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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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IMPACT OF VITAMIN D METABOLISM DISORDERS ON THE DEVELOPMENT OF AUTOIMMUNE KIDNEY DISEASES: A SYSTEMATIC REVIEW

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Abstract.

Introduction: Vitamin D plays a crucial role in immune regulation and renal physiology. It acts as a modulator of both the innate and adaptive immune systems. Vitamin D enhances the differentiation of monocytes into macrophages, thereby strengthening their phagocytic and chemotactic functions. Its deficiency has been associated with the pathogenesis of autoimmune renal diseases; however, the clinical and molecular mechanisms underlying this association remain insufficiently understood.

Aim: This systematic review focused on systemic lupus erythematosus with lupus nephritis (LN), IgA nephropathy (IgAN), and ANCA-associated vasculitis (AAV). The objective was to identify and synthesize available literature addressing the association between autoimmune kidney diseases and the dysregulation of vitamin D metabolism.

Methods: Studies published between 2020 and 2025 were retrieved from the Cochrane Library, Google Scholar, Scopus, Web of Science, and PubMed. The inclusion criteria emphasized investigations assessing vitamin D status and metabolism within the context of autoimmune kidney diseases.

Results: The analysis demonstrated that patients with LN, IgAN, and AAV exhibit a high prevalence of vitamin D deficiency, with rates frequently exceeding 70 percent. These values serve as descriptive summaries rather than as combined or pooled estimates.

Key words. Immune dysregulation, lupus nephritis, nephroimmunology, ANCA vasculitis, IgA nephropathy.

Introduction.

Vitamin D deficiency is prevalent in numerous autoimmune disorders, including renal autoimmune diseases such as lupus nephritis, IgA nephropathy, Goodpasture's syndrome, and ANCA-associated vasculitis [1]. In lupus nephritis, which is one of the most common complications of SLE, vitamin D deficiency is found to be inversely proportional to disease activity and renal involvement [1]. This indicates that low vitamin D levels in the body contribute to damage in the kidneys in autoimmune diseases. Moreover, vitamin D deficiency has been demonstrated to accelerate the development and progression of diabetic kidney disease [2]. Vitamin D is required in the metabolism process for the differentiation and functioning of T cells which plays a major role in the immune system. However, vitamin D deficiency can lead to an increase in pro-inflammatory T cells, which aggravates autoimmune attacks [3]. Increased sodium and water retention in vitamin D deficiency states has been associated with chronic renal inflammation and fibrosis which could trigger autoimmunity [4]. It has been shown that vitamin D might lower some inflammatory markers in patients with kidney autoimmune disorders, suggesting a possible therapeutic effect [5,6].

The regulatory functions of vitamin D on the immune system and the kidneys operates through diverse pathways. In the innate immune system, vitamin D augments the differentiation of monocytes to macrophages thus strengthening their phagocytic and chemotactic activity [7]. In addition, vitamin D enhances the synthesis of antimicrobial peptides which form parts of the first line defense of the body towards infectious agents [8]. Vitamin D affects blood vessel lining functions and podocyte cellular maintenance, moderates the renin-angiotensin-aldosterone axis, and has anti-inflammatory actions [9]. In chronic kidney disease (CKD), vitamin D deficiency is prevalent and postulated to stem from restricted absorption through the gut as well as reduced dietary intake [10]. Through the transcription of immune-response genes of macrophages, T cells, and dendritic cells, vitamin D modulates inflammatory processes [11]. Southeast Asia is one of the regions where vitamin D deficiency poses a significant health challenge. Results from 472 studies conducted on 746,564 subjects revealed an average 25-hydroxyvitamin D [25(OH)D] concentration of 49.39 nmol/L. The prevalence rates of deficiency (< 25nmol/L) varied by criteria as follows: 20.93%, 22.82%, 57.69%, and 76.85% [12]. It is estimated that SLE currently affects 3.41 million people worldwide, with an incidence rate of 5.14 per 100,000 person-years (8.82 in women; 1.53 in men) [13]. These figures further vary by region, exhibiting an incidence range of 0.3-8.7 and prevalence range of 3.2-159 per 100,000 [14].

Vitamin D deficiency occurs frequently with patients suffering from SLE because of lack of exposure to sunlight [15]. One significant factor that contributes to the development of autoimmune diseases is vitamin D deficiency [6,16]. The hypothesis of a Mendelian randomization study further reinforces the link between vitamin D levels and SLE, which reported that every standard deviation decreases in 25(OH)D3 levels is correlated with an increase in risk of SLE by 14.2% [17]. Another large cross-sectional study revealed that out of all patients suffering from autoimmune disorders, 82.9% were

vitamin D deficient. [18]. Due to lack of large-scale studies and over-reliance on small cross-sectional studies, there still exist critical gaps in knowledge regarding vitamin D's immune-modulatory function in autoimmune kidney diseases. This review synthesizes relevant literatures to clarify mechanistic pathways, hypothesize relationships with disease activity, evaluate polymorphisms within the VDR gene, and expose gaps in methodology with the aim of integrating clinical and experimental knowledge.

Methodology.

Search Sources: A systematic literature review was performed on four primary electronic databases: Scopus, PubMed, Web of Science (WoS), Google Scholar, and the Cochrane Library. These databases were chosen to achieve comprehensive coverage of the clinical and biomedical literature on autoimmune kidney diseases and vitamin D metabolism.

