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ЕЖЕМЕСЯЧНЫЙ НАУЧНЫЙ ЖУРНАЛ

Медицинские новости Грузии

საქართველოს სამედიცინო სიახლენი

GEORGIAN MEDICAL NEWS

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GMN: Georgian Medical News is peer-reviewed, published monthly journal committed to promoting the science and art of medicine and the betterment of public health, published by the GMN Editorial Board since 1994. GMN carries original scientific articles on medicine, biology and pharmacy, which are of experimental, theoretical and practical character; publishes original research, reviews, commentaries, editorials, essays, medical news, and correspondence in English and Russian.

GMN is indexed in MEDLINE, SCOPUS, PubMed and VINITI Russian Academy of Sciences. The full text content is available through EBSCO databases.

GMN: Медицинские новости Грузии - ежемесячный рецензируемый научный журнал, издаётся Редакционной коллегией с 1994 года на русском и английском языках в целях поддержки медицинской науки и улучшения здравоохранения. В журнале публикуются оригинальные научные статьи в области медицины, биологии и фармации, статьи обзорного характера, научные сообщения, новости медицины и здравоохранения. Журнал индексируется в MEDLINE, отражён в базе данных SCOPUS, PubMed и ВИНИТИ РАН. Полнотекстовые статьи журнала доступны через БД EBSCO.

GMN: Georgian Medical News – საქართველოს სამედიცინო სიახლენი – არის ყოველთვიური სამეცნიერო სამედიცინო რეცენზირებადი ჟურნალი, გამოიცემა 1994 წლიდან, წარმოადგენს სარედაქციო კოლეგიისა და აშშ-ის მეცნიერების, განათლების, ინდუსტრიის, ხელოვნებისა და ბუნებისმეტყველების საერთაშორისო აკადემიის ერთობლივ გამოცემას. GMN-ში რუსულ და ინგლისურ ენებზე ქვეყნდება ექსპერიმენტული, თეორიული და პრაქტიკული ხასიათის ორიგინალური სამეცნიერო სტატიები მედიცინის, ბიოლოგიისა და ფარმაციის სფეროში, მიმოხილვითი ხასიათის სტატიები.

ჟურნალი ინდექსირებულია MEDLINE-ის საერთაშორისო სისტემაში, ასახულია SCOPUS-ის, PubMed-ის და ВИНИТИ РАН-ის მონაცემთა ბაზებში. სტატიების სრული ტექსტი ხელმისაწვდომია EBSCO-ს მონაცემთა ბაზებიდან.

WEBSITE

www.geomednews.com

К СВЕДЕНИЮ АВТОРОВ!

При направлении статьи в редакцию необходимо соблюдать следующие правила:

1. Статья должна быть представлена в двух экземплярах, на русском или английском языках, напечатанная через **полтора интервала на одной стороне стандартного листа с шириной левого поля в три сантиметра**. Используемый компьютерный шрифт для текста на русском и английском языках - **Times New Roman** (**Кириллица**), для текста на грузинском языке следует использовать **AcadNusx.** Размер шрифта - **12**. К рукописи, напечатанной на компьютере, должен быть приложен CD со статьей.

2. Размер статьи должен быть не менее десяти и не более двадцати страниц машинописи, включая указатель литературы и резюме на английском, русском и грузинском языках.

3. В статье должны быть освещены актуальность данного материала, методы и результаты исследования и их обсуждение.

При представлении в печать научных экспериментальных работ авторы должны указывать вид и количество экспериментальных животных, применявшиеся методы обезболивания и усыпления (в ходе острых опытов).

4. К статье должны быть приложены краткое (на полстраницы) резюме на английском, русском и грузинском языках (включающее следующие разделы: цель исследования, материал и методы, результаты и заключение) и список ключевых слов (key words).

5. Таблицы необходимо представлять в печатной форме. Фотокопии не принимаются. **Все цифровые, итоговые и процентные данные в таблицах должны соответствовать таковым в тексте статьи**. Таблицы и графики должны быть озаглавлены.

6. Фотографии должны быть контрастными, фотокопии с рентгенограмм - в позитивном изображении. Рисунки, чертежи и диаграммы следует озаглавить, пронумеровать и вставить в соответствующее место текста **в tiff формате**.

В подписях к микрофотографиям следует указывать степень увеличения через окуляр или объектив и метод окраски или импрегнации срезов.

