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THE IMPORTANCE OF GUT MICROBIOTA IN PERSONALIZED APPROACH FOR OBESITY

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Summary.
Introduction. The impact of intestinal microflora on various processes, including metabolic, immune, and inflammatory processes, has been proven. Considering an individual's intestinal microbiome profile could influence the personalized approach to obesity management.
Materials and methods. A conducted literature review used the PubMed and Google Scholar search engines to select full versions of articles in English from 2019 to 2025.
Results. Numerous studies confirm the importance of maintaining the variability of the gut microbiome in obesity management. Particular attention is given to personalized nutrition for managing or preventing the disease. Intestinal dysbiosis develops as a result of a diet low in fiber and high in fat and animal protein; a sedentary lifestyle; exposure to toxic substances (pesticides), etc. Dysbiosis induces epigenetic changes, such as DNA methylation, non-coding RNA, and chromatin remodeling having negative effects on the epigenome and causing disturbances in carbohydrate and lipid metabolism. Individual restoration of intestinal balance is possible through molecular-genetic methods with the identification of intestinal microflora diversity and the subsequent implementation of personalized interventions.
Conclusion. The personalized approach to people with obesity is aimed at increasing the diversity of the intestinal microbiome through various methods. These interventions are based on personalized nutrition and on interventions such as activity regimens, administration of prebiotics, probiotics, postbiotics, or symbiotics, and fecal microbiota or bacteriophage transplantation, in addition to many other surgical, therapeutic, and behavioral interventions for the personalized treatment of obesity.
Keywords: *"*obesity", "gut microbiota" and "personalized medicine".

Rezumat. Importanța microbiotei intestinale în abordarea personalizată a obezității.
Introducere. Impactul microflorei intestinale în multiple procese este dovedit, inclusiv în procesele metabolice, imune și inflamatorii. Considerarea profilului microbiomului intestinal individual ar putea influența abordarea personalizată în managementul obezității.
Materiale și metode. O analiză a literaturii de specialitate a utilizat motoarele de căutare PubMed și Google Scholar pentru a selecta versiunile complete ale articolelor în limba engleză din perioada 2019-2025.
Rezultate. Multiple studii confirmă importanța menținerii variabilității microbiomului intestinal în managementul obezității. Atenție deosebită se acordă nutriției personalizate în managementul sau prevenția bolii. Astfel disbioza intestinală se dezvoltă în rezultatul unei diete sărace în fibre, bogate în grăsimi și proteina animalieră, sedentarismului, expunerea la substanțe toxice (pesticide), etc. Disbioza induce modificări epigenetice precum metilarea ADN-ului, ARN-ului non-codant, remodelarea cromatinei, având efectele epigenomice negative provocând dereglări ale metabolismului glucidic și lipidic. Restabilirea individuală a echilibrului intestinal este posibilă prin metode molecular - genetice cu identificarea diversității microflorei intestinale și implementarea ulterioră a intervențiilor personalizate.
Concluzii. Abordarea personalizată a persoanelor cu obezitate este îndreptată spre creșterea diversității microbiomului intestinal prin diferite metode. La baza acestor intervenții se află nutriția personalizată și intervenții precum regimul de activitate, administrarea de prebiotice, probiotice, postbiotice sau simbiotice, și transplant de microfloră fecală sau bacteriofagi, pe lângă multe alte intervenții chirurgicale, terapeutice și comportamentale pentru tratamentul personalizat al obezității.
Cuvinte cheie: „obezitate", „microflora intestinală", „medicina personalizată".

Резюме. Значение кишечной микробиоты в персонализированном подходе в борьбе с ожирением.
Введение. Влияние кишечной микрофлоры на многие процессы, в том числе на метаболические, иммунные и воспалительные процессы, доказано. Учет индивидуального профиля кишечного микробиома может повлиять на персонализированный подход к лечению ожирения.
Материалы и методы. В рамках анализа литературы с использованием поисковых систем PubMed и Google Scholar были отобраны полные версии статей на английском языке за период с 2019 по 2025 год.

