GEORGIAN MEDICAL NEWS

ISSN 1512-0112

NO 1 (370) Январь 2026

ТБИЛИСИ - NEW YORK

ЕЖЕМЕСЯЧНЫЙ НАУЧНЫЙ ЖУРНАЛ

Медицинские новости Грузии

საქართველოს სამედიცინო სიახლენი

GEORGIAN MEDICAL NEWS

Monthly Georgia-US joint scientific journal published both in electronic and paper formats of the Agency of Medical Information of the Georgian Association of Business Press. Published since 1994. Distributed in NIS, EU and USA.

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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Содержание:

GEORGIAN MEDICAL NEWS
No 1 (370) 2026

EVALUATION OF SERUM GALECTIN-3 LEVELS IN PATIENTS WITH HYPOTHYROIDISM AND HYPERTHYROIDISM IN AJMAN, UNITED ARAB EMIRATES

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Abstract.

Background: Galectin-3, a β-galactoside-binding lectin, is involved in fibrosis, inflammation, and carcinogenesis, and has been proposed as a biomarker in thyroid diseases. This study evaluated serum Galectin-3 levels in patients with hypothyroidism and hyperthyroidism and explored its association with thyroid function parameters. **Materials and Methods:** A cross-sectional analytical study was conducted on 96 participants categorized into three groups: hypothyroid (n=34), hyperthyroid (n=36), and euthyroid controls (n=26). Serum Galectin-3, TSH, and FT4 levels were measured using ELISA and standard biochemical methods. Data were analysed using SPSS software. ANOVA, t-tests, and Pearson correlation were applied to assess statistical significance and associations.

Results: Mean serum Galectin-3 levels were significantly higher in both hypothyroid and hyperthyroid patients compared with euthyroid controls ($p < 0.01$). However, no statistically significant difference was observed between the hyperthyroid and hypothyroid groups. Pearson correlation analysis demonstrated a significant negative correlation between FT4 and TSH ($r = -0.428$, $p < 0.01$), while no significant correlations were identified between Galectin-3 and FT4, TSH, or age. Chi-square analysis showed a significant association between thyroid disease category and Galectin-3 level groups ($p = 0.047$). **Conclusion:** Elevated Galectin-3 levels are associated with thyroid dysfunction in general but do not distinguish between hyperthyroidism and hypothyroidism. Therefore, Galectin-3 may reflect underlying inflammatory or fibrotic activity rather than serve as a differential diagnostic biomarker between thyroid functional states.

Key words. ELISA, Galectin-3, hyperthyroidism, hypothyroidism.

Introduction.

Hypothyroidism and hyperthyroidism are prevalent thyroid disorders that disrupt metabolic processes due to imbalances in thyroid hormone production [1,2]. Hypothyroidism, characterized by insufficient hormone production, leads to a slowdown in bodily functions [2], while hyperthyroidism, marked by excessive hormone production, accelerates metabolic activities [1]. Both conditions can significantly impact overall health and require appropriate medical management.

Galectin-3 is a β-galactoside-binding lectin involved in various biological processes, including cell proliferation, apoptosis, and immune responses. Its role in thyroid pathophysiology, particularly in autoimmune thyroid diseases and neoplasms, has garnered significant research interest [3].

A study by Ates et al. evaluated serum Galectin-3 concentrations in 46 patients with hypothyroidism compared to 37 healthy controls. The results indicated significantly elevated Galectin-3 levels in hypothyroid patients (median 2.89 ng/mL) versus controls (median 1.95 ng/mL, $p = .001$). Moreover, positive correlations were observed between Galectin-3 and TSH, anti-thyroglobulin, and triglyceride levels, while negative correlations existed with free T3 and free T4 levels. These findings suggest that increased Galectin-3 may contribute to thyroid gland hyperplasia and associated metabolic disturbances in hypothyroidism [4].

While direct studies on Galectin-3 levels in hyperthyroid patients are limited, its role in autoimmune thyroid diseases like Graves' disease has been explored. Elevated Galectin-3 levels have been associated with inflammatory processes in the thyroid, potentially influencing disease progression and severity. However, more targeted research is necessary to elucidate its specific role in hyperthyroid conditions [5].

Beyond functional thyroid disorders, Galectin-3 has been extensively studied in thyroid neoplasms.

