Metabolic syndrome and intestinal microflora: what is in common?

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Intestinal microbes may manipulate host's nutrition behavior. J. Alcock et al. [4] indicate that the microbes can seriously influence the formation of eating behavior through microbiome-gut-brain axis. Microbes produce a variety of neurochemical products. The exact hormone analogues of mammals that are involved in mood and behavior [29, 44, 56]. More than 50% of dopamine and serotonin in the vast majority of the body have an "intestinal" source [40, 73]. The appetite-regulating hormones, yet another potential means to manipulate intestinal microflora eating behavior in mammals, including humans. The evolutionary conflict between the intestinal microbiome and the host may become an important factor in the obesity epidemic.

In 1947, Jean Vague first proposed to assess the phenotype of android obesity is a common symptom of metabolic disorders associated with type 2 diabetes and cardiovascular disease [84]. Many years later, international organizations and expert groups [1, 3] identified the major components of metabolic disorders, which are commonly associated with obesity and metabolic syndrome called them. Although these symptoms are different in terms of the number and type of criteria, the definition of the metabolic syndrome include obesity, impaired glucose homeostasis (e.g., type 2 diabetes mellitus, impaired fasting glucose, impaired glucose tolerance and insulin resistance), disorders of lipid homeostasis and the high risk of cardiovascular disease.

Diseases associated with obesity such as atherosclerosis, diabetes, non-alcoholic fatty liver disease, and certain types of cancer are the leading causes of preventable death worldwide. More and more evidence is accumulated intestinal microbiota participate in the formation of this serious disease. Microbiota function is just as important as the function of metabolic "bodies" that affect energy homeostasis and body weight control. Moreover, changes in the intestinal microbiotic landscape leading to increased intestinal permeability, endotoxin, which plays a role in the development of chronic inflammation in the host organism, promoting the development of obesity and related chronic metabolic diseases, such as nonalcoholic fatty liver disease. As a result, we are in the midst of a paradigm shift in our approach in the fight against the obesity epidemic. In this article we will try to illustrate the pathophysiology of obesity and metabolic syndrome, their relationship with the intestinal microbiome, and propose options for a potential intervention for treatment.

We are linked to microbes in the environment, and each person has a unique set of organisms (microflora) [6]. The most studied bacteria are found in the distal part of the human colon, where flora density reaches 10^{11}-10^{12} CFU/g. Total number of bacteria in the human gut exceeds the number of somatic cells in the body on the order of intestinal microflora biomass may reach 1.5 kg [33]. Thus, the intestinal

flora can be regarded as the multicellular body, similar in size and metabolic capabilities of the liver [57]. Furthermore, the combined intestinal bacterial genomes contain 100 times more genes than is encoded in the human genome [49], and these genes make a significant contribution to our physiological processes, including the metabolism of [72, 83].

For many years it was thought that the intestinal microflora of the infant should resemble those of the mother, because most species of bacteria acquired during delivery [45].However, this paradigm has been challenged by recent evidence obtained by means of molecular techniques that showed that children's stool samples of feces resemble their parents no more than the feces of other adults [15]. Microbial bowel landscape remains remarkably constant after conversion into the adult type of microorganisms. However, temporal changes may occur, and as shown recently by Ley et al., dietary factors can lead to long-term changes in microbiome [52]. This overall stability is determined by the formation of the intestinal immune system by infant microbiota, which is very individual [59] In this case, the intestinal microflora of one man may differ markedly from each other; greater diversity also manifests itself in the lining (epithelium) of the intestine membrane [18]. Comparative studies of adults with varying degrees of kinship showed that the genotype of the host is more important than diet, age and lifestyle in determining the composition of the intestinal microflora [32, 77].

