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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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EPIDEMIOLOGY OF GENES ASSOCIATED WITH OBESITY IN ASIAN POPULATION. LITERATURE REVIEW

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Abstract.

Introduction: Among the general population, there is a category of individuals with an innate predisposition to obesity. In recent decades, the increase in the prevalence of obesity and related diseases worldwide indicates the need to study the etiological factors associated with its development. At present, it is still unknown how many genes are involved in the pathophysiology of obesity, and many studies are underway to identify "candidate genes" in clinical medicine. The detection of obesity by molecular genetic diagnostics and the implementation of appropriate preventive measures can significantly reduce the incidence. Considering that the majority of people suffering from metabolic diseases are members of the working population, the prevention of these diseases and early diagnosis are of both clinical and social and economic importance.

Aim: The aim of this study is to analysis of data on the most significant genetic markers in the development of obesity in the Asian population.

Material and methods: An online search for literature on genetic markers associated with the development of obesity was conducted in the databases Elibrary, Google Scholar, PubMed, Web of Science databases was carried out.

Results and discussion: The study of genetic markers and their determination has great prospects for the successful verification of the main clinical and biological markers of obesity. This will allow assessing the risks of developing obesity and developing standards for corrective measures for individuals with a high genetic risk.

Conclusion: Most of the GWAS studies have been conducted in the European population, the discovery of new genes in the Asian population has made a significant contribution to the identification of loci of predisposition to obesity. identification of polymorphic variants of candidate genes that are most significant in the development of this disease is an urgent task for both fundamental science and practical medicine.

Key words. obesity, gene polymorphism, genetics, GWAS, obesity, BMI, Asian population.

Introduction.

Among the general population, there is a category of individuals with an innate predisposition to obesity. In recent decades, the increase in the prevalence of obesity and related diseases worldwide indicates the need to study the etiological factors associated with its development. At present, it is still unknown how many genes are involved in the pathophysiology of obesity, and many studies are underway to identify "candidate genes" in clinical medicine. The detection of obesity by molecular genetic diagnostics and the implementation of appropriate preventive measures can significantly reduce the incidence. Considering that the majority of people suffering from metabolic diseases are members of the working population, the prevention of these diseases and early diagnosis are of both clinical and social and economic importance.

The aim of this study is to analysis of data on the most significant genetic markers in the development of obesity in the Asian population.

Search strategy.

An online search for literature on genetic markers associated with the development of obesity was conducted in the databases Elibrary, Google Scholar, PubMed, Web of Science databases was carried out. The objectives of the study correspond to 72 articles.

Obesity as a global problem of the modern world.

Obesity, a recurrent disease in which excess lipids accumulate in various fatty deposits due to a chronic imbalance between energy intake and expenditure, is associated with many diseases, such as type 2 diabetes, cardiovascular disease, osteoarthritis, dyslipidemia, fatty liver disease and some types of cancer [1,2]. In addition to the recognized role of environmental factors, such as sedentary lifestyles combined with high caloric intake and inadequate energy expenditure, the development of obesity probably also has a genetic component, as evidenced by both monogenic and common polygenic forms of obesity [3].

The prevalence of obesity is constantly increasing, and at the expense of its severe degree, which allows us to consider it as a worldwide epidemic associated with increased morbidity and mortality. According to epidemiological data, more than 1.7 billion people in the world are overweight, and more than 700 million of them are obese [4]. According to the WHO, overweight and obesity are the main factors in the development of coronary heart disease and stroke, which are the leading causes of mortality [5,6].

According to data from the National Bureau of Statistics, the population of Kazakhstan is 19 million, among which the distribution of overweight is about 29.7% for women and 33.9% for men; and the proportion of obesity is 25.8% for women and 15.3% for men over 18 years of age [7]. If current trends continue, epidemiologists predict that by 2025 40% of men and 50% of women will be obese. [8-10].

