

A Closer Look at 'Anti-inflammatory' Diets

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By David Lightsey M.S.

Just about every health guru on the planet, as well as many respected science organizations, has their advice regarding purported inflammatory and anti-inflammatory foods and their purported role in the prevention or development of a myriad of diseases. But is this advice based upon a clear understanding or a premature assumption of the biological reactions which are occurring with various foods after they have been digested?

Since this issue is far too expansive and thus virtually impossible to isolate all of the incredulous comments regarding anti-inflammatory diet plans, I am going to use a group who many would normally consider as being on “top of the food chain” so to speak regarding sound nutrition science advice. Most individuals would agree that the following group should retain a keen eye for data interpretation and the ability to draw warranted conclusions vs the tidal wave of unjustified rubbish in the uneducated culture at large. So, I will use the Department of Nutrition, Harvard T.H. Chan School of Public Health as my example here.

On November 7, 2018 in the *Harvard Health Publishing – Trusted advice for a healthier life*, which is rather an ironic comment as you will see, published “Foods that fight inflammation.” In this article they state the following:

Doctors are learning that one of the best ways to reduce inflammation lies not in the medicine cabinet, but in the refrigerator (they are not referring to the ice). By following an anti-inflammatory diet, you can fight off inflammation for good.

Further, in the same newsletter it states:

Choose the right anti-inflammatory foods, and you may be able to reduce your risk of illness. Consistently pick the wrong ones, and you could accelerate the inflammatory disease process.

The article goes on to identify the common culprits of “inflammation,” as:

- **refined carbohydrates**, such as white bread and pastries
- **French fries** and other fried foods
- **soda** and other sugar-sweetened beverages
- **red meat** (burgers, steaks) and processed meat (hot dogs, sausage)
- **margarine**, shortening, and lard

And anti-inflammatory foods as:

- **tomatoes**
- **olive oil**
- **green leafy vegetables**, such as spinach, kale, and collards
- **nuts** like almonds and walnuts
- **fatty fish** like salmon, mackerel, tuna, and sardines
- **fruits such as** strawberries, blueberries, cherries, and oranges

Now let's look at the evidence. After the ingestion of various types of purported inflammatory foods, such as refined carbohydrates, French fries, sodas, etc. as mentioned above, certain molecules (or biomarkers) known to be associated with the inflammatory process increase in concentration in the blood after the foods are consumed. Now, the false assumption is that this temporary increase of biomarkers normally associated with inflammation is a negative issue and not a normal, temporary physiological response after the ingestion of certain foods-especially high glycemic index foods. Now, are these known inflammatory biomarkers which are being released an indication of inflammation or an indication of another, yet unidentified role these biomarkers may have unrelated to inflammation?

Marc Donath M.D. is Professor and Head of the Clinic for Endocrinology, Diabetes and Metabolism at the University Hospital of Basel, Switzerland. His research focuses on the mechanisms and therapy of decreased insulin production in type 2 diabetes. He has shown that IL-6, one of the biomarkers of inflammation widely used to condemn certain foods as “inflammatory,” as the ones mentioned above, regulates another molecule production called GLP-1. So, is this “inflammatory” biomarker, IL-6, being mischaracterized as a negative reaction to the food being ingested, or just a separate normal biological role for this molecule?

GLP-1, which is stimulated by the “inflammatory” biomarker, is responsible for stimulating the B-cells of the pancreas to secrete more insulin, all of which is dependent upon blood sugar levels after a meal. GLP-1 also suppresses the hormone glucagon, which, if not suppressed, would stimulate the release of even more sugar into the blood. Thus, this reaction is a good thing and not a bad thing. The inflammatory biomarker initiates the initial necessary steps to maintain normal blood sugar levels after the ingestion of various high glycemic index foods. GLP-1 also induces satiety in the hypothalamus, which, if you pay attention to this signal from your brain, it will help prevent overindulgence and obesity. Again, so far this is all normal physiology, initially stimulated by the inflammatory biomarker. The inflammatory biomarker is simply an indication of your body's

normal physiological response to blood sugar levels and based upon Dr. Donath's research, is a necessary step of normal physiological reactions to maintain normal blood sugar homeostasis. So, what does all this mean in simplified functional terms?

