

Short Communication

The First Step to Solving any Problem is Recognizing there is one!

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Since the introduction of the Inflammation and Heart Disease Theory [1–3], a shift from a cholesterol only etiology for coronary artery disease (CAD) has occurred. Unfortunately, hundreds if not thousands of research studies – involving millions of dollars in vested funding – have focused on measuring changes in blood tests rather than measuring actual changes in CAD itself [4,5].

The consequence has been an amalgam of misinformation fueled by opposing factions of scientists and pseudo-scientists resembling more of a schoolyard brawl than scientific search for the truth. From this brawl both the media and social scientific neophytes vie for attention – a demonstration of true social desperation and not scientific discourse.

Fundamental questions about the impact of diet and drug treatment [6–9] remain poorly addressed due to this deeply flawed approach – thus the role diet and lifestyle play in preventing CAD remain unanswered.

While we have some information about what may be happening to people [8] who change their diets – the truth is we do not know and we will not know until we decide to scientifically **any** the question by measuring [5–7] the changes in CAD, which diets and medications [9] have on people with and without underlying CAD. The first step to solving this question of dietary epidemiology and prevention of CAD is to recognize we haven't been measuring the problem - CAD – itself. The first step to solving any problem is recognizing there is one.

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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].

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 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 [4].

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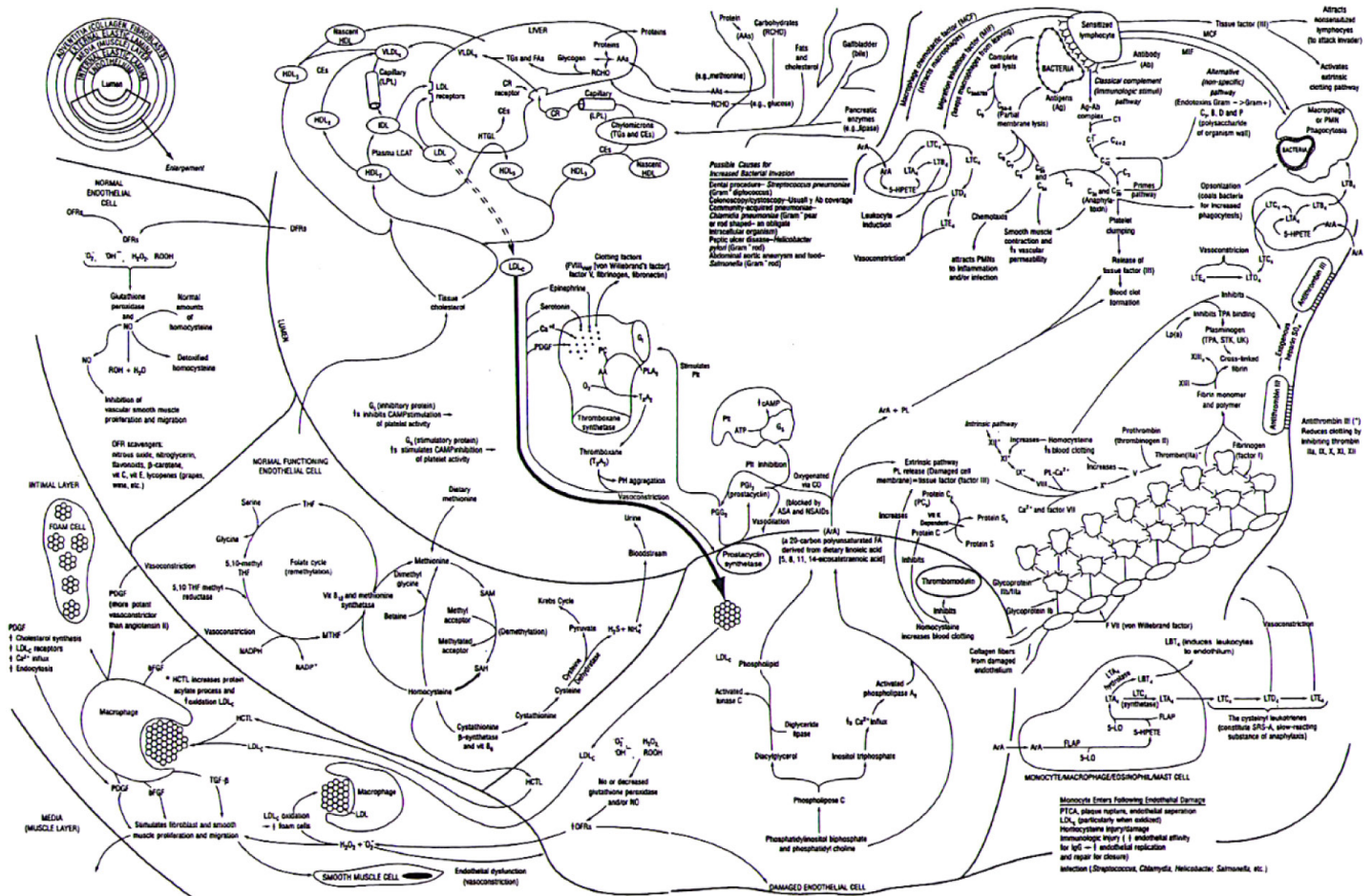


Figure 1. Fleming Inflammation and Vascular Disease Theory [2].

