



MEDICAL GROUP



JOURNAL OF
Cardiovascular Medicine and Cardiology

ISSN: 2455-2976

DOI: <https://dx.doi.org/10.17352/jcmc>

Short Communication

Recognizing the fundamental flaw in our dietary studies investigating the impact diets, drugs and lifestyle have on preventing or reversing CAD

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Received: 23 December, 2019

Accepted: 20 February, 2020

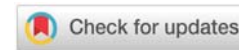
Published: 21 February, 2020

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Keywords: FMTVDM; Inflammation and heart disease theory; Angina theory; Primary prevention CAD; Secondary CAD treatment; Food fight; Heart disease; Cancer; LowCarb; KETO; Vegan; Ornish; Statins; PCSK9-Inhibitors

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Since the introduction of the (a) Inflammation and Heart Disease, and (b) Angina, Theories in the mid-1990s, a transition from a cholesterol only etiology for coronary artery disease (CAD) has taken place; resulting in an 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 [8-10]. Even the few dietary studies, which actually measured changes in CAD [11-14], provided only semi-quantified results – based upon the limited quantification methods available at the time of the studies [10].

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.

Consequently, fundamental questions regarding the impact of diet, drug, and lifestyle treatment effects on CAD [15-18] remain poorly addressed, due to the lack of actual measured effect these diets, drugs and lifestyle have on CAD itself [8-10]. 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 [10,19]. Thus the role diet, drugs and lifestyle play in preventing and treating CAD remains nothing more than a social media fight [20-23].

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 [9,10]. 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 [16,18] (Figures 1,2).

