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Foreword

To most scientists and to all of the general public, heart disease begins and ends with fat and cholesterol. Only those scientists who have done research in the field know that the earliest purely nutritional studies of experimental atherosclerosis were predicated on finding an atherogenic principle in animal protein. The early experiments did point to a connection between food of animal origin and atherosclerosis, but the observation that dietary cholesterol induced arterial lesions overwhelmed the earlier studies. It was assumed that the cholesterol content of the animal protein was responsible for the observed effects.

The obsession with dietary cholesterol exists to this day and the role(s) of animal and vegetable proteins have been investigated for brief periods and are rarely cited. There was a period of interest in the 1920s when Newburgh and Clarkson demonstrated the atherogenicity of animal protein even in the absence of cholesterol. The first comparison of the effects of animal and vegetable proteins was carried out by Meeker and Kesten in 1940, but this observation was not exploited for another two decades. There has been a steady output of papers in this field since the early 1970s but it has remained a trickle in comparison to the cholesterol and fat literature. This situation may change, now that we recognize risk factors such as homocysteine, C-reactive protein and the role of infection, and we realize that the disease goes beyond dietary fat.

In this book, Professor Debry has succinctly summarized and discussed the role that dietary protein may play in the etiology of atherosclerosis and atherosclerotic heart disease. The book is encyclopedic in scope and, remarkably for its size, summarizes virtually all aspects of the protein–atherosclerosis connection.

Data are presented on every animal species available for experimental work. The effects of proteins from different sources and of proteins within each source category are discussed, as are the additional effects of other dietary components (fat, fiber, minerals).

The possibility that levels or ratios of specific amino acids may exert special influences is addressed as well. The possible mechanisms of protein effects (which have still not been clarified) are also summarized. The effects of the nonprotein components of plant protein are also mentioned. Finally, there is a discussion of dietary protein effects in humans. Two of the conditions that play a role in atherosclerosis — hypertension and thrombosis — can also be affected by dietary protein and these, too, are discussed.

In the 1950s when the relationship between dietary fat and atherosclerosis was becoming established epidemiologically, Yerushalmy and Hilleboe used the same epidemiologic data to show that there was a protein effect that was as strong as that of fat. This book provides data that support and expand their observation.

Professor Debry has provided an invaluable source of data concerning dietary protein and atherosclerosis. It is not meant to displace the interest in fat and cholesterol in this disease, but rather to offer another area of interest and investigation that has been generally neglected. This book should become part of the library of every person working in atherosclerosis research or in public health. It will expand the horizons of research workers in the fields of biology and medicine related to atherosclerosis and heart disease. Combining the many new research tools now available with the long-neglected data presented in this volume should yield greater comprehension of the etiology of atherosclerosis and may lead to an earlier solution of this major health problem.

David Kritchevsky
Philadelphia, PA

Preface

Although food lipids are now recognized as the major nutritional factors in atherosclerosis, it is interesting to remember that the first experimental research projects investigating the possible relationships among nutrition, blood lipids, and atherosclerosis studied the effects of animal proteins and meat in particular. Indeed, dietary meat induces various forms of pathologic damage to hepatic tissues in rabbits (Garnier and Simon 1907) and dogs, and also liver necrosis and arterial lesions (D'Amato 1908). The results of these studies on rabbits were confirmed by Ignatowski (1908a, 1908b) and Fahr (1912). Later, investigators assessed the effects of different animal proteins: meat (Lubarsch 1909, 1910) or various associations such as meat, egg yolk, egg white, and milk (Starokadomsky and Ssobolew 1909; Stuckey 1911, 1912). Anitschkow and Chalatow established the cholesterol-fed rabbit as a model for atherosclerosis research (Anitschkow and Chalatow 1913; Finking and Hanke 1997). On the basis of their results, the investigators came to the conclusion that proteins were not the only factors responsible for arterial injuries and that the initially suspected effects of cholesterol should be accepted (Chalatow 1912; Wesselkin 1913; Anitschkow and Chalatow 1913; Wacker and Hueck 1913).

The respective roles of animal proteins and cholesterol already studied (Kon 1913, 1914; Steinbiss 1913; Knack 1915; Newburgh 1919; Newburgh and Squier 1920; Newburgh and Clarkson 1922, 1923a, 1923b; Diecke 1926; Clarkson and Newburgh 1926) are discussed later. A positive correlation has been established between animal protein consumption and serum cholesterol concentrations and the extent of atherosclerosis, despite the absence of cholesterol in the diet (Newburgh and Clarkson 1923a, 1923b). However, according to Diecke, no correlation can be established between atherosclerosis and hypercholesterolemia (Diecke 1926). The arterial hypertension observed in rabbits on a diet including meat could also be the cause of atherosclerotic lesions (Schmidtmann 1926). The prevalence of cardiovascular diseases, as assessed by epidemiologic studies over the same period, is positively and equally correlated with animal protein or fat consumption (Yerushalmy and Hilleboe 1957; Yudkin 1957).