Результаты. Многочисленные исследования подтверждают важность поддержания вариабельности кишечного микробиома в борьбе с ожирением. Особое внимание уделяется индивидуальному питанию в рамках лечения или профилактики этого заболевания. Таким образом, дисбактериоз кишечника развивается в результате диеты, бедной клетчаткой, богатой жирами и животным белком, сидячего образа жизни, воздействия токсичных веществ (пестицидов) и т. д. Дисбиоз индуцирует эпигенетические нарушения, такие как метилирование ДНК, некодирующей РНК, ремоделирование хроматина, что приводит к негативным эпигеномным эффектам, вызывающим нарушения углеводного и липидного обмена. Индивидуальное восстановление кишечного баланса возможно с помощью молекулярно-генетических методов с идентификацией разнообразия кишечной микрофлоры и последующим внедрением персонализированных мероприятий.

Выводы. Персонализированный подход к людям с ожирением направлен на увеличение разнообразия кишечного микробиома с помощью различных методов. В основе этих вмешательств лежат персонализированное питание и такие меры, как режим физической активности, прием пребиотиков, пробиотиков, постбиотиков или симбиотиков, а также трансплантация фекальной микрофлоры или бактериофагов, наряду с многими другими хирургическими, терапевтическими и поведенческими вмешательствами для персонализированного лечения ожирения.

Ключевые слова: *“ожирение”, “кишечная микробиота”, “персонализированная медицина”.*

Introduction.

In the 21st century, obesity is a global health problem, a multifactorial comorbidity influenced by genetics, the environment, and social determinants [1,2]. While an unhealthy diet and sedentary lifestyle, together with polygenic risk factors, are major causes of obesity, recent studies suggest that gut microbiota also plays an important role [3]. The gut microbiome contributes to several functions, including metabolism, adiposity, homeostasis, energy balance, and appetite regulation [4]. Similarly, the gut microbiota modulates innate and acquired immunity, both locally in the intestinal mucosa and outside the intestine [5]. In particular, it establishes a complex microbiota-gut-brain axis (MGBA), which enables communication between the gut and the central nervous system, via metabolites that cross the blood-brain barrier, or via the vagus nerve [6].

Actually, the human gut microbiota is a complex ecosystem that resides within the gut and the composition is highly influenced by diet [7]. Among the multiple macronutrients complex carbohydrates, in particular, oligo- and polysaccharides of plant origin, are one of the preferable strategies to modulate the gut microbiota [8]. Microorganisms can produce metabolites from both exogenous dietary substrates and endogenous host compounds, such as short-chain fatty acids (SCFAs), indole derivatives, polyamines, group B vitamins, secondary bile acids, adenosine triphosphate (ATP), and other [7,9]. The main regulators of energy balance and body weight that are derived from microflora are SCFAs which control satiety and hunger by promoting the release of peptide YY (PYY), ghrelin, insulin and glucagon-like peptide 1 (GLP-1), directly involved in regulating appetite and insulin secretion [10]. SCFAs also contributes to mediating the expression and activity of appetite-suppressing hormones such as glucagon-like peptide and YY peptides [11]. Increased intake of plant fiber has been reported to play a role in preventing obesity by increasing the intestinal production of SCFAs [12].

The resilience of the gut microbiota and symbiotic interaction with the host may be disrupted via intrinsic factors such as intestinal permeability, pH, mucus, and extrinsic factors including antibiotics, analgesics, psychotherapeutics and dietary components (trans fats, refined sugar, alcohol) [13]. Intestinal microflora dysbiosis in obesity impact adiposity and glucose metabolism, contribute to substantial reduction in microbial biodiversity, as evidenced by an increased Firmicutes-to-Bacteroidetes ratio [7,14,15]. Increased Firmicutes ratio (Ruminococcus, Clostridium, Eubacteria) is associated with production of pro-inflammatory cytokines, such as interleukins (IL), tumor necrosis factor (TNF) which are involved in low-grade chronic inflammation [6,16]. Chronic low-grade inflammation affects adipose tissue through the endless mobilization of immune cells, in particular macrophages, which infiltrate the tissue and secrete proinflammatory cytokines such as TNF-α, IL-1β and IL-6. These cytokines disrupt normal adipocyte function by inhibiting lipolysis, promoting fat accumulation and support insulin resistance [11,17]. So low-grade inflammation contributes obesity through a vicious circle between the immune system and metabolism [4].

