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COMMENTARY

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Role of Gut Microbiota Metabolites in Molecular Pathophysiology: Mechanisms, Pathways and Diseases

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About the Study

One of the most important modulators of host physiology and pathology is the gut microbiota, a dynamic and varied collection of bacteria that live in the human gastrointestinal tract. It is becoming better known that the metabolites generated by the gut microbiota have a significant impact on immunological responses, metabolic processes, and cellular pathways, hence changing molecular pathophysiology. These metabolites, which include polyamines, bile acids, short-chain fatty acids, and derivatives of amino acids, have systemic effects that can change molecular signaling pathways and influence health or disease states. Gut microbiota-derived metabolites act as signaling molecules that influence host molecular mechanisms through a variety of pathways, including the regulation of gene expression, post-translational modifications, and receptor activation. For instance, metabolites can bind to receptors such as G-Protein-Coupled Receptors (GPCRs) and nuclear receptors, which mediate downstream signaling events. This receptor activation results in the modulation of cellular processes like proliferation, apoptosis, autophagy, and differentiation. Additionally, microbiota metabolites can interact with host enzymes and signaling molecules to modify pathways involved in inflammation, energy metabolism, and oxidative stress, all of which are central to molecular pathophysiology.

The interplay between gut microbiota metabolites and host immune signaling pathways shows the importance of these molecules in maintaining immune homeostasis. For example, certain metabolites have been shown to influence the activation and differentiation of immune cells, such as T lymphocytes, macrophages, and dendritic cells, by modulating cytokine production and signaling pathways. Dysregulation of these processes can contribute to chronic inflammation, autoimmunity, or immunodeficiency. Microbial metabolites, through their interaction with immune receptors like Toll-Like Receptors (TLRs) and inflammasomes, develop the adaptive immune system and generate innate immunological responses. Therefore, a key factor in determining sensitivity to inflammatory conditions, cancer, and infectious diseases is the functional interaction between immune signaling networks and microbiota metabolites. Gut microbiota-derived metabolites are also deeply involved in metabolic regulation, which is important for maintaining cellular and systemic homeostasis. These metabolites modulate energy production, nutrient sensing, and lipid metabolism through pathways such as AMPK signaling, mTOR (mammalian Target of Rapamycin) activation, and insulin signaling. For instance, the regulation of host glucose and lipid metabolism by gut-derived metabolites has been linked to metabolic disorders such as obesity, type 2 diabetes, and fatty liver disease. Dysbiosis of the gut microbiota can change the production of specific metabolites, which in turn disturbs these metabolic pathways and contributes to disease progression. Microbial metabolites also affect mitochondrial function and biogenesis, thus regulating cellular energy production and redox balance.

Oxidative stress and redox signaling are additional mechanisms through which gut microbiota metabolites influence molecular pathophysiology. By modulating the production of Reactive Oxygen Species (ROS) and antioxidant responses, microbiota-derived metabolites regulate cellular redox homeostasis. While a certain level of ROS production is need for cellular signaling, excessive oxidative stress can damage cellular components, including lipids,

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proteins, and DNA, thereby contributing to disease pathogenesis. Microbial metabolites can either promote or alleviate oxidative stress, depending on their nature and concentration. Another important mechanism through which gut microbiota metabolites influence molecular processes is epigenetic regulation. Microbial metabolites can modulate histone modifications, DNA methylation, and non-coding RNA expression, thereby regulating gene expression in host cells. For example, certain short-chain fatty acids act as Histone De Acetylase (HDAC) inhibitors, leading to changes in chromatin structure and transcriptional activity. These epigenetic modifications play an important role in cellular differentiation, proliferation, and stress responses. Dysregulation of these processes, often due to imbalances in microbial metabolite production, has been implicated in the development of various diseases, including cancer, neurodegenerative disorders, and metabolic syndromes. By influencing epigenetic regulation, gut microbiota metabolites act as key modulators of host gene expression and contribute to the underlying molecular pathophysiology of diseases.