Search Strategy:

The approach of this search integrated the use of both free and controlled terms like MeSH in PubMed, which relate to the disorders of metabolism of vitamin D and autoimmune kidney diseases. Both the free and controlled vocabulary terms were combined using Boolean operators. This review was limited to the last five years, from 2020 to 2025, to reflect the most recent advancements in diagnostics and treatment, thus only peer reviewed English literature from this timeframe was included. The systematic methodology used adhered to rigorous standards in systematic reviewing which supports transparency and replication alongside thorough coverage of the literature. Such approaches have also been used successfully in other areas, including urology, showing the multidisciplinary applicability of some approaches to integration of evidence synthesis techniques [19].

Search Terms and Syntax:

The following search string were used across all the databases in other to ensure reproducibility: ("Vitamin D"[Mesh] OR "Vitamin D deficiency" OR "25-hydroxyvitamin D" OR "cholecalciferol" OR "calcitriol" OR "vitamin D metabolism") AND("Autoimmune Diseases"[Mesh] OR "autoimmune kidney disease" OR "systemic lupus erythematosus" OR "lupus nephritis" OR "IgA nephropathy" OR "ANCA-associated vasculitis" OR "autoimmune glomerulonephritis") AND("Kidney Diseases"[Mesh] OR "renal disease" OR "nephropathy" OR "glomerulonephritis"). This search was limited to only English articles published between the range of January 1, 2020 to May 1, 2025.

Inclusion Criteria:

Studies were included if they focused on autoimmune diseases with kidney involvement, evaluated the status and metabolism of vitamin D, and published within the date range.

Exclusion Criteria:

Research that did not consider vitamin D metabolism as well as its involvement in the pathophysiology of immune, or kidney systems was excluded together with purely editorial pieces, letters, commentaries, and non-autoimmune kidney disease focused works.

Study identification and Data Collection:

Identification of all articles through database searching allowed exporting to a reference managing software where duplicates were eliminated. Titles and abstracts were screened. All full texts of studies that met preliminary eligibility criteria were retrieved and assessed against inclusion/exclusion criteria. All disagreements were settled either through agreement or a third reviewer.

PRISMA Flowchart:

The entire selection procedure is captured in the PRISMA 2020 flow diagram which records the total count of records as they are identified, screened, included, excluded, and reasons for exclusions given at the full-text screening stage (Figure 1).

PRISMA Compliance:

This review adheres to the PRISMA 2020 framework for systematic investigations. To ensure a transparent process, the authors followed the corresponding PRISMA checklist at each stage. Items 1 and 2 appear in the title and abstract. The introduction presents the rationale cited in Item 3 as well as the specific objectives outlined in Item 4. Items 5 through 15 are covered in the methods section, including eligibility criteria (Item 6), information sources (Item 7), and study selection (Item 8). The authors evaluated risk of bias with the Newcastle-Ottawa Scale, fulfilling Item 11. The results section, in keeping with Items 16 through 22, summarizes study selection, characteristics, and synthesis. Finally, the discussion addresses Items 23 to 25, offering interpretation of findings, acknowledgment of limitations, and conclusions.

Risk of Bias Assessment.

The Newcastle-Ottawa Scale (NOS) was employed to assess bias in the observational studies, focussing on three key areas: how the study groups were selected, how comparable the groups remained, and how outcomes were recorded. Each study received a score ranging from 0 to 9 according to these criteria. Figure 2 below presents a summary of these NOS ratings for all included studies. Most investigations fell within the 6 to 8 bracket, suggesting overall moderate to high quality. Frequent shortcomings were the absence of participant blinding and uneven control for possible confounding variables. The NOS ratings then informed the strength-of-evidence evaluation included in the narrative synthesis. The NOS results were used to weigh the strength of evidence in the narrative synthesis.

Results.

Quantitative Overview of Study Characteristics:

Thirty-one studies met the inclusion criteria. Out of these, 11 (35%) used prospective cohort designs, 9 (29%) relied on retrospective cohorts, 5 (16%) were cross-sectional, 2 (6%) were randomized controlled trials, and 4 (13%) were laboratory experiments. Almost all observational studies were conducted within single hospitals and in a single center. The randomized trials employed parallel-group designs, with blinding and follow-up periods that varied across studies.

The sample size in individual articles ranged from 2 to 2,400 participants, with a median of 105 and an interquartile range of 45 to 288.