Thus, the gut microbiota is an extensive and dynamic area of research that requires a personalized strategy for effective clinical application. Over time, scientists have attempted to assess bacterial diversity using methods ranging from classical fecal cultures to advanced genetic and molecular techniques such as 16S rRNA PCR [14]. These methods form part of the foundation of personalized medicine, enabling health

professionals to determine, with greater precision, which patients may benefit more from specific interventions [8].

Therefore, a priority of maintaining a healthy gut microbiome, put in practice through individualized therapeutic strategies, plays a key role not only in supporting overall health, but also in preventing and managing obesity. This is achieved by microbiota-dependent modulation of host physiological functions, even including changing gene expression and epigenetic regulation [19].

Thus, **the purpose of this study** was to determine the impact of gut microbiota in personalized management of persons with obesity.

Materials and methods.

Literature search strategy: The type of the study is systematic review, conducted to identify relevant studies on the topic. A comprehensive search of two electronic databases, PubMed and Google Scholar, was conducted from June 2019 till June 2025. The search strategy used a combination of relevant keywords and controlled vocabulary terms. The search keywords included "obesity", "gut microbiota", "personalized medicine or precision medicine".

Eligibility criteria: full articles in English with free version available. The review excluded abstracts of articles, articles in other language than English and studies that did not focus on the gut microbiota and obesity. To ensure the review was comprehensive, the reference lists of the included studies were manually examined for any other studies that met the eligibility criteria but were not captured by the electronic database search. The initial number of selected studies was – 362, the number of sources for the final evaluation was 28.

Ethical considerations: This review exclusively used publicly available data and did not involve human or animal subjects. Limitations: The review may be limited by publication bias because it only included published studies available in the specified databases. Furthermore, the quality of different research may vary, affecting the overall robustness of the findings.

Results and discussions.

According to recent studies, microorganisms and viruses begin to colonize the human intestine shortly after birth and will influence the composition of the microbiome throughout life. Thus, factors that favor richer colonization include the type of birth (natural vs cesarean), the diversity of the mother's vaginal microflora, the type of infant feeding (breast milk vs formula), the age at which dietary diversification occurred, and geographical factors [20]. It has been shown that at the age of three, the diversity of the gut microbiome becomes stable with the species Firmicutes, Bacteroidetes, and Clostridium [4,7,20,21]. At the same time, reports on fiber intake indicate that the gut microbiota of African children who consume a high-fiber diet is significantly enriched in Bacteroidetes and reduced in Firmicutes, compared to European children who consume a Western diet [18]. The guardian of intestinal balance is represented by bacteriophages, namely the crAssphage family, which constitutes approximately 90% of the intestinal virome sequence. As a result, research has shown that in healthy children of normal weight, the abundance of the crAssphage Alpha subfamily is higher compared to obese children, in whom the Delta subfamily predominates [20].

Currently, obesity is a comorbidity that has reached pandemic proportions. As a multifactorial condition, each patient suffering from obesity requires a detailed examination, starting with their genetic profile and continuing with their metabolism, lifestyle, environmental and psychological factors, analysis of any associated conditions, and many other factors. The personalized approach, also known as precision medicine, is a set of interventions based on the patient's needs, taking into account individual variability, all genetic, epigenetic, metabolic, and environmental factors, as well as many other factors [8]. The gut microbiota is a biomarker that enables personalized treatment. When incorporated into obesity treatment strategies, it provides more effective management. Examining how the microbiota is connected to nearly every system in the human body reveals its value and importance for health. Although we cannot change our genetic profile or predisposition to obesity, we can modify our lifestyle, eating habits, and environmental factors to promote healthy intestinal microflora and bring about epigenetic changes [13].