The use of modern molecular techniques to study microbiome has shown that live in the human gastrointestinal tract is dominated by anaerobic bacteria three bacterial divisions: Gram-positive ***Firmicutes*** and ***Actinobacteria*** and gram ***Bacteroidetes. Firmicutes*** bacterium is the largest department, comprising over 200 genera, including species such as ***Lactobacillus, Mycoplasma, Bacillus*** and ***Clostridium.*** Departments ***Bacteroidetes,*** containing more than 20 genera of bacteria, and ***Actinobacteria*** also belong to the dominant intestinal microflora [31].

The increase in ***Firmicutes/Bacteroidetes*** ratio in obese patients remains a subject of debate. And esearch in humans have shown that the reduction in the number of ***Bacteroidetes*** (90%)***,*** and an increase in ***Firmicutes*** (20%) is directly linked to obesity and type 2 diabetes [12]. Reducing the number of ***Bifidobacterium*** observed in patients with overweight, obesity or type 2 diabetes [53]. Another type, which is reduced in diabetes mellitus type 2 is ***Faecalibacterium prausnitzi*** [17]. Interestingly, the level of ***Bifidobacterium*** and ***Faecalibacterium prausnitzii*** correlates with anti-inflammatory effects [8]. Moreover, subsequent studies have shown that the composition of microorganisms in childhood can predict the formation of metabolic syndrome [19]. The authors identified a higher level Staphylococcus aureus and lower levels of bifidobacteria in fecal samples of children who were recruited overweight. In a recent study, A. Vrieze et al. have shown that transplantation of intestinal microflora of healthy donor lean temporarily improves insulin sensitivity in patients with the metabolic syndrome [82]. These studies indicate that the composition and function of the intestinal microflora changes in obesity and type 2 diabetes before the researchers is the next question. Whether changes of intestinal microflora with diet or with the pathology of obesity? It should also be noted that the ratio of ***Firmicutes*** and ***Bacteroidetes*** is very inefficient,

because they contain large groups of pathogenic bacterial taxa such as ***Clostridium botulinum*** and ***Listeria,*** as well as species such as ***Eubacterium rectale*** and ***Faecalibacterium prausnitzii*** which is known to produce butyrate, being useful for the host. Therefore, a need for more standardized research, as well as taxonomically detailed description of modified microbial status.

In 2005 F. Bäckhead godu and colleagues have examined the role microbiome sterile mice [76], and found that the change in intestinal microbiome microbiota sterile mice with mice genetically determined obesity, led to the accumulation of them 60% fat, as well as development of insulin resistance during 2 weeks compared with sterile mice whose microbiome was unchanged. Subsequently, in 2006, Turnbaugh et al. confirmed these results, and further found that this feature can be inherited [5].

The composition and the microbiota metabolic effects play an important role in the energy component of the diet. In 2011, R. Jumpertz et al. identified the role of microbiota in regulating the absorption of nutrients by pyrosequencing of bacterial genes in faeces in 12 lean and 9 obese [25]. Researchers found that changes in the nutritional value of food received, against the background of changes in the microbiota, increased by approximately 150 kcal, i.e. microflora may play a significant role in the regulation of nutrient absorption. This phenomenon later became known as "lean genotype of the host."

Of interest are the results of studies by Ph.D. S.K. Panyushina et al. He argues that the main "energetically significant" metabolites of normal intestinal microflora are fatty acid (lactate, acetate, propionate, butyrate, succinate), alcohols, gases (hydrogen, carbon dioxide, methane). For example, the volatile fatty acids are substrates for energy eukaryotic cells (colonocytes intestinal mucosa, etc.) and other microorganisms. Since bifidobacteria (both types of bacteria predominant microflora microbiome) have heterofermentative lactic acid fermentation type and form 3 moles of acetate and 2 mol of lactate (average energy value of the organic acid 3 kcal/g) of 2 moles of recycled carbohydrate (energy value of 4 kcal/g). Thus, when disposing of 1 g prebiotic microflora and other substrates using an average of about 1 auxiliaries kcal (25%), and allocates the "common" in the form of metabolites about 3 kcal energy. Thus, the microflora of the digestive tract in the day only at the expense of "unrecorded" sources of energy creates about 2000 kcal. Furthermore, it should be added that the microflora can produce other forms of energy — the inclusion of additional gases and metabolite exchange water cycle of reactive oxygen species; energy potential difference between the total water and the border of the membrane with water; heat radiation (to warm the internal organs), mitogenetic radiation and others.

