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Successful application of a probiotic supplement in patients with type 2 diabetes mellitus

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Introduction

Type 2 Diabetes Mellitus (T2DM) is a progressive metabolic disorder characterized by persistent hyperglycemia resulting from the combined effects of insulin resistance in peripheral tissues, pancreatic β-cell dysfunction, and chronic low-grade systemic inflammation, which together impair glucose and lipid metabolism and predispose patients to long-term microvascular and macrovascular complications. Genetic predispositions and environmental factors such as obesity exacerbate insulin resistance and β-cell stress, while chronic inflammatory processes—driven by cytokines and immune cell infiltration in adipose tissue, liver, and pancreatic islets—further impair insulin signaling and β-cell viability [1-3]. In addition to conventional pharmacological and lifestyle therapies, modulation of the gut microbiota has emerged as a promising adjunctive approach to improve glycemic control and reduce cardiometabolic risk [4-6].

Probiotics, defined as "live microorganisms that, when administered in adequate amounts, confer a health benefit on the host," may favorably influence metabolic regulation. The beneficial effects of probiotics in the context of diabetes mellitus are mediated through multiple mechanistic pathways at the interface of the gut microbiota, intestinal barrier, metabolism, and systemic inflammation. A first key mechanism is the restoration of intestinal barrier function. In type 2 diabetes, dysbiosis and reduced abundance of beneficial lactic acid bacteria are associated with increased intestinal permeability and elevated serum Lipopolysaccharide (LPS) concentrations, leading to metabolic endotoxemia and low-grade systemic inflammation. Probiotic supplementation has been shown to enhance the expression of tight junction proteins such as occludin and zonula occludens-1, thereby reducing LPS translocation and improving insulin sensitivity [7]. A second mechanism involves the production of microbial metabolites, particularly Short-Chain Fatty Acids (SCFA) such as acetate, propionate, and butyrate. Lactic acid bacteria cross-feed other commensals, increasing SCFA production. SC-

FAs activate free fatty acid receptors (FFAR2/FFAR3) on entero-endocrine L-cells, stimulating Glucagon-Like Peptide-1 (GLP-1) secretion and thereby improving insulin secretion and sensitivity [8]. Furthermore, probiotics may modulate bile acid metabolism. Many strains possess bile salt hydrolase activity, leading to altered bile acid pools and activation of nuclear receptor FXR and membrane receptor TGR5. This signaling pathway has been implicated in the regulation of glucose homeostasis, lipid metabolism, and energy expenditure [9]. In addition, probiotics exert immunomodulatory effects. Clinical and preclinical studies indicate suppression of NF-κB signaling, downregulation of proinflammatory cytokines such as TNF-α and IL-6, and upregulation of the anti-inflammatory cytokine IL-10 [10]. This is of particular relevance given the established role of chronic low-grade inflammation in the development of insulin resistance and β-cell dysfunction.

Finally, probiotics may directly protect pancreatic β-cells. Experimental studies have demonstrated reduced oxidative stress, decreased apoptotic signaling, and improved insulin secretion in models supplemented with specific strains such as *Limosilactobacillus reuteri* [11]. These mechanistic insights collectively suggest that probiotic interventions could ameliorate both insulin resistance and β-cell dysfunction through integrated metabolic, vascular, and immune pathways.

A different and apparently more direct mechanism of action has recently been introduced with Beat-2 (in some countries also branded as “Sugarburner”, DeFaire Medical, Uppsala, Sweden). BeaT-2 is a probiotic nutritional supplement containing selected lactic acid bacterial strains (Bacillus subtilis and Bacillus coagulans) that preferentially metabolize glucose and complex carbohydrates into carbon dioxide and water in the upper small intestine. This process reduces the post-absorptive glucose load without directly lowering blood glucose, thereby avoiding hypoglycemia in normoglycemic individuals and similar to the action of other probiotic supplements [6]. In a recent randomized, double-blind, placebo-controlled pilot study involving 40 patients with T2DM, BeaT-2 supplementation for 6 weeks led to significant improvements compared with placebo in almost all observation parameters associated with diabetes and/or metabolic syndrome (e.g. glycemic control(BeaT-2 vs. Placebo): HbA1c: -0.3% vs. +0.1%, fasting glucose: -14 % vs. +8 % (both $p<0.05$), insulin resistance (insulin: -17% vs. +14%, HOMA-IR: -26% vs. +21%, both $p<0.05$), ß-cell dysfunction (intact proinsulin: -40% vs. -2 %, $p<0.05$) and chronic systemic inflammation (adiponectin: +8% vs. -8%; $p<0.05$, hsCRP: -31% vs. +27%, n.s.)). In addition, observed changes in the lipid profiles and other parameters of metabolic syndrome were more favorable with BeaT-2 than with placebo. The supplement was well tolerated, with fewer gastrointestinal complaints than placebo. These findings suggest that BeaT-2 may be a useful addition to standard diabetes therapy [12,13].

The participants of the study were offered to continue with the supplement after the study, which was accepted by the vast majority of the subjects. This allowed for observing long-term effects of the supplement in light of specific and individual efforts of the patients e.g. to further improve glycemic control by lifestyle measures and further weight loss. Here we report on two interesting patient cases that occurred in the aftermath of the clinical study.

