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SPECIAL REPORT

The Essential Debate: The Balance Between Omega-6 and Omega-3

The positive role of omega-3s, specifically eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), in promoting cardiovascular health has led to their increased acceptance and recommendation. Still at issue, though, is the added factor of omega-6 polyunsaturated fatty acid (PUFA) intake—and its potential to negatively impact beneficial omega-3 levels.

by Stuart Tomc and Eliza Leggatt

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During the last five decades, debate in the scientific literature has intensified regarding appropriate levels of omega-6 polyunsaturated fatty acid (PUFA) intake. While achieving high levels of omega-3 eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) in both diet and tissue is an unequivocal public health goal, researchers have been divided by recurring questions: Do omega-6 and omega-3 compete with one another, and, if so, what impediment does that pose to achieving higher omega-3 levels?

In 1961, the American Heart Association (AHA) made a cautious proclamation and started a PUFA dispute that persists to this day:

> "The reduction or control of fat consumption under medical supervision, **with reasonable substitution of polyunsaturated for saturated fats,** is recommended as a possible means of preventing atherosclerosis and decreasing the risk of heart attacks and strokes ... More complete information must be obtained before final conclusions can be reached."
>
> **AHA Recommendation, 1961**

Unfortunately, a crucial distinction was missing in that tentative recommendation: All PUFAs are not created equal. As Americans obediently abandoned animal fat in favor of vegetable oil, researchers began to delineate the metabolic pathways of omega-3 and omega-6 fats:

> **"...In simplification, the pattern of PUFA in tissue lipids is controlled by the concentrations of competing substrates in a common metabolic pathway."**
>
> **R. Holman, Ph.D., 1963**

Indeed, the formation and signaling action of bioactive mediators from longer-chain PUFAs, called highly unsaturated fatty acids (HUFAs), appear to be the critical difference between omega-3 and omega-6 fats. Metabolites of omega-3 HUFAs, specifically EPA and DHA, eventually work to retard inflammation, while excessive metabolites of omega-6 HUFAs, such as arachidonic acid, may act neutrally or promote inflammation. While the polyunsaturation craze took over in the context of blood cholesterol levels, a widening schism separated researchers into two camps: those who believed there was a competition between the families of essential fats, and those who did not.

Shortly after Americans began substituting margarine for butter, a revelation rocked the scientific world: Inuit Eskimos in Greenland were consuming exorbitant amounts of animal fat, yet were inexplicably unaffected by heart disease. In the early 1970s,

an analysis of the Inuit diet was published in the *American Journal of Clinical Nutrition,* theorizing that the marine oils omega-3 EPA and DHA uniquely conferred the cardioprotective element of their diet; soon, omega-3s became synonymous with cardiovascular health. Concurrently, omega-6 mechanisms were increasingly clarified, as demonstrated by the 1982 Nobel Prize in Physiology or Medicine, awarded for documenting a link between omega-6 fats and their largely proinflammatory prostaglandin metabolites.

These developments served only to intensify debate in the scientific community as to whether competition between omega-6 and omega-3 fats was physiologically significant. Some researchers continued to assert that if dietary (hence tissue) levels of omega-3 PUFAs exceeded levels of omega-6 PUFAs, competition would decrease the effects of pro-inflammatory omega-6 eicosanoid metabolites:

"Competition between the (omega-3) and (omega-6) types of highly unsaturated fatty acids can diminish the abundance of (omega-6) eicosanoid precursors in a tissue, which in turn can diminish the intensity of tissue responses that are mediated by (omega-6) eicosanoids."

W. Lands, Ph.D., 1992

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The debate continued to escalate as to whether omega-6s must decrease in order for omega-3s to increase, as did the studies unraveling the biochemical mechanisms underlying the now legendary "Inuit Paradox." These data formed part of the foundation for the 2002 AHA statement, emphasizing the potent role of omega-3 PUFAs in supporting cardiovascular health:

"Omega-3 fatty acids have been shown in epidemiological and clinical trials to reduce the incidence of CVD [cardiovascular disease]. Large-scale epidemiological studies suggest that individuals at risk for CHD [coronary heart disease] benefit from the consumption of plant- and marine-derived omega-3 fatty acids, although the ideal intakes presently are unclear."