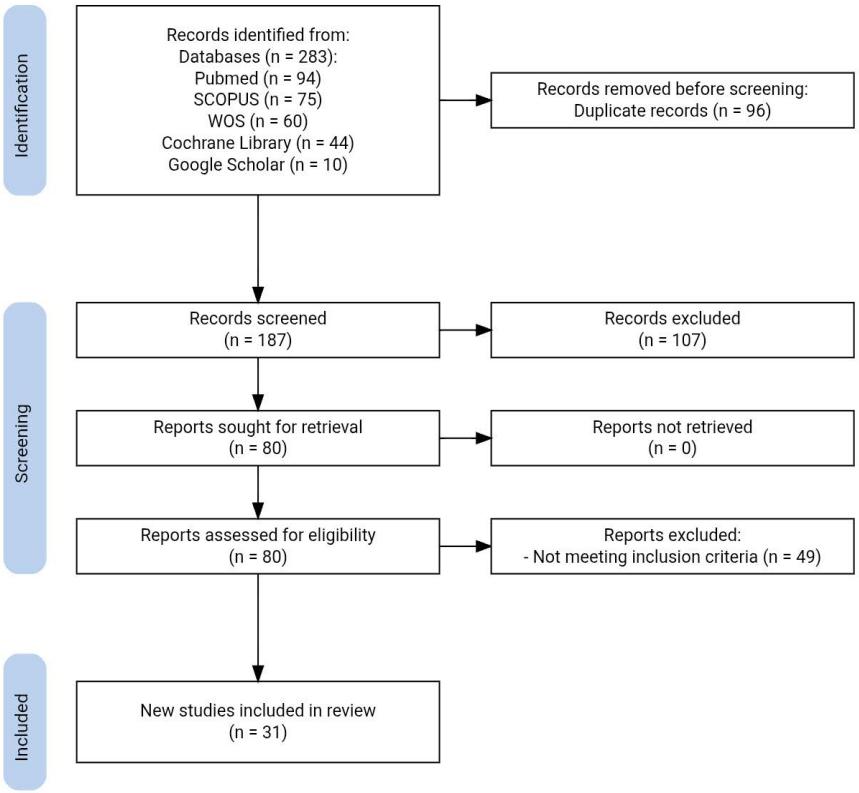

Figure 1. *PRISMA flowchart of selection process.*

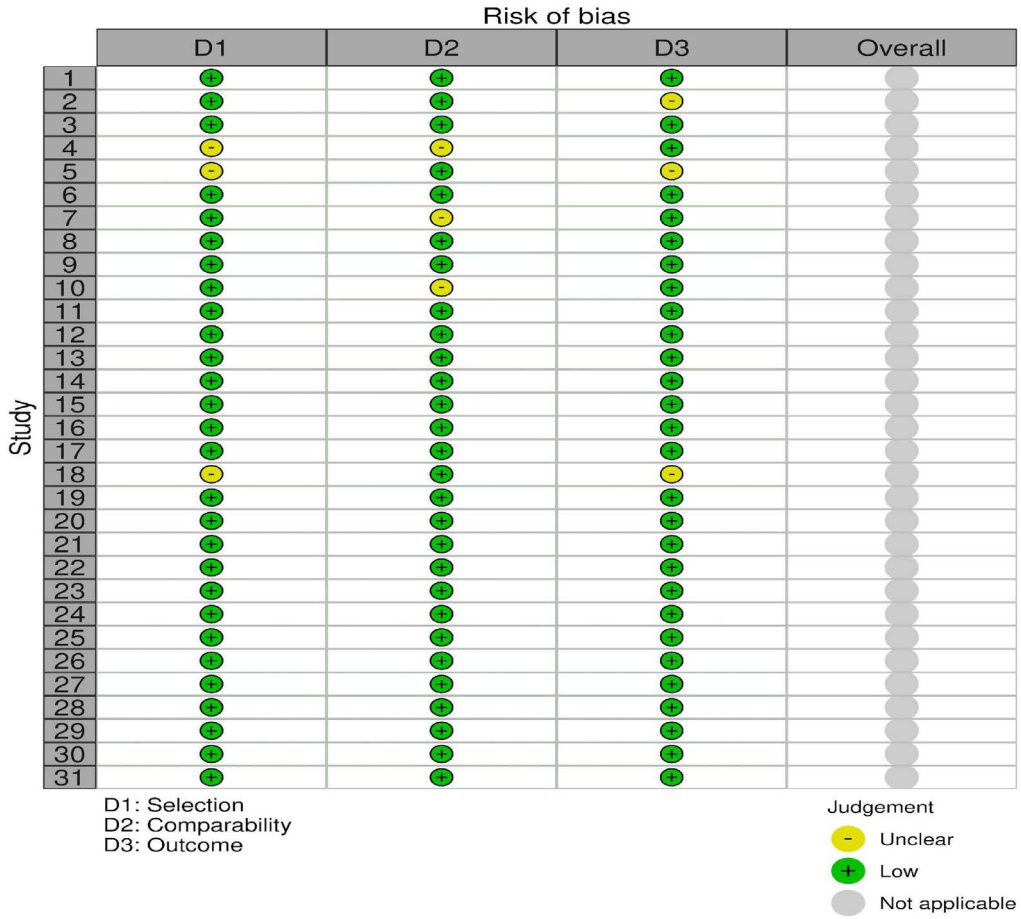

Figure 2. *Risk of Bias Assessment of Included Studies (Newcastle-Ottawa Scale).*

Geographically, 16 studies (52%) were based in Asia, 9 (29%) in Europe, and the remaining 3 (10%) each in North America and in South America combined with Africa.

Methodological quality of observational studies was evaluated using the Newcastle-Ottawa Scale. Scores fell between 5 and 9, with a median of 7, showing moderate to high quality overall; detailed scores are displayed in Figure 2. Lower ratings stemmed from weaknesses in outcome assessment or from follow-up that was too short or incomplete.

