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PECULIARITIES OF USING A NEUROVASCULARIZED FLAP ON THE SURAL ARTERY IN PLASTIC SURGERY OF GUNSHOT DEFECTS ON THE FOOT AND LOWER 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THE MICROBIOME AND METABOLIC DISORDERS: THE LINK BETWEEN THE GUT MICROBIOTA AND METABOLIC SYNDROME

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Abstract.

The diverse population of microbes that live in our digestive system, known as the gut microbiota, remains essential for many physiological processes. It plays a role in obtaining energy from food and controls both regional and overall immunity. In addition, changes in the microbiota of the digestive tract are connected to the emergence of an extensive variety of illnesses, such as cancer, gastrointestinal problems, and metabolic disorders. From a metabolic perspective, the gut microbiota can affect processes like lipid accumulation, lipopolysaccharide satisfaction, and short-chain fatty acid synthesis, all of which have an effect on food intake, inflammatory reactions, and insulin signaling. Prebiotics, probiotics, specialized anti-diabetic medications, and faecal microbiota implantation are a few of the ways that have been discovered to alter the gut microbiota; each has a different influence the human body's metabolism and the emergence of metabolic disorders. These therapies have been reported to be therapeutic strategies for enhancing general wellness and reestablishing a balanced gut flora.

Key words. Microbiota, Metformin, Gut microbiota (GM), obesity.

Introduction.

In recent years, the complex relationship between metabolic illnesses, notably metabolic syndrome, and GM has become one of the most fascinating areas of study. It was recently discovered that GM, a complex population of bacteria that live in our digestive system, has a significant impact on our metabolism, energy balance, and general health [1,2]. Fatness, insulin confrontation, dyslipidemia, and hypertension are all components of the metabolic syndrome. This is a significant risk indicator for the emergence of long-term conditions including type 2 heart attacks and diabetes. Discovering new therapeutic approaches and therapies to treat these pervasive and difficult illnesses offers great potential in light of the complex connection between metabolic syndrome and the gut microbiome [3,4]. The interesting interaction between the GM and metabolic syndrome is explored in this article, which also highlights recent research results, the mechanisms at play, and prospective directions for future studies and therapeutic approaches [5,6].

The host-microbiota interactions in both genders suffering from numerous forms of glucose metabolism disorders were examined in this pilot study [7]. In distinct metabolic states, they discovered that several bacteria species were either substantially increased or decreased.

In article [8], they explained about how the gut microbial community differs from that of healthy people, with potentially hazardous bacteria proliferating and helpful bacteria being inhibited. This difference is related to the clinical symptoms of MetS.

The current investigation [9], which was rendering to the test specimen dimension of about 7200 participants in an Eastern country, identified altered gut flora linked to MetS. These changes exhibited resemblance to those found in Western cultures and were phylogenetically preserved.

The purpose of the research was to look at how frequent kefir intake affected the makeup of the GM and how it related to the symptoms of the metabolic syndrome [11]. An examination of the GM revealed that frequent kefir intake only significantly increased the overall number of Actinobacteria (p=0.023). Kefir administration demonstrated positive impacts on some metabolic syndrome indicators, but more research is required to comprehend its effect on the genetic makeup of the GM.

This study [12] identified changes in the GM in a group of Mexican women who are healthy (CO), obese (OB), and metabolic syndrome (OMS) positive. By using high quantities DNA sequencing, they evaluated 68 women and described their anthropometric and biochemical characteristics as well as the variety of their gut microbes.

The present investigation [13] showed that LJPs prevented diet-induced obesity and enhanced body composition and lipid metabolism in mice receiving a high-fat diet (HFD). Such advantageous outcomes were linked to the manipulation of the GM, leading to a particular resembles typical microbiota in terms of composition. Changes in the GM may have an impact on how well nutrients are used and may control how lipids are metabolized via SCFA-dependent pathways.

In this investigation [14], the microbiota diversity of MetS patients receiving treatment was characterized, and the potential for employing GM as a marker of metabolic disorders was examined. A 16S rRNA metagenomic sequencing approach was used to analyses the GM of 111 MetS individuals from The Cohort of Individuals at a Significant Risk of Cardiovascular Events (CORE)-Thailand registries who had received treatment for the condition.
They constructed an inbred Wuzhishanminipig model of metabolic syndrome caused by an extended high-energy diet (MetS) [15], which is distinguished by its molecular support, small size, and resembling metabolism. The host transcriptome, GM, arterial plaques, metabolic variables, and abnormalities were all examined.