If you consume any food which may stimulate significant increases in blood sugar levels, which would be labeled as an "inflammatory food," such as potato products, refined carbohydrates, sodas, etc. what is going to happen? Your blood sugar will rise significantly. Then, the normal physiological response of the body is to secrete the insulin to control the level of blood sugar and keep it within normal ranges. Insulin will do this by stimulating the liver and muscle tissue to take up the excessive sugar. The two sites can either store it if possible as glycogen, or, in the liver's case, biosynthesize fatty acids from the excess sugar and store it in fat tissue. This is all perfectly normal metabolism of excessive calories, and the biomarkers for inflammation which have received so much negative attention, are simply assisting in this process by stimulating the necessary initial steps of it. Increased blood sugar stimulates the inflammatory biomarker, which then stimulates the GLP-1 molecule, which then stimulates the pancreatic beta cells to secrete insulin. This is not chronic inflammation, but a temporary secretion of a biomarker for inflammation, which obviously has other biological roles, to illicit the normal metabolic functions to control blood sugar levels.

Here is an email I sent to Dr. Donath on February 2, 2019 regarding this issue:

"It appears, the short-term inflammatory marker response is essentially a normal physiological response to increased blood sugar levels or excess calories, and the need to direct the appropriate hormonal responses to metabolize the sugars or excess calories. Is this correct or incorrect? Would you consider the current consumer and media reaction to the inflammatory marker response to food, similar to the misguided response to normal free radical production? In other words, under many conditions, such as post-exercise and during various immune responses, the increase free radical production is a normal metabolic response which acts as a signaling mechanism for various biological adaptations which need to take place, such as signaling the muscle tissue to adapt to the exercise load. Suppressing this free radical response hinders the adaptation. So, even though free radicals are often associated with negative biological activity by most consumers, during many physiological situations, their response is simply a signaling mechanism for further normal metabolic reactions in many situations. Is this also true for the markers of inflammation postprandial?"

His response on 2/10/19:

"Overall, I agree with your thinking."

To support all of this, in March 2017, Dr. Donath and his colleagues published their research findings in *Nature Immunology*, "Postprandial macrophage-derived IL-1B stimulates insulin, and both synergistically promote glucose disposal and inflammation." In the discussion section of this paper it states, "both insulin and IL-1B (biomarker for inflammation) regulated whole body glucose disposal by promoting glucose uptake in muscle and fat and fueled the immune system by stimulating the uptake of glucose into the immune-cell compartment." Also, their final comments were, "collectively, our findings have shown that IL-1B, a master regulator of inflammation, and

insulin, a key hormone in glucose metabolism, promoted each other. Both had potent effects on glucose homeostasis and on the activity of the immune system.”

On January 16, 2017, *Science Daily*, covered the same research of Dr. Donath in their review “Every meal triggers inflammation.” In this review they make the following points:

- When we eat, we do not just take in nutrients -- we also consume a significant quantity of bacteria. The body is faced with the challenge of simultaneously distributing the ingested glucose and fighting these bacteria. This triggers an inflammatory response that activates the immune systems of healthy individuals and has a protective effect.
- This inflammation does have some positive aspects. In healthy individuals, short-term inflammatory responses play an important role in sugar uptake and the activation of the immune system.

The bottom-line. Certain foods, due to their effect on blood sugar levels, precipitate the release of various molecules which are also associated with inflammation. However, this does not mean these foods are triggering inflammation or disease. Their presence is just a normal response and dual role these molecules have to assist in the maintenance of normal blood sugar homeostasis. Consuming refined carbohydrates, French fries, or sodas are not going to cause inflammation. Their excessive consumption will certainly be reflective of an LSD diet (lousy stinking diet), which is one low in plant-based foods and the subsequent absence of thousands of healthy plant chemicals, and a likely similar overall poor lifestyle habit. So of course, these individuals are going to have significant negative health issues, but it certainly is not going to be related to the occasional or limited daily use of the so-called “inflammatory” foods. To suggest so is junk-science and a lack of common sense.

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