Also human normal flora microorganisms (intestine, skin, mucous membranes) are involved in providing a variety of host needs — in synthesis, metabolism, recycling, reclamation vast range and physiologically important signaling molecules (vitamins, hormones, amino acids, steroids, and immunoglobulins et al.); operate to inactivate and remove toxins; for complete digestion to release and activation of plant bioactive substances, i.e. perform specific functions that are not able to fulfill the system of the human body. Consequently, this "indispensable" work microflora due

to the "unrecorded" energy sources should also be offset against the creation of its energy contribution to the total energy of the microorganism. However, these indirect cascading energy functions microbiocenosis difficult to quantify [26].

Many of the nutrients in the human diet and subjected to enzymatic treatment are absorbed in the small intestine. However, the intestinal flora plays a central role in the metabolism of dietary fibers, which are not cleaved by human enzymes. Comparative studies of the intestinal microflora of mammals have shown that some species of bacteria they are common and so their presence is dependent on diet and lifestyle [16]. Herbivorous mammals have a more diverse microbiota than carnivores, as the metabolism of plant polysaccharides is more complex and demanding, which is also reflected in the extension of the intestinal tract and increase the transit time from herbivores. Metagenomic sequencing intestinal microflora showed that herbivores are more nitrogen assimilation genes into proteins in comparison with predators as amino acids is significantly less present in the diet of herbivores [16]. Similarly intestinal microflora vegetarians and vegans unable to metabolize carnitine present in red meat [38]. In humans, bacteria react differently to dietary components, and long-term eating habits were associated with an abundance of microbial genera: ***Bacteroides*** positively correlated with protein-rich diet, while ***Prevotella*** associated with a diet rich in fiber [43]. Thus, the intestinal microflora is essential for normal metabolism with diet may modify the intestinal microbial community of the body [43].

Metabolites of dietary polysaccharides, namely monosaccharides and short chain fatty acids (SCFA) produced by intestinal microflora by hydrolysis and fermentation. These metabolites are absorbed in the intestine, as a source of energy to the host. In 2012, H. Lin et al. assessed the impact of the introduction of SCFA mice, arguing that the RCMP regulate the production of hormones in the gut via the free fatty acid receptor 2 (FFAR2) and 3 (FFAR3), protecting against diet-induced obesity and the development of insulin resistance [9]. They found that SCFA, namely, propionate and butyrate stimulate intestinal hormones and reduce the overall saturation independently through FFAR3. The authors concluded that the stimulation of intestinal hormones and reduced energy extraction from the diet via propionate and butyrate may be a novel mechanism by which a host microbiota will regulate metabolism.

Microbial fermentation is a complex process of SCFA production. Methanogens in the intestine is believed to play a key role in the process of fermentation and are eventually suppliers SCFA which lead to an increase in weight, power consumption by increasing meal [71]. In 2006 BS Samuel and JI Gordon assess the impact on energy production through the host bacterial synthesis of polysaccharides and RCMP [68].

L. Conterno et al. analyzed the intestinal flora fermentation activity and found that it increases in obesity [58]. Subsequently, A. Schwiertz and his colleagues studied the quantitative fecal SCFA volunteers of normal weight (BMI = 18.5-24.9 $kg/m^{2;}$ n = 30), patients who are overweight (BMI = 25-30 $kg/m^{2;}$ n = 35), and in patients with obesity (BMI = > 30 $kg/m^{2;}$ n = 33) [53]. Researchers found that fecal SCFA concentration was significantly higher in patients who are obese (103.9 ± 34.3 mmol/l) compared to patients who are overweight (98.7 ± 33.9 mmol/L) and normal

weight (84.6 ± 22.9 mmol/l). These data show that the synthesis of SCFA in the gut higher in obese and overweight.