Case presentations

Case 1

Patient AW (female, 60 years old, type 2 diabetes for approximately 12 years, current therapy: metformin 1000 mg morning and evening, plus insulin glargine 24 U at bedtime; weight 78.3 kg, BMI 30.6 kg/m², HbA1c 6.9%). During the study, the patient first received placebo, and after crossover was treated with BeaT-2. With the start of the supplement in the second part of the study, a period of marked weight loss began (-5.1 kg) accompanied by improvement in glycemic control (HbA1c: 6.5%), which motivated her to continue supplementation after the study ended. Over the following 12 months, she experienced further continuous weight reduction to 67 kg (BMI 26.2 kg/m²) and continued improvement in HbA1c to 6.1% (Figure 1).

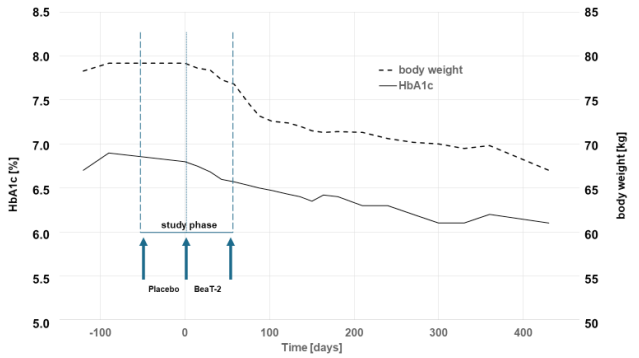

Figure 1: Patient case 1: Development of glycemic control and body weight after initiation of Beat-2 in the clinical study and in the following 12 months.

Case 2

Patient BF (female, 56 years old, newly diagnosed diabetes, therapy: lifestyle modifications; weight 98.6 kg, BMI 36.2 kg/m², HbA1c 6.4%). Immediately after the study, this patient began an extensive weight loss program supported by liraglutide (Saxenda) and BeaT-2. This combination, along with lifestyle changes (increased physical activity) and dietary adjustments (low-carbohydrate diet), led to a reduction in body weight to 60.2 kg within 9 months. After discontinuation of liraglutide, her weight remained stable for the next 12 months with BeaT-2 supplementation alone (Figure 2). Notably, this patient had previously undergone a comparable weight loss effort two years earlier (also with liraglutide but without BeaT-2), in which she reduced her weight from 105.5 kg to 60.9 kg within 9 months. However, after stopping liraglutide at that time, her weight rapidly rebounded to baseline within one-year years. In contrast, following the recent intervention, she had maintained her target weight for over two years now with ongoing BeaT-2 supplementation (Figure 2).

Discussion/conclusion

The present cases add to growing evidence that targeted modulation of the gut microbiota may improve metabolic control in T2DM [4-6,14]. Increasingly, the gut is recognized as an important regulator of glucose and energy metabolism, with microbial composition and metabolic activity influencing nutrient absorption, systemic inflammation, incretin release, and insulin sensitivity. The observed benefits in the larger BeaT-2 trial—reduced fasting glucose and HbA1c, improved insulin sensitivity, and favorable changes in adiponectin—are mechanistically consistent with reduced carbohydrate absorption and subsequent lowering of insulin demand [12,13]. Importantly,

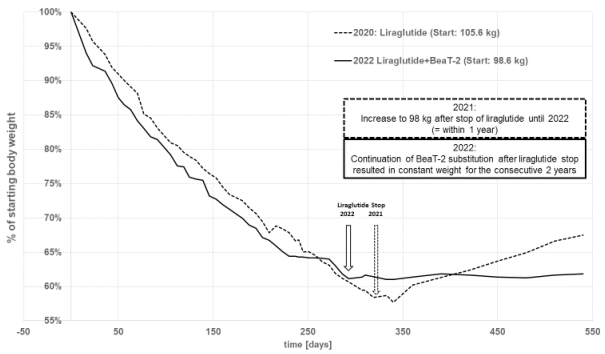

Figure 2: Patient case 2: Two efforts to losing weight in 2020 (with liraglutide) and 2022 (with liraglutide and Beat-2). While the patient immediately gained weight again back to baseline after stopping liraglutide in 2021, continuation of Beat-2 after the second successful weight loss attempt resulted in stable body weight for more than 2 years.

these improvements mirror known drivers of disease progression in T2DM, suggesting that modulation of microbial metabolism may address core pathophysiological processes rather than only symptoms.

Unlike α-glucosidase inhibitors, which retain carbohydrates in the intestinal lumen and often cause significant bloating and flatulence due to colonic fermentation [14,15], BeaT-2's enzymatic conversion of glucose to CO_2 and water in the upper ileum appears to minimize such gastrointestinal side effects [12,13]. By intervening earlier in the digestive process, BeaT-2 effectively reduces the glycemic burden without the drawbacks typically associated with delayed carbohydrate digestion, thereby offering a potentially more tolerable and patient-friendly approach to postprandial glucose control.

While the pilot trial was short in duration and included a relatively small cohort, its results are consistent with the hypothesis that probiotic supplements with defined metabolic activity may complement both pharmacological and lifestyle interventions [3,12-14]. Beyond improvements in glycemic parameters, the favorable changes in adiponectin and weight stabilization raise the possibility that such interventions could also mitigate associated cardiometabolic risk factors. In individual patients, as in the presented cases, BeaT-2 may therefore provide sustained improvements in metabolic control and support long-term weight management, thereby addressing two of the major challenges in T2DM care.

Ultimately, larger, long-term, placebo-controlled studies will be required to confirm these promising findings, clarify the durability of the effects, and better define the patient populations most likely to benefit. If validated, targeted microbiota modulation could emerge as a novel adjunctive therapy, positioned between lifestyle modification and conventional pharmacotherapy, contributing to a more personalized and mechanism-based approach to T2DM management.

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