

Proceedings

# The Interactions Between the Mechanistic Target of Rapamycin (mTOR) and the Microbiome

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**Abstract:** Type 2 Diabetes mellitus (T2DM) is one of the most prevalent diseases and is associated with several co-morbidities, including obesity, metabolic syndrome, and cardiovascular complications. The mechanistic Target of Rapamycin (mTOR) is a highly conserved protein kinase that integrates inputs from nutrients, insulin, growth factors, and environmental cues to control cellular metabolism. mTOR is dysregulated in insulin resistance and T2DM. Recent studies reveal that the gut microbiome interacts with mTOR to modulate nutrient absorption and homeostasis, but the mechanisms are not understood. mTOR nucleates two distinct complexes, namely mTORC1 and mTORC2, to regulate cell metabolism. mTORC1 receives inputs from nutrients and transmits signals to promote anabolic cell growth and inhibit catabolic autophagy. While mTORC2 phosphorylates Akt downstream of the insulin signaling pathway to regulate cell proliferation. Both mTORC1 and mTORC2 complexes network with multiple signaling pathways to control cellular growth, metabolism, and homeostasis. Recent advances in genomics, proteomics, and metabolomics research enabled identifying several gut microbiota species. These big data resources shed light on the interactions between the gut microbiome, dysbiosis, and mTOR in diabetes, metabolic syndrome, and obesity pathogenesis. Given the pleiotropic effect of mTOR on nutrient-sensing and utilization and the proposed interaction between mTOR and the gut microbiome, we hypothesized that mTOR crosstalks with the gut microbiome to modulate nutrient metabolism. This presentation will summarize the new advance in mTORC1 and mTORC2 research and the interactive crosstalk with the gut microbiome.

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