My cholesterols have always been normal, so you can imagine my surprise when I was told I had some plaque in my carotid arteries. How can I raise my HDL and lower my LDL—or, perhaps more important, does it really make much difference?

The cholesterol story is a long one, and though we have come a long way in our understanding, we can say unequivocally that there is a lot more to learn.

The cholesterol story began with concern with total cholesterol. We remember well the unraveling of cholesterol into the various categories of low-density, lipoprotein-bound cholesterol; high-density, lipoprotein-bound; ultra-low; and so forth.

Today, most laypeople know high density, lipoprotein (HDL)-bound cholesterols are “Healthy” or “Happy”; and low-density, lipoprotein (LDL)-bound cholesterols are “Lethal” or “Lousy”—but there is far more to it.

The ratio between these two is somewhat predictive of atheroma, but often more cases exist for which we just have no explanation. For about one third of individuals, sudden death is the first manifestation of heart disease. Obviously, we need better predictors of coronary heart disease that can be used—cost-effectively—in screening large populations.

HDL and LDL continue to be useful indicators, though not definitive. More than just the buildup of plaque in the arteries can cause heart attacks. Usually a “rupture,” or breakup of the plaque, precipitates the attack.

Much attention is being given to HDL. We know exercise raises HDL. Fiber from grains such as oats may lower LDL. Cholesterol-lowering medications reduce the risk of heart attack. Smoking more than doubles the risk.

Recently, a trial of a medication (torcetrapib) that raised HDL was abandoned after millions of dollars were spent on its development because Pfizer, Inc., a research-based pharmaceutical company, detected an increase in deaths and heart problems. Why would an elevated HDL be associated with such problems?

Research is now probing the complex nature of HDL. By breaking it into smaller fragments, numerous functions are being discovered. Not only may it wash LDL out of cells and plaque, but it may have anti-inflammatory effects. Besides its lipid metabolic effects, researchers have found it has an enzyme that may block the breakup of plaque (protease inhibitors). Additionally, it has regulatory enzymes involved in the inflammation reaction that may modify the response in the heart to
dying muscle.

Differences between the HDL of healthy people and those who have heart attacks were also found. A protein (ApoE) involved in lipoprotein transport was found in higher concentrations in patients than healthy controls.

We still have a lot to learn. It seems our understanding of these cholesterols is just beginning. The more deeply the complexity of creation is explored, the more reverent we become in the presence of such an awesome Creator. We will be wise to live as naturally as we can, and to walk humbly before such majesty.

Allan R. Handysides, M.B., Ch.B., F.R.C.P.(c), is director of the General Conference Health Ministries Department; Peter N. Landless, M.B., B.Ch., M.Med., F.C.P.(SA), F.A.C.C., is ICPA executive director and associate director of Health Ministries.

While this column is provided as a service to our readers, Drs. Landless and Handysides unfortunately cannot enter into personal and private communication with our readers. We recommend that you consult with your personal physician on all matters of your health.