Summary of Included Studies:

Research indicated that both low ambient UVB exposure and lower vitamin D levels were associated with AAV relapse and activity [20-22]. In lupus nephritis (LN), vitamin D suppressed major proinflammatory pathways in mice [23,24]. Moreover, high expression of VDR within the kidney tissue was associated with better outcomes, while low expression of CYP27B1 was a marker for severe disease [25,26]. There were also reports of altered VDR and enzyme patterns in the kidneys of patients with IgAN [27], and deficiencies were connected to hypoalbuminuria [28], although genetic studies did not strongly find these associations [29]. Vitamin D analogs were shown to help with steroid-induced osteoporosis [30], but many populations had high rates of vitamin D insufficiency [31], though some other studies found benefit with combined paricalcitol and IL-17 blockade [32] or with more severe pathology in deficient children [33]. FokI polymorphisms were shown to increase the risk of aplastic anemia and systemic lupus erythematosus [34], while a few systematic reviews confirmed the protective and immune regulatory roles of vitamin D on the kidney [35-38]. In Egypt, lower levels were found in nephritic SLE patients along with associations with lipids and metabolic derangements [39-41]; other reviews concentrated on responder status to treatment [42,43]. In one of the included studies, vitamin D deficiency was associated with immune dysregulation [44], and more recently, vitamin D's role in the progression of LN was highlighted [45,46]. In addition, deficiency was associated with downregulation of PD-1/PD-L1 alongside VDR expression with higher disease activity, notably among FokI polymorphism carriers [47,48]. Lastly, new studies revealed relationships connecting vitamin D with the modulation of ferroptosis and suppression of CDK1 in LN, which supports its modulatory role in autoimmunity [49-51]. Table 1 provides a comprehensive summary of the included studies.

Discussion.

This review examined how the disorders of vitamin D metabolism influence the pathogenesis and the clinical manifestations of autoimmune kidney diseases. The aim was to assess the available data concerning vitamin D deficiencies, polymorphisms in the receptors of vitamin D, immune system disorders, and their relationship with the severity of the disease and kidney function.

The studies reviewed showed that patients with LN, IgAN, and AAV had a high prevalence of vitamin D deficiency. This deficiency is likely associated with greater disease activity, poorer kidney outcomes, and heightened immune activation.

In AAV, there was a clear association of low vitamin D levels with increased relapse risk in patients with lower exposure to UVB radiation [20]. Lower levels of serum 25(OH)D were associated with greater activity of the disease and lower quality of life [21,22].

For LN, both preclinical and clinical studies demonstrated the inhibitory effects of vitamin D on pro-inflammatory signaling pathways. Administration of $1,25(OH)_2D_3$ led to the inhibition of NF-κB, MAPK, and NLRP3 pathways, resulting in diminished kidney inflammation [23,24]. In kidney tissues, VDR expression correlated with the responsiveness to treatment, suggesting its usefulness as a prognostic marker and backbone of kidney prognosis [25]. On the other side, lower levels of CYP27B1, the enzyme responsible for vitamin D activation, were found in patients with greater disease activity [26].

In IgAN, dysregulated expression of VDR and enzymes responsible for vitamin D metabolism were identified within kidney tissue, indicating potential dysregulation of vitamin D signaling in the kidneys [27]. Vitamin D-deficient patients exhibited lower serum albumin levels, but GFR and proteinuria exhibited inconsistent correlations [28]. One of the studies pointed to the absence of vitamin D as an initiator for IgAN, though it was not conclusively determined [29]. Notwithstanding, prevalence estimates were still quite high, with 87.1% prevalence in one Malaysian cohort study [31]. Paricalcitol, an IL-17 blockade vitamin D analog, demonstrated improvement in treatment-resistant cases [32]. In contrast, pediatric patients with lower 25(OH)D levels exhibited more severe biopsy findings [33].

Genetic research has shed light on the relation of vitamin D with immune functions. The FokI polymorphism in the VDR gene was associated with greater disease activity and lower receptor expression in lupus nephritis (LN) patients [34,48]. It is crucial to recognise that the results summarised here rely on observational research and genetic-correlational designs, and therefore cannot be taken as evidence of direct cause-and-effect links. To clarify their biological meaning, further experiments that directly assess gene function are still necessary. This polymorphism is hypothesized to alter the functional form of the VDR protein and may affect its ability to modulate immune responses, although this relationship is yet to be established.

Another proposed mechanism is that vitamin D modulates ferroptosis-associated immune dysregulation in LN [49]. In addition, low serum vitamin D was shown to inversely relate to CDK1 gene expression and also correlate with greater disease severity [50,51].

The role of vitamin D in immunology expands beyond these disorders. It includes the enhancement of regulatory T cells, suppression of the immune Th1/Th17 responses, and the stabilization of immune equilibrium [52,53]. Moreover, vitamin D is important for the survival and functioning of immune cells [54], and its absence has shown correlation with increased susceptibility to infectious and autoimmune diseases [55].

Sufficient concentrations of serum vitamin D work by activating regulatory T cells (Tregs) and curbing pro-inflammatory T helper 17 (Th17) cells. Tregs induce immune tolerance and immune-protective autoimmune damage, whereas Th17 stimulates inflammation and autoimmune injury. Imbalance between these