Several studies emphasize the importance of diet in obesity management [11, 23, 25]. However, diet plays a particularly significant role in developing the gut microbiome, which influences individual metabolism. This was observed in a group of 48 adults with a body mass index (BMI) greater than 25 kg/m² who consumed 21 g/day of oligofructose (inulin) for 12 weeks. There was reported weight loss, decreased ghrelin expression, increased PYY levels, low energy consumption, and low plasma glucose and insulin levels [11]. Another study reveals that subjects who had a high P: B (Prevotella: Bacteroides) ratio after implementing a high-fiber diet for 6 months lost more weight compared to subjects with a low P:B ratio. Moreover, after consuming a high-fiber diet for at least 3 days, scientists found

that people with the Prevotella enterotype improved their enzymatic capacity for fiber degradation and glucose metabolism, an effect that was not observed in subjects with the Bacteroides enterotype [22]. At the same time, comparing the effectiveness of whole grains with refined wheat (fiber intake: 33g/day vs. 23g/day), a weight loss of 1.8 kg was observed among participants with high levels of Prevotella [22].

It is noteworthy that the Bacteroides enterotype has been associated with a diet rich in animal protein and fat, while the Prevotella enterotype is linked to a diet rich in carbohydrates [18]. Bifidobacterium, especially Bifidobacterium adolescentis, has been seen to lower triglyceride and glucose levels in plasma, and it helps lower BMI and blood pressure by making more SCFAs [23]. In addition, SCFAs contribute approximately 10% of daily energy requirements and are responsible for nearly 75% of energy metabolism in the colonic epithelium [11]. Symbiotics are known to have a positive effect on blood sugar, lipids, and blood pressure control. Studies showed that in prediabetic patients, treatment with symbiotics improved FPG (fasting plasma glucose), FIL (fasting insulin levels), HbA1c (glycated hemoglobin) and lipid profile compared to the placebo group, while probiotics only influenced HbA1c levels [2]. According to recent studies, inulin-type prebiotics induce satiety, influence intestinal peptides involved in appetite regulation, and promote the growth of beneficial bacteria such as Bifidobacterium and Lactobacillus [1,2].

The gut microbiota is considered as “the second genome” and when it is unbalanced, the composition of microbial metabolites (such as SCFAs, LPS) influences the epigenetic mechanisms in a bad way like DNA methylation, non-coding RNAs and chromatin remodeling [13]. These epigenetic modifications can lead to host genome reprogramming by altering the transcriptional machinery of the cell in response to environmental stimuli, with potential implications on health status and disease development [18]. It has been reported that DNA methylation patterns are associated with gut microbiota profiles, especially the differential methylation of gene promoters linked to lipid metabolism and obesity [13]. Gut dysbiosis can disrupt DNA methylation by decreasing the production of SCFAs, which normally regulate gene expression through epigenetic mechanisms. It also induces low-grade inflammation and inhibits the uptake of key nutrients (e.g., folic acid, B12), leading to abnormal methylation patterns that affect genes involved in metabolism and immune function [11].

A close link has been found between the severity of obesity (BMI>35 kg/m2) and reduced numbers of intestinal bacterial cells and microbial genes potentially involved in biotin metabolism [9]. A cohort study of 1,500 subjects shows a decrease in genes involved in biotin biosynthesis and transport, as well as a reduction in microbial biotin producers (dominated by Proteobacteria and Bacteroidetes strains) and consumers (dominated by Firmicutes strains) [3]. However, biotin biosynthesis has been described as a distinctive feature of the microbiome enriched with Bacteroides [9]. The absolute potential for biotin biosynthesis strongly correlates with the abundance of bacterial species with complete biotin biosynthesis pathways and without genes involved in biotin transport, and this group is dominated by Proteobacteria and Bacteroidetes. In contrast, the absolute potential for biotin consumption correlates strongly with the abundance of bacterial species without biotin biosynthesis genes and with biotin transport genes (BioY), represented by Firmicutes [3,9].