Its overexpression is noted in malignant thyroid tissues, particularly papillary thyroid carcinoma (PTC). A meta-analysis demonstrated that Galectin-3 is a highly sensitive marker for PTC diagnosis, with higher expression rates in patients with lymph node metastasis, indicating its potential prognostic value [6].

Additionally, combining Galectin-3 with other markers like cytokeratin 19 (CK-19) and Hector Battifora mesothelial cell-1 (HBME-1) enhances diagnostic accuracy. For instance, a meta-analysis reported that Galectin-3 had a pooled sensitivity of 84.2% and specificity of 83.3% for PTC diagnosis, with an area under the curve (AUC) of 0.9128, indicating high diagnostic performance [5].

Galectin-3 emerges as a significant biomarker in thyroid pathology. Its elevated levels in hypothyroidism and potential involvement in hyperthyroid conditions underscore its relevance in autoimmune thyroid diseases. Furthermore, its diagnostic and prognostic utility in thyroid neoplasms, especially PTC, highlights its broader clinical significance. Future research should focus on longitudinal studies to validate these findings and explore therapeutic implications.

This study aimed to evaluate the association between Galectin-3 levels and thyroid function (TSH and free T4) in patients with hypothyroidism and hyperthyroidism and explore the potential of Galectin-3 as a biomarker for differentiating between hypothyroidism and hyperthyroidism.

Materials and Methods.

This cross-sectional case–control study was conducted at Thumbay Hospital, Ajman, UAE, between January and June 2025 to evaluate the association between serum galectin-3 levels and thyroid function in patients with hypothyroidism and

hyperthyroidism. A total of 96 adults aged 19–65 years were enrolled and subclassified into three groups: healthy controls (n = 26), patients with overt hypothyroidism (n = 34), and patients with overt hyperthyroidism (n = 36). Hypothyroidism was defined by elevated thyroid-stimulating hormone (TSH > 5.6 mIU/L) with reduced free thyroxine (FT4 < 9.01 pmol/L), while hyperthyroidism was defined by suppressed TSH (< 0.34 mIU/L) with elevated FT4 (> 19.05 pmol/L). Participants with pregnancy, malignancy, chronic liver or kidney disease, autoimmune or systemic inflammatory disorders, previous thyroidectomy or radioactive iodine therapy, or medications affecting thyroid function or galectin-3 levels were excluded. Venous blood samples were collected into serum separator tubes, allowed to clot at room temperature, centrifuged at 4000 rpm for 10 minutes, and sera were stored at −80 °C until analysis. Serum TSH and FT4 concentrations were measured using an automated Chemiluminescence immunoassay (CLIA) analyzer (Beckman Coulter DxI 800, Brea, CA, USA), with analytical ranges of 0.006–33.80 mIU/L and 3.47–65.77 pmol/L, respectively, and intra- and inter-assay coefficients of variation below 9%. Serum galectin-3 levels were quantified using a commercially available enzyme-linked immunosorbent assay (ELISA) kit (Human Galectin-3 ELISA, eBioscience, Franklin Lakes, NJ, USA), with an analytical range of 0.47–30 ng/mL, a detection limit of 0.29 ng/mL, and intra- and inter-assay coefficients of variation below 12% and 10%, respectively. Statistical analysis was performed using one-way ANOVA, independent samples t-tests, Pearson correlation, and Chi-square tests. Results were expressed as mean ± standard deviation, and a p-value < 0.05 was considered statistically significant. Ethical approval was obtained from the Institutional Review Board of Gulf Medical University, and written informed consent was signed by all participants.

Results.

The study included 96 patients, categorised into three groups: 36 patients in the Study group, indicating hyperthyroidism, 34 patients in the hypothyroidism group, indicating hypothyroidism, and 26 patients in the control group, comprising all healthy individuals. All individuals recruited for the study were between the ages of 21 and 60. The two groups were age-matched (p-value = 0.166) and gender-matched (p-value = 0.281).

Figure 1 illustrates the distribution of thyroid disease by gender. Among male participants, 25 (26.04%) had hyperthyroidism, 19 (19.79%) had hypothyroidism, and 19 (19.79%) were in the normal control group. Among females, hypothyroidism was more common (15.63%), followed by hyperthyroidism (11.46%) and normal status (7.29%). This suggests a gender difference in disease prevalence, with males more frequently affected by hyperthyroidism and females by hypothyroidism.

