Cholesterol and Coronary Heart Diseases- A Review Article

ABSTRACT

Atherosclerosis is a disease whose causes are multifactorial, and alterations in plasma lipids and lipoproteins are one facet of the problem. A major achievement of research has been to show how the various risk factors interact in atherogenesis. Low density lipoprotein levels in the plasma are highly correlated with levels of plasma cholesterol. The role of LDL-cholesterol for atherosclerosis growth has been exaggerated, a finding with consequences for the prevention of cardiovascular disease. For instance, as the statins exert their beneficial influence on the cardiovascular system by several mechanisms, it may be wiser to search for the lowest effective dose instead of the dose with maximal effect on LDL-cholesterol. While there is considerable evidence that high density lipoprotein levels are negatively correlated with risk, there are as yet no data on the effects of altering these levels in individuals or in populations.

Introduction

Atherosclerosis is a disease whose causes are multifactoral, and alterations in plasma lipids and lipoproteins are one facet of the problem. A major achievement of research has been to show how the various risk factors interact in atherogenesis. To focus solely on lipids and specifically on dietary cholesterol provides a narrow view of the problem. Various factors are cooperative and operate together to produce the disease.

According to the low-density-lipoprotein (LDL) receptor hypothesis, development of atherosclerosis is caused by a high concentration of LDL-cholesterol in the blood, and lowering LDL cholesterol reverses, or at least retards, atherosclerosis, thus preventing cardiovascular disease. As a scientific hypothesis, it is open to falsification: if the correlation of LDL-cholesterol or total cholesterol and the degree of atherosclerosis do not correlate, or if there is no exposure-response, e.g. if there is no association between the cholesterol changes and atherosclerosis progression.

Very low density lipoproteins have been described in the Food and Nutrition Board Report as neutral, but the meaning of neutrality is not clear. High density lipoproteins are now in vogue and are unique in that their levels in the plasma are negatively correlated with coronary heart disease.

Low density lipoprotein levels in the plasma are highly correlated with levels of plasma cholesterol. Since the latter is far easier to measure, most clinical and epidemiologic data are in terms of total cholesterol which, however, reflects low density lipoprotein levels. I have taken data from the Lipid Research Clinics Prevalence Study and plotted low density lipoprotein cholesterol versus plasma cholesterol. The role of LDL-cholesterol for atherosclerosis growth has been exaggerated, a finding with consequences for the prevention of cardiovascular disease. For instance, as the statins exert their beneficial influence on the cardiovascular system by several mechanisms, it may be wiser to search for the lowest effective dose instead of the dose with maximal effect on LDL-cholesterol. While there is considerable evidence that high density lipoprotein levels are negatively correlated with risk, there are as yet no data on the effects of altering these levels in individuals or in populations.

Key Words

Atherosclerosis, Cholesterol, Low density lipoprotein

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through the liver, although not necessarily by the high affinity receptor mechanism. These studies clearly demonstrate that a genetic defect in receptor production leads to elevated plasma low density lipoprotein levels.

It has already been pointed out that low density lipoprotein levels are affected by levels of saturated fatty acids in the diet so that reducing saturated fatty acids in the diet will lead to reduction of low density lipoproteins. In the Indians studied by Dr. Connor in Mexico, he found a good correlation between dietary cholesterol and plasma cholesterol. It is pointed out that these subjects ate diets with a high P:S ratio, and it could well be argued that this may not be applicable to the American population. Dr. Ahrens and his associates demonstrated clearly that substitution of polyunsaturated fat for saturated fat in the diet lowered serum cholesterol. Substitution of coconut oil, which does not contain saturated fatty acids, causes the cholesterol level to return to baseline levels and corn oil caused cholesterol to decrease. This is an unequivocal proof that by altering the diet we can change levels of low density lipoproteins. Studies have been performed in free living Scandinavian populations on a diet low in saturated fat and high in polyunsaturated fatty acids. In almost all instances, serum cholesterol levels in these individuals fell.

There is considerable disagreement as to the significance of very low density lipoprotein as a risk factor. Epidemiologists using multivariate analysis state that hypertriglyceridemia is not a primary risk factor, and some do not believe it useful to measure plasma triglycerides. However, it has long been known that patients with coronary heart disease have, on average, higher levels of triglyceride. Thus, in the study of Goldstein et al the patients with coronary heart disease had higher levels of triglycerides than did the control subjects. In some populations, such as in Sweden, prospective studies have shown a high correlation between plasma triglyceride concentrations and the incidence of myocardial infarction.

Conclusion
‘The more LDL there is in the blood, the more rapidly atherosclerosis develops.’ Low density lipoprotein is clearly a major risk factor for coronary heart disease. There are less definitive data regarding the role of very low density lipoprotein, but it does have a strong association with coronary heart disease.