Table 1. *Systematic Summary of Included Studies.*

Study ID	Disease	Study Design Type	Key Findings	Sample Size	Geographic Location
[20]	AAV	Ecological cohort	Low UVB/vitamin D associated with relapse risk.	2400 patients (Ireland 439; UK 1,961)	UK and Ireland
[21]	AAV	Cross-sectional	Deficiency associated with higher disease activity	125 patients (GPA 50; EGPA 50; MPA 25)	US and Canada
[22]	AAV	Observational Cohort	Lower 25(OH)D serum levels than healthy controls	104 (AAV: 54, controls: 50)	South Korea
[23]	LN	Experimental	$1,25(OH)_2D_3$ reduced NF-κB and MAPK activation	48 mice	China
[24]	LN	Experimental	Vitamin D suppressed inflammasome activity	N/A	China
[25]	LN	Biomarker analysis	VDR expression predicted treatment response	66 (responders: 50, non-responders: 16)	South Korea
[26]	LN	Molecular study	Low CYP27B1 associated with higher disease activity	112 (77 SLE patients + 35 controls)	Not specified
[27]	IgAN	Histological study	Altered VDR and vitamin D enzyme expression	17 (12 IgAN patients + 5 controls)	Croatia
[28]	IgAN	Prospective observational	Deficient patients had lower serum albumin	105 (IgAN patients)	India
[29]	IgAN	Mendelian randomization	Weak causal link via Mendelian randomization	N/A (Mendelian randomization)	China
[30]	IgAN	Retrospective cohort	Vitamin D slowed bone loss progression	48 (IgAN patients)	Japan
[31]	IgAN	Cross-sectional	87.1% patients had insufficient/deficient vitamin D	70 (IgAN patients)	Malaysia
[32]	IgAN	Case series	Paricalcitol + IL-17 therapy improved outcomes	7 (refractory IgA nephropathy patients)	Spain
[33]	IgAN	Paediatric observational	Low vitamin D associated with worse biopsy scores	N/A	China
[34]	LN	Genetic association	VDR polymorphisms associated with SLE risk	266 (SLE 128; controls 138)	Brazil
[35]	GN	Narrative review	Reviewed vitamin D's role in glomerulonephritis	N/A	Global review
[36]	Nephrolithiasis	Clinical safety study	High-dose vitamin D deemed safe short-term	2	Brazilnamd Russia
[37]	LN	Cross-sectional	Inverse relation of vitamin D to disease activity	166 (SLE: 100; controls: 66)	Egypt
[38]	Autoimmune	Narrative review	Vitamin D supported immune regulation	N/A	Global review
[39]	LN	Observational	Deficiency correlated with LN presence	100 (SLE patients)	Paraguay
[40]	Autoimmune	Review	Reviewed immunopathology and vitamin D	N/A	UK
[41]	LN	Metabolomics	Vitamin D deficiency disrupted lipid metabolism	31 (SLE patients)	China
[42]	LN	Cross-sectional observational study	Lower serum vitamin D levels were significantly associated with higher disease activity	150 (LN: 50; non-LN: 50; controls: 50)	Egypt
[43]	LN	Narrative review	Vitamin D supplementation may help reduce corticosteroid dosage and attenuate disease severity in SLE	N/A	Taiwan
[44]	Autoimmune disease	Narrative review	Vitamin D deficiency disrupts immune regulation	N/A	UK
[45]	LN	Review	Vitamin D and its analogs can modulate innate and adaptive immune responses by targeting VDR-expressing immune cells,	N/A	Canada
[46]	LN	Review	Vitamin D impacts lupus nephritis	N/A	Not specified
[47]	LN	Molecular study	55% deficient, 100% with >6 yr disease duration	72 (SLE patients)	Egypt
[48]	LN	Cross-sectional	Deficiency associated with low PD-1/PD-L1, high activity	50 (LN: 30; controls: 20)	Egypt
[49]	LN	Genetic association	FokI polymorphism associated with high activity	390 (SLE 194; controls 196)	Mexico
[50]	LN	Immunological review	Vitamin D regulated ferroptosis and T cells	N/A	China
[51]	LN	Case control	Low vitamin D correlated with CDK1, activity	70 (LN: 50; controls: 20)	Egypt

subsets has been associated with autoimmune kidney disease.

In turn, Vitamin D modulates important aspects of kidney physiology. It controls the renin-angiotensin-aldosterone system, protects podocyte function, and decreases kidney fibrosis [56,57]. The role of vitamin D on the immune system is very beneficial. The intersection of these pathways emphasizes the renal and immunologic protective roles of vitamin D. Assessing functionality of the VDR could be done through potential biomarkers beyond just serum concentration 25(OH) D, which may increase precision in diagnosis and therapy [58].

Polymorphisms of VDR genes, especially FokI, BsmI, and TaqI polymorphisms, influence the risk of developing autoimmune kidney disease. These polymorphisms change the VDR protein either by modifying its structure or its expression, which affects the immune system and the response to therapy [59]. The impact of these polymorphisms is heterogenous among different ethnic groups, but some studies show defined populations with corresponding distributions and impacts on disease prevalence [60,61]. This calls for tailored treatment in addition to highlighting the importance of pharmacogenomics in nephrology.

Besides genetic determinants, other more global molecular factors have been defined. Genetic studies have shown associations between vitamin D pathway genes and the risk of autoimmune kidney disease [62]. In addition, social, cultural, and ethical dimensions impact clinical outcomes. Even intervention strategies, like supplementation with vitamin D or other more comprehensive measures, are not decided independently of the context [63]. Thus, genetic research needs to be framed within ethical boundaries and patient-oriented frameworks [64]. This is further supported by the individual paradigm by the development of VDR-related biomarkers and multi-omic approaches.

However, methodological inconsistencies limit the generalizability of these findings. Most studies were observational, with a small number of randomized controlled trials (RCTs). The definitions of vitamin D deficiency were disparate and ranged from <25 to <50 nmol/L, making a lot of them difficult to compare directly. Some studies did not attempt to stratify patients based on genetics, comorbid disease severity, or concurrent treatments. Moreover, the protocols for vitamin D supplementation differed in terms of dosage, length, and type, which restricted the conclusions that could be drawn on assessment of efficacy. However, associations between VDR gene polymorphisms and disease outcomes should be interpreted with caution, as most studies were observational and may be influenced by certain factors.

