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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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ASSOCIATION OF NESFATIN-1 AND INSULIN RESISTANCE IN OBESE ADOLESCENTS OF IRAQ POPULATION

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Abstract.

Background: Nesfatin-1 reduces body weight and the intake of food, it is also tangled in setting insulin release. This study aims at comparing the levels of serum of Nesfatin-1 with the insulin resistance in obese adolescent of iraqi population with other nations around the world predicating development of diabetes mellitus later.

Method: 90 participants were needed for this cross-sectional study, including 30 control participants (17 men and 13 women) and 60 obese adolescents (36 men and 24 women). Serum glucose, insulin, and glycated hemoglobin in starved participants were estimated, using an ELISA kit, the serum level of Nesfatin-1 was measured, and insulin resistance was calculated.

Results: obese adolescents aged 12 to 18 and the control group, who were between 13 and 18 years old. The level of nesfatin-1 was significantly lower in the group of obese adolescents than in the controls. The ranges of Nesfatin-1 were (1.22±0.39 n/ml vs 2.54±0.64 n/m P = 0.001). In the control and obese groups respectively. In comparison to the results of the non-obese adolescent group, the obese group has significantly lower insulin sensitivity. Serum Nesfatin-1 is negatively associated with insulin sensitivity, lipid profile, and body mass index.

Conclusions: In general, our study revealed that there is no effect of food culture and eating intake on the role of Nesfatin -1 inducing obesity.

Key words. Obese adolescents, Nesfatin-1, Insulin resistance.

Introduction.

Obesity and overweight have become a global problem, affecting people of all ages, genders, and socioeconomic backgrounds. Among adolescents, the prevalence of obesity and overweight has been increasing in the last decades in whole world spacialy in developing countries. This fact is concerning, as adolescent obesity leads to a variety of health issues including chronic diseases and this could occur as they get older [1]. The pathogenesis of obesity is complex and involves multiple factors such as genetic, environmental, and lifestyle factors. The hypothalamus is the regulatory center for the balance of energy, and it plays a vital role in body weight homeostasis [2]. The gut and adipose tissue are examples of peripheral sites where peptides that regulate satiety and hunger can be found, these peptides play crucial roles in maintaining body weight homeostasis and also promote the pathogenesis of obesity, which has adverse impacts on the lipid, insulin, and blood pressure levels, in addition, many recent studies and researches have suggested that adipokines activity may perform a task in overweight etiology, due to all the previous reasons we are focusing in this study to a new adipokines that secret by adipose tissues constituting a link between food intake , body mass index and glucose level disturbances [3]. Nesfatin-1 have been recently identified as a neuropeptide that originates from the precursor protein nucleobindin-2 (NUCB2) [4]. It is produced by different organs, including the hypothalamus, white adipose tissue, pancreas, and ovaries. Nesfatin-1 has important anorexigenic effects and regulates food intake through a leptin-independent mechanism with the aid of a mechanism which depends on the melanocortin receptor. It also reduces blood glucose levels and performs a role within the regulation of glucose metabolism. Furthermore, researches have proven that nesfatin1 regulates cardiac functions, acts as a neuroendocrine regulator, decreases blood glucose levels, and causes loss of weight along with energy intake reduction. also, it reduces gastric emptying and gastric motility [4,5].

Another frequently discussed issue in the literature is whether or not obese children and adolescents secrete nesfatin-1. According to some studies, obese subjects' nesfatin-1 concentrations are lower than those of control subjects. In contrast, other studies have found that the overweight group had higher concentrations of nesfatin-1 than the control group did. These discrepancies can be the result of differences in methods and protocols used to measure levels of nesfatin-1 in various studies [6]. Overall, the role of nesfatin-1 in the pathogenesis of obesity and how it changes with different feeding behavior and eating habits is still not fully understood, so the study of such newly discover peptide like nesfatin-1 can deliver new insights into the pathogenesis of obesity which might give us potential therapeutic targets for the treatment and prevention of this worldwide issue.

