Gut Microbiota and Glucose Homeostasis

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Introduction

The involvement of gut microbiota in type 2 diabetes has been recognized\cite{1,2}. Several *Clostridium or lactobacilli* species are reportedly enriched in type 2 diabetes\cite{1,2}. A study by Forslund et al. showed that gut microbes mediate the therapeutic effects of metformin through certain types of short-chain fatty acid production\cite{3}. Gut microbiota may therefore influence insulin sensitivity. Serum metformin levels of type 2 diabetes patients are positively correlated with *Escherichia* abundance, whereas they are negative correlation with *Intestinibacter* abundance\cite{3}.

Serotonin (5-hydroxytryptamine, 5-HT) is mainly synthesized, stored, and released from enterochromaffin cells within mucosal epithelia of the gut\cite{4}. Gut microbes regulate 5-HT levels in the colon and blood. Spore-forming bacteria (Sp) from the mouse and human microbiota promote 5-HT biosynthesis from colonic enterochromaffin cells, which supply 5-HT to the mucosa, lumen, and circulating platelets\cite{4}. The microbiota-dependent changes in gut 5-HT impact gastrointestinal motility and platelet function\cite{4}.

5-HT1B and 5-HT4 receptors, which are distributed in the enteric nervous system and smooth muscle in the gut, may be involved in the gut-mediated glucose homeostasis\cite{5,6}. Pharmacologic stimulation of serotonin 5-HT1B or 5-HT4 receptors increases plasma active glucagon-like-peptide-1 levels independently of feeding and improves glucose tolerance under the dipeptidyl peptidase-4 inhibition in mice\cite{5,6}. Although it remains unclear whether gut microbiota influence insulin secretion, the gut microbiota-5-HT axis may be a novel therapeutic target for type 2 diabetes in future.

References