To examine the possibility of the connection between metabolic syndrome and GM, with a particular focus on how changes in the GM affect the onset and progress of metabolic illnesses. The human GM is extremely diverse and varies greatly from person to person. It is difficult to pinpoint consistent microbial patterns linked to particular metabolic diseases because of this heterogeneity. The issues of standardizing microbiome analysis approaches and taking into consideration individual variance are ongoing. The goal of the research is to identify relationships among the microbiome's makeup and the onset or progression of metabolic illnesses. This entails researching how alterations in the microbiota may affect metabolic and to create focused therapies that control the gut microbiota to enhance metabolic health. This could entail using probiotics, prebiotics, dietary changes, microbial transplants, or even pharmaceuticals that directly target gut bacteria or their metabolites.

Modifications in the constitution of intestinal microbiota related with overweight.

The control of the metabolism of the crowd and the absorption of liveliness from consumed nutrition are both significantly influenced by the GM. In especially whenever it is due to obesity and other metabolic problems, the GM interacts with the host in a pathogenic manner in addition to serving the host's positive purposes as shown in figure 1. Recent research proposes which varies in the gut flora can motivate to the progression of diabetes and obesity. Since rats raised in germ-free environments have lower stages of "fasting-induced adipose factor (Fiaf), a lipoprotein lipase (LPL) inhibitor", they are safeguarded against the obesity and metabolic dysfunction brought on by high fat diets (HFD), including glucose intolerance [17]. Moreover, beneficiary mice developed much higher levels of body fat and insulin sensitivity after being colonized by GM obtained from traditionally bred obese donors. The phyla Bacteroidetes and Firmicutes contain the majority of the species of bacteria found in the mouse and human guts. Leptin-deficient ob/ob mice demonstrated a reduction in Bacteroidetes and a matching rise in Firmicutes comparing to their lean counterparts.

Interesting human investigations have also revealed that the configuration of the GM varies between overweight and slim patients. Recent studies, figure 2 [27] demonstrate that butyrate-producing bacteria could safeguard towards type 2 diabetes by reducing the amount of butyrate-producing Clostridiales (Roseburia and Faecalibacterium prausnitzii) and increasing the relation of non-butyrate-producing Clostridiales in subjects with T2DM [18].

Since decreased movement of the gastrointestinal tract and small intestine colonies of bacteria are frequently observed in T2DM, it is still unknown if these modifications in the arrangement of the intestinal microbiota are a result of those conditions. However, certain intestinal bacteria strains may work in hospitals as initial warning signs for recognizing obese people who may be susceptible to developing T2DM and provide a unique therapeutic approach, Prevention method for T2DM or fat.

These include glucagon-like peptide (GLP)-1 and PYY, which decreases hunger and consumption of calories, and the reduced production of the gut peptide ghrelin, which stimulates food consumption by impacting the hypothalamus and reward-related systems in the brainstem (Figure 3) [19]. Insulin signaling in fat

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**Figure 1.** “(A) Body length, (B) body weight, and (C) Body mass index (BMI) were traced weekly. Total cholesterol (E)and Insulin (INS) (D) were evaluated at week 4. CUG-SGA (n=13) and NCUG-SGA (n=12). **p<0.01, *p<0.5, ***p<0.001 (NCUG-SGA vs CUG-SGA)”.
cells was suppressed as a consequence of GPR43 activation by SCFAs [28].

**Metabolic endotoxemia and gut permeability.**

Endotoxemia originating from the GM and disturbance of the intestinal barrier's performance have both been linked to the aetiology of obesity and T2DM, according to a number of recent research. An HFD dramatically reduced the synthesis of stringent junction proteins including zonula occludens-1 and occludin in the skin chambers of mouse digestive tract while increasing intestinal permeability (Figure 4) [20]. Diminishing of the gut barrier increased gastrointestinal permeability in either naturally or Mice with HFD-induced obesity were exposed to lipopolysaccharide at the region where blood flow enters (Figure 4) [29]. This hypothesis is backed by the findings

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**Figure 2.** AMY1 CN prevalence in the studied population.