The novel integrative approach of this review is what makes it unique. Rather than analyzing a single disease or mechanism as previous analyses did, this review conducted a wholistic analysis of LN, IgAN, and AAV, revealing shared and unique features of the diseases. It integrates immune regulation, kidney function, and genetics, offering a coherent framework while also introducing novel pathways like ferroptosis and CDK1 signaling into discussions concerning the therapeutic potential of vitamin D.

This review draws on a small pool of randomized controlled trials (RCTs), leaving most included research observational in nature, and that imbalance naturally raises concern about bias. Observational designs, especially when conducted retrospectively, are vulnerable to confounding, selection bias, and misclassification errors. RCTs, by contrast, aim to minimize these problems through random assignment and, when feasible, through blinding and placebo arms. Yet even the two RCTs featured here were small, and neither consistently documented allocation concealment or blinding, weakening their internal validity. To appraise quality, the Newcastle–Ottawa Scale (NOS) was reserved for the observational work and returned an overall rating of moderate to high; however, domains like outcome ascertainment and control for confounders displayed notable inconsistency. As a result, although the observational evidence adds valuable context, findings as such must be interpreted cautiously especially when drawing firm causal conclusions.

This review underlines the clinical relevance of vitamin D beyond that of a simple nutritional supplement. It reveals its utility as a marker for certain biological processes and as a risk factor which can be altered through appropriate intervention. Incorporating polymorphic analysis of VDR and functional tests into clinical workflows could enhance the precision and efficacy of the therapy [66,67].

Conclusion.

To conclude, the evidence on the role of vitamin D in autoimmune kidney diseases is quite diverse. Its deficiency is associated with increased disease activity and poor kidney outcomes: immune dysregulation in systemic lupus erythematosus nephritis and antineutrophil cytoplasmic antibody associated vasculitis (AAV). The evidence in IgA nephropathy tends to be more variable, but not unreasonably, supports a pathogenic role. The genetic variants of the vitamin D receptor (VDR) gene modulates the risk and responsiveness to treatment to a given disease further. These gaps, particularly the lack of longitudinal and interventional studies, strengthen the argument for the inclusion of vitamin D levels and VDR genotyping in the routine evaluation of patients with autoimmune kidney disease. Additionally, the role of vitamin D in immune modulation is far beyond renal autoimmunity to endocrine and reproductive disorders, including hypothyroidism in adolescents and preeclampsia in pregnancy.

REFERENCES

1. Athanassiou L, Kostoglou-Athanassiou I, Koutsilieris M, et al. Vitamin D and autoimmune rheumatic diseases. Biomolecules. 2023;13.

2. Souza CS, Deluque AL, Oliveira BM, et al. Vitamin D deficiency contributes to the diabetic kidney disease progression via increase ZEB1/ZEB2 expressions. Nutr Diabetes. 2023;13:9.

3. Grigg JB, Shanmugavadivu A, Regen T, et al. Antigen-presenting innate lymphoid cells orchestrate neuroinflammation. Nature. 2021;600:707-712.

