



CALL FOR PAPERS

Gastrointestinal Inflammation is a complex biological response to injury as a result of different stimuli such as pathogens, damaged cells, or irritants. Symbiotic microbiome in digestive tract is considered to protect gut by removing harmful stimuli and to enhance healing process. Altered microbiome or absence can lead to exacerbated type 2 immunity and allergic/infectious inflammations including parasites. Thus, the microbiota regulates type 2 responses and acts as a key element in harmonizing immune responses at mucosal surfaces. While the mechanism by which microbiota regulates type 2 immunity is unclear, it is known as a strong inducer of proinflammatory T helper 17 cells and regulatory T cells (Tregs) in the intestine.

The signals at the sites of inflammation mediate rapid cell recruitment and differentiation in order to remove inflammatory inducers and promote tissue homeostasis restoration. However, persistent inflammatory stimuli or dysregulation of mechanisms of the restoration can lead to chronic inflammation. Different stressors can affect immune system and increase risk for infectious diseases, such as gastritis in post infectious IBS, and vice versa. As IBS patients have increased susceptibility to develop infectious gastroenteritis. Various viral (e.g., norovirus), microbial (e.g., *Campylobacter jejuni*, *Clostridium*, *Mycobacterium*), and parasitic agents (e.g., *Giardia*, helminths) are known to be involved in the development of chronic inflammatory bowel diseases. Yet, the mechanisms of action are not well known and there is no available cure. Additionally, nutritional elements, such as antioxidants, probiotics, and prebiotics directly and indirectly modulate GI immunity. Diets high in fat change the populations of innate microbiome in digestive tract and alter signaling to the brain and satiety, leading to obesity and inflammation.

We are inviting investigators to submit original research papers and review articles that will stimulate the continuing efforts in following areas.

Potential topics include, but are not limited to:

- ▶ Studies of the mechanisms of inflammatory pathways and microbiome involved in GI diseases (e.g., gastritis colitis, hepatitis, and pancreatitis) in patients and experimental models
- ▶ The role of altered microbiome/ infectious agents and mechanisms of interactions between various mediators involved in regulating the immune response in GI inflammation
- ▶ Breakthroughs in GI repair management and anti-inflammatory properties: biological, chemical, pharmaceutical, and nutraceutical ones
- ▶ New challenges in the effect of antibiotics and antibiotic resistance on GI microbiome and inflammation
- ▶ Translational preclinical and clinical pilot studies regarding the feasibility and safety profiles of anti-inflammatory compounds, biological agents relevant to GI inflammatory disease
- ▶ Elucidation role/mechanism of microbiome, nutrients, and metabolites during acute or chronic GI inflammation and malignancies
- ▶ New findings relevant to a specific nutrient or food component intervention alone or in combination on the modulation of GI immunity in health and disease

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First Round of Reviews

Friday, 22 July 2016

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Friday, 16 September 2016