The highest frequency of disease occurred in the 26–45-year age group, with 22 hyperthyroid (22.92%), 20 normal (20.83%), and 17 hypothyroid cases (17.71%). The second most affected group was aged 46–65 years. Minimal cases were observed in individuals under 25 or over 66 years. This distribution suggests thyroid disorders are most prevalent in early to mid-adulthood.

Figure 2 shows that the majority of hyperthyroid patients (35 out of 36; 97.2%) exhibited elevated FT4 levels, falling into the high FT4 category, consistent with the expected biochemical profile of hyperthyroidism. In contrast, most hypothyroid patients (23 out of 34; 67.6%) had FT4 levels within the normal range, while 10 patients (29.4%) showed low FT4 values, reflecting the variability often seen in hypothyroid states. Nearly all individuals in the control group (25 out of 26; 96.2%) had FT4 levels within the normal range, indicating accurate classification. These distributions support the expected hormonal patterns across the groups, with elevated FT4 in hyperthyroidism, reduced FT4 in hypothyroidism, and stable normal FT4 values among controls.

Figure 3 illustrates that the majority of hypothyroid patients (29 out of 34; 85.3%) exhibited elevated TSH levels, while most hyperthyroid patients (31 out of 36; 86.1%) had suppressed TSH values, reflecting the typical hormonal profiles associated with these conditions. In the control group, TSH levels were predominantly within the normal range (24 out of 26; 92.3%), indicating appropriate classification. This strong alignment between clinical diagnosis and TSH levels reinforces the reliability of the group categorization used in this study. The clear bimodal distribution of TSH between hypothyroid and hyperthyroid patients highlights the continued diagnostic utility of TSH and supports the internal consistency and validity of the dataset.

Figure 4 illustrates the distribution of Galectin-3 levels (low, normal, high) by disease group. The majority of hyperthyroid

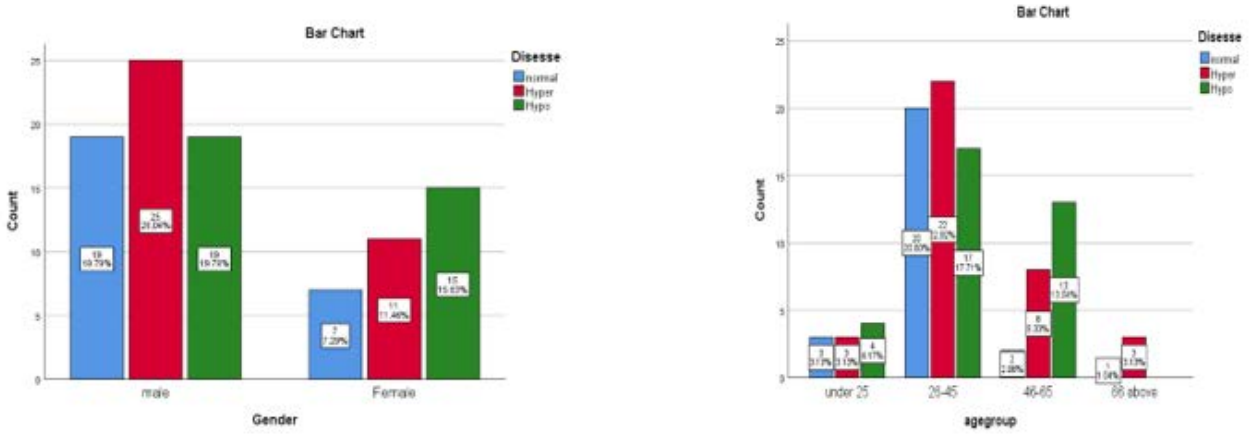

Figure 1. *Distribution of thyroid disease types by gender, age, disease status.*

