



Food Allergies, Intolerances and Sensitivities: A Link to Cardiovascular Issues

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Approximately 15-20% of adults have a food intolerances or sensitivity.¹ These patients report a wide range of symptoms, including gastrointestinal distress including abdominal pain, gas/belching/burping, IBDs (like IBS, Crohn's, ulcerative colitis), peptic ulcer, migraines, chronic fatigue, arthritis, rashes, respiratory distress, skin issues, weight gain, increased illnesses/immune/autoimmune imbalances, and chronic pain issues.^{2,3} Of interest to this article, cardiovascular issues have also been linked to chronic inflammation – metaflammation - that results from a food sensitivity/intolerance.⁴

Current literature supports the fact that food allergies, intolerances and sensitivities are directly tied to metainflammatory sequelae and the release of GUT mucosal IgE and IgG type antibodies.^{5,6,7} This inflammatory signaling “drives” your patient’s trend toward cardiovascular diseases.

How you address this metaflammation dictates the patients ability to decrease the risks of cardiovascular diseases.

One of the main targets of metainflammatory signaling is the GUT, including mucosal integrity and microbiome issues.⁸ Food allergies/sensitivities and intolerances play a significant role in mitigation of this inflammatory response and avoidance of the offending antigens has been shown to reduce symptoms.

¹ Lomer MC. Review article: the aetiology, diagnosis, mechanisms and clinical evidence for food intolerance. *Aliment Pharmacol Ther.* 2015;41(3):262-275.

² Neuendorf R, et al. Impact of food immunoglobulin IgG-based elimination diet on subsequent food immunoglobulin G and quality of life in overweight/obese adults. *J Allergy Clin Immunol.* 2019;25(2):241-48.

³ Schafer T, et al. Epidemiology of food allergy/food intolerance in adults: Associations with other manifestations of atopy. *Allergy Eur J Allergy Clin Immunol.* 2001;56:1172-79.

⁴ Tsantsaridou A, et al. Association of food intolerance with coronary artery disease. *J Food Nutr Res.* 2019;7(1):71-81.

⁵ Ohtsuka Y. Food intolerance and mucosal inflammation. *Pediatr Int.* 2015;57(1):22-9.

⁶ Chahine BG, et al. The role of the GUT mucosal immunity in the development of tolerance versus development of allergy to food. *Curr Opin Allergy Clin Immunol.* 2010;10(4):394-9.

⁷ Berlin MC. Mucosal antibodies in the regulation of tolerance and allergy to foods. *Semin Immunopathol.* 2012;34(5):633-42.

⁸ Lobionda S, et al. The role of gut microbiota in intestinal inflammation with respect to diet and extrinsic stressors. *Microorganisms.* 2019;7(8):271.

An altered microbiome is not only a marker of disease but also actively contributes to multiple disease pathogenesis...⁹

Cardiovascular Diseases (CVDs) and the GUT

Cardiovascular disease is the leading cause of death for men, women, and people of most racial and ethnic groups in the United States. One person dies every 36 seconds in the United States from a CVD related event. About 655,000 Americans die from CVDs each year—that's 1 in every 4 deaths.¹⁰ By 2030, 40.5% of the US population is projected to have some form of CVD.¹¹ Between 2010 and 2030, the real, total direct medical costs of CVDs are predicted to triple from \$273 billion to \$818 billion, and the real, indirect costs (owing to lost productivity) for all CVDs are estimated to increase by 61% (\$172 billion to \$276 billion).¹²

The heart is intimately connected to the GUT – known as the HEART-GUT Axis. Studies over the last decade suggested a potential role for the gut microbiome in the pathogenesis of atherosclerosis, CVD and heart failure.¹³ Disturbances in the GUT microbiome and epithelial function contribute greatly to chronic inflammatory sequelae through the release of endotoxin (lipopolysaccharide or LPS) and the upregulation of inflammatory cytokines. High serum LPS activity is directly associated with cardiometabolic disorders.¹⁴