4. Makin S. Cracking the code of autoimmunity. Nature. 2021;595.

5. Eisenstein M. Nature. Homing in on an oral link to inflammatory disease. 2021

6. Vahdat S. Vitamin D and kidney diseases: A narrative review. Int J Prev Med. 2020;11:195.
7. Caballero-García A, Córdova-Martínez A, Vicente-Salar N, et al. Vitamin D, its role in recovery after muscular damage following exercise. Nutrients. 2021;13:2336.
8. Hamza FN, Daher S, Fakhoury HMA, et al. Immunomodulatory properties of vitamin D in the intestinal and respiratory systems. Nutrients. 2023;15:1696.
9. Huang H-Y, Lin T-W, Hong Z-X, et al. Vitamin D and diabetic kidney disease. Int J Mol Sci. 2023;24.
10. Cianciolo G, Cappuccilli M, Tondolo F, et al. Vitamin D effects on bone homeostasis and cardiovascular system in patients with chronic kidney disease and renal transplant recipients. Nutrients. 2021;13:1453.
11. Tomaszewska A, Rustecka A, Lipińska-Opałka A, et al. The role of vitamin D in COVID-19 and the impact of pandemic restrictions on vitamin D blood content. Front Pharmacol. 2022;13:836738.
12. Jiang Z, Pu R, Li N, et al. High prevalence of vitamin D deficiency in Asia: A systematic review and meta-analysis. Crit Rev Food Sci Nutr. 2023;63:3602-11.
13. Tian J, Zhang D, Yao X, et al. Global epidemiology of systemic lupus erythematosus: a comprehensive systematic analysis and modelling study. Ann Rheum Dis. 2023;82:351-6.
14. Fatoye F, Gebrye T, Mbada C. Global and regional prevalence and incidence of systemic lupus erythematosus in low-and-middle income countries: a systematic review and meta-analysis. Rheumatol Int. 2022;42:2097-107.
15. Magro R, Saliba C, Camilleri L, et al. Vitamin D supplementation in systemic lupus erythematosus: relationship to disease activity, fatigue and the interferon signature gene expression. BMC Rheumatol. 2021;5:53.
16. Hysa E, Gotelli E, Campitiello R, et al. Vitamin D and Muscle Status in Inflammatory and Autoimmune Rheumatic Diseases: An Update. Nutrients. 2024;16:2329.
17. Ren YQ, Liu JP, Cui Y. Associations between vitamin D levels and systemic lupus erythematosus risk: a Mendelian randomized study. Zhonghua Yu Fang Yi Xue Za Zhi. 2023;57:891-8.
18. Kondratyeva EI, Loshkova EV, Odinaeva ND, et al. Seasonal fluctuations in vitamin D levels in children with various diseases. Eksp Klin Gastroenterol. 2022;6:5-13.
19. Veliyeva A, Omarova G, Mustafazade T, et al. Risk factors for postpartum stress urinary incontinence: An updated systematic review and meta-analysis. Electron J Gen Med. 2024;21:em595.
20. Scott J, Havyarimana E, Navarro-Gallinad A, et al. The association between ambient UVB dose and ANCA-associated vasculitis relapse and onset. Arthritis Res Ther. 2022;24:147.
21. Doubelt I, Cuthbertson D, Carette S, et al. Vitamin D status in ANCA-associated vasculitis. Rheumatol Adv Pract. 2023;7:rkad021.
22. Yoon T, Ahn SS, Pyo JY, et al. Serum vitamin D level correlates with disease activity and health-related quality of life in antineutrophil cytoplasmic antibody-associated vasculitis. Z Rheumatol. 2022;81:77-84.
23. Li X, Liu J, Zhao Y, et al. 1,25-dihydroxyvitamin D3 ameliorates lupus nephritis through inhibiting the NF-κB and MAPK signalling pathways in MRL/lpr mice. BMC Nephrol. 2022;23:243.
24. Huang J, An Q, Ju B-M, et al. Role of vitamin D/VDR nuclear translocation in down-regulation of NF-κB/NLRP3/caspase-1 axis in lupus nephritis. Int Immunopharmacol. 2021;100:108131.
25. Park DJ, Joo YB, Nam E, et al. Exploring potential multiple molecular biomarkers that predict treatment response in patients with lupus nephritis. Sci Rep. 2024;14:31422.
26. Luo M, Liu J, Yuan Y, et al. The role of vitamin D-synthesizing enzyme CYP27B1 in systemic lupus erythematosus. Turk J Med Sci. 2022;52:984-9.
27. Arapović A, Vukojević K, Glavina Durdov M, et al. Expression of renal vitamin D receptors and metabolizing enzymes in IgA nephropathy. Acta Histochem. 2021;123:151740.
28. Farooqui N, Subbiah A, Chaturvedi P, et al. Association of vitamin D status with disease severity and outcome in Indian patients with IgA nephropathy. BMC Nephrol. 2023;24:15.
29. Shi C, Cao H, Zeng G, et al. Mendelian randomization analyses explore the effects of micronutrients on different kidney diseases. Front Nutr. 2024;11:1440800.
30. Takiguchi R, Nishi S, Goto S, et al. Effect of bisphosphonate and active vitamin D analog on glucocorticoid-induced osteoporosis in patients with IgA nephropathy: A retrospective observational study. Kobe J Med Sci. 2023;69:E9-15.
31. Mustafar R, Nesam T, Kamaruzaman L, et al. Serum vitamin D levels among immunoglobulin A nephropathy patients and the associated parameters. Med J Malaysia. 2023;78:87-92.
32. Uriol-Rivera MG, Obrador-Mulet A, Juliá MR, et al. Sequential administration of paricalcitol followed by IL-17 blockade for progressive refractory IgA nephropathy patients. Sci Rep. 2024;14:4866.
33. Yu P, Zhang P, Gao C-L, et al. Clinicopathological significance and prognostic value of serum 25-hydroxyvitamin D3 level in children with IgA vasculitis nephritis. Zhongguo Dang Dai Er Ke Za Zhi. 2025;27:55-61.
34. De Azevêdo Silva J, de Lima SC, Fragoso TS, et al. Differential distribution of vitamin D receptor (VDR) gene variants and its expression in systemic lupus erythematosus. Int J Immunogenet. 2022;49:181-192.
35. Gembillo G, Siligato R, Amatruda M, et al. Vitamin D and glomerulonephritis. Medicina (Kaunas). 2021;57:186.
36. de Carvalho JF, Churilov LP. Safety of megadose of vitamin D in patients with nephrolithiasis. Nutrition. 2021;87-88:111201.
37. Khairallah MK, Makarem YS, Dahpy MA. Vitamin D in active systemic lupus erythematosus and lupus nephritis: a forgotten player. Egypt J Intern Med. 2020;32.
38. Bellan M, Andreoli L, Mele C, et al. Pathophysiological role and therapeutic implications of vitamin D in autoimmunity: Focus on chronic autoimmune diseases. Nutrients. 2020;12:789.
39. Acosta-Colman I, Morel Z, Paats A, et al. Association between vitamin D deficiency and disease activity in Paraguayan patients with systemic lupus erythematosus. Rev Colomb Reumatol. 2022;29:19-25.

