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Gut Microbiota and Glucose Homeostasis

Katsunori Nonogaki*

Department of Diabetes Technology, Tohoku University of Graduate School of Biomedical Engineering, 6-6-11, Aramakiaza, Aoba, Aoba-ku, Sendai, Miyagi 980-8579, Japan

***Corresponding Author:** Katsunori Nonogaki MD, PhD, Professor, Department of Diabetes Technology, Tohoku University of Graduate School of Biomedical Engineering, 6-6-11, Aramakiaza, Aoba, Aoba-ku, Sendai, Miyagi 980-8579, Japan, Tel: +81-22-795-3865; Fax: +81-22-795-3865; E-mail: katsu@trc.med.tohoku.ac.jp

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Introduction

The involvement of gut microbiota in type 2 diabetes has been recognized[1,2]. Several *Clostridium or lactobacilli* species are reportedly enriched in type 2 diabetes[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[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[3].

Serotonin (5-hydroxytryptamine, 5-HT) is mainly synthesized, stored, and released from enterochromaffin cells within mucosal epithelia of the gut[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[4]. The microbiota-dependent changes in gut 5-HT impact gastrointestinal motility and platelet function[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[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[5,6]. Although it remains unclear whether gut microbiota influence insulin secretion, the gut microviota-5-HT axis may be a novel therapeutic target for type 2 diabetes in future.

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