

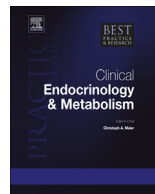


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Impact of diet and exercise on lipid management in the modern era



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Unfortunately, many patients as well as the medical community, continue to rely on coronary revascularization procedures and cardioprotective medications as a *first-line strategy* to stabilize or favorably modify established risk factors and the course of coronary artery disease. However, these therapies do not address the root of the problem, that is, the most proximal risk factors for heart disease, including unhealthy dietary practices, physical inactivity, and cigarette smoking. We argue that more emphasis must be placed on novel approaches to embrace current primary and secondary prevention guidelines, which requires attacking conventional risk factors and their underlying environmental causes. The impact of lifestyle on the risk of cardiovascular disease has been well established in clinical trials, but these results are often overlooked and underemphasized. Considerable data also strongly support the role of lifestyle intervention to improve glucose and insulin homeostasis, as well as physical inactivity and/or low aerobic fitness. Accordingly, intensive diet and exercise interventions

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can be highly effective in facilitating coronary risk reduction, complementing and enhancing medications, and in some instances, even outperforming drug therapy.

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Numerous studies have now shown that acute myocardial infarctions (AMI) often evolve from plaque rupture with thrombus formation, generally at mild-to-moderate coronary artery stenosis [1,2]. Fortunately, the plaque components responsible for vulnerability are amenable to prophylactic intervention via intensive coronary risk factor modification. Accordingly, the soft lipid-rich component appears to be more tractable, with greater potential to regress under intensive risk factor modification than the preponderant calcium-collagenous component of atherosclerotic plaque. This presumably occurs by defusing triggers of arterial inflammation, improving endothelial function, or both [3], which may account for the early reduction in clinical cardiac events observed in contemporary cholesterol lowering trials [4]. Regardless of the precise mechanisms, it appears that intensive risk factor modification may stabilize plaques, leaving them less likely to rupture. Collectively, these findings suggest a new paradigm for preventing and managing coronary heart disease (CHD) (Table 1) [5].

Preventing or favorably modifying traditional and nontraditional (emerging) risk factors through a combination of lifestyle interventions and pharmacologic therapy, complemented by other public laws or policies to further reduce the burden of CHD (e.g., taxes on sugary beverages, bans on trans fats, smoking bans, and regulated decrements in salt content of processed foods), are now widely recognized as the cornerstone of initiatives aimed at the primordial, primary and secondary prevention of CHD [6,7]. Although many patients, as well as the medical community, continue to rely on costly coronary revascularization procedures and/or medications as a first-line strategy to stabilize or favorably modify established risk factors and the course of CHD, these interventions do not address the root or foundational causes, including poor dietary habits, physical inactivity, and cigarette smoking (Fig. 1) [6,7]. Notably, these unhealthy lifestyle practices strongly influence not only blood pressure, lipid/lipoprotein levels, triglycerides, and glucose-insulin homeostasis, but also nontraditional risk indices such as endothelial function, oxidative stress, inflammation (e.g., C-reactive protein), thrombosis/coagulation, and other modulators of acute cardiovascular events (e.g., secondhand smoke, psychosocial stressors, air pollution) [6].

This review provides a compendium of important advances in our understanding of the impact of structured exercise/lifestyle physical activity and/or aggressive dietary modification on lipid/lipoprotein management in the modern era, with specific reference to potential mechanisms of action, associated risk factor reductions, research-based counseling strategies to facilitate lifestyle change, and the independent and additive role that exercise and diet play in complementing the pharmacologic treatment of dyslipidemias.

Impact of physical activity/structured exercise on lipid/lipoprotein levels

The impact of physical activity and regular exercise participation on plasma lipid/lipoprotein levels has been more clearly defined in regard to the interactions between lipids, lipoproteins, apolipoproteins (apo), lipoprotein enzymes, and various genetic and environmental modulators such as aging, body fat distribution, dietary composition, and cigarette smoking [8–10]. This section summarizes present information regarding the impact of exercise training/physical activity on lipid/lipoprotein metabolism.

Endurance exercise training

Lower exercise-induced plasma triglyceride (TG) concentrations are widely reported, with exceptions in some cross-sectional and longitudinal exercise training studies [11,12]. Significant plasma TG reductions are noted after exercise training among previously inactive individuals who have higher baseline concentrations [12,13], whereas more modest TG decreases are observed in subjects having

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