

GUT DYSFUNCTIONS UNLEASH BACTERIAL TOXINS INTO CIRCULATION

Source: Caradonna L, Amati L, Magrone T, Pellegrino NM, Jirillo E, Caccavo D. Enteric bacteria, lipopolysaccharides and related cytokines in inflammatory bowel disease: biological and clinical significance. *J Endotox Res* 2000;6(3):205-212.

Toxic overload in the body - triggered by increased intestinal permeability and an imbalanced microflora in the gut - may be a precipitating factor that triggers and drives Inflammatory Bowel Disease (IBD), according to a new review study.

When the gut lining's normally tight junctions become wrenched loose, increased intestinal permeability, sometimes called "leaky gut," may allow bacterial toxins to penetrate the gut and enter the circulation. These toxins, including dietary antigens, bacterial pathogens, toxic by-products, and bacterial cell wall lipopolysaccharides, can then ignite an intricate chain of chronic inflammatory immune reactions that damage the intestine and set off symptoms associated with IBD, the researchers note.

A viable, healthy population of "friendly" bacteria in the gut, such as *Lactobacilli*, can help block the translocation of potential gut pathogens, prevent "bad" bacteria from "sticking" to the gut lining, and dampen an overactive immune response. Yet in many patients with IBD, concentrations of these protective *Lactobacilli* have been shown to be abnormally low.

As a potential result of these gut dysfunctions, endotoxemia occurs in as many as 94% of patients with Crohn's Disease, one of the major forms of IBD, and appears to correlate with the active stage of the disease.

Because gastrointestinal dysfunctions such as intestinal permeability are common both among patients with IBD and their relatives, IBD appears to arise from a combination of both genetic and environmental triggers, the researchers postulated.

They based their view on over 125 studies, both experimental and clinical, examining the pathogenesis of IBD in relation to bacterial translocation and gastrointestinal function.

NOTE: For over a decade, Great Smokies' gastrointestinal assessments have served as pioneering clinical tools for delineating the pivotal role of gut function in health and disease.

Intestinal Permeability Assessment is an important analysis of gut permeability function, an accurate predictor of relapse in asymptomatic patients with IBD and other gastrointestinal disorders, and an important clinical index in patients suffering from toxic overload, maldigestion, or food allergies.

The Comprehensive Digestive Stool Analysis evaluates digestive function and the balance of intestinal microflora as an overall indication of gut innate defense against gastrointestinal-related disorders and other health conditions. This comprehensive panel includes butyrate and lactoferrin, agents cited by the authors for their important roles in modulating toxic immune reactivity in IBD.

COULD "LEAKY GUT" TRIGGER CARDIAC DYSFUNCTION?

A patient might think twice before habitually popping a pill to relieve that minor ache or pain. Besides possibly damaging their gut's protective lining, some drugs may eventually impair their heart's innate ability to "keep on ticking."

The use of nonsteroidal antiinflammatory drugs (NSAIDs) significantly increases the risk of congestive heart failure in older patients, especially those with a prior history of heart disease, according to a new case-control study in the *Archives of Internal Medicine*.

Researchers tracked NSAID use in 365 patients with moderate or severe congestive heart failure and 658 controls. They excluded individuals taking low doses of aspirin, which is believed to benefit cardiovascular health.

Patients recently taking NSAIDs had twice the risk of being hospitalized with congestive heart failure. For those with a previous history of heart disease the risk associated with NSAID use was even greater, reaching over ten-fold. For those with a history of heart disease suffering first-time congestive heart failure, non-aspirin NSAID use was associated with a 26.3 times greater risk of congestive heart failure than for reference subjects with no heart disease and no NSAID use.

NSAIDs appear to increase vascular resistance and may also interfere with the actions of drugs such as diuretics and ACE inhibitors used to treat heart disease, the researchers said. In this way, NSAID use may accelerate congestive heart failure, rather than cause it.

If the evidence holds, it suggests that as many as 20% of first cases of congestive heart failure could be attributable to NSAID use. That would make the overall incidence of first-time congestive heart failure from NSAID's comparable to the rate of gastrointestinal complications such as ulcers and bleeding. What's more, the cardiac and gastrointestinal complications may be closely related. In a follow-up correspondence appearing in the Archives, Mathias Rauchhause, MD, and his colleagues note that nonaspirin NSAIDs can damage protective cells lining the gut, increasing intestinal permeability and allowing the translocation of toxic bacterial lipopolysaccharides into the bloodstream. The ensuing endotoxemia, a state in which the body becomes overloaded with bacterial toxins and their products, is often seen in patients with congestive heart failure, they point out. This may explain why chronic NSAID use could potentially trigger cardiac dysfunction.

NOTE: Because "leaky gut" has been implicated in the etiology of diverse conditions, Intestinal Permeability Assessment has a wide range of clinical applications, including patients suffering from food allergy, inflammatory bowel disease, arthritis, premature aging, cognitive disorders, HIV/AIDS, alcohol/drug addiction, autoimmune disease, fatigue, and chronic dermatological conditions.

Sources: Page J, Henry D. Consumption of NSAIDs and the development of congestive heart failure in elderly patients: an under-recognized public health problem. Arch Intern Med 2000;160:777-784.