In clinical practice, obesity is traditionally estimated using body mass index (BMI). It is calculated by taking a person's weight in kilograms divided by their height, in meters squared (kg/m^2). These BMI classifications are used by the WHO for the European population. BMI values underestimate the risk of obesity in Asian and South Asian populations, so their classifications have little variation.

A study in the United States from 2010 to 2016 confirmed the low prevalence of overweight/obesity in Asians, after adjusting for WHO recommended BMI values for Asia (overweight: 23-

Table 1. *Grades of obesity according to BMI [13].*

Grades of obesity	BMI (kg/m2)	
	Europeans	Asians
1	30-34,9	25-28,9
2	35-39,9	29-32,9
3	40 and above	33 and above

24,9 kg/m^2; obese: ≥25 kg/m^2). Asians are more susceptible to obesity-associated comorbidities than Caucasians, even at lower BMI and waist circumference values [11,12].

Obesity is both an independent risk factor for cardiovascular disease and closely related to a number of other risk factors. Multiple clinical and epidemiological data link obesity with a wide range of CVDs, also with obstructive sleep apnea and other hypoventilation syndromes, which adversely affect cardiovascular function [14-16].

It is well known that 90% of patients with type 2 diabetes mellitus are overweight or obese. Today, both of these diseases have become a noninfectious epidemic. People who are genetically predisposed to develop type 2 diabetes are at high risk for obesity because of insulin resistance in their muscle and islet α-cells, which contributes to increased glucose and insulin release. This resistance leads to increased glucose production in the liver and increased insulin levels, which are the cause of obesity [17].

Cancer is the second leading cause of death worldwide, with 14.1 million cases and 8.2 million deaths annually [18] In addition to well-known risk factors for cancer such as genetic predisposition, ionizing radiation, tobacco use, infections, unhealthy diet, alcohol consumption, sedentary lifestyle and other environmental exposures, obesity is an established risk factor for several malignancies [19-21]. The incidence of cancer will continue to increase due to the increasing prevalence of risk factors, mainly obesity and metabolic syndrome [22].

The outbreak of coronavirus infection (Covid-19) has been a developing world health crisis. Interest in the interaction between obesity and infection was sparked by the H1N1 pandemic in 2009 [23-25], published data suggesting that obese people were more susceptible to respiratory viral infection, greater disease severity and adverse outcomes following infection, including higher rates of hospitalization, USA admission and death [26]. In this Covid-19 epidemic, higher rates of obesity and severe obesity were also reported among patients with Covid-19 compared with historical non-Covid-19 controls. A report from the United States also showed that among patients with Covid-19 younger than 60 years of age with BMIs between 30 kg/m^2 and 35 kg/m^2 and greater than 35 kg/m^2, 1.8 and 3.6 times as often were admitted to the ORIT, respectively, compared with patients with BMIs <30 kg/m^2 [27].

Genetic markers of predisposition to obesity.

The problem of obesity is becoming increasingly relevant and is beginning to pose a social threat to the lives of people regardless of their social and professional affiliation, area of residence, age, and gender. At present, it is still unknown how many genes are involved in the pathophysiology of metabolic diseases; in clinical medicine, there are many studies to identify «candidate genes» [28]. Recent advances in high-throughput genotyping technologies have enabled the development of powerful analytical tools, such as the Genome-wide association study (GWAS), to study novel genes and loci contributing to genetic susceptibility to complex diseases. Over the past decade, large-scale GWASs have identified hundreds of genetic loci, making significant progress in genetics.

