

Your Gut Microbiota Defines Cardiovascular Disease Risk!

by Guy Schenker, DC

THERE IS AN EXCLAMATION POINT at the end of this article's title. Why? This title belongs in the "believe it or not" category. Your "gut reaction" when reading it (pun intended) is likely, "Oh, come on. You're kidding," or, "Nah, how can that be?" But the truth in that claim will be revealed in this article.

Whether it is myocardial degeneration or arteriosclerosis, cardiovascular disease is primarily associated with inflammation of sufficient intensity and duration to cause inflam-aging in the cardiovascular system. What is a major driver of this chronic low-grade inflammation? Dysbiosis that causes a deranged microbiota-immune axis.¹

Research shows there are direct axes (communication systems) from your microbiota-controlled gut lining, including:

- A gut-brain axis²
- A gut-hypothalamus axis³
- A gut-liver axis⁴
- A gut-adipose axis⁵
- A gut-pancreas axis⁶
- A gut-muscle axis⁷
- A gut-immune-system axis⁸

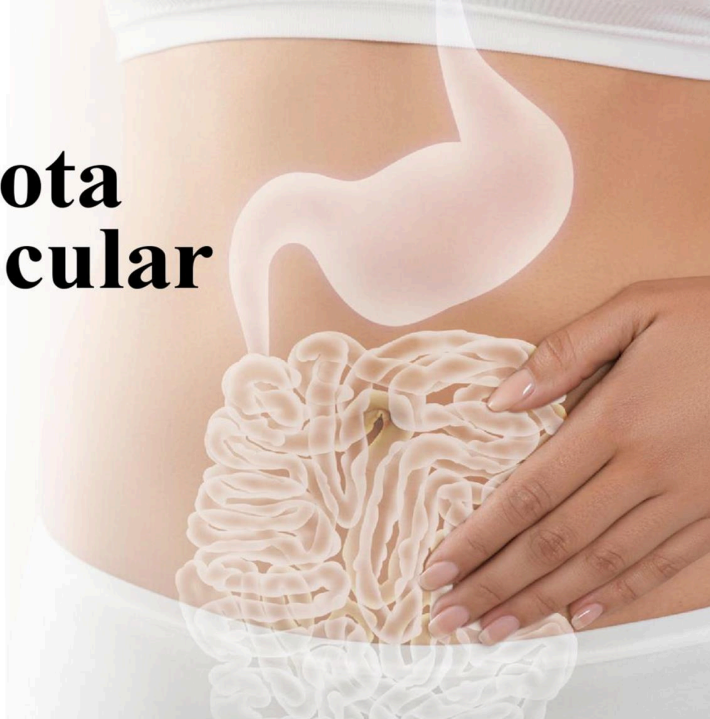
Yes, there is no "gut-cardiovascular axis." None is needed to explain inflam-aging-mediated cardiovascular disease. When stressed by microbiota dysbiosis, these seven axes place a huge inflammatory burden on the heart.

A diverse and prolific gut microbiota is essential for your health. Amazingly, 100 trillion microbes reside in and on your body, including over 1,000 different species in your gut. There are 10 times as many bacterial cells as human cells in your body. The complex interactions of these gut microbes with each other, with their metabolites, and with the seven axes, largely control the adaptative capacity of your immune system, nervous system, and hormone system.

By mechanisms that are still being researched, your gut microbiota produces several bioactive metabolites that impact cardiovascular health. The influences are exerted largely through two metabolic pathways originating in the gut — the SCFA pathway and the TMAO pathway.⁹

The anti-inflammatory short-chained fatty acid (SCFA) pathway is initiated by the short-chained fatty acids produced as metabolic products by certain beneficial probiotics. By far, the most important of these SCFAs is butyrate, which is powerfully anti-inflammatory via several of the gut-organic axes previously listed.

The pro-inflammatory TMAO pathway is initiated by a metabolite derived from noxious gut microbiota and has emerged as a major factor in the risk of fatty liver disease via



the gut-liver axis. More recently, this toxic substance has been shown to damage the cardiovascular system.

Fortunately, an overwhelming volume of research shows that consumption of a high-fiber diet along with synbiotic (probiotic and prebiotic) supplementation supports an abundant production of SCFAs at the same time it inhibits the toxic load of TMAO. Research clearly establishes that synbiotic supplementation can be potentially used for prevention and management of cardiovascular disease.

