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Fat or fiction: the diet-heart hypothesis

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Setting the Record Straight on Saturated Fat and LDL-cholesterol. Ignorance is not bliss.

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Like so many purported pundits, DuBroff R, de Lorgeril M [1] have attempted to dispute the significance of the role of saturated fat (triglycerides) and LDL-cholesterol in the development of coronary artery disease, while noting the importance of inflammation itself [1,2]. In law, ignorance of the law is not a defense - the same is true for medicine. Not understanding something does not make you an expert [2] and it does not make your argument valid. Appealing to the court of public opinion does not make it so either. Accordingly, we present a brief explanation of why the authors [1,2] – and others – have presented an invalid discussion of the role fat and LDL-cholesterol plays in coronary artery disease.

In the mid-1990s, as one of the reviewers for the American Heart Association, the first author of this letter, Dr Richard M Fleming (RMF) introduced a then controversial theory stating that Coronary Artery Disease (CAD) is the result of an inflammatory process, which builds up within the walls of the arteries impairing their ability to dilate and increase coronary blood flow when needed; thus producing regional blood flow differences resulting in angina [3-6] and ultimately myocardial infarction (MI) and death.

In recent years, people promoting various dietary and lifestyle practices – particularly those promoting LowCarb-Keto diets, have used the obesity epidemic to focus attention on obesity and weight loss. These same individuals have not demonstrated the actual impact their diets have on CAD, anymore than BigPharma has by reporting changes in lipid levels using their drugs. To demonstrate such change in CAD - either by drug or dietary intervention - requires more than the mere showing of changes in weight or serum blood tests as discussed infra. It requires the actual measurement of the changes occurring within the walls of the coronary arteries themselves – not some other artery – where the

actual inflammation and resulting change in coronary artery function exists [3,6].

The arguments presented by DuBroff [1] and Ravnskov [2] erroneously use studies measuring lipid and inflammatory surrogate markers - blood tests - to support their position, while others [7] use this same approach to support their dietary recommendations by showing weight loss, and occasionally reductions in cholesterol levels – at least initially in some people. As more studies have been done, it has been shown that these initial reductions in lipid levels either do not occur for everyone or are followed by a subsequent increase. This has forced the purported diet pundits to support the position that LDL-cholesterol and saturated (triglycerides) fat do not clog the (coronary or other) arteries [7].

For the authors [1,2,7] to declare that saturated fat and LDL-cholesterol have nothing to do with the development of inflammatory CAD demonstrates a complete failure to understand the “Inflammation and Heart Disease” Theory [5,8], or a failure to have read it, and therefore cannot be taken seriously.

The specific claims made by Malhotra [7] introduces yet another major misconception into the discussion of CAD. Specifically, the process of “clogging of the coronary arteries.” The narrowing or “clogging” of the coronary artery lumen – where the blood flows - so frequently referred to as CAD, is actually a late process in the development of the inflammatory changes that are CAD [9-15].

CAD begins with an inflammatory process, which first distends the wall of the artery outward away from the lumen – impairing the function of the artery - and only later encroaches upon the coronary lumen itself [3,5,9]. Recognition that the rupture of this inflammatory process may occur following minimal or no coronary lumen narrowing [3-5,9] has resulted in the recent acknowledgement by the Cardiology community that infarction of myocardium may occur with (Type I) or without (TYPE II) coronary lumen obstruction.

Fleming and Harrington’s research published in 2008 [16] demonstrated that the relationship between weight loss, and changes in lipids and other blood tests reflecting inflammatory processes [5], are only mildly-to-moderately correlated with actual changes occurring within the coronary arteries themselves. Thus further exposing the erroneous artery “clogging” statement - using the results of blood tests – to declare that saturated fat and cholesterol are not involved in CAD.

To understand the impact LowCarb-Keto diets - or for that matter any diet or drug treatment - has on CAD, one needs to measure what is actually happening to the coronary arteries themselves [17-19]; quantitatively now made possible using FMTVDM [6].

To state that Saturated fat and LDL-cholesterol has nothing to do with CAD and do not result in the “clogging” of coronary arteries or CAD itself, and then to state that CAD is a chronic inflammatory condition - raises serious concerns about the motivation and integrity of their arguments. It also raises serious questions about their actual understanding of the “Inflammation and Heart Disease” and “Angina” Theories [3,5,20]. Ignorance is not bliss - “we can teach it to you, but we cannot understand it for you.”

We are sadly reminded of these words from Billy Madison:

“Mr. Madison, what you just said is one of the most insanely idiotic things I have ever heard. At no point in your rambling incoherent response were you even close to anything that could be considered a rational thought. Everyone in this room is now dumber for having listened to it. I award you no points and may God have mercy on your soul.”

Acknowledged potential COI: FMTVDM (The Fleming Method for Tissue and Vascular Differentiation and Metabolism) [6] is issued to the first author. The first author authored the “Inflammation and Heart Disease” and “Angina” Theories.

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Conflict of Interest:

FMTVDM (The Fleming Method for Tissue and Vascular Differentiation and Metabolism) [6] is issued to the first author. The first author authored the "Inflammation and Heart Disease" and "Angina" Theories.

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