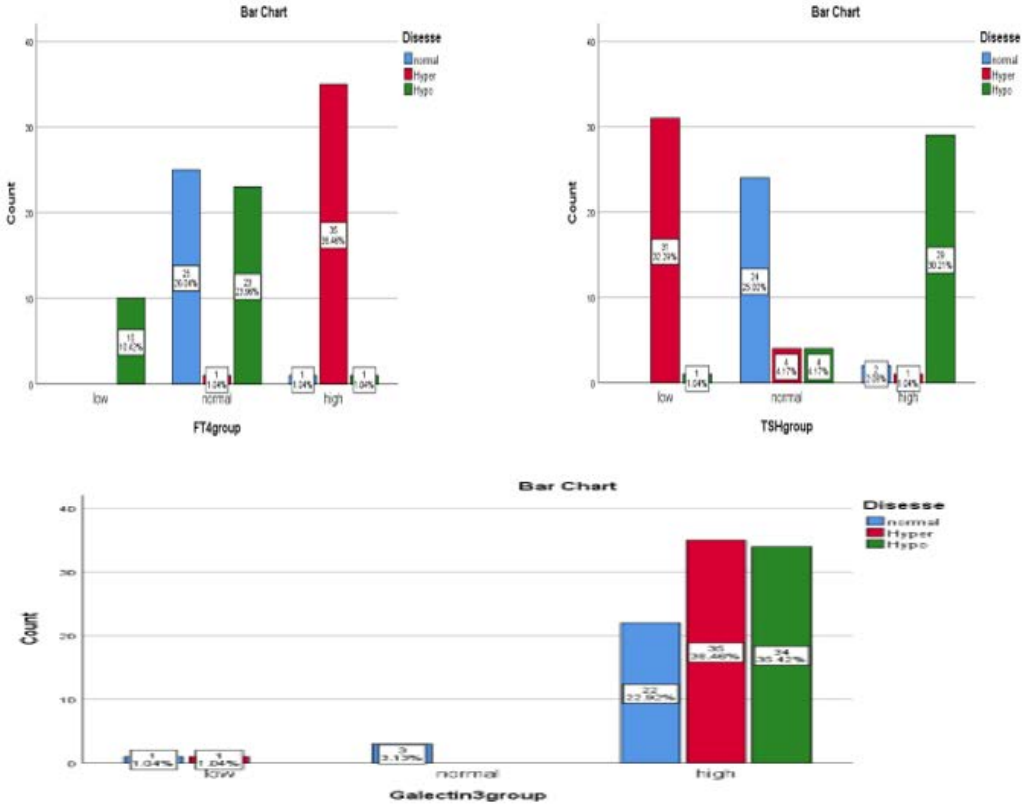

Figure 2. *Bar chart showing the distribution of disease status by FT4, TSH, and Galectin-3 values.*

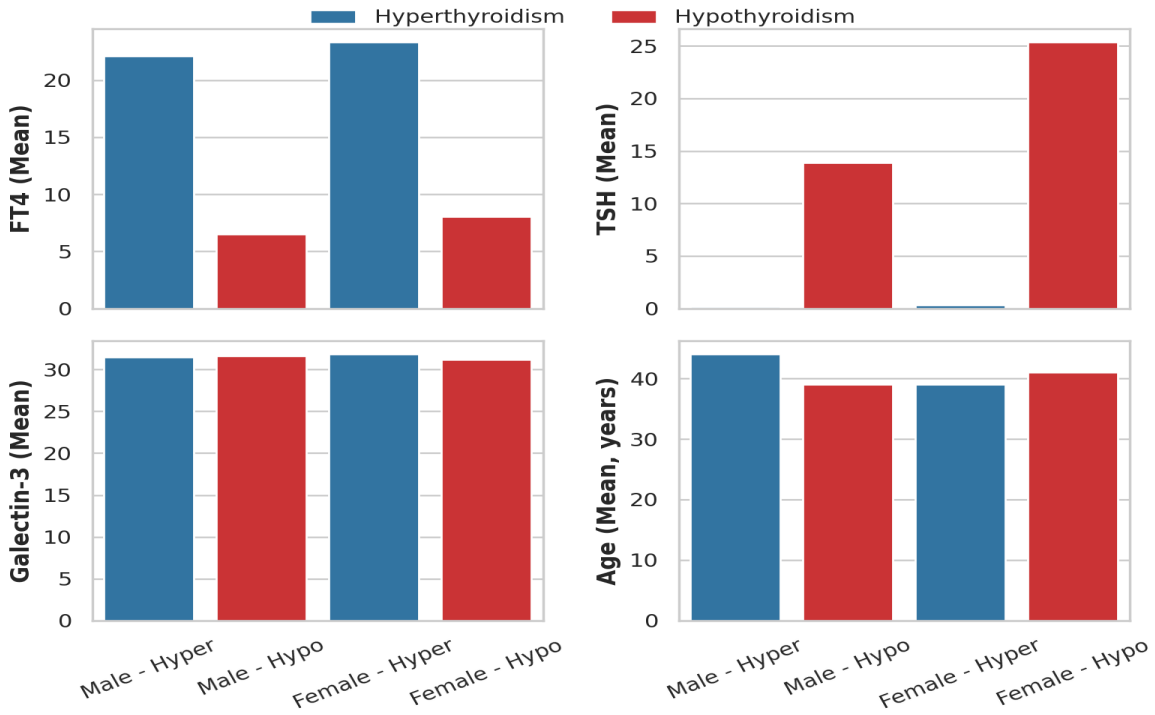

Figure 3. *Mean values of FT4, TSH, Galectin-3, and Age by Gender and Disease Type.*