7. Фамилии отечественных авторов приводятся в оригинальной транскрипции.

8. При оформлении и направлении статей в журнал МНГ просим авторов соблюдать правила, изложенные в «Единых требованиях к рукописям, представляемым в биомедицинские журналы», принятых Международным комитетом редакторов медицинских журналов - http://www.spinesurgery.ru/files/publish.pdf и http://www.nlm.nih.gov/bsd/uniform_requirements.html
В конце каждой оригинальной статьи приводится библиографический список. В список литературы включаются все материалы, на которые имеются ссылки в тексте. Список составляется в алфавитном порядке и нумеруется. Литературный источник приводится на языке оригинала. В списке литературы сначала приводятся работы, написанные знаками грузинского алфавита, затем кириллицей и латиницей. Ссылки на цитируемые работы в тексте статьи даются в квадратных скобках в виде номера, соответствующего номеру данной работы в списке литературы. Большинство цитированных источников должны быть за последние 5-7 лет.

9. Для получения права на публикацию статья должна иметь от руководителя работы или учреждения визу и сопроводительное отношение, написанные или напечатанные на бланке и заверенные подписью и печатью.

10. В конце статьи должны быть подписи всех авторов, полностью приведены их фамилии, имена и отчества, указаны служебный и домашний номера телефонов и адреса или иные координаты. Количество авторов (соавторов) не должно превышать пяти человек.

11. Редакция оставляет за собой право сокращать и исправлять статьи. Корректура авторам не высылается, вся работа и сверка проводится по авторскому оригиналу.

12. Недопустимо направление в редакцию работ, представленных к печати в иных издательствах или опубликованных в других изданиях.

При нарушении указанных правил статьи не рассматриваются.

REQUIREMENTS

Please note, materials submitted to the Editorial Office Staff are supposed to meet the following requirements:

1. Articles must be provided with a double copy, in English or Russian languages and typed or compu-ter-printed on a single side of standard typing paper, with the left margin of **3** centimeters width, and **1.5** spacing between the lines, typeface - **Times New Roman** (**Cyrillic**), print size - **12** (referring to Georgian and Russian materials). With computer-printed texts please enclose a CD carrying the same file titled with Latin symbols.

2. Size of the article, including index and resume in English, Russian and Georgian languages must be at least 10 pages and not exceed the limit of 20 pages of typed or computer-printed text.

3. Submitted material must include a coverage of a topical subject, research methods, results, and review.

Authors of the scientific-research works must indicate the number of experimental biological species drawn in, list the employed methods of anesthetization and soporific means used during acute tests.

4. Articles must have a short (half page) abstract in English, Russian and Georgian (including the following sections: aim of study, material and methods, results and conclusions) and a list of key words.

5. Tables must be presented in an original typed or computer-printed form, instead of a photocopied version. **Numbers, totals, percentile data on the tables must coincide with those in the texts of the articles.** Tables and graphs must be headed.

6. Photographs are required to be contrasted and must be submitted with doubles. Please number each photograph with a pencil on its back, indicate author's name, title of the article (short version), and mark out its top and bottom parts. Drawings must be accurate, drafts and diagrams drawn in Indian ink (or black ink). Photocopies of the X-ray photographs must be presented in a positive image in **tiff format**.

Accurately numbered subtitles for each illustration must be listed on a separate sheet of paper. In the subtitles for the microphotographs please indicate the ocular and objective lens magnification power, method of coloring or impregnation of the microscopic sections (preparations).

7. Please indicate last names, first and middle initials of the native authors, present names and initials of the foreign authors in the transcription of the original language, enclose in parenthesis corresponding number under which the author is listed in the reference materials.

8. Please follow guidance offered to authors by The International Committee of Medical Journal Editors guidance in its Uniform Requirements for Manuscripts Submitted to Biomedical Journals publication available online at: http://www.nlm.nih.gov/bsd/uniform_requirements.html
http://www.icmje.org/urm_full.pdf
In GMN style for each work cited in the text, a bibliographic reference is given, and this is located at the end of the article under the title "References". All references cited in the text must be listed. The list of references should be arranged alphabetically and then numbered. References are numbered in the text [numbers in square brackets] and in the reference list and numbers are repeated throughout the text as needed. The bibliographic description is given in the language of publication (citations in Georgian script are followed by Cyrillic and Latin).