Acknowledgments

FMTVDM issued to first author. Inflammation and Heart

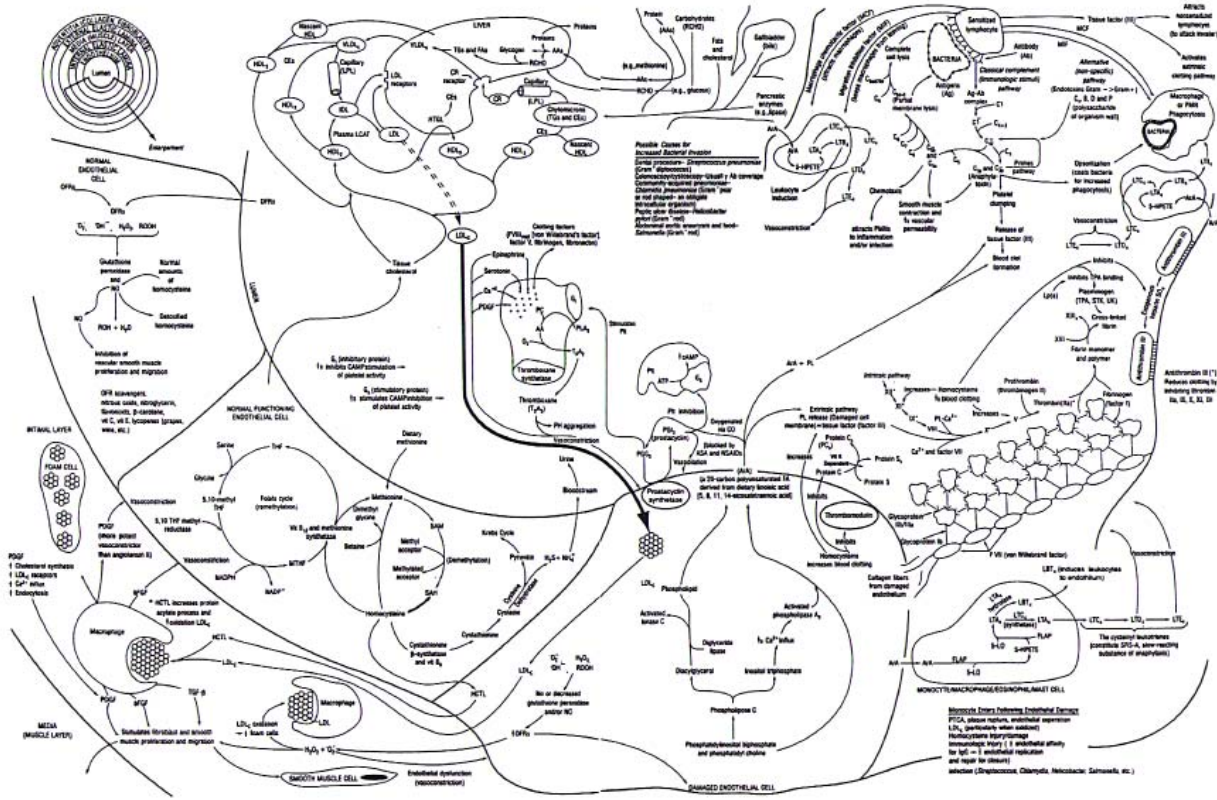


Figure 1: Coronary artery disease is an inflammatory process precipitated by more than a dozen variables. Each variable contributes to inflammation within the blood vessels of the body, including the coronary arteries to varying degrees in different individuals [1].

Blood Flow Image Change vs Blood Chemistry Changes

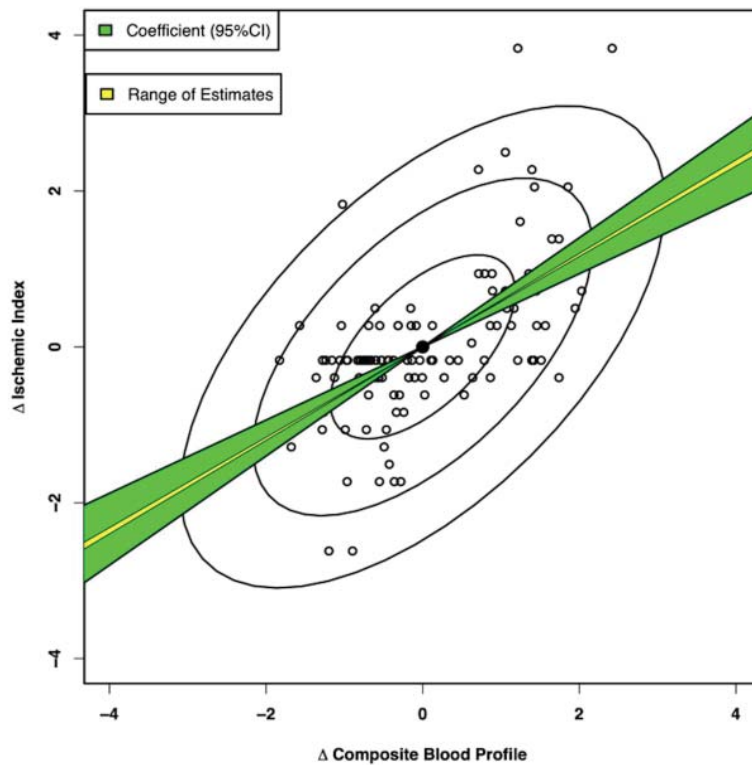


Figure 2: The X-axis displays the composite blood profile including TC, fat, low HDL, IL-6, Lp, and Fib. The Y-axis displays changes in ischemia as measured by semi-quantitative nuclear imaging. The standard regression analysis shows both the range of estimates (yellow) and the 95% confidence intervals (green). HDL, high-density lipoprotein; IL-6, interleukin-6; Lp-a, lipoprotein-a; Fib, fibrinogen; Tc, total cholesterol [8].



Disease and Angina Theories authored by first author. Figures expressly reproduced with permission of first author.

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Citation: Fleming RM, Fleming MR, Chaudhuri TK (2020) Recognizing the fundamental flaw in our dietary studies investigating the impact diets, drugs and lifestyle have on preventing or reversing CAD. J Cardiovasc Med Cardiol 7(1): 036-038. DOI: <https://dx.doi.org/10.17352/2455-2976.000109>