Diabetes Updates

Opinion

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A possibility to develop the innovative screening tool for risk group of type 2 diabetes mellitus (T2DM) onset using fecal microbiome metagenomic analysis

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By analyzing microorganisms which promote glycolysis, glucose metabolism modified transferase, glucose metabolism and energy metabolism and microorganisms involved in the expression of succinate dehydrogenase which activate mitochondrial function using fecal microbiome metagenome analysis, it may be a possible to develop the screening tool for risk group of type 2 diabetes mellitus (T2DM) onset. Developments of the screening tool for preliminary stage group of T2DM onset will be groundbreaking.

Currently, there are approximately 430 million people with type 2 diabetes mellitus (T2DM) worldwide, and it is estimated that there are approximately 800 million patients with T2DM and impaired glucose tolerance (IGT) worldwide.

Patients with T2DM and IGT are diagnosed by HbA1C and glucose tolerance tests. Advances in diagnostic methods such as serum glucoalbumin measurement and urinary inositol measurement have made it possible to more accurately diagnose IGT and postprandial hyperglycemia (So-called hidden T2DM) [1,2].

However, once T2DM onset, T2DM cannot be completely cured even if the blood glucose level and the condition of T2DM can be controlled by diet or drug treatments. The currently diagnostic methods for T2DM can diagnose patients with T2DM early stage and accurately, but it cannot be a preventive method for the onset of T2DM. To prevent the onset of T2DM, it is essential to develop a tool which can reliably diagnose the stage immediately before the onset of T2DM. The tool for screening a risk group of T2DM onset (preliminary T2DM group) has not yet been developed. If this diagnostic tool can be developed, it will be an epoch-making which can completely suppress T2DM onset.

I had reported the relationship between T2DM and intestinal microbiome [3]. In above report, microorganisms' analyses in feces were performed up to the genus level and functional analysis of these microorganisms were examined by metagenomic analysis. The report is summarized below; when the hyperglycemic state continues, microorganisms which promote glycolysis, glucose metabolism modified transferase, glucose metabolism and energy metabolism become predominantly recognized in the intestinal microbiome analyze. Then, when the hyperglycemic state becomes severe, the appearance of microorganisms involved in the expression of succinate dehydrogenase is observed. Succinate dehydrogenase activates the mitochondrial function of brown adipose tissue. Once blood glucose control by only microorganisms becomes difficult, the mitochondrial function of brown adipose tissue activates. By activating the mitochondrial function of brown adipose tissue, energy metabolism is promoted and it works to control blood glucose. It has been reported that succinic acid enters the mitochondria of brown adipose tissue and increases energy cost [4]. The dysfunction of mitochondria of brown adipose tissue has been reported in patients with T2DM [5,6]. I had reported that intestinal microbiome may play a role in maintaining homeostasis in living organisms [3]. It is conceivable that T2DM onset is triggered by mitochondrial dysfunction of brown adipose tissue. However, it is unclear whether patients with IGT have mitochondrial damage to brown adipose tissue.

I had proposed an idea of the screening tool for preliminary T2DM group using fecal microbiome metagenomic analysis in the previously report [3]. It is conceivable that these findings in fecal microbiome metagenomic analysis appear before diagnosis of T2DM by blood tests. In conclusion, I would like to propose to examine the above-mentioned microorganisms such as promote glycolysis, glucose metabolism modified transferase, glucose metabolism and energy metabolism, and microorganisms involved in expression of succinate dehydrogenase for the screening tool of preliminary T2DM group using fecal microbiome metagenomic analysis. I believe the development of this tool is feasible, and the development of this tool will be groundbreaking.

By the way, it has been reported that there is a transition from T2DM to dementia [7,8]. Developments of the screening tool for preliminary stage of T2DM onset will lead to the prevention of dementia, and it will become increasingly important or necessary in the future.

Conclusion

I proposed an idea of the screening tool for the risk group of T2DM onset (the preliminary stage group of T2DM) using fecal microbiome metagenomic analysis. Developments of this tool is epoch-making, which will lead not only to the prevention of T2DM but also to the prevention of dementia in the future.

References

1. Freitas PAC, Ehlert LR, Camargo JL (2017) Glycated albumin: a potential biomarker in diabetes. *Arch Endocrinol Metab* 61: 296-304. [Crossref]

2. Hong JH, Jang HW, Kang YE, Lee JH, Kim KS, et al. (2012) Urinary chiro- and myo-inositol levels as a biological marker for type 2 diabetes mellitus. *Dis Markers* 33: 193-192.

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3. Ohara T (2021) Type 2 diabetes mellitus (T2DM) and intestinal microbiota. *Diabetes Updates*.

4. Mills EL, Pierce KA, Jedrychowski MP, Garrity R, Winther S, et al. (2018) Accumulation of succinate controls activation of adipose tissue thermogenesis. *Nature* 560: 102-106.

5. Lee JH, Park A, Oh KJ, Lee SC, Kim WK, et al. (2019) The role of adipose tissue mitochondria: Regulation of mitochondrial function for the treatment of metabolic diseases. *Int J Mol Sci* 20: 4924. [Crossref]

6. Pinti MV, Fink GK, Hathaway QA, Durr AJ, Kunovac A, et al. (2019) Mitochondrial dysfunction in type 2 diabetes mellitus: an organ-based analysis. *Am J Physiol Endocrinol Metab* 316: E268-E285.

7. Lyu F, Wu D, Wei C, Wu A (2020) Vascular cognitive impairment and dementia in type 2 diabetes mellitus: An overview. *Life Sci* 254: 117771.

8. Arnold SE, Arvanitakis Z, Macauley-Rambach SL, Koenig AM, Wang HY, et al. (2018) Brain insulin resistance in type 2 diabetes and Alzheimer disease: concepts and conundrums. *Nat Rev Neurol* 14: 168-181.