AHA Recommendation, 2002

The requirement of omega-3s, specifically EPA and DHA, for cardiovascular health became a widely embraced and uncontroversial recommendation, although to this day, a recommended daily intake has yet to be established.

But for many lipid scientists, the background diet rife with omega-6 was of equal, if not greater, concern than setting an omega-3 intake recommendation.

The indiscriminate "polyunsaturation" of the nation craze had increased omega-6 consumption threefold in the form of cheap, shelf-stable vegetable oils, as corn, soy and safflower oils now dominated processed foods and animal feeds. Yet advocates of omega-6 PUFA intake maintained the massive shift in oil consumption was beneficial providing sufficient EPA and DHA were maintained—much to the alarm of prominent biochemists.

In 2009, the AHA attempted to quell the controversy with an inquiry into appropriate omega-6 levels:

> **"The data also suggest that higher intakes appear to be safe and may be even more beneficial (as part of a low-saturated-fat, low-cholesterol diet). In summary, the AHA supports an omega-6 PUFA intake of at least 5 percent to 10 percent of energy in the context of other AHA lifestyle and dietary recommendations."**
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> **AHA Recommendation, 2009**

This statement, intended to extinguish the ongoing debate, merely served as a spark to ignite further controversy. While the "other AHA lifestyle and dietary recommendations" did advocate proportionate omega-3 intake, the acknowledgment of any competition was dismissed; mainstream researchers purported human data was insufficient. Furthermore, they claimed, omega-6 consumption was responsible for the decline in CVD, not state-of-the-art technological intervention or increasingly sophisticated prescription drugs.

What were the potential ramifications of unchecked omega-6 consumption?

Meanwhile, researchers such as Joseph Hibbeln, M.D., methodically charted a 1,000-fold increase in soybean oil consumption since 1909. Miriam Garland pointed out that omega-6 levels had soared from 6 percent of adipose tissue in 1961 to a whopping 15 percent in 1998—was this the "reasonable substitution" the AHA initially called for? What were the potential ramifications of unchecked omega-6 consumption?

> **"This analysis found no benefit, and a relatively consistent signal toward harm from selectively increasing LA. We conclude that evidence from RCTs and prospective observational cohorts, the top two tiers of evidence-based medicine, does not support current population-wide advice to maintain or increase consumption of the (omega-6) PUFA LA."**
>
> **C. Ramsden et al., 2012**

With increasing evidence that high omega-6 consumption may be detrimental, the schism between those arguing for omega-6 reduction and those calling for maintenance of its high levels broadened. Such division can

amplify vigorous debates into trenchant battles—and the body of scientific literature has the scars to prove it. Further research is needed to facilitate another evolution in thinking.

Omega-3s seem to have limitless benefit, but do not seem to have a limitless supply. If indeed the radical increase of omega-6 throughout the past several decades simply requires proportionate omega-3 EPA and DHA intake, the question then becomes: How much is enough? Will we always have enough? How can we ensure our omega-6 overload does not also overburden the resources of the ocean?

Perhaps more than any other group, omega-3 manufacturers receive the brunt of sustainability inquiries surrounding ocean-harvested food. Addressing sustainability concerns solely to the fish oil industry—which constitutes a mere 3 percent of the total oceanic catch—is a small fraction of the answer. Ethical fish oil manufacturers are uniquely aware of the inestimable value of this natural resource, and uniquely aware of sustainability challenges that arise from excess omega-6 intake. The challenge becomes meeting the growing needs of consumers—and the growing rate of consumption. Responsible manufacturers seek creative sustainable solutions by sourcing with integrity and promoting viable alternatives to fish, such as algal oils. What else can be done? We know that consumers instigate change; yet, without awareness, no change will come.