Lipoprotein lipase (LPL) plays a key role in the hydrolysis of triglycerides and the release of fatty acids for their transportation into adipocytes. After logging into adipocytes, these fatty acids are re-esterified in triglycerides and stored as fat. The secreted by adipose tissue, liver and intestines angiopoietin-4 antagonist activity of LPL (FIAF) prevents the accumulation of triglycerides and deposited as fat. F. Bäckhead et al. showed an increase in LPL activity in adipose tissue of 122% and the simultaneous reduction of expression FIAF with high statistical significance, leading to an increase in fat sterile mice [76]. Subsequently, F. Bäckhead et al. appreciated FIAF effect on limiting catabolism comparing susceptibility sterile FIAF-deficient mice with sterile mice wild-type [38]. In sterile FIAF-deficient mice were not resistant to western diet induces obesity compared with sterile mice wild type. Researchers were able to demonstrate a model in which the intestinal microflora FIAF inhibits expression in response to the excessive power, thereby increasing the LPL activity and the deposition of fat in adipocytes.

AMP-activated protein kinase (AMPK) is an enzyme that plays an active role in energy homeostasis. It is expressed primarily in the brain response, liver and skeletal muscle ratio changes to AMP (adenosine monophosphate)/ATP (ATP) or NAD^+ (nicotine adenine dinucleotide)/NADH (reduced nicotine adenine dinucleotide) that define metabolic stress. As a result, it increases the activity of AMPK, to compensate for power shortages condition by stimulating fatty acid oxidation, ketogenesis, glucose uptake and insulin secretion and, at the same time inhibited the synthesis of cholesterol, triglycerides, and lipogenesis [48]. Modified host microbiome inhibits AMPK, influencing the oxidation of fatty acids and is a predisposing factor in the formation of obesity and insulin resistance [85].

As previously discussed, the fermentation of carbohydrates of SCFA formed, which ultimately leads to the regulation of intestinal hormones such as glucagon-like peptide (GLP) and peptide YY (PYY). These intestinal hormones are responsible for the onset of satiety through the regulation of the synthesis of digestive enzymes [58]. Pharmacological and genetic studies have shown that the Y-2 receptor provides anorectic effects PYY3-36 [62] at the level of the hypothalamus, and brainstem vagal [61]. PYY3-36 modulates the activity of neurons in the hypothalamus of the brain stem, and which are involved in saturation. Thus, overeating may result from changes in the microflora and inhibiting the secretion of PYY3-36. Some data suggest that low levels of circulating PYY predispose to the development and maintenance of obesity [39] and the rapid saturation level and circulating PYY, negatively correlated with obesity markers. in addition, mice lacking giperfagikami and PYY are becoming obese. In contrast, chronic administration PYY3-36 obese rodents reduced degree of obesity. Transgenic mice with increased circulating PYY are resistant to diet-induced obesity. The sensitivity of obese subjects exposed to PYY-36 shows that administration of PYY microbiome changes may determine a new therapeutic strategy for the treatment of obesity.

