Gut microbiota metabolites in human health and disease

Guest Editor

Assoc. Prof. Robert A. Olek
Department of Athletics, Strength and Conditioning, Poznan University of Physical Education, Krolowej Jadwigi 27/39, 61-871, Poznan, Poland
robert.olek@aol.com; olek@awf.poznan.pl

Message from the Guest Editor

Dear Colleagues,

Metabolites produced by the gut microbiota can modulate the health status of the host, and thus alterations in the microbiome are increasingly recognized as a major pathogenic factor in the development and progression of various diseases. Several pathways and potential mechanisms which may regulate the gut microbiome are therefore currently under investigation.

One of the metabolites produced by the microbiome is trimethylamine (TMA). TMA is derived from dietary nutrients such as choline, betaine, or carnitine, and is further metabolized by hepatic flavin monooxygenases into trimethylamine N-oxide (TMAO). In healthy subjects TMAO is efficiently excreted in the urine, hence the level of circulating TMAO is regulated by intestinal microbiome TMA production, hepatic conversion, and renal filtration.

Elevated serum levels of TMAO were first associated with an increased risk of cardiovascular disease. Many subsequent studies showed that TMAO promotes inflammation and induces oxidative stress. Recently, TMAO has been proposed to act as a gut microbiota metabolite involved in the development of metabolic and neurological diseases, as well as cancer.

Although some data indicate that TMAO is deleterious, other studies support it having beneficial effects. TMAO may prevent the aggregation of proteins associated with neurodegenerative diseases, including Parkinson’s and Alzheimer’s disease. Moreover, the effects of TMAO may differ between healthy and diseased conditions.

The goal of this special issue is to collate clinical and animal studies that link oxidative stress or inflammation with gut microbiota metabolites. Investigators are encouraged to submit original research and reviews aimed at increasing our knowledge in regard to TMA and TMAO function in health and disease.
Assoc. Prof. Robert A. Olek

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Submission Deadline: 31 December 2023
Submission: https://www.fbscience.com/Landmark
Science Citation Index Expanded: 4.009 (2020)
Contact us: Front.Biosci.Landmark@fbscience.com