9. To obtain the rights of publication articles must be accompanied by a visa from the project instructor or the establishment, where the work has been performed, and a reference letter, both written or typed on a special signed form, certified by a stamp or a seal.

10. Articles must be signed by all of the authors at the end, and they must be provided with a list of full names, office and home phone numbers and addresses or other non-office locations where the authors could be reached. The number of the authors (co-authors) must not exceed the limit of 5 people.

11. Editorial Staff reserves the rights to cut down in size and correct the articles. Proof-sheets are not sent out to the authors. The entire editorial and collation work is performed according to the author's original text.

12. Sending in the works that have already been assigned to the press by other Editorial Staffs or have been printed by other publishers is not permissible.

Articles that Fail to Meet the Aforementioned Requirements are not Assigned to be Reviewed.

ავტორთა საყურადღებოდ!

რედაქციაში სტატიის წარმოდგენისას საჭიროა დავიცვათ შემდეგი წესები:

1. სტატია უნდა წარმოადგინოთ 2 ცალად, რუსულ ან ინგლისურ ენებზე,დაბეჭდილი სტანდარტული ფურცლის 1 გვერდზე, 3 სმ სიგანის მარცხენა ველისა და სტრიქონებს შორის 1,5 ინტერვალის დაცვით. გამოყენებული კომპიუტერული შრიფტი რუსულ და ინგლისურენოვან ტექსტებში - **Times New Roman (Кириллица)**, ხოლო ქართულენოვან ტექსტში საჭიროა გამოვიყენოთ **AcadNusx.** შრიფტის ზომა – 12. სტატიას თან უნდა ახლდეს CD სტატიით.

2. სტატიის მოცულობა არ უნდა შეადგენდეს 10 გვერდზე ნაკლებს და 20 გვერდზე მეტს ლიტერატურის სიის და რეზიუმეების (ინგლისურ, რუსულ და ქართულ ენებზე) ჩათვლით.

3. სტატიაში საჭიროა გაშუქდეს: საკითხის აქტუალობა; კვლევის მიზანი; საკვლევი მასალა და გამოყენებული მეთოდები; მიღებული შედეგები და მათი განსჯა. ექსპერიმენტული ხასიათის სტატიების წარმოდგენისას ავტორებმა უნდა მიუთითონ საექსპერიმენტო ცხოველების სახეობა და რაოდენობა; გაუტკივარებისა და დაძინების მეთოდები (მწვავე ცდების პირობებში).

4. სტატიას თან უნდა ახლდეს რეზიუმე ინგლისურ, რუსულ და ქართულ ენებზე არანაკლებ ნახევარი გვერდის მოცულობისა (სათაურის, ავტორების, დაწესებულების მითითებით და უნდა შეიცავდეს შემდეგ განყოფილებებს: მიზანი, მასალა და მეთოდები, შედეგები და დასკვნები; ტექსტუალური ნაწილი არ უნდა იყოს 15 სტრიქონზე ნაკლები) და საკვანძო სიტყვების ჩამონათვალი (key words).

5. ცხრილები საჭიროა წარმოადგინოთ ნაბეჭდი სახით. ყველა ციფრული, შემაჯამებელი და პროცენტული მონაცემები უნდა შეესაბამებოდეს ტექსტში მოყვანილს.

6. ფოტოსურათები უნდა იყოს კონტრასტული; სურათები, ნახაზები, დიაგრამები - დასათაურებული, დანომრილი და სათანადო ადგილას ჩასმული. რენტგენოგრამების ფოტოასლები წარმოადგინეთ პოზიტიური გამოსახულებით **tiff** ფორმატში. მიკროფოტოსურათების წარწერებში საჭიროა მიუთითოთ ოკულარის ან ობიექტივის საშუალებით გადიდების ხარისხი, ანათალების შეღებვის ან იმპრეგნაციის მეთოდი და აღნიშნოთ სურათის ზედა და ქვედა ნაწილები.

7. სამამულო ავტორების გვარები სტატიაში აღინიშნება ინიციალების თანდართვით, უცხოურისა – უცხოური ტრანსკრიპციით.

