

Rebuilding Intestinal Mucosa

Can Improve Overall Immunity

Tom Bayne, DC



The mucosal system is a very important part of the human immune system. Not many people think of mucus as being protective, but it acts as the main interface between the human body and the outside world. The mucosal system actually contains 150 times more surface area than skin, which makes it one of the most important immune barriers in the body. Furthermore, new studies indicate that the health of the intestinal mucosa can actually determine how the body interacts with antigens.¹ In this way, the integrity of the intestinal mucosa can dictate overall immune function.

Research suggests that the intestinal mucosal barrier plays a significant role in the pathogenesis of many chronic conditions, including inflammatory bowel diseases (IBD), depression, acquired immunodeficiency syndrome (AIDS), gastroesophageal reflux disease (GERD), acute pancreatitis, and more.²⁻⁶ Some researchers even believe that mucosal damage may be the initial injury that leads to many of these chronic conditions. Unfortunately, the Western lifestyle is quite damaging to the intestinal mucosa. Antibiotics, NSAIDs, stress, alcohol, gut infections, and other factors can easily degrade the intestinal mucosa, weakening the immune system and allowing unwanted toxins to enter circulation and trigger an inflammatory response.⁷

One of the most pervasive toxins is an endotoxin known as lipopolysaccharide (LPS). This endotoxin is a component of the outer cell membrane of gram-negative bacteria in the gut. When these bacterial cells die, they release LPS into the intestinal lumen. If the mucosal barrier becomes damaged, LPS can easily enter circulation and trigger the release of inflammatory cytokines, like tumor necrosis factor-alpha (TNF- α), interleukin-6 (IL-6), IL-8, and IL-1beta.

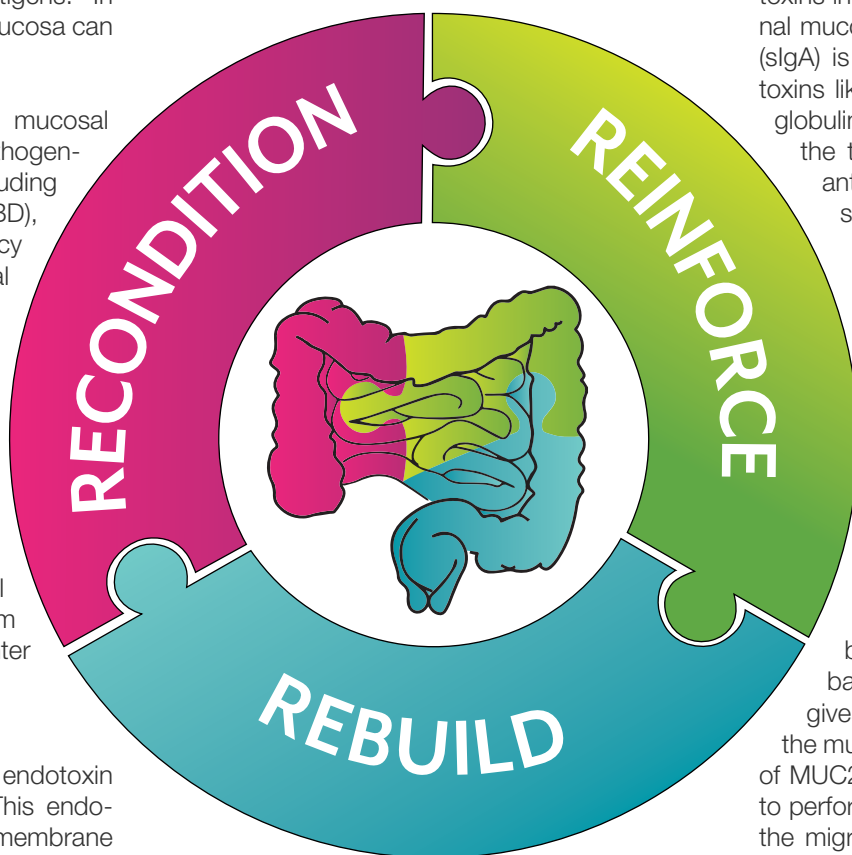
Studies have found that TNF- α is one of the main effectors of intestinal inflammation in inflammatory bowel diseases, depression, acute pancreatitis, and more.^{3, 6, 8} TNF- α appears to modulate the transcription of tight junction proteins that tightly connect intestinal epithelial cells, or enterocytes.⁹ When tight junctions are functioning properly, they affix enterocytes to one another like super glue, forming a strong intestinal lining. An overabundance of TNF- α can cause tight junctions to loosen up between enterocytes, creating a more permeable intestinal lining. In some cases, too much TNF- α can even trigger apoptosis, or cell death, of enterocytes, leaving open gaps between intestinal cells.¹⁰ The result is a hyperpermeable intestinal lining, otherwise known as leaky gut. When the gut is hyperpermeable, or leaky, it allows unwanted toxins and even

undigested food particles to leak directly into the bloodstream. Once toxins like LPS enter the blood, they can accumulate and induce chronic inflammation just about anywhere in the body, including the brain. Short-chain fatty acids (SCFAs), like butyrate, are normally responsible for regulating the production of inflammatory cytokines in the intestines. Butyrate can regulate the production of TNF- α by reducing the activity

of histone deacetylase (HDAC), an enzyme that promotes its expression.¹¹ In this way, butyrate has a strong anti-inflammatory effect in the intestines. In a healthy gut, butyrate comes from butyrate-producing bacteria, like *Bifidobacteria* and *Faecalibacterium prausnitzii*. When these bacteria die, from antibiotics or poor diets, the availability of butyrate in the gut begins to fade. Not surprisingly, low levels of *Bifidobacteria* and *F. prausnitzii* appear to be common signatures of many chronic diseases.¹²