The genetic aspects of obesity lead to mutations in various genes responsible for controlling appetite and metabolism. Over the past two decades, several strategies have been developed to identify the genetic determinants of obesity. It includes severe obesity studies, genome-wide linkage studies (GWLSS) and GWAS and candidate gene analysis. About 127 sites in the human genome have been known to be associated with the development of obesity due to GWAS results [29-30]. The greatest success in the search for candidate genes of predisposition to obesity is associated with the technology of large-scale genotyping of GWAS using hundreds of thousands and millions of polymorphisms (SNPs). Large-scale GWAS studies and meta-analyses have identified hundreds of genetic loci that threaten the spread of obesity. GWAS has been conducted in European and East Asian populations, and individual gene polymorphisms associated with the development of obesity have been studied in Central Asian populations, but no large multicenter studies of these polymorphisms have been conducted. Current research tools and extensive studies will lead to an understanding of the genes and their interactions that cause obesity, which may aid in successful diagnosis [31,32].

Among these GWAS results, the first locus identified that was sensitive to obesity was the **FTO gene** (associated with obesity with fat mass). This gene has the greatest influence on the risk of obesity phenotypes to date.

According to the results of several meta-analyses, the FTO rs9930506 polymorphism has shown a dominant pattern of association with obesity, especially in the European population [33-35]. The small allele of the FTO gene increases BMI by 0.39 kg/m^2 and the risk of obesity by 1.20-fold. This association has been confirmed in populations of different ages and backgrounds. FTO gene polymorphisms affect obesity symptoms slightly less in African and Asian populations than in European populations. Among populations in Western and Central Europe and West Africa, 46-51%, and in China, only 16% [36-37].

The association with FTO rs9939609 and physical activity was observed in a cohort study that followed 17,400 people from 17 countries in six ethnic groups, including South Asian, European, East Asian, Hispanic, African, and Native American populations over three years [38-40].

The relationship of the FTO rs9939609 polymorphism with metabolic syndrome and obesity among the population of different regions of Russia was investigated. The study concluded that the FTO rs9969309 gene polymorphism is associated not only with abdominal obesity, but also with other MS components such as hyperglycemia, arterial hypertension [41].

Ning-Ning Zhao et al. [42] showed that rs9939609, rs6499640, rs8050136, rs1421085, and rs17817449 showed a significant association in the European SNP population, allowing rs9939609 as the TagSNP for the other four polymorphisms. Similar results were obtained in a number of European, East Asian, and Gujarat Indians, with four of the five SNPs rs9939609, rs8050136,

rs1421085, and rs17817449 showing strong LD, indicating that rs9939609 can be used as a TagSNP for the other three polymorphisms [11,42].

Adipose tissue plays a crucial role in the regulation of energy homeostasis, insulin sensitivity, and lipid and carbohydrate metabolism and is considered as an endocrine organ that secretes a number of adipocytokines (e.g., adiponectin and tumor necrosis factor-a) [43].

Adiponectin is a protein secreted by adipose tissue that is encoded by the adiponectin gene (**ADIPOQ**), consists of three exons and two introns, and is located on chromosome 3q27 [44]. The biological functions of adiponectin are diverse: from antidiabetic, anti-atherogenic, anti-inflammatory to anticancer. Numerous studies have shown the protective role of adiponectin in obesity-related diseases [20,45]. The relationship of adiponectin, including leptin, with obesity and insulin resistance and other metabolic risk factors has been studied in different populations [46].

The rs2241766 ADIPOQ polymorphism is a «candidate gene» for obesity. A number of studies have been published worldwide examining the association between the ADIPOQ rs2241766 polymorphism and obesity [43,47,48]. However, inconsistent results have been obtained in different populations. These discrepancies may be related to racial or regional differences in the frequency of the ADIPOQ polymorphism.

Adiponectin has also been considered as a marker of the metabolic syndrome [49,50]. A meta-analysis involving 13 studies with 2,684 cases and 2,864 controls in a Chinese population was conducted to identify the association of the rs2241766 variant with MS [51,52], and the results confirmed that the frequency of the G allele was significantly higher in obese patients than in controls [51].