RCMP signaling cascades are mediated by G-proteins, namely FFAR2 and FFAR3. Propionate, butyrate and acetate-derivative RCMP have a high affinity for

FFAR2 receptors [13].Role FFAR2 is advantageous conservation of energy, due to the stimulation of lipogenesis, inhibition of lipolysis and reducing energy consumption [71]. In the colon, FFAR2 and FFAR3 work in tandem, regulating intestinal peristalsis and saturation through GLP-1 (glucagon-like peptide-1) [12]. M. Bjursell et al. noted that FFAR2-deficient mice ($Grp41^{-/-}$), which are fed a high-fat diet had a significantly lower fat content, low weight, increase lean body mass, greater insulin sensitivity, lower triglyceride levels and cholesterol than their wild-type counterparts [36]. Researchers marked reduction of lipids in brown adipose tissue histologically FFAR2-deficient mice, which led to an increase in energy consumption and a high body temperature. Thus, reduction FFAR2 has a protective effect against obesity and dyslipidemia by increasing energy expenditure. BS Samuel et al., Evaluated the effect of intestinal microflora obesity comparing FFAR3-deficient mice to wild type mice ($Grp41^{+/+}$) [22]. The researchers noted that FFAR3-deficient mice had a significantly lower body fat mass and an increase in lean body mass relative to wild-type mice, an effect researchers attribute PYY expression reduction in FFAR3-deficient mice. The result of this was to stimulate gut motility and an increase in power consumption in conjunction reduced food intake, thereby created a dual effect on the energy balance. This has led investigators to conclude that a regulator FFAR3 energy homeostasis caused microbiome individual host.

Conventionalization of sterile mice leads to a sharp increase in hepatic lipogenesis, a process in which an excess of glucose is converted to fat [71]. In sterile mice increased glucose consumption activates protein binding carbohydrate-sensitive element (ChREBP), acetyl-CoA-carboxylase (Acc1), fatty acid synthase (FAS), and sterol-regulated enhancer element protein-1 (SREBP-1), which in turn increases adipogenesis and insulin concentrations [58]. It is important to further distinguish those paths which lead to the inhibition of lipogenesis and negative caloric balance as the potential for therapy.

Congenital disorders of immunity have an ambiguous impact on the formation of the metabolic syndrome. Toll-like receptors (TLRs) are a class of cellular receptors with a transmembrane fragment, which recognize conserved microbial structures and activate a cellular immune response. TLRs are involved in the pathogenic process of formation of diabetes by increasing blood sugar levels and non-esterified free fatty acids, release of cytokines and reactive oxygen species, resulting in a pro-inflammatory state, which leads to diabetes [14]. Toll-like receptor 5 (TLR5) is a protein that plays a key role in the activation of innate immunity through recognition of the pathogen through the microbe-associated molecular patterns (mAMPs) bacteria, viruses and fungi [78]. Pathogen-associated molecular pattern (PAMPs) through TLR5 leads to the induction of inflammatory cascades by transcription of various cytokines and inflammatory mediators. Thus, the interaction between the microflora and TLR5 is vital for intestinal homeostasis.

The majority of intestinal epithelial cell lines to respond to flagellin (bacterial protein that is able to self-organize into hollow cylindrical structure forming filaments bacterial flagella represented in considerable quantities in all flagellated bacteria.) To which TLR5 has high affinity. In response to TLR5 flagellin includes inflammatory cascade through a number of transcription factors, primarily the

transcription factor NF-κB (nuclear factor kappa-light-chain-enhancer of activated B cells), which controls the expression of immune response genes to enhance the immune protection and improve survival [66]. M. Vijay-Kumar et al. showed that TLR5-deficient mice ($TLR5^{-/-}$) are susceptible to the development of metabolic syndrome, including, insulin resistance, hypertension, and hyperlipidemia [51]. In addition, TLR5-deficient mice had hyperphagia, which resulted in increased obesity. Transplant microflora from TLR5-deficient mice to their sterile wild-type congeners led to the phenotypic manifestations of the metabolic syndrome. It is interesting to note that the limitation of power in TLR5-deficient mice prevented obesity, but insulin resistance has remained unchanged. The researchers concluded that the intestinal microflora contributes to the development of metabolic syndrome due to congenital disorders and the system may further promote its development.

