ALIVE!

AN EFFORT OF NATURE

THROUGHOUT MUCH OF the 20th century, atherosclerosis was thought of as a disease of the inside space of arteries, the lumen. Most of us considered this hardening and thickening of the arteries to be the result of excess lipids in the bloodstream. Fat build up resulted in clogged arteries and clogged arteries don't make for free-flowing blood to the brain and heart. As a result, fat-free was the way to be. Low-fat became the health slogan emboldened on plastic packaging in an attempt to ease fat conscious consciences seeking to avoid the #1 killer: cardiovascular disease. Unfortunately, consuming all of those low-fat, processed foods didn't make much of a dent in our ever-escalating disease and death rate.

Yes, elevated blood lipids (fat-like compounds) contribute to heart disease in a big way. LDL cholesterol especially is strongly associated with atherosclerosis, and reducing saturated fat in the diet does lower LDL. In fact, current knowledge is that the higher your blood cholesterol level, the greater your risk for developing heart disease or having a heart attack. This is no inconsequential matter when you remember that heart disease is the number one killer of women and men in the United States. Each year, more than a million Americans have heart attacks, and about a half million people die from heart disease. But while this is the case, it is significant to realize that the body produces the majority of blood cholesterol. It is not necessarily a direct deposit from what we eat.

More recent evidence has shown atherosclerosis to be a disease of the wall of the artery rather than the inside space. While it is true that blood fats are involved in the development of plaque and clogging of the arteries, the progression of cardiovascular disease is intimately connected with inflammation within the blood vessel wall. Instead of a disease of excess blood lipids, atherosclerosis is now regarded as an inflammatory disease.

Inflammation is a response of the immune system to the presence of infection, irritation, trauma, or damage.

Inflammation is a word we throw around a lot. We are familiar with its manifestation of heat, pain, redness, and swelling. Inflammation is quite the wanderlust, traveling throughout the body. Medicine has identified its presence in various locales with the suffix –itis: bursitis, dermatitis, gingivitis, gastritis, colitis, vasculitis, arthritis, pancreatitis, cholecystitis, etc. Inflammation is a response of the immune system to the presence of infection, irritation, trauma, or damage. It is intended to be protective and focused on eliminating the root of what is causing harm. Inflammation is created by our own body's defense.

Immune cells secrete molecules called cytokines that produce inflammation. These cytokines are the

communicators of the immune system and call the shots to immune cells. We make cytokines that are pro-inflammatory and we make cytokines that are anti-inflammatory. When things are in balance, inflammation is temporary and aids in maintaining health. The flame gets put out. When things become unbalanced, inflammation can become chronic and promote pain and damage to bone, destroy cartilage, and cause trauma to blood vessels. As mentioned earlier, inflammation can manifest in many different systems and organs of the body. The reason why this can occur is because every organ has immune cells poised in close proximity, ready to respond to a perceived threat by pumping out these cytokine communicators that create inflammation.

From its very inception, atherogenesis has a strong inflammatory component. Individuals with elevated levels of inflammation have an increased risk for sudden cardiac events and mortality. The buildup of fatty plaque appears to be more of a compensatory response to the invasion, damage, and inflammation occurring in the arterial wall. You might say atherosclerosis is an effort of nature to free the system from conditions that result from a violation of the laws of health.[1] Viewing the process of cardiovascular disease in this way, we are led to look farther upstream for causative factors.

Since inflammation is a response to something threatening, it would be smart to ask the question, What could be continuously threatening? There could be various drivers of inflammation, many of which are not dietary related. However, for many, breakfast, lunch, and dinner provide ample sustenance of inflammation.

The highly processed, calorie-dense, nutrient-depleted standard American diet leads to exaggerated post-meal spikes in lipids and blood sugar. This state has been labeled postprandial dysmetabolism. This post-meal increase in blood sugar and triglycerides induces immediate stress and damage to the blood vessels. Significantly high blood sugar after a meal is correlated with increased incidence of cardiovascular events and mortality, independent of fasting blood sugar levels. The older we get the more sensitive we become to these fluctuations. Large epidemiological studies have shown that elevated, post-meal triglyceride levels are an additional risk factor for the development of cardiovascular disease. For 14.6 years, 24,535 Norwegian women aged 35-49 years old were followed. Researchers found that death from coronary heart disease, cardiovascular disease and all causes, steadily increased with increasing non-fasting triglyceride concentrations. Another study conducted in the U.S. with 26,509 initially healthy women followed for 11.4 years indicated that both fasting and non-fasting triglycerides levels were predictors of such cardiovascular events as nonfatal heart attack, stroke, and cardiovascular death. Worth noting is the observation that obese individuals, and especially those with abdominal visceral obesity, have an increased response to elevated triglycerides after the consumption of a fatty meal compared to non-obese subjects.[2]

Disease free, normal weight individuals under 50 years old consumed an Egg McMuffin, Sausage McMuffin, and two hash brown patties after fasting overnight. The researchers found that this single high-fat meal negatively impaired the lining of blood vessels and promoted atherogenesis, independent of cholesterol levels. After high carbohydrate, high-fat meals, a transient increase in free radicals is experienced along with elevated blood sugar and blood fat. This triggers changes in the blood vessels including inflammation, endothelial dysfunction, and increased clotting. "Post-prandial dysmetabolism is an independent predictor of future cardiovascular events even in non-diabetic individuals. Improvements in diet exert profound and immediate favorable changes in the post-prandial dysmetabolism. Specifically, a diet high in minimally processed, high-fiber, plant-based foods such as vegetables and fruits, whole grains, legumes, and nuts will markedly blunt the post-meal increase in glucose, triglycerides, and inflammation."[3]

We are all immersed in a world that is in many ways out of sync with nature. Trammeled nature will put forth effort for restoration and ultimately, it "will be delivered from the bondage of corruption into the glorious liberty . . ." (Romans 8:21).

1. Ellen G White, *Ministry of Healing*, p. 127.

2. Christos Pappas, et al., "Postprandial dysmetabolism: Too early or too late?," *HORMONES* 2016, http://www.hormones.gr/8653/article/article.html.

3. J.H. O'Keefe, N.M. Gheewala, J.O. O'Keefe, "Dietary strategies for improving post-prandial glucose, lipids, inflammation, and cardiovascular health," *J Am Coll Cardiol*, Jan. 22, 2008, https://www.ncbi.nlm.nih.gov/pubmed/18206731.

RISË RAFFERTY, RDN

Risë is a Registered Dietitian Nutritionist. Her understanding of how significantly diet and lifestyle impact one's health and happiness fuels her passion to help, educate, and inspire others.