Cholesterol Insights: A New, 

Life-Systems Engineering Science Discovery

**Brief History:** I thank my colleague, EFA/eicosanoid specialist Paul Beatty, Toronto, Canada for this brief history regarding cholesterol: In 1958, Ancel Keys published the historic “7-Countries Study,” showing a linearly increasing cholesterol level / linearly increasing heart disease connection. *He misled the world* by not including many other countries where this correlation does not exist and would have contradicted his hypothesis. Furthermore, *he made no distinction in the quality* of fats / oils being critical to human health — he incorrectly stated that all fats raise cholesterol — especially, saturated fats. **To the contrary, unprocessed / fully functional EFA-containing plant- and seed- based oils lower LDL-Cholesterol.**

Keys’ misleading and wrong conclusions were opposed by at least 3 prominent researchers — Hugh Sinclair and Kinsell & Groen in 1952. Sadly, Keys ill-founded argument won the day. This was the beginning of the “no/low fat hypothesis” (*guess*) and the beginning of calling the absolutely required cholesterol [*Textbook of Medical Physiology*], “the enemy.” To this day, the majority of healthcare practitioners in the medical community are still misled in this area, as are their patients.
Key Insights:

1. **There is no cholesterol sensor in the bloodstream.** No, this is not a “genetic defect.” There are sensors for glucose (0.1% tolerance across all patients unless diabetic), sodium level, potassium level, etc. Given a system of n-1 linear equations with independent variables (and no other conditions), there must be 1 degree of freedom; LDL-C will be dependent on virtually everything else. Utilizing a biological systems approach, cholesterol can be viewed as a dependent variable. Its absolute number, therefore, is irrelevant. This explains lack of a required sensor. Furthermore, in addition to healing cardiovascular abnormalities, cholesterol has no less than 10 critical functions in the body, (*Textbook of Medical Physiology*). Pre-1990 LDL-C was never considered “bad.”

2. **Other than healing an impaired vascular issue, does cholesterol have other important functions?** YES. LDL-C is the transporter of EFAs (linoleic acid (LA) and alpha-linolenic acid (ALA) — the only 2 essential fatty acids). “Esterification” is a condensation reaction (the water is removed) — think of the EFAs being “magnetized” to the cholesteryl molecule for transport in the bloodstream.
3. Why is this esterification of the cholesteryl molecule to the EFAs critical to the understanding of the action of statins? Because, due to ubiquitous food processing, all cooking oils contain significant amounts of highly chemically processed nonfunctional LA (transformed from active to functionally impaired via harmful trans fats / interesterified fat formation, etc.). Therefore, when LDL-C is reduced, its nonfunctional / adulterated LA it transports is also REDUCED, which is very good. HOWEVER, at the same time, the fully functional, critically important LA transported is REDUCED — very, very bad. In fact, “all-cause” mortality would be expected to significantly increase in statin users; and it does increase (e.g., increased cancer and diabetes).

4. Fully functional LA is critical to each of our 100 trillion cells. Fully functional LA in the cell membranes allow hormones — including insulin — to be more effectively utilized (less insulin resistance). Fully functional LA is also critically important for maximizing CELLULAR OXYGENATION (Campbell) — and lack of / impairment of (cellular) oxygen transport is known to be highly cancer-causing (Warburg).
5. **Does reduction of LDL-C’s (esterified) LA cause additional problems?** Yes. Statins are sometimes claimed to possess mild anti-inflammatory action; however, LA’s long-chain metabolite, PGE$_1$ is the body’s most potent natural anti-inflammatory. Statin use decreases its production. PGE$_1$ is also known to reverse existing arterial occlusions/thromboses and is a potent natural vasodilator which increases blood flow—all helping to prevent CVD. Statins decrease both of these positive effects.

6. **Why is LA adulterated in the diet?** To obtain long shelf-life and allow long-term use in commercial restaurant baking and frying. Extended use of cooking oils causes shorter lifespan in humans because of significantly impaired functionality of LA.

7. **The intima — inner lining of the artery is 100% exclusively made of LA.** If there is less of the raw substrate (LA), the patient has a defective arterial lining. If the patient is consuming processed foods (as the vast majority do), he will also have an increase in defective intima and defective/functionally impaired cell membranes throughout all organs/tissues.
Furthermore, virtually all medical textbooks, including the medical standard—*Textbook of Medical Physiology*—state the intima is a mere 1 cell thick. This is incorrect; the intimal structure is actually 8-10 layers thick. [Note: an esteemed pathologist, Dr. Subbotin, came across my work and sent me a superb journal article with high-resolution electron photography confirming the multi-layer structure of the intima].

Therefore, with defective / adulterated/ nonfunctional LA there is in actuality up to 10x more chance of impairment to the arterial wall and its structure. **Cholesterol transports fully functional LA to the arterial wall in an attempt to repair defects in the arterial wall.**

8. **Exactly, what is oxidizing in LDL-C – the cholesteryl molecule itself or something else?** The answer is “something else.” Cholesterol / cholesteryl molecule and LA are each HIGHLY RESISTANT to OXIDATION in the body. There is only 1 anti-oxidant per 165 cholesterol molecules (1:165) — far too
few “protectors to oxidation. [Note: Three double bonds are required before \textit{in vivo} oxidation becomes an issue and LA has only 2 double bonds] — it is the adulterated / processed / nonfunctional LA that the cholesteryl molecule is transporting that is oxidized. This oxidized LA is NOT becoming oxidized in the body; it is \textit{exogenous} – coming in from consumed food already oxidized. Yes, high glucose auto-oxidizes in the bloodstream, too, but it is less significant an issue than the consumed EFAs from food already in a non-functional / oxidized state.

9. \textbf{What comprises an arterial occlusion (clog)?} High resolution chromatography confirms the occlusion is approx. 85% nonfunctional / adulterated / processed LA.

10. \textbf{Can increasing HDL solve the problem.} Given the analysis above, no, not at all.

\textbf{SOLUTION:} Consuming fully functional LA&ALA (the EFAs) each day – it’s easy and the excessive oxidization problem is solved. \textbf{Note, Keto followers:} Since LA and ALA are long-chain (18-carbon), not medium- or short-chain fatty acids, you are likely deficient in fully functional EFAs, and long-term you will suffer CVD issues if this issue isn't addressed.
Remarkable Clinical Results with EFAs

2008: “I previously wrote you about the remarkable cause / effect relationship in reversing plaque volume in a (smoking) patient taking conventional treatment (i.e. statins, aspirin, Co-Q10, etc.). In reading over [the patient’s] scans I have never seen such a remarkable result.

When he [the patient] stopped the EFAs the plaque came back.

As you can see, for the first time from 2007 to 2008, the volume of plaque decreased from 39 to 30, which is a decrease of 22% when annualized on a yearly basis. I have never seen a decrease of coronary artery plaque volume by more than 5% in one year.”

Robert Kagan, MD
Radiologist, USA

Former Chairman of the Board of Nuclear Medicine Resource Committee of the College of American Pathologists / President Clinton appointee as the sole physician commissioner on the White House Fellowship Commission / Past President of the Florida Association of Nuclear Physicians.