Recent studies have shown that intestinal bacteria can initiates obesity and insulin resistance due to LPS activity that can cause inflammation by forming CD14 complex with TLR-4 (CD14 — membrane glycosylphosphatidylinositol-linked protein expressed on the surface of the myeloid series of cells, especially macrophages, receptor component complex CD14/TLR4/MD2, recognizing LPS on the surface of innate immune cells). The relevance TLR-4 due to metabolism was confirmed by the finding that reduction of TLR-4 reduces obesity caused by insulin resistance [80].

In another study of TLR-2-deficient mice was studied obesity, insulin resistance, and impaired glucose tolerance. Intestinal microflora in TLR-2-deficient mice had increased number of bacteria card ***Firmicutes*** and reduction ***Actinobacteria*** of the genus ***Bifidobacterium*** [56]. Amount Decrease TLR-2 in mice led to changes in the composition of their gut bacteria, which cause a high risk of diabetes. Thus d efitsit TLR-2 in the diabetic mice leads to an increase in the development of diabetic complications, such as diabetic nephropathy [41]. Perhaps intestinal microbiome plays a crucial role in regulating diabetic vasculopathies, these findings could be an important area of future research.

An important role in the formation of the metabolic syndrome, insulin resistance plays, which we have already mentioned in the article. Insulin is an important hormone for the regulation of glucose homeostasis and initiates its biological effect through the activation of the insulin receptor (IR) [55], which leads to the transport of glucose into muscle and adipose tissue, glycogen synthesis in muscle and liver, as well as lipogenesis in adipose tissue. This process may be disturbed by several mechanisms. One of the major mechanisms by which activates JNK (c-Jun-N-terminal mitogen-activated protein kinase), which plays an important role in the prevention of apoptosis in response to oxidative stress. In adipose tissue, and liver of mice continuously fed a high fat diet, JNK activity was significantly increased compared to lean animals [24].

Insulin activity can also be reduced by the modified secretion of cytokines and chemokines. For example, patients with type 2 diabetes, circulating T-cells produce higher levels of IL-17 and TNF-γ, which leads to proinflammatory effects in the body [23]. Other cytokines such as tumor necrosis factor (TNF)-α and interleukin (IL)-6, are also associated with insulin resistance. TNF-α increased in adipose tissue in

obesity and diabetes, and inhibition of its expression leads to an increase in the peripheral uptake of glucose. Similarly, the modulation of TNF-α-releasing cell surface leads to disruption of glucose metabolism and vascular inflammation [35]. IL-6 may also influence the insulin secretion: its plasma concentration is inversely proportional to the sensitivity to insulin [74].

High uptake of fat is also correlated with an increase in gram-negative/gram-positive bacterial factor [50]. These data also support the conclusion that the intestinal microbiota can be responsible for changes in metabolic state, and its disturbances lead to endotoxemia, and metabolic diseases. A diet with high fat in conjunction with antibiotics in mice reduced the plasma parameters LPS, reducing the appearance of the adipose tissue, inflammation, oxidative stress markers and macrophages, as well as hypertrophy of adipocytes prevented and improved metabolic parameters of diabetes and obesity [11]. Thus, dietary fats may be associated with increased absorption of LPS, which are caused by changes in intestinal microflora characterized by a decrease in the amount of Gram ***Bacteroides***-like bacteria representatives group ***Eubacterium rectale-Clostridium coccoides*** and bifidobacteria [81].

Although it is assumed that the insoluble fiber cereals are beneficial to the subsequent fermentation in the colon [64], an explanation of its reduced risk of forming diabetes is still unclear. Study by M.O. Weickert et al. showed that within the first six weeks on the diet with a high content of cereal fibers in overweight patients, observed increased sensitivity to insulin. However, in 18 weeks, no significant difference was observed as compared with the control and a diet high in protein [21]. Furthermore, grain diet was not accompanied by changes in the composition of the intestinal microflora or after 6 weeks, or 18 weeks after diet observations [10]. Thus, these effects may be other mechanisms are responsible.