As studies have shown, there was a significant genotypic association between the obese and non-obese groups in Malaysian. The frequencies of AG and AA genotypes were significantly higher in the obese group (11%) than in the non-obese group. The probability of having the A alleles was twice as high in the obese group than in the non-obese group (OR 2.15; 1.13-4.09). Independent t-test analysis showed that sex, age, height, weight, waist circumference, hip circumference, BP, systolic and diastolic BP, BMI, triacylglycerol, HDL cholesterol, and LDL cholesterol were significantly higher in the main group (with obesity) than in the control group. As a result, ADIPOQ rs17366568 polymorphisms were significantly associated with BMI [53].

Many studies have shown that the pathogenesis of obesity has a genetic basis, with polymorphisms in the **TMEM18** gene region being an important risk factor [54,55], which plays a significant role in eating behavior; however, subsequent studies among different ethnic groups and age groups have shown conflicting results. Natalia Koj et al. [56] examined the relationship between TMEM18 polymorphisms and the risk of obesity as a function of age group and ethnicity. They found a significant association between rs6548238 and the risk of obesity (OR=1.25). Regarding population type, a significant association was found among European c and Mexican groups. However, a lack of statistical significance was noted in Asians with OR=1.11. The results show that there are differences between ethnic populations, which confirms the results of the study. The researchers concluded that polymorphisms near TMEM18 play a role in the development of obesity [56].

Studies by Scott M Williams [57] provided strong evidence that eight (SEC16B, TMEM18, ETV5, GNPDA2, TFAP2B, BDNF, FTO, MC4R) of the 21 loci associated with high BMI in populations of European origin were successfully replicated in African American populations.

Genetic epidemiology of NEGR1, SEC16B, NRXN3 in an Asian population.

The **SEC16B gene** encodes the Sec16 protein, which is expressed mainly in brain tissues and is responsible for the transport of appetite-regulating peptides.

According to a meta-analysis by Lu Y. et al. [58], the rs543874 polymorphism of the SEC16B gene is associated with an increase in body fat percentage, which is likely to be a more accurate prognostic criterion for obesity development than BMI scores. This meta-analysis found associations between the SEC16B rs543874 polymorphism and increased percent body fat, with increased BMI in several European, Asian, and African American populations.

SEC16B is not only a susceptibility locus for obesity in people of African and European descent but is also associated with BMI in people of Asian descent [59].

Along with this, there is an opposite view, so replicate studies of 27 polymorphisms in 14 genes of 1129 Japanese with obesity and 1736 subjects with normal weight, confirmed the association for only five polymorphisms, rs10913469 in SEC16B and four SNPs in the TMEM18 gene. rs543874 SEC16B showed little association with obesity in the Japanese population. The results suggest that only some SNPs identified in genome wide GWAS association studies in European populations are predisposition polymorphisms for obesity in other ethnic populations [9].

Five GWAS from Chinese, Malay and Indian ethnic groups were analyzed. As a result, in addition to the FTO gene, genes that had statistically significant associations with obesity in these populations were also found, one of which was SEC16B [60].

The **NEGR1** gene is a protein-coding gene localized in chromosome 1p31.16 and is among the predisposition loci for obesity [10].

The association of rs2815752 NEGR1 with overweight/obesity in different populations was studied. In order to identify the association of rs2815752 with overweight, obesity, and related traits in Pakistanis, Sobia Rana et al [61] conducted a study, as no such study has ever been conducted in the Pakistani population. The study found a significant gender-specific association of rs2815752 with obesity (OR 3.03; CI 1.19-7.72) and some abnormal anthropometric characteristics associated with obesity (weight, BMI, waist circumference, hip circumference, thickness of abdominal and iliopsoas folds) in women according to the dominant model. The researchers concluded that NEGR1 rs2815752 may be associated with the obesity phenotype and some related anthropometric traits in Pakistani women [61].

However, a number of independent replicative studies in other ethnic populations have failed to reproduce a reliable association of rs3101336, rs2568958, and rs2815752 of the NEGR1 gene with obesity traits [62].