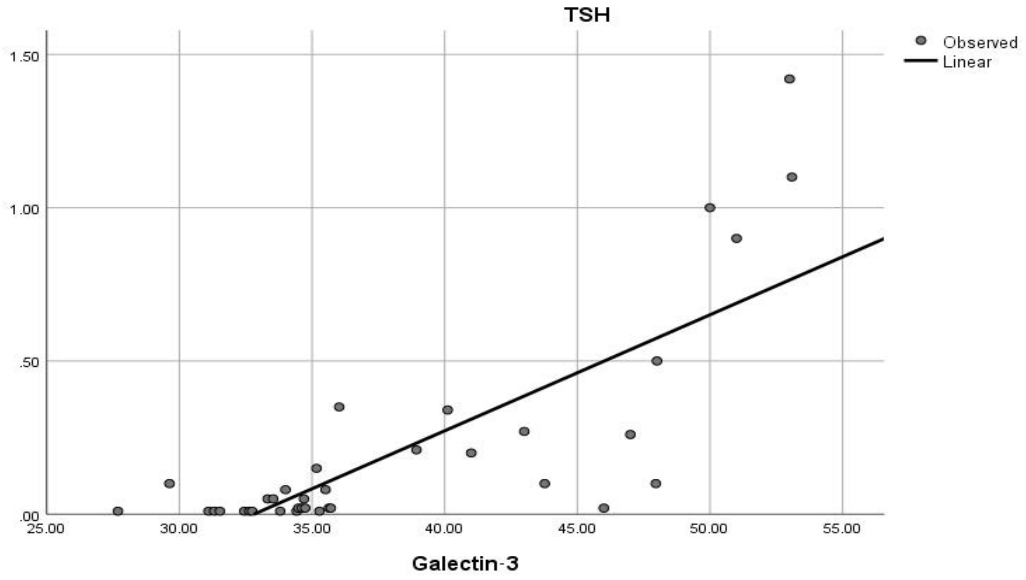

Figure 4. *Scatter Plot Showing the Linear Relationship Between Galectin-3 and TSH Levels.*

Table 1. *Comparison between FT4, TSH, Galectin-3, and Age Across Thyroid Functional Groups (Normal, Hypothyroid, and Hyperthyroid).*

Parameter	Source	Mean Square	F-value	Sig. (p-value)
FT4	Between Groups	2186.862	39.229	< 0.001 ***
	Within Groups	55.747		
Age	Between Groups	171.870	1.065	0.349
	Within Groups	161.358		
TSH	Between Groups	3565.466	44.290	< 0.001 ***
	Within Groups	80.502		
Galectin-3	Between Groups	112.734	6.670	0.002 **
	Within Groups	16.903		

*- ***p < 0.001 = highly significant*
*- **p < 0.01 = significant*

Table 2. *Relationships Among FT4, TSH, Galectin-3, and Age.*

	FT4	Age	TSH	Galectin-3
FT4	1	0.027	-0.428**	0.042
Age	0.027	1	0.075	0.143
TSH	-0.428**	0.075	0.07	0.02
Galectin-3	0.042	0.143	0.102	1

Note: p < 0.05 for the significant correlation between FT4 and TSH.
***. Correlation is significant at the 0.01 level (2-tailed).*

Table 3. *The association between Galectin-3 level groups and thyroid disease type.*

Test	Value	df	p-value
Pearson Chi-Square	9.660	4	0.047
Likelihood Ratio	10.052	4	0.040
Linear-by-Linear Association	4.758	1	0.029

P-value <0.005 indicates statistical significance.

(36.46%) and hypothyroid (35.42%) patients had high Galectin-3 levels. In contrast, only 22.92% of normal individuals had high Galectin-3. This further supports the association between elevated Galectin-3 and thyroid dysfunction.