It is also appropriate to say that most of the results using diet or drugs tend to get out of short-term studies, which makes it difficult to formulate conclusions for the person with the eating habits, which are present throughout most of his life. For example, metabolic guar gum consumption effects can be fully opposite, depending on the duration of its administration [20]. Moreover, it is unclear whether the results of studies using animal models to be applicable to humans, since they represent differences in composition of the intestinal microflora and the diet.

The best non-surgical strategy to reverse obesity in patients with metabolic syndrome should be small, but long-term changes in diet and physical activity, which will create a negative energy balance [42]. Scheme treatments that regulate energy balance, suggest that microorganisms can have a significant cumulative effect in the treatment of pathologies discussed. Integrated use of antibiotics, probiotics and prebiotics can lead to non-specific modulation of the gut microflora. The use of antibiotics is justified in severe change in the microbial landscape of the intestine, especially in combination with the syndrome of excessive intestinal bacterial contamination (SIBOS). To this end, antibiotics-enteroseptic wide spectrum of action are recommended, which include rifaximin. Studies show that rifaximin is highly effective at SIBOS in 80% of patients [1]. High doses of rifaximin (1200 or 1600 mg/day) are more effective than standard ones (600 or 800 mg/day) [30].

After obtaining encouraging results in animals [28, 69], the effects of the use of protected enteric probiotics (live bacteria that can carry out the colonization of the colon) and prebiotics (indigestible oligosaccharides such as inulin and oligofructose, which can enhance the growth of beneficial commensal organisms such as ***Bifidobacterium*** and ***Of Lactobacillus***) have been investigated in a number of controlled trials. In the randomized controlled trials (RCTs), do not exceed the duration of 6 months, with the participation of a small number of subjects (<50 participants) evaluated surrogate markers rather than clinical endpoints. In these trials investigated grounded mechanisms of action of probiotics and prebiotics, such as early satiety and decrease caloric intake, decreasing glucose and manifestations of systemic inflammation [37, 60, 75].

A new study by the company Danisco found that the probiotic strain of ***Bifidobacterium animalis*** (B420) can significantly increase metabolism, counteracting negative diet effects a high-fat diet. The results of this study showed that probiotic treatment resulted in a significant reduction in systemic inflammation and metabolic endotoxemia. M nogotsentrovoe double blind, randomized, placebo-controlled trial was conducted with the participation of 87 patients with a high body mass index, which received ***Lactobacillus gasseri*** (LG2055) in this study, the probiotic LG2055 was presented as more culture in yogurt, which has been added to the usual yoghurt cultures ***Streptococcus thermophilus*** and ***L. Delbrueckii*** (subspecies ***Bulgaricus)***; yogurt without LG2055 was used as placebo. The results of this study showed that the use of probiotic strain significantly reduced abdominal obesity, body weight, showing its beneficial effects on metabolic disorders. In a subsequent study, the same researchers evaluated degree and the level of visceral obesity SICAM-1 (soluble intercellular adhesion molecule-1) in blood as a marker of inflammation, which increases in obesity [34]. The results showed that the probiotic strain to inhibit the increase of visceral adipocytes and SICAM-1 regulates the blood.

In another study, patients with obesity were appointed ***L. rhamnosus*** PL60 and ***L. Plantarum*** PL62. [63]. In 8 weeks of ***L. rhamnosus*** PL60 intake subjects had decreased body weight due to a significant decrease in white adipose tissue, and the patients did not change feeding behavior. Polyphenol supplements for probiotics in the diet have also been suggested for weight loss [47]. These dietary strategies with probiotics and polyphenols may in the future be used to achieve and maintain a normal body weight in obese people.