An altered intestinal barrier function can arise from multiple factors, including as a consequence of a hypo-perfused gut that allows gut microbiota components to be translocated into the bloodstream.¹⁵ Epithelial dysfunction impairs absorption of sugars, protein, and fat, which may have an adverse effect on nutritional status and contribute to cachexia, further complicating cases of advanced cardiovascular diseases such as heart failure.¹⁶ Also, imbalances in the microflora of the GUT can create elevated levels of TMAO – or trimethylamine N-oxide – which is implicated in increasing the risk of cardiovascular diseases, including myocardial infarction, stroke, left ventricular diastolic dysfunction and risk of death.^{17,18} TMAO is being used as a novel biomarker for predicting CVD risk. In patients admitted to the hospital with acute decompensated heart failure (HF), elevated plasma TMAO levels correlate with decreased renal function and can

⁹ Wang F, et al. Gut homeostasis, microbial dysbiosis and opioids. *Toxicol Pathol.* 2017;45(1):150-56.

¹⁰ Centers for Disease Control (CDC). www.cdc.gov. Accessed Feb 2021.

¹¹ Heidenreich P.A., et al. Forecasting the Future of Cardiovascular Disease in the United States: A Policy Statement from the Am. Heart Association. *Circulation.* 2011;123:933–944.

¹² Heidenreich P.A., et al. Forecasting the Future of Cardiovascular Disease in the United States: A Policy Statement from the Am. Heart Association. *Circulation.* 2011;123:933–944.

¹³ Kamo T, et al. Novel concept of a heart-gut axis in the pathology of heart failure. *Korean Circ J.* 2017;47(5):663-669.

¹⁴ Kallio KAE, et al. . Endotoxemia, nutrition, and cardiometabolic disorders. *Acta Diabeto.* 2015;52:395-404.

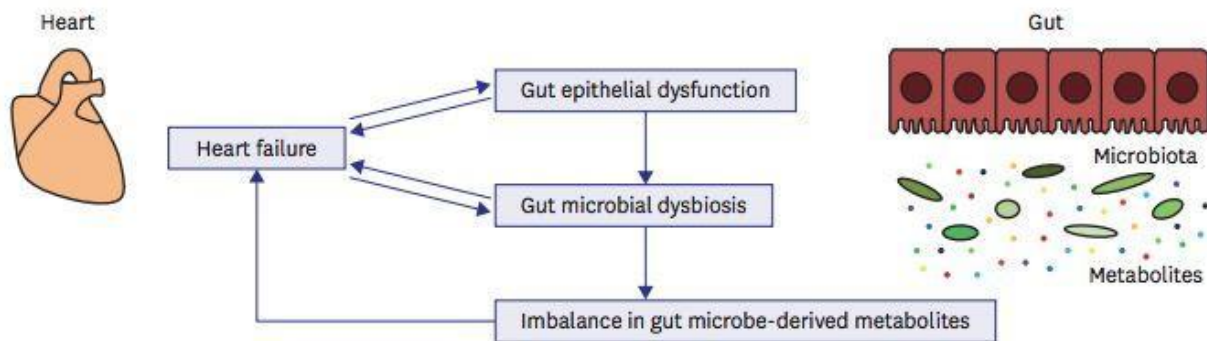
¹⁵ Tang WHW, et al. Dietary metabolism, the gut microbiome and heart failure. *Nat Rev Cardiol.* 2019;16(3):137-54.

¹⁶ Sandek A, Bauditz J, Swidsinski A, et al. Altered intestinal function in patients with chronic heart failure. *J Am Coll Cardiol.* 2007;50:1561–1569.

¹⁷ Tang WH, Hazen SL. The contributory role of gut microbiota in cardiovascular disease. *J Clin Invest.* 2014;124:4204–4211.

