

Effect of Dietary Cholesterol and Cholesterol Oxides on Blood Cholesterol and Lipids, and the Development of Atherosclerosis in Rabbits

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Cholesterol is an important component in human body, but is also considered as a major risk factor related to the development of atherosclerosis and coronary heart disease (CHD). Literature on atherosclerosis and CHD is dominated by two compelling hypotheses, the "lipid hypothesis" and the "response-to-injury hypothesis". Cholesterol plays a pivotal role in the lipid hypothesis, whereas cell damages by oxidized cholesterol initiate atherosclerosis and CHD in the response-to-injury theory. The lipid hypothesis suggests that atherosclerosis and CHD are caused by hypercholesterolemia-induced deposition of lipid in vessel wall. The objectives of this study were to determine the effect of dietary cholesterol and cholesterol oxides on the development of atherosclerotic lesions, and changes in fatty acid and blood characteristics in rabbits. Two studies were conducted to determine the effects of dietary cholesterol (CHO) and cholesterol oxides (COPs) on the development of atherosclerosis, and the changes in fatty acid and blood characteristics in rabbits. In the first study, a total of 40 male New Zealand White rabbits were divided into 5 groups and fed commercial rabbit chow added with none, 1g CHO, 0.9 g CHO+0.1 g COPs, 0.8 g CHO+0.2 g COPs, or 0.5 g CHO+0.5 g COPs per kg diet. In the second study, a total of 24 male New Zealand White rabbits were divided into 3 groups and fed a diet containing 2 g CHO, 1.6 g CHO+0.4 g COPs, or 1.2 g CHO+0.8 g COPs per kg diet. All diets except for control diet induced atherosclerotic lesions in aorta of rabbits. The contents of total cholesterol and LDL-cholesterol in plasma ($P < 0.05$) increased significantly with dietary cholesterol, but the presence of dietary COPs at 20% or higher of total dietary cholesterol (CHO+COPs) dramatically reduced them. The level of plasma triglyceride also increased with high-level cholesterol (Study 2) treatments, but COPs reduced it. The amount of plasma HDL-cholesterol increased with age, but was not influenced by dietary CHO or COPs. The ratio of saturated fatty acid (SFA) to unsaturated fatty acid (USFA) decreased as the amount of dietary total cholesterol (CHO + COPs) increased. The results suggested that the presence of COPs in diet inhibited the absorption of cholesterol in the guts and, thus, dietary COPs had less detrimental effect on the development of atherosclerosis than CHO in rabbits.