Chloe YY, et al. [63] conducted a replicative study looking for associations of 13 previously identified SNPs with obesity and BMI in 1170 Chinese subjects. Statistically significant associations with obesity were found for 7 SNPs in the Chinese population, indicating the presence of common genetic variants with previously studied European populations. The authors attributed this to differences in the genetic composition and LD patterns between Chinese and European populations and the lower prevalence of these SNPs in the Chinese population [63].

A study was conducted on the effect of genetic variants affecting appetite on obesity in the population of Saudi Arabia. These variants included rs2815752 NEGR1 and rs543874 SEC16B. The result of the study showed that markers affecting fat mass through increased appetite affect obesity in Saudi residents, perhaps to a greater extent than in Europeans [64].

The results of GWAS analysis to search for associations with obesity found 16 significant genomic signals in the FTO gene, as well as quantitative signs of obesity (total body weight, waist circumference, and waist-to-hip ratio) in the **NRXN3** rs11624704 gene. It has been suggested that the NRXN3 gene is associated with the distribution of body fat [65]. Assessing the genetic contribution of the NRXN3 gene to the development of obesity is particularly challenging because NRXN3 is an extremely large gene (~1.5 Mb) [10], is controlled by two promoters, and has multiple transcripts. SNP polymorphisms related to weight and fat distribution are in different parts of the gene and are likely to involve different transcripts with potentially different functions.

Obesity impairs nervous system abilities such as memory and fine motor skills. Common biological mechanisms involved in obesity and neurodegenerative/neurodegenerative diseases are insulin resistance, proinflammatory cytokines, and oxidative damage, among others, leading to impaired brain development or cell death [66]. Environmental conditions contributing to obesity are not the only factors influencing neurodegenerative and neurodegenerative diseases. Moreover, in recent decades, the discovery of new genes related to obesity (FTO, NRXN3, NPC1, NEGR1, etc.) has opened new ways to understand the general mechanisms involved in these diseases. It should be noted that environmental conditions contributing to obesity, genes and gene-environment interactions will lead to a better understanding of the etiology of these diseases [66,67].

The minimal G allele rs10146997 in the NRXN3 gene is associated with increased waist circumference but is not associated with BMI scores, as reported in a recent meta-analysis of 249,796 individuals [9]. The authors attribute the reason for these discrepancies to the lack of statistical power of the study because of the relatively low population frequency of unfavorable alleles.

There is evidence that waist circumference and waist-to-hip ratio are associated with alterations in several genes, including NRXN3. To investigate the relationship between visceral fat and subcutaneous fat area and genes in the Japanese population, 8 single nucleotide polymorphisms (SNPs) were genotyped. Multiple regression analysis showed that NRXN3 was not significantly associated with BMI, visceral fat and subcutaneous fat area. These inconsistent results may be related to racial or regional differences in the frequency of this polymorphism [68].

Conclusion.

In the course of writing this article, we analyzed the results of GWAS and obesity meta-analysis from worldwide information databases and selected the most significant polymorphisms associated with obesity. Over the past decade, GWAS has demonstrated a large number of loci associated with BMI, providing an effective way to better understand the mechanisms of obesity that are needed in our journey to improve obesity treatment. Although large-scale GWAS has largely been performed on individuals of European ancestry, significant progress has been made, which is now closing the gap between our knowledge of obesity genetics in European and Asian ancestors.

Current evidence suggests that there are clear differences in the distribution of fat by ethnicity. In general, Asians accumulate more visceral fat than subcutaneous fat compared to Africans, Hispanics, and Caucasians. Current literature suggests that these differences are largely due to intrinsic genetic differences and physical traits that limit fat accumulation in certain areas. However, over the millennia, biological genetic differences have increasingly changed and become dependent on the environment (diet, physical activity, lifestyle and nutrition, stress, etc.) [69].

It should be noted that GWAS studies performed on individuals of Asian descent not only confirmed the potential role of previously linked obesity loci, but also revealed new loci.

