

Impact of Diet, Drugs and Lifestyle on Coronary Artery Disease

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ABSTRACT

Coronary artery disease develops when the major blood vessels that supply heart become damaged or diseased. Cholesterol-containing deposits (plaques) in your coronary arteries and inflammation are usually to blame for coronary artery disease. Once the inner wall of an artery is damaged, fatty deposits (plaque) made of cholesterol and other cellular waste products tend to collect at the site of injury.

KEYWORDS: Coronary Artery Disease; Atherosclerosis; Blood Cells; LDL Cholesterol

Coronary artery disease develops when the major blood vessels that supply heart become damaged or diseased. Cholesterol-containing deposits (plaques) in your coronary arteries and inflammation are usually to blame for coronary artery disease. Once the inner wall of an artery is damaged, fatty deposits (plaque) made of cholesterol and other cellular waste products tend to collect at the site of injury. This process is called atherosclerosis. If the plaque surface breaks or ruptures, blood cells called platelets clump together at the site to try to repair the artery. This clump can blockage of the artery.

The coronary arteries supply blood, oxygen and nutrients to your heart. A buildup of plaque can narrow these arteries, decreasing blood flow to heart. Eventually, the reduced blood flow may cause chest pain (angina), shortness of breath, or other coronary artery disease signs and symptoms. A complete blockage can cause a heart attack.

Since the introduction of the Inflammation and Heart Disease and Angina, Theories in the mid-1990s, a transition from a cholesterol only etiology for coronary artery disease (CAD) has taken place, resulting in understanding that CAD is the result of an Inflammatory process precipitated by a number of factors-including, but not limited to LDL cholesterol and saturated fat [1-5], which impair coronary blood flow.

Unfortunately, hundreds if not thousands of research studies-involving millions of dollars in vested funding – have focused on measuring changes in weight and blood tests [6,7] rather than measuring actual changes in CAD itself [8]. Even the few dietary studies, that actually measured changes in CAD, provided only semi-quantified results that are based upon the limited quantification methods available at the time of the studies.

The consequence has been a fusion of misinformation, fueled by opposing factions of scientists and pseudo-scientists resembling more of a schoolyard brawl than an actual scientific search for the truth. From this, physicians, the media, and social scientific neophytes have vied for attention in support of their positions – demonstrating more social consternation than scientific discourse [9].

Consequently, fundamental questions regarding the impact of diet, drug, and lifestyle treatment effects on CAD remain poorly addressed. This is due to the lack of actual measured effect these diets, drugs and lifestyle have on CAD itself [10]. The concept of confusion about what constitutes true quantification has further confounded investigations of the impact of diet and drug treatments for both primary and secondary prevention and treatment of CAD. Thus the role diet, drugs and lifestyle plays a major role in preventing and treating CAD remains nothing more than a social media fight [11].

The first step to solving any problem is recognizing there is one. To date, studies looking at the primary or secondary prevention of CAD using dietary, drug, or lifestyle intervention, have been severely limited by the absence of quantified measurements of the changes in CAD following treatment intervention [12]. Measurements of blood tests and weight do NOT and have NOT answered this question-resulting in a need for new studies to be done to address these deficiencies and answer the fundamental question of what impact various diets, drugs and lifestyle interventions truly have on CAD.

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