Table 1 displays the mean values of FT4, TSH, Galectin-3, and age among male and female patients with hyperthyroidism and hypothyroidism. The table shows that FT4 levels were elevated in hyperthyroid patients across both genders, while TSH levels were markedly increased in individuals with hypothyroidism. Galectin-3 levels appeared slightly higher in hyperthyroid patients, though overall variability was limited. Additionally, the age distribution showed minimal differences among the study groups.

To compare the means of FT4, TSH, Galectin-3, and Age across normal, hyperthyroid, and hypothyroid groups, one-way ANOVA was conducted (Table 2)

Statistical analysis revealed significant differences in FT4 levels among the groups ($p < 0.001$), aligning with the expected

hormonal variations between hypothyroid and hyperthyroid states. TSH levels also differed significantly ($p < 0.001$), further supporting the validity of the diagnostic groupings. Additionally, Galectin-3 levels showed a statistically significant difference across the groups ($p = 0.002$), suggesting a possible association between Galectin-3 expression and thyroid dysfunction. In contrast, age did not exhibit a significant difference between groups ($p = 0.349$), indicating that age is unlikely to be a confounding factor in the observed biochemical patterns. The Tukey HSD test was applied to identify pairwise differences between the normal, hypothyroid, and hyperthyroid groups for FT4, TSH, and Galectin-3 levels (Table 3).

These results show that FT4 and TSH levels vary significantly across groups, as expected. Galectin-3 levels were markedly higher in both hyperthyroid and hypothyroid groups compared to normal, suggesting its potential role as a general biomarker for thyroid dysfunction. However, Galectin-3 levels did not significantly differ between hyperthyroid and hypothyroid patients. An independent samples t-test was performed to compare means between the normal and hyperthyroid groups for Age, FT4, TSH, and Galectin-3.FT4 levels were significantly higher in the hyperthyroid group compared with controls (control minus hyperthyroid mean difference = −11.32, $p < 0.001$), indicating markedly elevated FT4 concentrations in hyperthyroidism.

This analysis further supports the trend observed in previous tests. FT4 is significantly lower, and TSH is considerably higher in hypothyroid patients. Galectin-3 is also significantly elevated compared to the normal group, reinforcing its association with thyroid dysfunction. As with the comparison of hyperthyroidism, age differences between groups were not statistically significant.

Pearson correlation analysis demonstrated a significant negative correlation between FT4 and TSH ($r = −0.428$, $p < 0.01$), indicating physiological feedback regulation. No statistically significant correlations were identified between Galectin-3 and FT4, TSH, or age, which is consistent with the weak correlation coefficients observed.

To examine the association between Galectin-3 level groups (low, normal, high) and thyroid disease type (normal, hyperthyroid, hypothyroid), a chi-square test was conducted.

The chi-square test demonstrated a statistically significant association between disease type and Galectin-3 group ($\chi^2 = 9.660$, $p = 0.047$), suggesting that thyroid disease status is related to Galectin-3 level. Most disease cases, particularly hyperthyroid and hypothyroid, were associated with high Galectin-3 levels.

High Galectin-3 levels were prevalent across all study groups, with particularly high frequencies observed in hyperthyroid (97.2%; 35 out of 36) and hypothyroid patients (100%; 34 out of 34). Among euthyroid individuals, 84.6% (22 out of 26) also exhibited elevated Galectin-3 levels, while a small number had normal (3 patients) or low (1 patient) levels. Notably, none of the hypothyroid patients had Galectin-3 levels within the normal or low range. Overall, only 2 patients (2.1%) in the entire sample had low Galectin-3 levels, and just 3 patients (3.1%) fell within the normal range. These findings suggest a strong trend toward elevated Galectin-3 expression in both thyroid dysfunction and, to a lesser extent, in euthyroid individuals.

The average age of male participants was slightly lower than that of females. TSH and FT4 values were similar across genders, while Galectin-3 levels showed a small increase in females.

These summaries align with earlier inferential statistics. Hyperthyroid patients had the highest FT4 and lowest TSH levels, while hypothyroid patients had markedly elevated TSH. Both disease groups exhibited higher Galectin-3 concentrations compared to the normal group, reaffirming Galectin-3's potential role as a marker in thyroid dysfunction.

These detailed case summaries support the statistical findings and add context by showing consistent patterns across demographic and clinical variables.