Relieving chronic intestinal inflammation is a multi-pronged approach that aims to neutralize toxins in the intestines and **REBUILD** the intestinal mucosal barrier. Secretory immunoglobulin A (sIgA) is the body's first line of defense against toxins like LPS in the intestinal lumen. Immunoglobulins, also known as antibodies, can reduce the toxic load on the system by binding to antigens and removing them from the system. When sIgA levels are low, the adaptive immune response is weakened. However, sIgA levels can be revived with the supplementation of serum-derived bovine immunoglobulins (SBI). As a dairy-free alternative to colostrum, SBI powders can bind a variety of pathogens, including bacteria, viruses, and fungi, as well as their toxic by-products in order to remove them from the body.¹³

The mucosa is a key barrier that protects LPS from entering into the basolateral layer. The inner mucosal barrier contains a protein called MUC2 that gives this layer its thick, gel-like quality. When the mucosa suffers from inadequate production of MUC2 mucin and inadequate viscosity, it fails to perform its barrier function and thus allows for the migration of LPS into circulation. Increasing MUC2 mucin production can help prevent LPS and other toxins from migrating towards the intestinal epithelial and triggering an innate immune reaction. Butyrate is a SCFA that not only feeds enterocytes but also acts as one of the most potent stimulators of MUC2 mucin production. However, a healthy mucosal barrier needs more than an ample supply of butyrate – it also needs the essential amino acid building blocks. These building blocks include L-threonine, L-serine, L-proline, and L-cysteine. These four amino acids have been shown to drastically increase MUC2 production and stimulate mucin synthesis in the colon, resulting in a thicker and healthier mucosal barrier.¹⁴ Once the gut has been reconditioned and reinforced, the third and final step is to **REBUILD** the intestinal mucosa with the necessary building blocks in order to restore the health of the gut microbiome.



of histone deacetylase (HDAC), an enzyme that promotes its expression.¹¹ In this way, butyrate has a strong anti-inflammatory effect in the intestines. In a healthy gut, butyrate comes from butyrate-

Dr. Tom Bayne is a Chiropractic Physician and public speaker dedicated to understanding and improving the gut microbiome. As the President of Microbiome Labs, Dr. Bayne travels around the world to educate other healthcare practitioners on the connection between the gut microbiome and many chronic diseases.



<https://bit.ly/2F54Yrr>



RECONDITION



REINFORCE



REBUILD

REFERENCES: 1) Citi S. Intestinal barriers protect against disease. *Science*. 2018;359(6380):1097-1098. 2) Michielan A, et al. Intestinal Permeability in Inflammatory Bowel Disease: Pathogenesis, Clinical Evaluation, and Therapy of Leaky Gut. *Mediators Inflamm*. 2015; 2015:628157. 3) Maes M, et al. The gut-brain barrier in major depression: intestinal mucosal dysfunction with an increased translocation of LPS from gram negative enterobacteria (leaky gut) plays a role in the inflammatory pathophysiology of depression. *Neuro Endocrinol Lett*. 2008;29(1):117-24. 4) Paiardini M, et al. Mucosal immune dysfunction in AIDS pathogenesis. *AIDS Rev*. 2008;10(1):36-46. 5) Kandulski A, et al. Gastroesophageal reflux disease--from reflux episodes to mucosal inflammation. *Nat Rev Gastroenterol Hepatol*. 2011;9(1):15-22. 6) Liu H, et al. Early gut mucosal dysfunction in patients with acute pancreatitis. *Pancreas*. 2008;36(2):192-6. 7) de Punder K, et al. Stress Induces Endotoxemia and Low-Grade Inflammation by Increasing Barrier Permeability. *Front Immunol*. 2015;6:223. 8) Schmitz H, et al. Tumor necrosis factor alpha (TNF α) regulates the epithelial barrier in the human intestinal cell line HT-29/B6. *Journal of Cell Science*. 1999;112(1):137-146. 9) Coskun M. Intestinal epithelium in inflammatory bowel disease. *Front Med*. 2014;1:24. 10) Su L, et al. TNFR2 activates mlck-dependent tight junction dysregulation to cause apoptosis-mediated barrier loss and experimental colitis. *Gastroenterology*. 2013;145(2):407-415. 11) Vinolo MAR, et al. Regulation of Inflammation by Short Chain Fatty Acids. *Nutrients*. 2011; 3(10): 858-876. 12) Matsuoka K, et al. The gut microbiota and inflammatory bowel disease. *Semin Immunopathol*. 2015; 37: 47-55. 13) Detzel CJ, et al. Bovine immunoglobulin/protein isolate binds pro-inflammatory bacterial compounds and prevents immune activation in an intestinal co-culture model. *PLoS one*. 2015;10(4):e0120278. 14) Faure M, et al. Specific Amino Acids Increase Mucin Synthesis and Microbiota in Dextran Sulfate Sodium-Treated Rats. *J Nutr*. 2006;136(6):1558-64.