8. სტატიას თან უნდა ახლდეს ავტორის მიერ გამოყენებული სამამულო და უცხოური შრომების ბიბლიოგრაფიული სია (ბოლო 5-8 წლის სიღრმით). ანბანური წყობით წარმოდგენილ ბიბლიოგრაფიულ სიაში მიუთითეთ ჯერ სამამულო, შემდეგ უცხოელი ავტორები (გვარი, ინიციალები, სტატიის სათაური, ჟურნალის დასახელება, გამოცემის ადგილი, წელი, ჟურნალის №, პირველი და ბოლო გვერდები). მონოგრაფიის შემთხვევაში მიუთითეთ გამოცემის წელი, ადგილი და გვერდების საერთო რაოდენობა. ტექსტში კვადრატულ ფჩხილებში უნდა მიუთითოთ ავტორის შესაბამისი N ლიტერატურის სიის მიხედვით. მიზანშეწონილია, რომ ციტირებული წყაროების უმეტესი ნაწილი იყოს 5-6 წლის სიღრმის.

9. სტატიას თან უნდა ახლდეს: ა) დაწესებულების ან სამეცნიერო ხელმძღვანელის წარდგინება, დამოწმებული ხელმოწერითა და ბეჭდით; ბ) დარგის სპეციალისტის დამოწმებული რეცენზია, რომელშიც მითითებული იქნება საკითხის აქტუალობა, მასალის საკმაობა, მეთოდის სანდოობა, შედეგების სამეცნიერო-პრაქტიკული მნიშვნელობა.

10. სტატიის ბოლოს საჭიროა ყველა ავტორის ხელმოწერა, რომელთა რაოდენობა არ უნდა აღემატებოდეს 5-ს.

11. რედაქცია იტოვებს უფლებას შეასწოროს სტატია. ტექსტზე მუშაობა და შეჯერება ხდება საავტორო ორიგინალის მიხედვით.

12. დაუშვებელია რედაქციაში ისეთი სტატიის წარდგენა, რომელიც დასაბეჭდად წარდგენილი იყო სხვა რედაქციაში ან გამოქვეყნებული იყო სხვა გამოცემებში.

აღნიშნული წესების დარღვევის შემთხვევაში სტატიები არ განიხილება.

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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 satisfied, 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 faecalmicrobiota 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 research provided compelling evidence that the GM regulates a number of characteristics and metabolites that are important for cardio-metabolic health [10]. They have discovered a variety of new connections between gut microorganisms and various mingling metabolites, and some of these relationships may be able to detect the presence of pre-diabetes.

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 rRNAmetagenomic 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 Faecalibacteriumprausnitzii) 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