Meeker and Kestens (1940, 1941) demonstrated that in contrast to animal proteins, vegetable proteins do not induce an increase in serum cholesterol concentrations. Atherosclerosis is not observed in rabbits fed maize oil and casein or wheat gluten. Nevertheless, plasma cholesterol concentrations were higher when the rabbits were fed casein rather than wheat gluten (Enselme et al. 1963).

The experiments by Kritchevsky et al. (1959) and Howard et al. (1965) confirmed the results obtained by Meeker and Kestens. When cholesterol was added to the diet of rabbits, the nonpurified soybean protein provided decreased serum cholesterol concentrations, and not only was harmless but prevented atherosclerosis. Two general reviews have summarized the effects of dietary proteins on plasma

cholesterol concentrations and atherosclerosis in animals (Yudkin 1957; Enselme et al. 1962).

In young women, the capacity of vegetable proteins compared to animal proteins to lower plasma cholesterol levels was first observed by Walker et al. (1960). In men, Hodges et al. (1967) showed that vegetable proteins decrease the serum cholesterol concentrations induced by diets containing various proportions of fats and simple or complex carbohydrates. In spite of these results, investigations on the relationship between proteins and atherosclerosis have been replaced by others focusing on the rapid development of the lipidic theory of atherosclerosis.

Nevertheless over the past 30 years, there has been renewed interest in the protein theory (Connor and Connor 1972; Carroll and Hamilton 1975; Hermus 1975; Kritchevsky 1976; Hamilton and Carroll 1976; Debry 1976; Carroll 1978a, 1978b; Kritchevsky 1980a, 1980b; Carroll 1981b; Kritchevsky and Czarnecki 1982, 1983; Laurent 1983, Terpstra et al. 1983a, 1983b; Kritchevsky 1983a, 1983b; Kritchevsky et al. 1983, 1984; Debry et al. 1984; Goldberg and Schonfeld 1985; West and Beynen 1986; Forsythe et al. 1986; Kritchevsky et al. 1987; Kritchevsky and Klurfeld 1987; Debry 1987a, 1987b; Guzman and Strong 1987; Kritchevsky 1987; Foley et al. 1988; West and Beynen 1988; Barth and Pfeuffer 1988; Kritchevsky 1990; Debry 2001a, 2001b). The history of these relationships was reviewed by Kritchevsky and Czarnecki (1983) and Terpstra et al. (1983a, 1983b), and more recently by Kritchevsky (1993, 1995). However, in one general review on diet, plasma cholesterol, and coronary heart disease by Smith and Pickney (1989), none of the 1700 references quoted dietary proteins as a possible factor involved in atherosclerosis, and in a recent review on coronary heart disease risk factors (Ferns and Lamb 2001), the eventual role of proteins is not mentioned.

The results of experiments with varying diet components have shown that serum cholesterol concentrations are positively correlated with animal protein content, and casein in particular. However, according to the authors, these effects could also be due to other diet components (Carroll and Hamilton 1975; Hamilton and Carroll 1974, 1976; Carroll 1978a), and cannot be generalized since the serum cholesterol levels induced by certain animal and vegetable proteins are not significantly different. This is the case, for example, with pork protein or raw egg white, wheat gluten, and peanut protein (Carroll and Hamilton 1975) or with beef and vegetable proteins (Kritchevsky et al. 1981). Moreover, since animal growth depends on the biological value of proteins, this value should be taken into account (Hermus 1975).

Although the results of epidemiologic studies and clinical trials support the notion of a positive correlation between the consumption of dietary proteins and the prevalence of atherosclerosis or coronary thrombosis, this theory cannot be attested with certainty since the collection of data on animal and vegetable protein consumption remains relatively inaccurate in humans. In contrast, the numerous experimental studies carried out in animals of various species as well as in humans have partly identified, although only in a limited number of studies, the pathophysiologic mechanisms of the effects of proteins on atherosclerosis.

The damage caused by some animal proteins and the protective effects of certain vegetable proteins have been clearly demonstrated. Although dietary proteins are considered to be of minor importance in the etiology of hypercholesterolemia and

atherosclerosis in humans (Carroll 1978a; Kris-Etherton et al. 1988), their effects on plasma lipids, various factors of blood coagulation, endocrinologic balance, arterial hypertension, and arterial wall properties have been demonstrated. The effects of nonprotein substances associated with vegetable proteins in plants have been the subjects of numerous studies over recent years. Results have shown that the direct influence of these proteins on plasma lipid levels and the development of atherosclerosis remains unclear and further studies are still required to establish the real effects (Smith 1998).