Nesfatin-1 performs a fundamental part in glucose homeostasis, which is rationalised by the presence of NUCB2/nesfatin-1 and insulin together in human body and rodent pancreas [7], since glucose concurrently released with nesfatin-1 from the pancreatic cells [8,9]. Nesfatin-1 stimulates the flow of calcium involving the L-type channels, increasing the expression of pre-proinsulin mRNA as well as the secretion of glucose induced insulin, according to studies conducted in vitro. Studies that looked into the coincidence of nesfatin-1 and diabetes pathogenesis have shown discrepant finings [10]. Numerous studies have shown that patients with Type 1 DM have high nesfatin-1 levels, while those with Type 2 DM have low nesfatin-1 levels [6,7]. In the literature, there are some conflicts in the data related to nesfatin-1 secretion in obese children and adolescents. In control participants, the serum nesfatin-1 levels were dramatically higher than overweight participants [7]. Conversely, Anwar et al. found that the overweight group had a higher concentration of nesfatin-1 than the control group [5]. The aim of the study is to investigate the role of nesfatin-1 in adolescents suffering from obesity and evaluate its relation with insulin resistance.

Materials and Methods.

Study design: This prospective study was given approval, and it was confirmed that all participants provided written consent forms and they were collected. Obese and non-obese adolescents of secondary schools' students in Tikrit city included in this study in April and June 2022 divided into two groups study group and control group respectively.

BMI was calculated as well and Obese adolescent was defined as BMI > 95th percentile. After fasting for the previous night, a sample of venous blood was taken. Each blood sample was centrifuged at 3,000 rpm at +25 C for 15 minutes before being placed in an Eppendorf tube. Samples were moved on ice and stored at -20° C for the duration of the investigation, which took three months to complete. Insulin resistance was calculated using the homeostasis model of insulin resistance (HOMA-IR), which is equal to [fasting insulin (U/ml)] [fasting glucose (mmol/l)]/22.5. The enzyme-linked immunosorbent assay (ELISA) technique used for detection of serum nesfatin-1. Chemiluminescence was used to measure the plasma insulin levels. The photometric method was used to measure common biochemistry parameters like serum creatinine, glucose, cholesterol, and triglycerides.

Statistical analysis: All statistical analyses used SPSS for Windows version 18. Data expressed as mean ± standard deviation. P<0.05 considered as a significant.

Results.

Thirty healthy controls and a total of 60 obese adolescents (36 male and 24 female) enrolled in the present study. Table 1 contains participant demographic and laboratory information. Due to small group sizes and non-significant difference in serum nesfatin-1 in the male versus female participants in the two groups they are treated the same. The obese adolescent group's median age was 15.5 years, and 14.8 years for the healthy controls.

Table 1 summarizes the laboratory data that obtained from the groups studied. obese adolescents had significantly higher BMI, cholesterol, LDL, and triglyceride levels than the control group. The HDL level was significantly higher in control group versus overweight group. The obese group have higher insulin levels and insulin sensitivity measurements (HOMA-IR) than the control group. This study confirmed that levels of nesfatin-1 and

Table 1. *Comparison of study variables serum levels between obese and non-obese adolescent groups.*

Variable (mean±SD)	Control group (n=30)	Obese adolescent (n=60)	P value
BMI (kg/m^2)	21.3± 2.4	31.1±4.2	0.001
FBG (mg/dl)	85.5±15.3	92.4±18.4	0.06
Total cholesterol (mg/dl)	151.4±24.4	183.4± 16.4	0.001
HDL-C (mg/dl)	43.2 ± 12.5	34.4± 10.3	0.001
LDL-C (mg/dl)	84.8 ± 15.8	106.3± 10.5	0.001
Triglyceride (mg/dl)	72.6± 15.3	101.4± 20.8	0.001
Insulin (µlU/mL)	8.0± 1.81	12.13± 2.13	0.052
Nesfatin-1 (ng/ml)	2.54±0.64	1.22± 0.39	0.013
HOMA-IR	1.55± 0.5	2.62± 0.81	0.002
Liptin(ng/ml)	8.67± 1.81	13.3 ± 2.5	0.05