> Until new data is available, researchers may continue to be divided by the omega-3/6 gap in the literature.

"Effective preventive nutrition interventions may help people choose foods to develop a balance of (omega-3) and (omega-6) HUFA in their tissues in a way that may lower the severity of the disorders and lower their personal risk for health-care expenses."

W. Lands, Ph.D.

We can begin to understand our own personal dietary intake by utilizing valuable free resources such as the omega-3/6 balance scores, which can be downloaded to any mobile device from fastlearner.org.

"Before long, your omega-3 index just could be the new cholesterol—the number you want to brag about."

B. Healy, M.D.

We can ascertain the omega-3/6 balance of HUFA in our blood and modify our diet accordingly to ensure highest levels of protection.

"Dietary omega-6 PUFA lowering for 12 weeks did produce significant increases in the EPA (+51%) and DHA (+19%) suggesting that high omega-6 (LA) diets may interfere with the synthesis and/or accumulation of long-chain omega-3 EPA and DHA in human tissues."

C. Ramsden et al., 2013

If research continues to confirm decreasing omega-6 (LA) could increase EPA and DHA tissue levels in humans, reducing vegetable oil consumption may be the most effective method of ensuring adequate tissue levels of omega-3 worldwide.

Yet until new data is available, researchers may continue to be divided by the omega-3/6 gap in the literature. However, it has often been said that necessity is the mother of invention, and in lieu of new data, resourceful researchers such as Christopher Ramsden, M.D., have applied modern technology to old data with impressive results. With a recent analysis of the Sydney Heart Trial (often praised for reducing unhealthy cholesterol levels), a new perspective emerges as well:

"In this evaluation of data from the Sydney Diet Heart Study, selectively increasing the (omega-6) PUFA LA from safflower oil and safflower polyunsaturated margarine increased rates of death from cardiovascular disease, coronary heart disease, and all cause mortality compared with a control diet rich in SFA from animal fats and common margarines. This is the first published report to show an increase in mortality from cardiovascular disease and coronary heart disease, comparing this LA intervention to the control group, and demonstrating that the magnitude of increased (omega-6) LA intake was associated with higher risk of death. Although increased all cause mortality was reported in a 1978 publication, cardiovascular disease and coronary heart disease clinical outcomes, rather than all cause mortality, are the most relevant endpoints considered when evaluating the evidence base and formulating dietary guidelines for cardiovascular risk reduction. **Therefore, recovery of these missing data has filled a critical gap in the published literature archive, allowing for a more comprehensive risk-benefit assessment for (omega-6) LA, including all known datasets from randomized controlled trials."**

C. Ramsden et al., 2013

Such an impactful revelation may begin to bridge the gap between opposing schools of thought. The question is, will we likely continue to be preoccupied by blood cholesterol levels, with this new evidence demonstrating potential harm from high omega-6 intake? And what are the potential consequences of inaction?

"Brain and heart disorders resulting from LC-omega-3 (EPA & DHA) deficiency are the biggest challenges to the future of humanity."

"Tissue concentrations of LC-omega-3 (relative to LC-omega-6) are the key variable for health – not dietary intakes."

"To make tissue targets feasible, we urgently need to reduce LA ... in human and animal diets while increasing the availability of LC-omega-3 (especially DHA) for human consumption in a sustainable, environmentally responsible way."

-Extracted from Unanimous Consensus Statement
Global Summit on Nutrition Health and Human Behavior

We know that omega-3s are essential—that failing to correct the global omega-3 deficiency jeopardizes our future and that of our children as well. We also know that the oceans' supply is finite—that omega-3s are an invaluable natural resource that demands stewardship.

If omega-3s can be increased specifically by reducing omega-6, would that be good news for the planet? If there is a plausible method of increasing omega-3 in human tissue independent of marine-based omega-3 oil consumption, a sustainable solution may be achieved. We must ask the hard questions, and prepare ourselves for uncomfortable answers. We must reject the notion of consumption without question, without responsibility, without evidence ... but certainly not without consequence. ❑

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