

Atherosclerosis Update

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Physical activity and the risk of coronary heart disease

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Physically active people have less risk of coronary heart disease (CHD) than sedentary ones.¹⁻³ Nevertheless, the respective contributions of genetic predisposition for and level of physical activity to the difference in CHD risk are not known. Possibly some people are more active because their constitutional and metabolic characteristics allow them to exercise comfortably. However, it has not been determined if increased physical activity in sedentary people reduces the risk of CHD. Such a study would be enormously costly and require thousands of subjects.

However, the associations between plasma lipoprotein levels and CHD⁴ strongly suggest that these levels are important risk factors for CHD. Thus, the highly favourable plasma lipoprotein profile of endurance-trained runners⁵ has stimulated interest in physical activity as a means of reducing CHD risk. It is well known that increased physical activity lowers the plasma triglyceride and very-low-density lipoprotein cholesterol levels and raises the high-density lipoprotein cholesterol levels.^{6,7} Prolonged low-intensity physical activity seems to induce greater changes than short, high-intensity exercise, apparently though changes in enzymatic activity associated with lipoprotein metabolism.^{6,7}

Although some subjects are highly sensitive to exercise training, others show very little change in their lipoprotein levels.⁸ Since our study sample comprised monozygotic twins, we concluded that this heterogeneous response reflected different genotypes. A major task will be to identify early those genotypes that are resistant to changes in lifestyle and may require more stringent treatment.

Also, we recently observed that sedentary, moderately obese young men subjected to prolonged low-intensity exercise (2 hours per day for

100 days) had a substantial weight loss and significant changes in plasma lipoprotein levels, including an increase in the high-density lipoprotein cholesterol concentration, although the maximal oxygen consumption was not significantly increased. Thus, the effect on plasma lipid transport — and, presumably, CHD risk — was independent of the training-related change in physical fitness (unpublished observations); according to Wood and colleagues' findings,⁹ much of that effect would have been due to the weight loss. The recommendations of the Canadian Consensus Conference on Cholesterol⁴ for enough physical activity to maintain cardiovascular fitness and for adjustment of the balance between energy intake and expenditure so as to maintain an acceptable body weight are therefore justified.

References

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