40. Harrison SR, Li D, Jeffery LE, et al. Vitamin D, autoimmune disease and rheumatoid arthritis. Calcif Tissue Int. 2020;106:58-75.
41. Yan Y, Yu F, Li Q, et al. Metabolic alterations in vitamin D deficient systemic lupus erythematosus patients. Sci Rep. 2024;14:18879.
42. Elhosiny H, Kassem E, Ahmed A, et al. Study of Vitamin D in Patients with Systemic Lupus Erythematosus and Its Association with Lupus Nephritis. Egypt J Hosp Med. 2023;92:6664-71.
43. Ho L-J, Wu C-H, Luo S-F, et al. Vitamin D and systemic lupus erythematosus: Causality and association with disease activity and therapeutics. Biochem Pharmacol. 2024;227:116417.
44. Fletcher J, Bishop EL, Harrison SR, et al. Autoimmune disease and interconnections with vitamin D. Endocr Connect. 2022;11.
45. Artusa P, White JH. Vitamin D and its analogs in immune system regulation. Pharmacol Rev. 2025;77:100032.
46. Roveta A, Parodi EL, Brezzi B, et al. Lupus Nephritis from pathogenesis to new therapies: An update. Int J Mol Sci. 2024;25:8981.
47. Motawei N, Tawfik G, Omar H, et al. Vitamin D Deficiency in Patients with Systemic Lupus Erythematosus in Suez Canal University Hospital. Suez Canal Univ Med J. 2022;25:114-24.
48. Youssry S, Hussein A, Moaaz M. The immunoregulatory axis (programmed death-1/programmed death ligand-1) on CD4+ T cells in lupus nephritis: association with vitamin D and chemokine C-X-C motif ligand 12. Microbiol Immunol. 2021;65:392-9.
49. Meza-Meza MR, Vizmanos B, Rivera-Escoto M, et al. Vitamin D receptor (VDR) genetic variants: Relationship of FokI genotypes with VDR expression and clinical disease activity in systemic lupus erythematosus patients. Genes (Basel). 2022;13:2016.
50. Fan Y, Ma K, Lin Y, et al. Immune imbalance in Lupus Nephritis: The intersection of T-Cell and ferroptosis. Front Immunol. 2024;15:1520570.
51. Osman EMA, Abu El Nazar SY, Maharem DA, et al. Relation between vitamin D level and cyclin-dependent kinase-1 gene expression in Egyptian patients with lupus nephritis and their impact on disease activity. Indian J Nephrol. 2021;31:163-168.
52. Voiculescu VM, Nelson Twakor A, Jerpelea N, et al. Vitamin D: Beyond traditional roles-insights into its biochemical pathways and physiological impacts. Nutrients. 2025;17.
53. Sîrbe C, Rednic S, Grama A, et al. An Update on the Effects of Vitamin D on the Immune System and Autoimmune Diseases. Int J Mol Sci. 2022;23:9784.
54. Magro R, Saliba C, Camilleri L, et al. EP36 Vitamin D and interferon signature gene expression in systemic lupus erythematosus: a cross-sectional cohort study. Rheumatology (Oxford). 2020;59.
55. Wimalawansa SJ. Infections and autoimmunity-the immune system and vitamin D: A systematic review. Nutrients. 2023;15.
56. Lu M, Zhan Z, Li D, et al. Protective role of vitamin D receptor against mitochondrial calcium overload from PM2.5-Induced injury in renal tubular cells. Redox Biol. 2025;80:103518.
57. Zappulo F, Cappuccilli M, Cingolani A, et al. Vitamin D and the kidney: Two players, one console. Int J Mol Sci. 2022;23:9135.
58. Al-Ghafari AB, Balamash KS, Al Doghaither HA. Serum vitamin D receptor (VDR) levels as a potential diagnostic marker for colorectal cancer. Saudi J Biol Sci. 2020;27:827-32.
59. Agliardi C, Guerini FR, Bolognesi E, et al. VDR Gene Single Nucleotide Polymorphisms and Autoimmunity: A Narrative Review. Biology. 2023;12:916.
60. Trefilio LM, Bottino L, de Carvalho Cardoso R, et al. The impact of genetic variants related to vitamin D and autoimmunity: A systematic review. Heliyon. 2024:e27700.
61. Ruiz-Ballesteros AI, Meza-Meza MR, Vizmanos-Lamotte B, et al. Association of Vitamin D Metabolism Gene Polymorphisms with Autoimmunity: Evidence in Population Genetic Studies. Int J Mol Sci. 2020;21:9626.
62. Huang SU, Kulatunge O, O'Sullivan KM. Deciphering the Genetic Code of Autoimmune Kidney Diseases. Genes. 2023;14:1028.
63. Omarova G, Sultanova Z, Aimbetova A, et al. Cesarean Section: Medical, Social and Moral and Ethical Factors. Salud Cienc Tecnol. 2024;4:1337.
64. Becker AL, Carpenter EL, Slominski AT, et al. The role of the vitamin D receptor in the pathogenesis, prognosis, and treatment of cutaneous melanoma. Front Oncol. 2021;11:743667.
65. Kamri AM, Utami AN, Hasnawati A, et al. Comparison of the Safety Effects of Antiplatelets on the Kidneys in Patients with Vascular Disease. Futurity Medicine. 2024;3.
66. Donayeva A, Kulzhanova D, Amanzholkyzy A, et al. Relationship between vitamin D and adolescents' hypothyroidism – a cross-sectional study. Menopause Rev. 2023;22:186-190.
67. Franchuk U, Malanchuk L, Malanchyn I, et al. Vitamin D status: approaches to diagnosis and prevention in pregnant women at high risk of moderate preeclampsia. Rom J Diabetes Nutr Metab Dis. 2023;30:292-7.