No physical activity or exercise is not an option

The incidence of coronary artery disease (CAD) increases with increasing plasma low-density lipoprotein-cholesterol concentrations and decreases with increasing high-density lipoprotein-cholesterol (HDL-C) concentrations. Plasma triglycerides are an accepted risk factor for peripheral vascular disease and for CAD in women and for individuals with diabetes, but in the past, plasma triglycerides were believed to add little to the overall prediction of CAD risk. Nonetheless, a recent meta-analysis of 17 studies provides strong evidence that plasma triglyceride levels are an independent CAD risk factor (6). After adjusting for other risk factors, including HDL-C, an 88 mg/dl increase in plasma triglyceride levels was associated with a 14% increase in CAD risk for men and a 37% increase for women (3).

There is strong agreement that physical activity lowers the risk of CAD and that part of this risk reduction involves positive changes in plasma lipids and lipoproteins (5). Presently, many factors are known to influence lipid and lipoprotein synthesis and catabolism by altering the interactions between lipids, lipoproteins, apolipoproteins, and the enzymes associated with lipoprotein metabolism. Although in recent years much has been learned regarding the impact of exercise on plasma lipids and lipoprotein levels, the mechanisms responsible for these changes in lipid and lipoprotein metabolism and their interaction with genetic and environmental factors are not completely understood (2).

Factors such as aging, body fat distribution, dietary composition, and cigarette smoking status are known factors that modify lipoprotein metabolism and composition (6). For example, intervention programs that reduce fat and increase carbohydrate dietary intake while increasing daily physical activity positively influence plasma lipid and lipoprotein profiles and reduce CAD risk. Because of continued scientific evaluation, we better understand the positive impact that exercise training has on the lipid and lipoprotein profile, and in recent years we have gained a greater appreciation for the necessary amount or volume of exercise needed to cause these changes (8, 9). In addition, intermittent exercise or several short exercise sessions performed on a single day are known to positively change plasma lipid and lipoprotein levels (1). One recent publication reported lower postprandial plasma triglycerides and attenuated postprandial lipemia following acute exercise (1).

Besides regularly practiced physical activity, a single exercise session can also positively alter plasma lipid and lipoprotein profiles. Furthermore, these effects are different for sedentary and trained subjects and can last up to 3 days after the exercise. Crouse et al. (4) and Visich et al. (12) both reported that in sedentary subjects, lipid and lipoprotein changes can occur after a single exercise session when one exercises at least 350 kcal. On the other hand, Ferguson et al. (8) reported that a single exercise session with an energy expenditure of at least 1,100 kcal was necessary to improve HDL-C levels in trained subjects.

The study by Slentz and colleagues (11) in the Journal of Applied Physiology focuses not only on these lipid, lipoprotein, and exercise training relationships but considers detraining or the cessation of exercise. The present study is part of a larger project titled Studies of a Targeted Risk Reduction Intervention Through Defined Exercise (STRRIDE) project, which previously reported that exercise volume rather than exercise intensity is a more important consideration for positively enhancing plasma lipid and lipoprotein concentrations (9). The present study by Slentz et al. (11) builds on these earlier findings by assessing the impact of the cessation of exercise training and finds important information that adds to the ever-expanding list of the ill effects of being physically inactive.

The most important findings of this study are the results relative to plasma lipid and lipoprotein changes during a 15-day detraining period. Subjects were divided into three exercise groups based on exercise volume and exercise intensity, with a fourth group as a control. After 6 mo of exercise training, all exercise groups improved their HDL profile (HDL-C concentrations, large HDL, and HDL size). This improved HDL-C profile was then maintained during a 15-day detraining period. This finding is important because HDL possesses many antiatherogenic properties that are postulated as having anti-inflammatory effects (10). These positive lipid and lipoprotein changes were maintained for 15 days with no exercise.

Total plasma triglycerides and the triglyceride associated with the very low density lipoprotein particles were also reduced in all groups after exercise training. However, these levels remained lower after 15 days only in the low-volume, moderate-intensity exercise group. Thus, when exercising only at lower volumes and at moderate intensities, these beneficial effects still existed after detraining. This information may prove an important finding because individuals starting an exercise program who start with a low volume and exercise at a moderate intensity may be more likely to remain physically active. Again, the single most important aspect of these findings is that the beneficial effects of exercise training on plasma lipids and lipoproteins is maintained for up to 15 days. Thus no physical activity or not exercising is not an option.

Current treatment guidelines for the management of plasma lipids and lipoproteins have been provided by the National Cholesterol Education Program Adult Treatment Panel II (NCEP) (7). Dietary modification, weight loss, and exercise are recommended as initial therapies for at least 6 wk. However, when these strategies are not successful, present guidelines recommend pharmacological intervention as primary therapy for reducing plasma lipid and lipoprotein levels, with diet, exercise, and weight loss interventions as secondary but important adjunct therapies (7). Clinical approaches presently use a combination of these therapies in conjunction with an individual risk assessment developed from the Framingham Study data to enhance patient success (13).

Future studies that examine exercise and other lifestyle changes can benefit from the knowledge obtained from the study of Slentz et al. (11). These findings will help in the development of exercise programming options that can optimize lipid and lipoprotein changes in conjunction with lifestyle programming. Again, the important finding from this study is that moderate-intensity exercise regimens can have a positive impact on lipids and lipoprotein profile and that these benefits are maintained for 15 days after stopping the exercise. Mod-
erate-intensity exercise programming is much more tolerable for individuals who have been physically inactive and can aid in optimizing blood lipid and lipoproteins especially in these individuals.

The study by Slentz et al. (11) has several strengths, including that it was part of the STRRIDE project, which is a nationally recognized, multicenter, randomized controlled study investigating different volumes and intensities for exercise training. The study had a large sample size, which is a tremendous improvement over past studies having small sample sizes. Also, all measurements were closely monitored and clearly documented. This study reported results that add to the list of negative effects related to physical inactivity. Finally, the exercise training volumes in this study are manageable for most physically inactive or sedentary persons who are starting an exercise program and can lead to favorable benefits that are maintained after 15 days of detraining.

In the future, large, randomized, controlled studies evaluating the combined effects of medications and exercise would provide valuable information not presently available to evaluate the interaction between lipid-lowering agents and exercise. Data in this area would prove most helpful in designing exercise and medication interventions to optimize plasma lipid and lipoprotein concentrations.

REFERENCES