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The Author

Professor G. Debry is currently Professor Emeritus at the Department of Nutrition at the University Henri Poincaré in Nancy, France. He achieved the degree of M.D. in 1956 and has been a professor of human nutrition and metabolic diseases at University Henri Poincaré since 1967. He founded the Training for Dietitians at French Technological Universität Institutes in 1968 and was the Director of the INSERM Unit of Human Nutrition from 1963 to 1983. In 1984 he founded the Human Nutrition Center of the University Henri Poincaré, which he also managed until 1996. From 1970 to 1994, Professor Debry held a joint appointment at the University Hospital as the head of the Diabetes, Nutrition and Metabolic Diseases and of the Enteral Nutrition Departments.

Professor Debry has served and headed several French ministerial committees, notably, the Ministries of Health, of Research, and of Agriculture. He was an expert in nutrition for the World Health Organization from 1970 to 2000, and has been a corresponding member of the French Academy since 1986 and a member of the European Academy of Nutritional Science since 1988. He has won several research awards from the French Academy of Medicine and the French Academy of Sciences, as well as the Research Prize in Nutrition from the French Nutrition Foundation in 1980 and the André Mayer Award in 1983. He has also received several medals including Officer of the Legion of Honour in 1991.

Professor Debry has written numerous publications, including 37 contributed chapters to edited volumes and nine monographs. He has also served as editor for five International Congresses.

Introduction: Epidemiologic and Clinical Data

EPIDEMIOLOGIC DATA

In some epidemiologic surveys, a strong positive correlation has been observed between the consumption of animal proteins and the incidence of coronary morbidity and mortality (Yudkin 1957; Yerushalmy and Hilleboe 1957; Connor and Connor 1972; Armstrong et al. 1975; Kritchevsky 1976; Stamler 1979; Kritchevsky and Czarnecki 1982; Debry et al. 1984, 1987a, 1987b). Data from epidemiologic studies regarding the relationships between protein consumption, plasma lipids, and atherosclerosis in vegetarians and vegetalians cannot be considered significant since these diets contain few saturated fats and a high quantity of dietary fibers (Hardinge and Stare 1954; Sacks et al. 1975; Burslem et al. 1978). In the same way, the results of several epidemiologic surveys in Western countries cannot be considered significant even though the consumption of animal proteins is high because, in these foods, the proteins are associated with animal fats. In addition, these foods are cooked with animal fats. The nature of the fats associated with the proteins must also be taken into account. As an example, the plasma cholesterol concentrations in the Masaï population who drink considerable quantities of milk (Mann et al 1964) and in Eskimos who consume large quantities of fish fats (Bang et al. 1971) are significantly lower than those of Western populations. Nevertheless, more accurate surveys are still necessary to provide a correct interpretation of these results.

In conclusion, epidemiologic data relating to a possible effect of certain food proteins on the development of atherosclerosis in humans are still inadequate for any conclusive association to be confirmed. This explains the absence of consensus statements in the international literature (Dawber 1980; Bulpitt 1985; Lee et al. 1990).

CLINICAL DATA

The influence of malnutrition and the incidence of cardiovascular diseases with atheroma remain unclear. The consumption of proteins, animal fats, minerals, vitamins, and antioxidant substances are indeed reduced. During starvation the incidence of coronary heart disease and of myocardial infarction is reduced, whereas that of hypertension and congestive heart failure remains unchanged. Plasma cholesterol concentrations do increase during the progression of kwashiorkor but the disease duration is too short, invariably interrupted by death or nutritional rehabilitation, to allow any progression of the atherosclerosis.

Kwashiorkor may be induced by an unbalanced diet containing adequate energy supplies or by a diet of poor protein quality whereas marasmus is induced by either a balanced or an unbalanced diet with inadequate energy supplies (Sidransky 1990). In both of these diseases, plasma lipid concentrations are very likely reduced via a mechanism of reduced protein synthesis that diminishes lipoprotein formation (Truswell and Hansen 1969). Total heparin lipolytic activity is reduced, particularly as a result of reduced hepatic lipase activity in children with kwashiorkor (Agbedana et al. 1979a, 1979b). In infants with kwashiorkor, plasma cholesterol concentrations are restored to normal levels by a low-fat diet enriched with amino acids or various proteins (Schendel and Hansen 1958). These diets have the same effects in undernourished adults (Tripathy et al. 1970). In patients with chronic renal insufficiency, the Kempner diet, which is low in proteins and fats, reduces plasma lipid concentrations (Kempner 1948; Loschiavo et al. 1988; Coggins et al. 1994). However, Olson et al. (1958b) suggested that this reduction may not be due to reduced protein intake alone.

With the exception of the anatomopathologic study by Moore et al. (1981), we have not found any other anatomopathologic work relating to the association between protein consumption and atherosclerosis. In this study, however, the correlations that were established between protein consumption and the extent of atherosclerotic lesions cannot be interpreted since the confounding influence of the consumption of fats is too great.

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