As an expanding body of research shows that inflam-aging underlies heart failure, arteriosclerosis, hypertension, and cardio-renal disease, the mechanism by which inflammation takes its toll is being identified. An inadequate or unbalanced microbiota floods the body with four destructive inflammatory. These include the three cytokines, tumor necrosis factor-alpha, interleukin-1-beta, and interleukin-6, along with endotoxin. These instruments of inflam-aging are associated with the gut-immune axis.¹⁰

Recall that more than 70% of your immune cells reside in the gut lining.¹¹ From there, macrophages, mast cells, and inflammatory cytokines are activated inappropriately, contributing to the chronic inflammation that underlies all the "diseases of aging," including cardiovascular disease. As research identifies the mechanisms of cardiovascular inflammation, it also brings us the good news that inflammation can be significantly inhibited by synbiotic supplementation.

However, not just any prebiotic or probiotic supplement will do. You need to account for specificity of pre- and probiotic supplementation. You specifically want the probiotics that inhibit TNF-alpha, IL-1-beta, and IL-6, and that decrease circulating endotoxin. Some probiotics powerfully inhibit the inappropriate activation of macrophages and mast cells that results in those three inflammatory cytokines. Some of them do so by their direct action, and some do so by generating the production of the anti-inflammatory SCFA, butyrate. No ordinary, randomly blended probiotic product will do the job.

Macrophages are the major cell type of inflammatory mediators involved in inflam-aging diseases, such as arteriosclerosis. When excessively activated, these white blood cells (among the

monocyte analyte reported in the WBC differential of a complete blood count) produce large amounts of TNF-alpha, IL-6, inducible nitric oxide, and inflammatory prostaglandins (omega-6 fatty acid derivatives).

IL-6 is of particular significance. Macrophages excessively triggered from an unhealthy colon microbiota produce IL-6, which does direct inflammatory damage to the arterial intima. Part of the inflammatory response in the arterial wall includes increasing deposits of both calcium plaques and cholesterol. The cholesterol is oxidized, which explains why total cholesterol is not an independent risk factor for heart attacks and strokes, but oxidized LDL cholesterol is. The one blood test paralleling this arteriosclerotic inflammatory reaction is c-reactive protein.¹²

“While dysbiotic gut microbiota inappropriately initiates arteriosclerotic degeneration, only a healthy microbiota can inhibit it.”

While dysbiotic gut microbiota inappropriately initiates arteriosclerotic degeneration, only a healthy microbiota can inhibit it. The SCFA butyrate is by far the most potent inhibitor of arterial inflammation. It decreases macrophage chemoattractant protein-1 (MCP-1), even when MCP-1 production is exacerbated by the presence of gut-derived endotoxin. Butyrate, along with its team of other SCFAs, dose-dependently inhibits IL-6 and suppresses NF-kB, one of the most destructive pro-inflammatories of the inflam-aging process. By promoting SCFA production, synbiotic supplementation controls the whole gamut of vascular-related inflammatory mediators, including endotoxin, NF-kB, and the three major inflammatory cytokines.

Butyrate and its team of other SCFAs also have an indirect effect that inhibits cardiovascular disease. SCFAs regulate insulin resistance and reduce the risk of type 2 diabetes, which research identifies as an important promoter of CVD. Experimentally, mice consuming a high-fat (corn oil) diet develop insulin resistance and obesity, while butyrate supplements reverse that progression.¹³

Furthermore, not only is chronic low-grade inflammation a major factor in abdominal obesity that accompanies insulin resistance, that inflammatory pathway involves the same destructive cytokines associated with macrophage activation — IL-6, TNF-alpha, IL-1-beta, and others. These further disturb metabolism and aggravate the gut-pancreas axis, eventually leading to beta-cell dysfunction and insulin resistance. Additionally, unhealthy gut microbiota increases the level of endotoxin, which research also shows is a primary cause of type 2 diabetes. Now we have come full circle. The inflammation associated with abdominal weight gain and even-

tually type 2 diabetes is the very same inflam-aging pathway that causes cardiovascular degeneration.

Other experiments on mice show that mice on a healthy diet supplemented with endotoxin develop obesity and type 2 diabetes, similarly to those fed the high-fat diet. Abdominal obesity derived from insulin resistance (which is typically accompanied by an elevated triglyceride-to-HDL cholesterol ratio) is the number one risk factor for heart attacks and strokes. These studies suggest the dysbiotic gut is the primary cause, not poor eating habits. Both weight gain and cardiovascular disease are secondary to unhealthy microbiota.

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Obviously, prevention involves healthy eating, but as these research studies have shown, cardiovascular disease will develop even with a healthy diet if the microbiota is producing too much endotoxin and not enough anti-inflammatory SC-FAs. There is a gut-cardiovascular axis.¹⁴ Your microbiota truly does define your CVD risk.



Dr. Guy Schenker, a Pennsylvania chiropractor since 1978, developed the Nutri-Spec System of Clinical Nutrition, which eschews symptom-based nutrition in favor of individualized metabolic therapy. Reach us at 800-736-4320, email NutriSpec@Nutri-Spec.net, or visit nutri-spec.net.

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