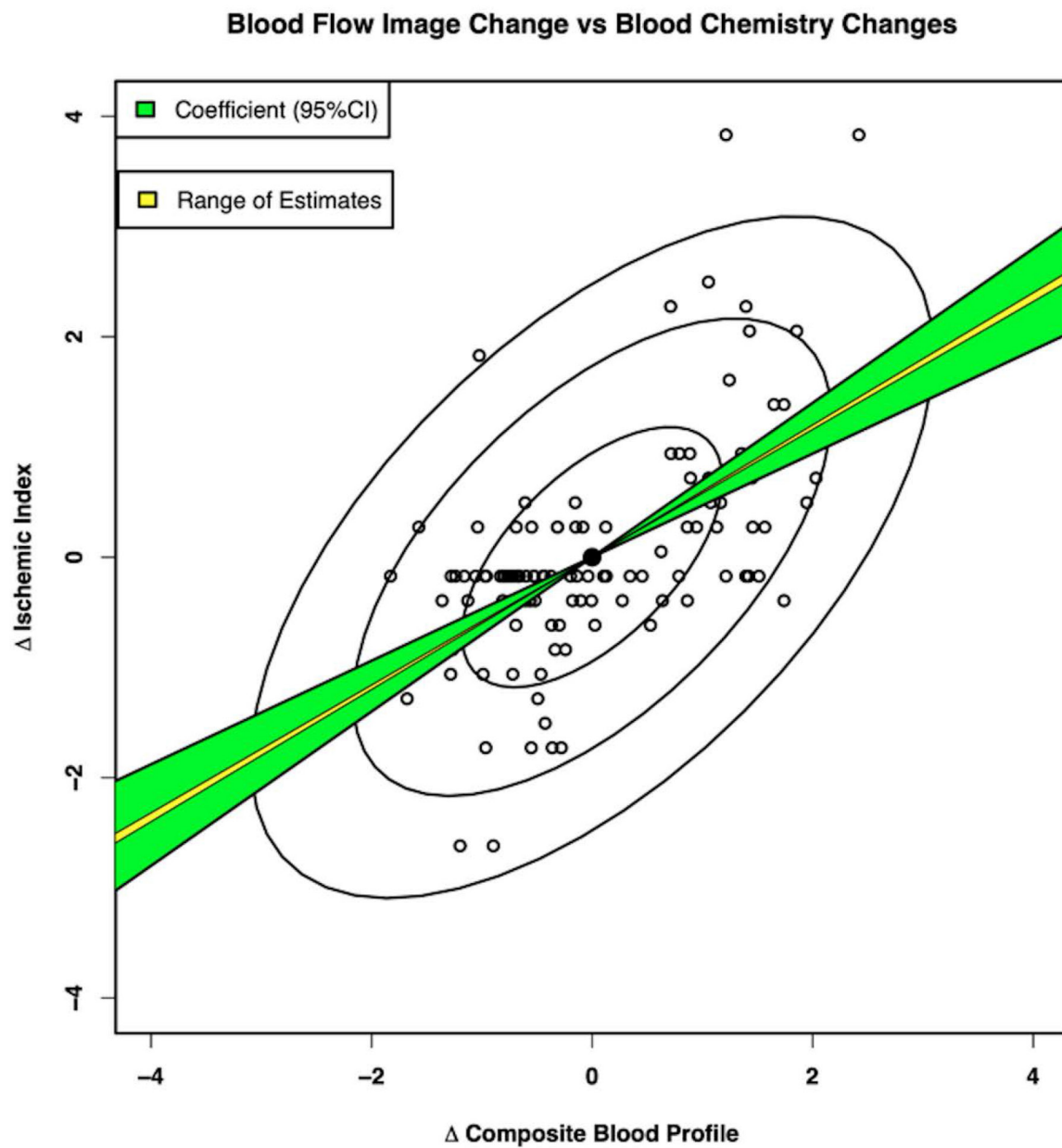


Figure 2. Standardized regression of coronary blood flow on composite blood profile [6].