Thus, it is possible to formulate probiotic effects on body weight, glucose homeostasis, lipid profile of the plasma and associated risk factors of cardiovascular disease [67] :

- modulation of intestinal microflora;
- induction of proliferation of enteroendocrine L cells and modulation of glucagon-like peptide-1, peptid-YY and greline;
- reduction of systemic inflammation and metabolic endotoxemia;
- modulation of systemic inflammation in obese people;
- significant reduction in abdominal obesity, body weight, beneficial effects on metabolic disorders;
- inhibition of accumulation of body weight and visceral fat;

- prevention of the increase in body weight and fat in the diet, causing weight gain;
- improvement of lipid profile.

When choosing a probiotic preparation, there are several problematic issues, the first of which is the survival of the bacteria. As mentioned above, only living microbes have probiotic properties. Moreover, a number of works have shown that a sufficient minimum dose capable of providing significant effect, the dose may be regarded as at least 10^7 CFU [67]. The survival rate of the bacteria depends on the production technology and product storage conditions. For example, the addition of bifidobacteria in yogurt can't guarantee their safety and ability to vegetation; viable microflora, both liquid and dry forms in simple formulations may be lost before an official date. For most probiotics, especially for liquid formulations require special storage conditions such as temperature. It is necessary to take into account the devastating effect of gastric juice on an unprotected flora. It is proved that only a small number of strains of lactic acid bacteria (***L. reuteri, L. NCIB8826 plantarum, S. boulardii, L. acidophilus, L. casei Shirota***) and bifidobacteria has acid resistance, while the majority of microbes are killed in the stomach. Therefore, probiotics are preferably enclosed in acid-resistant capsule. According to A. Bezkorovainy [7], only 20-40% of selective strains survive in stomach. D. Pochart [46] demonstrated that of 10^8 CFU lactobacillus taken acid resistant capsule in the intestine detected 10^7 CFU, after receiving the same amount in yoghurt — 10^4 CFU and after administration of the same dose in the clear powder microbes in the intestines are not detected at all.

In the small intestine probiotics are exposed to bile acids and pancreatic enzymes. Consequently, many microbes, for example, ***L. fermentum KLD, L. lactis MG1363*** are almost completely killed. This may be due to increased permeability of the cell membrane of bacteria, which occurs in response to bile acids. Survival of bacteria is most dependent on the manner in which they are taken: in a protective capsule, in the form of yogurt, milk or without any protection. Thus, according to K. Kailasapathy [70], many strains such as lactobacillus from dairy products do not reach the intestines or survive therein only a few days. These data cast doubt on the effectiveness of unprotected and non-acid resistant probiotics. Therefore, for correction of the intestinal microflora it is necessary to select only proven quality probiotics, which have all the protection degrees and "work" only in those sections of the intestine where it is needed.

Obesity and its related complications cause most of the damage of human health and have significant economic consequences on a global scale. The evidence presented by us, suggest that intestinal flora plays a key role in the regulation of energy homeostasis, development and progression of obesity and related metabolic disorders. Manipulations with intestinal flora may be a means for the potential of targeted therapy of metabolic syndrome. Regular consumption of a balanced set of substances pro- and prebiotic effects allows to restore qualitative and quantitative composition of microflora in the digestive tract, and as a result to reduce the risk of obesity, stabilize metabolism and energy exchange in people who are overweight due to intestinal dysbiosis and interrelated health problems.

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Metabolic syndrome and intestinal microflora: what is in common?

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Key words: intestinal microflora, metabolic syndrome, diabetes, obesity, probiotics

The article highlights the problem of the correlation between human intestinal microbiome and metabolic syndrome. Changes in bacterial intestinal proportions upon obesity captured the attention of scientists around the world, especially in relation to their effect on the metabolism. Increasing proportion of Firmicutes and Actinobacteria and decrease of Bacteroidetes are associated with increased levels of serum lipopolysaccharides, insulin resistance, weight gain, and other co-morbid manifestations of the metabolic syndrome. The mechanisms, underlying this interdisciplinary problem, are actively studied to optimize the prevention and treatment of obesity and type 2 diabetes.