Thus, multi-ethnic studies have enormous potential not only to better understand the complex etiology of obesity in humans, but also potentially ethnic differences in the prevalence of obesity, which may ultimately open up new possibilities for more targeted and personalized treatment of obesity. In addition, follow-up GWAS research strategies on multi-ethnic studies are worthwhile.

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РЕЗЮМЕ.

ЭПИДЕМИОЛОГИЯ ГЕНОВ, АССОЦИИРОВАННЫХ С ОЖИРЕНИЕМ В АЗИАТСКОЙ ПОПУЛЯЦИИ. ОБЗОР ЛИТЕРАТУРЫ.

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Введение: Среди общей популяции существует категория лиц с врожденной предрасположенностью к ожирению. В последние десятилетия рост распространенности ожирения и связанных с ним заболеваний во всем мире свидетельствует о необходимости изучения этиологических факторов, связанных с его развитием. В настоящее время еще неизвестно, сколько генов участвует в патофизиологии ожирения, проводится множество исследований по выявлению «генов-кандидатов» в клинической медицине. Выявление ожирения методами молекулярно-генетической диагностики и проведение соответствующих профилактических мероприятий может значительно снизить заболеваемость. Учитывая, что большинство людей, страдающих метаболическими заболеваниями, являются представителями трудоспособного населения, профилактика этих заболеваний, ранняя диагностика имеют как клиническое, так и социальное и экономическое значение.

Цель: анализ данных о наиболее значимых генетических маркерах в развитии ожирения в азиатской популяции.

Материалы и методы. Проведен онлайн поиск литературы о генетических маркерах, ассоциированных с ожирением в базах данных Elibrary, Google Scholar, Pubmed, Web of Science.

Результаты и обсуждение. Изучение генетических маркеров и их определение имеет большие перспективы для успешной верификации основных клинических и биологических маркеров ожирения. Это позволит оценить риски развития ожирения и разработать стандарты коррекционных мероприятий для лиц с высоким генетическим риском.

Вывод. Большинство GWAS исследования было проведено в европейской популяции, раскрытие новых генов у азиатской популяции внесли значительный вклад в выявление локусов предрасположенности к ожирению. Выявление полиморфных вариантов генов-кандидатов, наиболее значимых в развитии этого заболевания, представляет собой актуальную задачу, как для фундаментальной науки, так и практической медицины.

Ключевые слова: ожирение, полиморфизм генов, азиаты, genetics, GWAS, obesity, BMI, Asian population.

Summary.

EPIDEMIOLOGY OF GENES ASSOCIATED WITH OBESITY IN ASIAN POPULATION. LITERATURE REVIEW.

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Introduction. Among the general population, there is a category of individuals with an innate predisposition to obesity. In recent decades, the increase in the prevalence of obesity and related diseases worldwide indicates the need to study the etiological factors associated with its development. At present, it is still unknown how many genes are involved in the pathophysiology of obesity, and many studies are underway to identify “candidate genes” in clinical medicine. The detection of obesity by molecular genetic diagnostics and the implementation of appropriate preventive measures can significantly reduce the incidence. Considering that the majority of people suffering from metabolic diseases are members of the working population, the prevention of these diseases and early diagnosis are of both clinical and social and economic importance.

The aim of this study is to analysis of data on the most significant genetic markers in the development of obesity in the Asian population.

Material and methods. An online search for literature on genetic markers associated with the development of obesity was conducted in the databases Elibrary, Google Scholar, PubMed, Web of Science databases was carried out.

Results and discussion. The study of genetic markers and their determination has great prospects for the successful verification of the main clinical and biological markers of obesity. This will allow assessing the risks of developing obesity and developing standards for corrective measures for individuals with a high genetic risk.

Conclusion. Most of the GWAS studies have been conducted in the European population, the discovery of new genes in the Asian population has made a significant contribution to the identification of loci of predisposition to obesity. identification of polymorphic variants of candidate genes that are most significant in the development of this disease is an urgent task for both fundamental science and practical medicine.