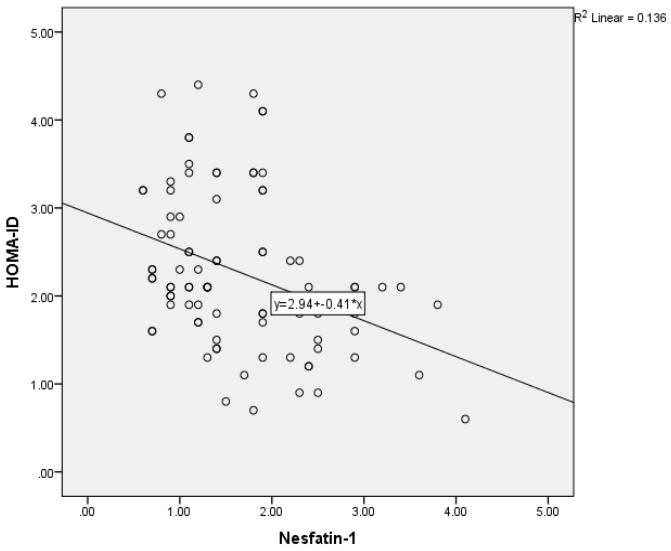

Figure 1. *The correlation analysis of serum nesfatin-1 versus HOMA-IR.*

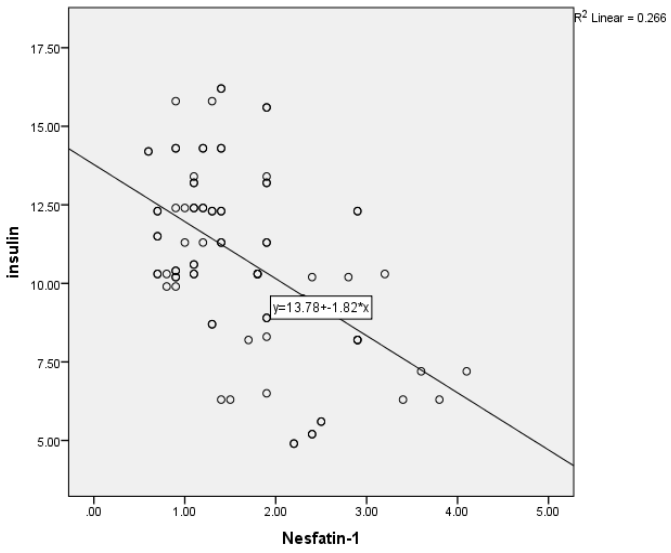

Figure 2. *The correlation analysis of serum nesfatin-1 versus insulin levels.*

HOMA-IR in both groups did not significantly differ between male and female.

Serum nesfatin-1 levels in the obese group were significantly lower than those in the control group. (1.22±0.39 n/ml vs 2.54±0.64 ng/ml, P =0.0013).

The results of the correlation between nesfatin-1 and various variables across the entire group of this study are shown in the figures below. We observed a negative correlation between nesfatin-1 and BMI, as well as HOMA-IR (Figure 1), insulin (Figure 2), and leptin (Figure 3) serum levels, also there was a negative correlation with cholesterol. TG and LDL-C while a positive corelation was found with HDL-C.

Discussion.

The findings of the present study revealed that serum nesfatin-1 concentration in overweight adolescents were

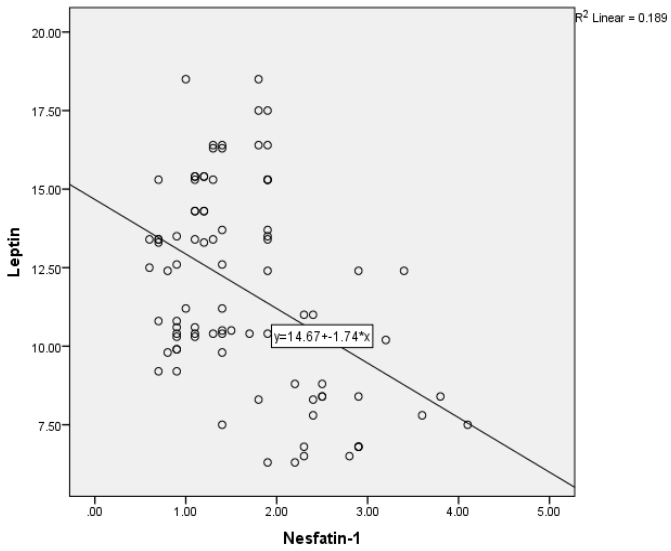

Figure 3. *The correlation analysis of serum nesfatin-1 versus leptin.*

significantly lower than those of normal weight adolescents and confirmed the reciprocal association of serum nesfatin-1 levels and overweight in adolescents. According to the study's data, serum nesfatin-1 levels were negatively correlated with BMI. In children and adolescents who are obese, low levels of nesfatin-1 may play a role in inadequately controlled food intake, Similarly, a number of animal studies strongly suggest that nesfatin-1 may contribute to energy homeostasis [11]. According to Shin-Hee et al, in vivo nesfatin-1 activity deficiency can lead to overweight and obesity [9]. Nesfatin-1 was chronically infused into the third ventricle of rats, and this consistently reduced body weight gain. Additionally, the deficiency of the prohormone convertase enzyme, which is necessary for the synthesis of nesfatin-1, causes the early onset of obesity [12].