A scatter plot with a fitted linear regression line was constructed to explore the relationship between Galectin-3 levels and TSH concentrations. As shown in Figure 4, the data reveal a positive linear association between the two variables. While most data points cluster around lower Galectin-3 and TSH values, a noticeable upward trend is evident. This suggests that increased Galectin-3 levels may be associated with elevated TSH, which supports the hypothesis that Galectin-3 plays a role in thyroid dysfunction, particularly in hypothyroid states where TSH is typically elevated. However, the presence of some variability implies that other regulatory mechanisms may also influence this relationship.

Discussion.

The present study investigated serum levels of free thyroxine (FT4), thyroid-stimulating hormone (TSH), and Galectin-3 in patients diagnosed with hyperthyroidism and hypothyroidism, with additional consideration of gender-related differences. The results revealed statistically significant differences in FT4 and TSH levels across the disease groups ($p < 0.001$), reaffirming well-established endocrine patterns associated with thyroid dysfunction. Specifically, patients with hyperthyroidism exhibited elevated FT4 levels due to increased thyroid hormone production, while those with hypothyroidism showed suppressed FT4 levels consistent with decreased thyroid gland activity. Conversely, TSH levels were markedly elevated in hypothyroid individuals as a compensatory response by the pituitary gland to low circulating thyroid hormone levels and significantly suppressed in hyperthyroid patients due to negative feedback from excessive hormone levels.

These hormonal profiles reflect the fundamental physiological mechanisms underlying thyroid regulation and serve as a validation of the diagnostic accuracy and classification used in this study. Furthermore, these findings are in line with previous research, such as that by Biondi and Cooper (2008), who detailed the biochemical and clinical features of thyroid dysfunction, and the clinical guidelines outlined by Garber et al. (2012), which emphasize the diagnostic role of TSH and FT4 measurements in thyroid disease. The consistency of the current results with the broader literature supports the reliability of the data and supports the use of these biomarkers in the clinical evaluation and differentiation of thyroid disorders [7,8].

Importantly, Galectin-3 also demonstrated a statistically significant variation among the study groups ($p = 0.002$), highlighting its potential relevance in the context of thyroid

disease beyond malignancy. While Galectin-3 is most widely recognized as a diagnostic marker for thyroid carcinoma—particularly in distinguishing malignant from benign thyroid nodules—it is increasingly being explored for its role in non-neoplastic thyroid conditions. In this study, elevated Galectin-3 levels were observed in both hyperthyroid and hypothyroid patients compared to the control group, suggesting that this protein may reflect broader pathological processes associated with thyroid dysfunction.

Galectin-3 is a multifunctional β-galactoside-binding lectin involved in a wide range of biological activities, including the regulation of cell proliferation, apoptosis inhibition, angiogenesis, tissue fibrosis, and immune system modulation. Its expression is known to increase in response to cellular stress and inflammation, which are common features in autoimmune thyroid diseases such as Hashimoto's thyroiditis and Graves' disease. The protein's involvement in tissue remodelling and fibrosis also suggests a role in chronic thyroid inflammation and long-standing hormonal imbalance. Studies by Yang et al. (2008) and Liu et al. (2012) have further emphasized the role of Galectin-3 in inflammatory signalling pathways and its ability to influence the immune response, which may contribute to thyroid tissue alteration even in the absence of malignancy.

These findings support the hypothesis that Galectin-3 could serve not only as a marker for malignancy but also as a potential indicator of inflammatory or autoimmune activity in thyroid disorders. Therefore, its clinical application may extend into the diagnostic and prognostic assessment of benign thyroid dysfunction, meriting further investigation in future longitudinal and mechanistic studies [9,10].

The elevated Galectin-3 levels observed in both the hyperthyroid and hypothyroid groups—particularly with slightly higher values noted in hyperthyroid patients—may indicate the presence of underlying subclinical inflammatory activity or tissue remodeling processes within the thyroid gland. This finding is consistent with the emerging view that Galectin-3 is not only a marker of malignancy but also plays a broader role in the pathophysiology of benign thyroid disorders. Inflammatory processes, extracellular matrix remodelling, and fibrosis are common in autoimmune thyroid diseases such as Hashimoto's thyroiditis and Graves' disease, both of which can occur alongside hypothyroid and hyperthyroid states, respectively. Galectin-3's known involvement in these biological processes—especially its roles in promoting fibrosis, inhibiting apoptosis, and modulating immune responses—suggests that its elevated serum levels may reflect ongoing immune-mediated damage or repair mechanisms within thyroid tissue, even in the absence of neoplasia.