Keywords: obesity, gene polymorphism, genetics, GWAS, obesity, BMI, Asian population.

შემაჯამებელი

სიმსუქნესთან ასოცირებული გენების ეპიდემიოლოგია აზიურ პოპულაციაში.

ბაზარგალიევი ე.შ., მაკაშოვა მ.ს., კუდაბაევა ხ.ი., კოსმურატოვა რ.ნ.

დასავლეთ ყაზახეთის სამედიცინო უნივერსიტეტი მარატ ოსპანოვის სახელზე, აქტობე, ყაზახეთი

შესავალი: ზოგად პოპულაციაში არის ინდივიდების კატეგორია სიმსუქნისადმი თანდაყოლილი მიდრეკილებით. ბოლო ათწლეულების განმავლობაში, სიმსუქნისა და მასთან დაკავშირებული დაავადებების გავრცელების ზრდა მთელ მსოფლიოში მიუთითებს მის განვითარებასთან დაკავშირებული ეტიოლოგიური ფაქტორების შესწავლის აუცილებლობაზე. დღეისათვის ჯერ კიდევ უცნობია რამდენი გენი მონაწილეობს სიმსუქნის პათოფიზიოლოგიაში და მიმდინარეობს მრავალი კვლევა კლინიკურ მედიცინაში „კანდიდატური გენების“ იდენტიფიცირებისთვის. სიმსუქნის გამოვლენა მეთოდოლოგიური გენეტიკური დიაგნოსტიკის მეთოდით და პროფესიული პროფილაქტიკური ინტერსტიციების პროგრამით შეიძლება იყოს გამაღიზიანებელი. იმის გათვალისწინებით, რომ მეტაბოლური დაავადებებით დაავადებულთა უმრავლესობა მშრომელი მოსახლეობის წევრია, ამ დაავადებების პრევენციას, ადრეულ დიაგნოზს აქვს როგორც კლინიკური, ასევე სოციალური და ეკონომიკური მნიშვნელობა.

ამ კვლევის მიზანია აზიის მოსახლეობაში სიმსუქნის განვითარების ყველაზე მნიშვნელოვანი გენეტიკური მარკერების შესახებ მონაცემების ანალიზი.

მასალები და მეთოდი. გამოქვეყნებულია ონლაინ ლიტერატურის შესახებ გენეტიკურ მარკერებში, ასოცირდება ზოგად ბაზარში.

ედეგები და დისკუსია. გენეტიკური მარკერების შესწავლას და მათ განსაზღვრას დიდი პერსპექტივა აქვს სიმსუქნის ძირითადი კლინიკური და ბიოლოგიური მარკერების წარმატებით გადამოწმებისთვის. ეს საშუალებას მისცემს შეფასდეს სიმსუქნის განვითარების რისკები და შემუშავდეს მაკორექტირებელი ზომების სტანდარტები მაღალი გენეტიკური რისკის მქონე პირებისთვის.

დასკვნა. კვლევების უმეტესობა ჩატარდა ევროპულ პოპულაციაში, აზიის პოპულაციაში ახალი გენების აღმოჩენამ მნიშვნელოვანი წვლილი შეიტანა სიმსუქნისადმი მიდრეკილების ლოკების იდენტიფიცირებაში. კანდიდატი გენების პოლიმორფული ვარიანტების იდენტიფიცირება, რომლებიც ყველაზე მნიშვნელოვანია ამ დაავადების განვითარებაში, გადაუდებელი ამოცანაა როგორც ფუნდამენტური მეცნიერებისთვის, ასევე პრაქტიკული მედიცინისთვის.

საკვანძო სიტყვები: სიმსუქნე, გენის პოლიმორფიზმი, აზიელები, გენეტიკა, სიმსუქნე, სხეულის მასის ინდექსი, აზიური პოპულაცია.