Among sedentary adults with type 2 diabetes (T2D) or prediabetes, participation in a physical training program increased the Bacteroidetes phylum and reduced the Firmicutes/Bacteroidetes ratio. In addition, a decrease in the Clostridium and Blautia genera was observed. Systemic and intestinal markers of inflammation were also reduced, indicating a reduction in endotoxemia, which appears to be associated with a healthier microbiota [24].

It should be noted that fecal microbiota transplantation (FMT) is still in the clinical research stage. A meta-analysis from China (2022) reported that FMT in obese individuals has a limited effect on carbohydrate and lipid metabolism, insulin resistance, cholesterol levels, blood pressure, and inflammatory responses. Therefore, FMT may be a more suitable therapeutic strategy for patients infected with Clostridium difficile [25]. Treatment with FMT achieved a cure rate of 78,1%–94,8% among patients with CDI (Clostridium difficile infection) [20]. Similarly, another group reported that improving effects could be observed in approximately 83.1% of patients with CDI after FMT treatment during the 3-month follow-up period [26]. Although there is still limited clinical research and approximately 14.3% of cases fail, FMT treatment offers a promising personalized treatment option for obesity and metabolic syndrome, liver disease, dermatological conditions, systemic lupus erythematosus and immunodeficiency states [20,26].

One of the major factors determining the alteration of the intestinal microflora is diet, as observed in an experiment involving pregnant laboratory mice that became obese after two weeks of feeding a diet rich

in fat/sugar. This diet-induced obesity was associated with reduced levels of Bifidobacterium and Lactobacillus spp. and increased levels of Clostridium and Methanobrevibacter spp. Diet-induced obese females also had higher levels of blood glucose, plasma insulin, and fasting plasma leptin (produced in proportion to fat mass) and lower levels of PYY [11]. A high-fat diet (HFD) has a similar effect on the microbial metabolism of biotin. Significant decreases in plasma biotin levels were associated with obesity induced by a HFD (60% of calories from lipids), despite additional biotin intake in the diet compared to animals consuming standard feed (0.65 vs. 0.26 µg biotin/day for these diets, respectively) [9]. Exposure to pesticides for 12 weeks increased body fat and insulin resistance in laboratory mice, causing low-grade inflammation and metabolic syndrome. These changes also led to an increase in Firmicutes and Proteobacteria and a decrease in Bacteroidetes [4].

In recent decades, intestinal dysbiosis has been associated with type 2 diabetes, high levels of Bacteroidetes and Escherichia coli, and low levels of Clostridium, Roseburia, and Faecalibacterium. Since butyrate producers are reduced in patients with type 2 diabetes, it has been suggested that Clostridium, Roseburia, and Fecalibacteria species may protect against type 2 diabetes [19]. The abundance of Firmicutes is significantly reduced in type 2 diabetes mellitus, while the variety of Bacteroidetes is increased compared to the control group [17].

The leading view currently is that a healthy microbiota community is characterized by high taxonomic diversity, microbial gene richness, and a stable core microbiota. With the rapid development of new technologies, such as high-throughput sequencing, more attention has been given to the relationship between microbiome functions [7]. The gut microbiota ferment food ingested by the host to produce metabolites. At the same time, metabolites derived from the microbiota serve as substrates, regulate epigenetic modification enzyme activities, influence host gene expression, and trigger immune inflammation in intestinal epithelial cells (IECs), resulting in several metabolic disorders [13,28].

From a personalized medicine perspective, current research indicates that the gut microbiota could act as a predictor for weight loss, serving as a biomarker to predict successful clinical interventions in the future [8]. Also, to improve biomarker predictions, it's important to consider factors like gender, age, and eating habits [13]. The reviewed literature allows us to hypothesize that we can influence the microbiota with certain interventions such as high-fiber diets, calorie restriction, administration of prebiotics, probiotics, or postbiotics, fecal or bacteriophage transplantation to modify the microbiota and improve the outcomes of proposed therapies [26]. Overall, the integration of such covariates into predictive models will allow the creation of more rigorous associations and the definition of tailored treatments for individuals based on their gut microbiota configuration [8].