¹⁸ Tang WH, Wang Z, Levison BS, et al. Intestinal microbial metabolism of phosphatidylcholine and cardiovascular risk. *N Engl J Med.* 2013;368:1575–1584.

predict an increased risk of death or rehospitalization due to HF.¹⁹ Alterations in secondary bile acid production and short chain fatty acids production (SFCAs - namely propionate, malonate, butyrate, 2-hydroxyisobutyrate (2-HIBA), β -hydroxybutyrate, crotonate, succinate, and glutarate) and their cognate acylations (propionylation, malonylation, butyrylation, 2-hydroxyisobutyrylation, β -hydroxybutyrylation, crotonylation, succinylation, and glutarylation) are also correlated with an increased risk of HF.²⁰



Research reports that IgG antibodies produced by foods and improper dietary habits directly affects the circulatory systems and are directly linked to an increase in CVDs.²¹

Food Allergies/Sensitivities and CVDs

There is a biochemical and immunological basis of atherosclerosis and vascular inflammation linked to IgG food sensitivities.²² Gluten free diets have mixed reports of risks in CVDs, but it's reasonable for patients with predisposition to CVDs and IBDs to delete gluten from the diet.²³

A 2019 study (n=63 patients /with CVDs) reported those w/ CVDs have more frequent intolerances to banana, dairy and eggs.²⁴ In another study (n=60 - 30 obese children, 30 normal weight), the researchers wanted to answer the question “Do IgG antibodies increase risk of

¹⁹ Suzuki T, Heaney LM, Bhandari SS, Jones DJ, Ng LL. Trimethylamine N-oxide and prognosis in acute heart failure. *Heart*. 2016;102:841–848.

²⁰ Jia Q, et al. Role and effective therapeutic target of GUT microbiota in heart failure. *Cardiovasc Ther*. 2019; 2019:5164298.

²¹ Tsantsaridou A, et al. Association of food intolerance with coronary artery disease. *J Food Nutr Res*. 2019;7(1):71-81.

²² Tsantsaridou A, et al. Association of food intolerance with coronary artery disease. *J Food Nutr Res*. 2019;7(1):71-81.

²³ Potter MDE, et al. Effect of the gluten-free diet on cardiovascular risk factors in patients with coeliac disease: a systematic review. *J Gastroenterol Hepatol*. 2008;33(4):781-91.

²⁴ Tsantsaridou A, et al. Association of food intolerance with coronary artery disease. *J Food Nutr Res*. 2019;7(1):71-81.

vascular inflammation and atherogenesis?”²⁵ The results of the study reported that obese juveniles, compared to normal weight, reported significant increase in:

- Intima media thickness IMT
- Elevated CRP values
- **Anti-food IgG antibody concentrations**

The authors concluded that IgG food antibodies positively associate with intima media layer (IMT) thickness of carotid arteries in obese juveniles and is associated with increased risk of atherosclerosis.

Conclusion

Testing for IgE, IgG, Ig G4 and complement is very important for clinicians treating ~~with~~ cardiovascular patients. Certain foods may trigger GUT microbiome issues that then disrupt HEART-GUT signaling and lead to chronic inflammation, increasing the risk of CVDs.

Recommending a diet that minimizes foods that provoke these responses will decrease many types of inflammation and symptoms and is foundational to wellness. When we are recommending eating a less inflammatory diet, we are optimizing our patient's chance for success and improved health and vitality. Remember that HPA axis dysregulation due to chronic stress and imbalanced cortisol levels is directly related to GUT issues, so take that into consideration when issuing a treatment plan. Proper diet, stress control and sleeping 7-8 hours a night results in stronger immunity and better protection for the body from many factors including foods that irritate the GUT. Also, the prudent use of dietary supplements for those with CVD's is imperative, especially considering the DIND (drug-induced nutrient depletion) AND the DIMD (drug induced microbiome disruption) that most cardiovascular and ancillary drugs can cause.

²⁵ Wilder-Trusching M, et al. IgG antibodies against food antigens are correlated with inflammation and intima media thickness in obese juveniles. *Exp Clin Endocrinol Diabetes*. 2008;116(4):241-5.