, been achieved experimentally. It is now possible to produce ruminant meats and dairy products in which the fat contains greatly elevated concentrations of the essential fatty acids, particularly linoleic acid. It would still be desirable to induce the further modification of reducing the cholesterol content of these foods - this is a task for the future. In spite of this limitation, it is now probable that the modified foodstuffs can be incorporated into diets that are acceptable for consumption over indefinitely long periods and that meet the chemical criteria desired by medical authorities. It will, however, be self-evident that the proof of this statement will have to await the passage of at least another 50 years - and more likely, a century.

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