**Figure 3.** The transformation of unprocessed carbohydrates into short-chain fatty acids (SCFAs), chiefly acetate, propionate, and butyrate, by the GM controls human metabolism. SCFAs communicate with G protein-joined receptors 41 (GPR41) and 43 (GPR43), which trigger the release of the peptide YY (PYY), improve the response to insulin, reduce the function of the fasting-induced adipose factor (Fiaf), activate intestinal gluconeogenesis (IGN), and have an impact on glucose metabolism and fat storage.
that prebiotics or antibiotics enhanced intestinal permeability, lowered metabolic endotoxemia, lowered inflammation, and reduced tolerance to glucose through modifying the composition of the GM.

Modulation of intestinal microbiota as new therapeutic approaches for diabetes and obesity.

Prebiotics.
Prebiotics are polysaccharides that are fermented but indigestible, like inulin, fructo-, oligo, galato-, or lactulose. Prebiotics are nutrients that have been intentionally enhanced with these fibres because they encourage the formation of SCFA and the expansion of good microorganisms, particularly Bifidobacterium and Lactobacillus [21]. Prebiotic ingestion decreases hunger and increases fullness, according to studies in healthy people and rats. As previously mentioned, alterations in gut peptide production brought on by SCFAs are a contributing factor in this modification of ingestive behaviour. Prebiotics also enhance the function of intestinal barriers by fostering Bifidobacterium populations.

Probiotics.
Living bacteria known as probiotics are said to benefit human health when given in the appropriate doses. It has been demonstrated that some strains of probiotic bacteria, particularly those of the families Lactobacillus and Bifidobacterium, can reduce overweight and metabolic problems. A few of the proposed processes are stabilization of the community of microbes, enhancement of mucosal preservation and barrier function, and prevention of pathogen attachment to gut mucosa. The activities of the SCFA byproducts of bacterial fermentation may explain the improvements in gut barrier function. According to a new investigation, Lactobacilli has direct positive effects on epithelial cells and the nervous system of the enteric tract, which regulates gut elasticity [22].

Drugs.
According to a recent study, the drug metformin, which is frequently prescribed to treat T2DM, can reduce the ageing process in Caenorhabditis worms by changing the metabolism of Escherichia coli, with which it is combined [23]. It was...
discovered that this impact was brought on by metformin's change of E. coli's folate and homocysteine synthesis. S-adenosylmethionine (SAMe) and S-adenosylhomocysteine levels rose while the methionine cycle was suppressed by metformin. SAMe functions as a co-repressor of the enzymes involved in methionine synthesis and suppresses the folate cycle, slowing the ageing process in the parasites.

Mammal gastrointestinal microbiome can be impacted by metformin as well. In the stomach of laboratory mice given an HFD, we have shown that metformin has the ability to alter the mice microbiota and enhance the number of Akkermansiamuciniphila, a G (-) anaerobe that degrades mucin (Figure 2) [24]. Additionally, we noticed that the consumption of A. muciniphila has related favourable metabolic impacts to the treatment of metformin: (1) More mucin-producing goblet cells were observed soon after metformin or A. muciniphila administration; (2) Reduced regulatory T (Treg) cell numbers and elevated interleukin 1 (IL-1) or IL-6 mRNA expression in The abdominal fat of rats that were fed an HFD were exactly changed later metformin or muciniphila management.

In addition to its traditional function as an external barrier, mucin was recently demonstrated to improve the transmission of tolerogenic immunoregulatory information to the intestinal epithelium by producing galectin-3-dectin-1-FcRIIB complex [25]. This work emphasizes the possibility that.

**Fecal microbiota transplantation.**

Faecalmicrobiota transplantation (FMT)-related publications in poetry have recently generated a lot of attention. FMT is said to be an extremely active management for recurring Clostridium difficile infection [26]. These findings also revealed a possible therapeutic role for FMT in T2DM or metabolic syndrome.

According to the latest research, FMT administered through a gastroduodenal tube to obese those who have metabolic syndrome caused a considerable development in their insulin sensitivity. After 42 days of therapy, FMT led to a decrease in faecal SCFA levels while increasing the variety of microbes and the fraction of the butyrate producing Roseburiaintestinalis by 2.5 times. Although this initial proof suggests that this kind of strategy may be appealing, larger, more thorough investigations are required to determine whether these methods are generally advantageous for people with metabolic syndrome or T2DM.

**Conclusion.**

By affecting weight gain, immunological movement, and insulin sensitivity, the microbiota of the intestines may be crucial in the aetiology of T2DM. Future research is necessary to deepen our comprehension of the intricate interactions between the microbiota of the intestines and the mass in T2DM and to allow the creation of novel, active cures for the disease.

**REFERENCES**