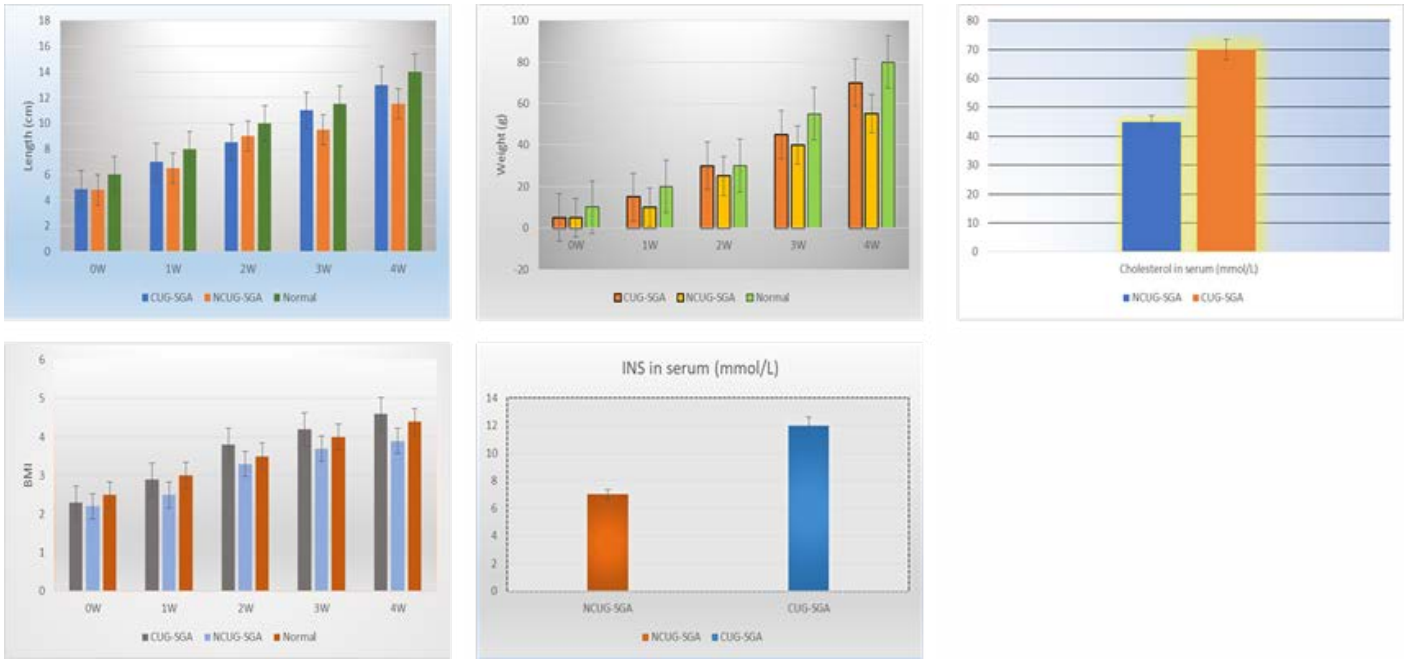

Figure 1. *"(A) Body length, (B) body weight, and (C) Body mass index (BMI) were traced weekly. Total cholesterol (E)and Insulin (INS) (D) were evaluated at week 4. CUG-SGA (n=13) and NCUG-SGA (n=12). **p<0.01, *p<0.5, ***p<0.001 (NCUG-SGA vs CUG-SGA)".*