The epigenetic changes necessary for resetting the human genome in response to environmental stimuli are more relevant in childhood and may be linked to the colonization and development of the gut microbiota through birth type, breastfeeding, introduction of solid foods, infections, and antibiotic treatments [18]. Moreover, nutritional modulation of the gut microbiota can influence total energy intake, nutrient absorption, transport, and storage, which is further reflected in the host's overall metabolism, ultimately improving the person's health or, on the contrary, promoting weight gain. This is where the term "targeted health maintenance" comes in, when we can influence a person's health through the gut microbiota. For example, people who regularly eat a diet rich in fiber and polyphenols have a greater diversity of microbiota compared to consumers of a Western diet [8,14,16]. The types of protein consumed and the balance of amino acids can also influence gut microbial diversity. For instance, plant proteins are linked to higher concentrations of Bifidobacterium, Ruminococcus bromii, Lactobacillus, and Roseburia. In contrast, Bacteroides, Alistipes, Bilophila, and Clostridium perfringens are predominantly found in animal proteins. Concurrently, an increase in Bacteroidetes, Bifidobacterium, and a decrease in LPS (lipopolysaccharides) have been observed in response to soy protein intake [16]. It is known that Bacteroides and Propionibacterium species can convert protein into amino acids and their derivatives. Aromatic amino acids can be fermented into phenolic compounds similar to those produced by the decomposition of vegetables by intestinal microbiota [18].

Bifidobacterium and Lactobacillus can also produce lactate serving as a substrate for neuronal cells and prolonging postprandial satiety. In contrast, acetate and butyrate are produced by bacterial fermentation of non-digestible dietary fiber. Acetate is capable of activating the citric acid cycle in the hypothalamus and further changing the expression profile of neuropeptides that regulate satiety. Butyrate can influence the host's appetite and eating behavior by activating the vague nerve and hypothalamus, as butyrate has the ability to cross the blood-brain barrier [17].

From a scientific perspective, a low-carbohydrate diet leads to reduced levels of butyrate-producing

bacteria, whereas butyrate contributes to the regulation of food intake and energy expenditure by inducing the secretion of GLP-1 and PYY [27]. In addition, the decrease in Bacteroides and Lactobacillus in obesity leads to a reduction in bile acids, which inactivates the TGR5 (Takeda G protein-coupled receptor 5)/FXR (Farnesoid X Receptor) signaling pathway in brown adipose tissue, thereby reducing mitochondrial function, thermogenesis, and browning of white adipose tissue. At the same time, the presence of SCFA suppresses the secretion of FIAF (fasting-induced adipose factor) in the intestines, which inhibits certain catabolic processes, such as β-oxidation. Thus, intestinal dysbiosis in obesity results in higher energy intake and absorption and lower energy expenditure, which contributes to the progression of obesity [17].

Contemporary medicine is taking a personalized approach to obesity through microbiome sequencing. This creates an "internal biological map" that allows treatment to be tailored to the needs of each patient. Fecal microbiota transplantation is at the center of this approach [2]. However, insufficient and inconsistent evidence of FMT's direct effect on anthropometric parameters in obese populations does not prevent researchers from moving forward with promising discoveries regarding changes in glucose metabolism, insulin resistance, and inflammatory responses [25]. Various epigenetic drugs, histone deacetylase (HDAC) inhibitors, dietary supplements, probiotics, prebiotics, and FMT are currently being targeted for personalized obesity treatment [13].

Conclusion.

The personalized approach to people with obesity is aimed at increasing the diversity of the intestinal microbiome through various methods tailored to the patient's needs. These interventions are based on personalized nutrition and on interventions such as activity regimens, administration of prebiotics, probiotics, postbiotics, or symbiotics, and fecal microbiota or bacteriophage transplantation, in addition to many other surgical, therapeutic, and behavioral interventions for the personalized treatment of obesity.

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