Numerous studies looking into the reciprocal association of obesity with nesfatin-1 and proved that overweight participants have lower fasting nesfatin-1 levels than normal weight participants. Moreover, BMI and lipid parameters showed a negative correlation with nesfatin-1 levels [13-15].

Nesfatin-1 demonstrates anorexigenic activity through its effect on hypothalamus by mechanism independent of the release of leptin. in addition to various neurotransmitters regulating pituitary hormone and stress. Also, it reduces gastric motility and emptying [15].

Many previous studies demonstrate there is relationship between nesfatin-1 blood levels with fasting blood glucose, insulin resistance and blood insulin levels. Nesfatin-1's anti-hyperglycaemic effect following peripheral administration was clearly observed, and it was discovered that blood nesfatin-1 levels in diabetic patients were decreased by their anti-diabetic treatment.

According to an observation done by Nakata et al. (2011), that the relatively high CSF/plasma nesfatin-1 ratio can be used to explain the variations within nesfatin-1 levels between obese and non-obese subjects which suggests that a large amount of nesfatin-1 may originate from central neurons and therefore this may account for difference. Furthermore, it's likely that nesfatin-1 binds to proteins, and variations in protein binding between obese and lean subjects could also contribute to the explanation of these results. Last but not least, obese people may have a reduced nesfatin-1 uptake into CSF [12]. In disagreement with our study there are other study confirmed that the relationship between nesfatin-1 and BMI is a positive correlation [9,12,13].

Serum nesfatin-1 and BMI are positively correlated in obese adolescents, according to Anwar et al. Variation in gene polymorphism of nesfatin-1 of population, assessment methods, experimental conditions, commercial kit. Could contribute to these discrepancies [5].

Previous research had demonstrated a conflicting result regarding to the association between nesfatin-1 serum level and insulin sensitivity. According to this study, insulin resistance and nesfatin-1 levels in obese adolescents had a significant negative correlation. In the same study it was also seen that injection of nestafin-1 increases insulin secretion, so it effects on insulin sensitivity [15,17]. The effect of NES-1 in glucose metabolism and food intake could explain this negative correlation. The results of a study conducted by Niyan and his college agrees with this conclusion. Also, the anti-inflammatory effect of nesfatin-1 that has been recently discovered [6,18].

it has been confirmed by previous study that nesfatin-1 activates L-type Ca+2 channels in pancreatic islet beta cells which leads to an increase in insulin secretion , Nesfatin-1 raises the level of intracellular Ca+2 stimulated by glucose in pancreatic islet beta cells, leading to an increase in insulin secretion. Increase in calcium level inside the cells, occurs independently of protein kinase A and phospholipase A2, resulting in an increase in postprandial plasma glucose level [19,12].

Regarding to leptin levels, high levels of leptin can be explained as a response to high weight gain in obese adolescent compared with control group. Leptin hormone terminates eating. As a result, the high levels of leptin hormone circulating in obese individuals are most likely caused by the fact that leptin is meant to provide satiety in order to prevent further weight gain. Leptin inhibits NPY and food intake but stimulates energy expenditure and thus reduces body weight [20,21]. In addition to these measured parameters, tissue localized milieu including cellular trophic factors [22,23] should be considered as a contributor in pathogenesis of inflammatory reaction and their associated diseases.

Conclusion.

In conclusion, the results of this study indicate that BMI and serum nesfatin-1 levels were negatively correlated in the adolescent populations of Iraq. It implies that nesfatin-1 may play a significant role in regulating food intake in overweight/obese adolescents. Also, the strong negative correlation between serum level of nesfatin and insulin resistance that has been confirmed by this study shows the significance of Nesfatin-1 in the increase of insulin resistance in Iraqi obese adolescents. In general, our study revealed that there is no effect of food culture and eating intake on the role of Nesfatin -1 inducing obesity.

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