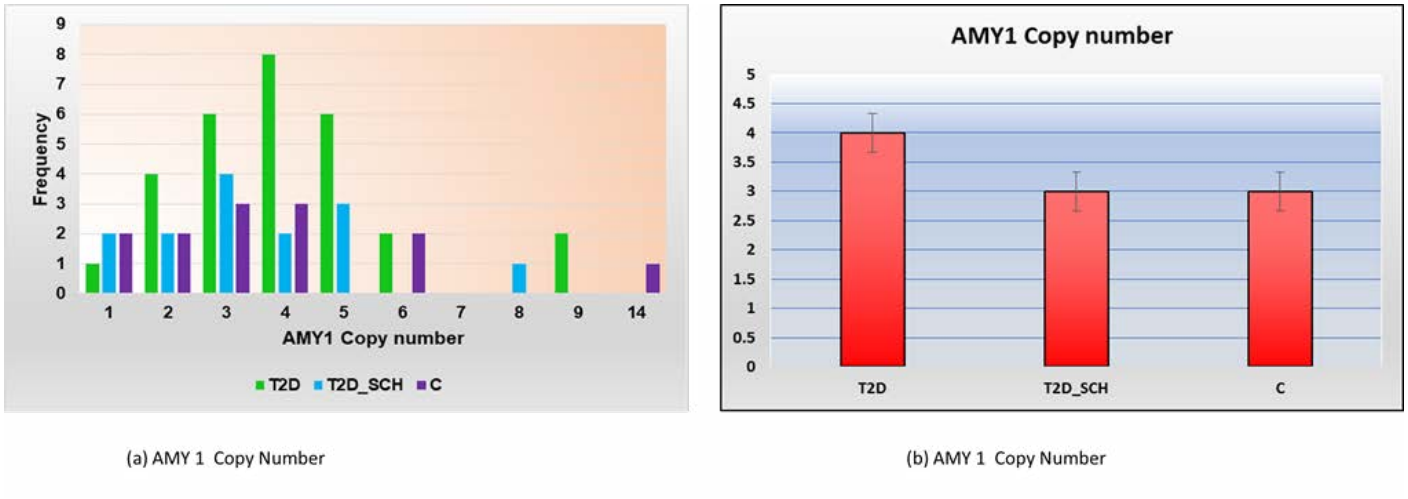

Figure 2. *AMY1 CN prevalence in the studied population.*

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.*

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

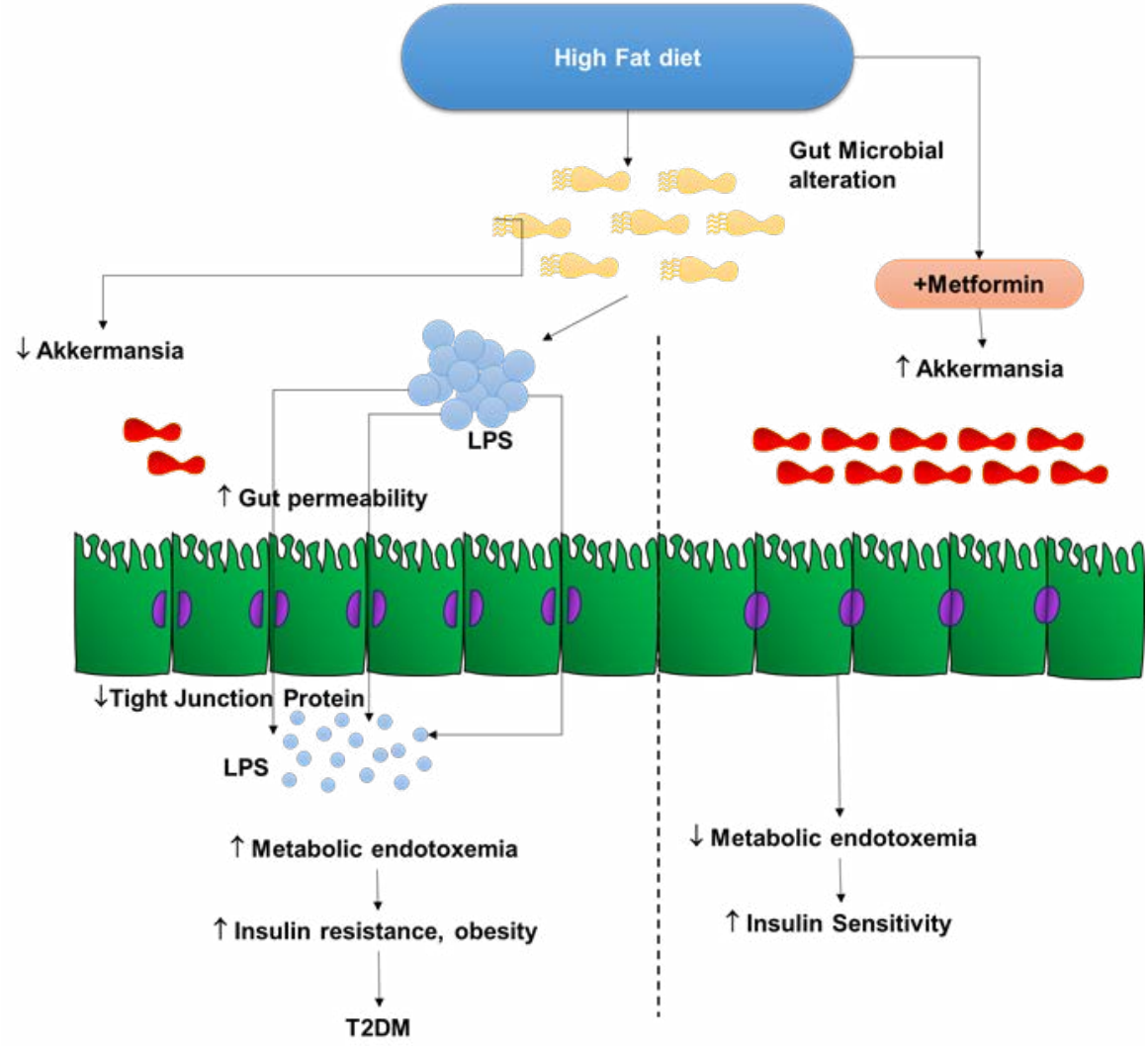

Figure 4. *A high-fat diet (HFD) causes gut microbial changes, raises intestinal permeability, lowers the expression of ZO-1 and occludin, two tight junction proteins, and allows lipopolysaccharide (LPS) to enter the portal vein circulation. Obesity, glucose resistance, and type 2 diabetes mellitus (T2DM) may be influenced by disruptions in gut barrier function and endotoxemia produced from the gut microbiota. In mice fed an HFD, metformin increases the quantity of Akkermansiamuciniphila, an anaerobe that breaks down mucin, and has similar favourable metabolic effects.*

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-adenosylhomocystein 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.

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