This interpretation is supported by prior research. Bartolazzi et al. (2001) demonstrated that Galectin-3 expression is frequently elevated in thyroid tissues undergoing pathological changes, including inflammation and fibrosis, regardless of malignant transformation. More recently, Kim et al. (2021) reported associations between Galectin-3 and the degree of fibrotic remodelling in autoimmune thyroiditis, proposing it as a possible marker of chronic tissue injury. Together, these studies suggest that Galectin-3 may serve as a sensitive indicator of subclinical thyroid damage or immune activity and could have diagnostic or prognostic value in monitoring disease progression or treatment response in thyroid dysfunction [11,12].

Interestingly, the analysis revealed no statistically significant difference in age among the study groups ($p = 0.349$), suggesting that age is not a confounding variable in the observed variations in serum Galectin-3, FT4, or TSH levels. This is a remarkable finding, as it implies that the changes in Galectin-3 levels are more likely attributable to thyroid functional status rather than to age-related physiological or metabolic alterations. Since aging is known to influence endocrine function and inflammatory markers in general, ruling out its effect in this context strengthens the association between Galectin-3 and thyroid dysfunction itself.

This observation supports the growing body of evidence that Galectin-3 is more than just a marker for thyroid malignancy. Traditionally recognized for its role in identifying thyroid carcinoma, Galectin-3 is now being explored as a potential biomarker in non-neoplastic thyroid conditions. The findings from this study contribute to that evolving perspective by showing that Galectin-3 levels are elevated even in functional thyroid disorders such as hyperthyroidism and hypothyroidism, independent of age. This raises the possibility that Galectin-3 could serve as a supplementary tool in the clinical evaluation of thyroid disease, potentially aiding in the assessment of disease activity or severity alongside conventional thyroid function tests.

However, to fully establish Galectin-3's clinical relevance in benign thyroid pathology, further research is warranted. Future studies should involve larger, more diverse cohorts and incorporate correlations with histopathological findings, thyroid imaging (such as ultrasound or scintigraphy), and clinical outcomes.

Such investigations could provide deeper insights into the pathophysiological mechanisms linking Galectin-3 to thyroid tissue changes and help define its role as a diagnostic or prognostic biomarker in routine clinical practice.

Limitations.

A key limitation of this study is the absence of thyroid autoantibody measurements, including anti-thyroid peroxidase (anti-TPO) and anti-thyroglobulin (anti-Tg) antibodies. Since Galectin-3 is closely associated with inflammatory and fibrotic processes, correlating Galectin-3 levels with antibody titers would have provided important insight into whether the observed elevation reflects autoimmune activity, tissue remodeling, or other non-immune mechanisms. Consequently, conclusions regarding the underlying pathophysiological pathways remain speculative and should be interpreted with caution. In addition, the cross-sectional design and relatively modest sample size limit causal inference and generalizability. Future longitudinal studies with larger cohorts, antibody profiling, and imaging or histopathological correlations are recommended to better clarify the clinical and mechanistic significance of Galectin-3 in thyroid dysfunction.

Conclusion.

This study found that Galectin-3 levels were noticeably higher in both hypothyroid and hyperthyroid patients compared to healthy individuals, suggesting that this protein could be a

useful marker for detecting thyroid dysfunction. Its consistent elevation across both disease types points to a broader role in thyroid pathology, possibly linked to inflammation or autoimmune activity that often accompanies these conditions.

While Galectin-3 has already been recognized for its role in cancer and heart disease, its potential use in thyroid disorders is a newer and exciting development. These findings add to growing evidence that Galectin-3 may support traditional thyroid tests, especially in cases where diagnosis is uncertain or when a deeper understanding of disease activity is needed.

That said, this was a cross-sectional study with a relatively small sample size, so more research is needed to confirm these results. Future studies should follow patients over time to see how Galectin-3 levels change with disease progression or treatment. It would also be valuable to explore the biological mechanisms behind Galectin-3's involvement in thyroid disorders to better understand how it might be used in clinical practice.

In summary, Galectin-3 shows real promise as a diagnostic and possibly even a prognostic tool in managing thyroid disease. With further research, it could become a valuable part of how we understand and treat thyroid conditions.

Acknowledgment.

We would like to express our gratitude to all participants involved in the study.

Funding.

This research received no external funding.

Conflict of interest.

The authors declare that there is no conflict of interest.

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