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REVIEW ARTICLE

Interaction between genetic, immunological, and environmental factors in the development of vitiligo

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ABSTRACT

Introduction: vitiligo is the most common depigmenting disorder and represents a complex cutaneous disease with significant clinical, immunological, and psychosocial repercussions, whose origin is clearly multifactorial.
Objective: to describe the interaction of genetic, immunological, and environmental factors involved in the pathogenesis and progression of vitiligo.
Methods: a bibliographic review was conducted in international databases such as Medline, Embase, PubMed, Cochrane, and DARE, including studies published between 2019 and 2024. Selection criteria were applied to identify and select relevant sources for subsequent analysis.
Development: the literature demonstrates a strong genetic basis associated with genes related to innate and adaptive immunity, as well as melanocyte-specific genes that act as autoantigens. Multiple autoimmune comorbidities are described, with particular emphasis on thyroid diseases, alopecia areata, and psoriasis. Associations with hepatitis C, metabolic syndrome, and mild cardiovascular alterations are also identified. Environmental factors, such as exposure to phenols, smoking, certain medications, and inadequate diet, influence the development of intestinal dysbiosis and oxidative stress. The psychosocial impact is significant, with high prevalences of anxiety, depression, and neuropsychiatric disorders, especially in cases of early onset.
Conclusions: vitiligo results from a complex interaction between genetic predisposition, immune dysfunction, and environmental factors. Its clinical management should be comprehensive, taking into account systemic comorbidities and psychological support, which may optimize early diagnosis and guide more personalized therapeutic strategies.

Keywords: Autoimmunity; Risk Factors; Genetics; Disease Susceptibility; Vitiligo.

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INTRODUCTION

Vitiligo is understood as a depigmenting skin disorder characterized by the selective loss of melanocytes, leading to pigment dilution in affected skin areas. It presents in two main forms: non-segmental vitiligo (NSV) and segmental vitiligo (SV). The hallmark lesion is completely amelanotic—non-scaly, chalk-white in color, with well-demarcated borders, the appearance of which depends on its location and the type of vitiligo manifesting in the patient.[1]

Vitiligo is the most frequent cutaneous depigmentation disorder, with an estimated global prevalence of 0,5–2 %, affecting individuals of all ages and ethnic groups. Most cases appear before the age of 30, although onset in older adults is associated with autoimmune comorbidities such as thyroid disease, diabetes mellitus, rheumatoid arthritis, and alopecia areata. It is recognized as a multifactorial autoimmune disease influenced by genetic and environmental factors, as well as metabolic disturbances and oxidative stress. Although repigmentation therapies exist, no curative treatment is currently available. Vitiligo can affect any skin region, with predilection for the face, body folds, and bony prominences, as well as mucous membranes and other melanin-rich structures such as hair follicles and the iris.[2]

Vitiligo patches can have a significant psychosocial impact, especially in patients with dark or tanned skin or when lesions affect the face or hands. Affected individuals may experience stigma, often resulting in low self-esteem and lack of confidence. Children with vitiligo may be subjected to teasing and bullying at school. Despite this, only one study has evaluated psychological therapy for vitiligo, highlighting the profound psychosocial burden associated with the condition.[3]

It is important to consider specific risk factors, such as exposure to chemical phenols, which can act as tyrosine analogs and trigger antimelanocyte autoimmunity, a mechanism linked to vitiligo. Paracetamol (N-acetyl-p-aminophenol) is an over-the-counter phenolic analgesic. Although the risk of vitiligo from systemic paracetamol exposure has not yet been fully assessed, long-term use (>5 years) has been associated with high prevalence. Additionally, numerous drugs suspected of accelerating vitiligo development continue to be investigated.[4]

Genetic factors play a crucial role in vitiligo pathogenesis, as demonstrated by the discovery of multiple associated genes involved in innate and adaptive immunity. Genes related to innate immunity include *TICAM1*, *IFIH1*, *NLRP1*, *C1QTNF6*, *CASP7*, *TRIF*, and *FOXP3*; those linked to adaptive immunity include *CD80*, *IL2R*, *BACH2*, *CCR6*, *CTLA4*, and *PTPN22*. Furthermore, melanocyte-specific genes—such as *MC1R*, *TYR*, and *OCA2*—encode proteins that function as autoantigens, potentially triggering an immune response against melanocytes and thereby contributing to disease development.[5]

Among commonly associated autoimmune conditions, thyroid disorders are the most prevalent (22 %) in this population, particularly in patients whose vitiligo onset occurred after age 30—reflecting the autoimmune nature of the disease and underscoring its relevance for early diagnosis and appropriate management. Given that autoimmune diseases encompass a broad spectrum of pathologies, their comprehensive evaluation is essential.[6,7]

Understanding the clinical evidence surrounding vitiligo—including its risk factors—is fundamental for guiding future research and enabling accurate interpretation in patient care. Therefore, this work focused on synthesizing current knowledge through bibliographic reviews regarding its incidence, mortality, and characterization, due to the scarcity of previously published investigations. Based on this rationale, the present review was conducted with the

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objective of describing the interaction among genetic, immunological, and environmental factors involved in the pathogenesis and evolution of vitiligo.

METHODS

A bibliographic review was carried out in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines. The methodological objective was to identify, evaluate, and synthesize the available scientific evidence on the topic. The search period spanned from January 2010 to December 2024 to ensure inclusion of current and relevant literature, reflecting conceptual and methodological advances in the field.

Information sources included internationally recognized electronic databases: PubMed/MEDLINE, SciELO, ScienceDirect, Google Scholar, LILACS, and BVSALUD. Additionally, reference lists of selected articles were manually reviewed to identify potentially relevant studies not captured in the initial search. Grey literature—including theses and institutional academic documents—was also considered, provided it met established quality and thematic relevance criteria.

The search strategy was designed using standardized DeCS and MeSH descriptors combined with Boolean operators. The algorithm included key terms related to the central theme: ("vitiligo" OR "vitíligo") AND ("genetic factors" OR "immune factors" OR "environmental factors" OR "risk factors" OR "pathogenesis"). Search strings were adapted to the specific syntax of each database. Publications in Spanish, English, and Portuguese were included to broaden coverage and minimize language bias.

Inclusion criteria encompassed original articles, systematic reviews, and observational studies published within the defined timeframe, with full-text availability and direct relevance to the research topic. Excluded were duplicate studies, publications outside the temporal range, articles without full-text access, isolated case reports, and those providing no relevant information. The selection process was conducted in three phases: title screening, abstract evaluation, and full-text analysis. Initially, 55 records were identified; 10 were removed as duplicates. After title and abstract screening, 16 articles were excluded, and 29 full texts were analyzed—all of which were included in the final synthesis. This process was documented using a PRISMA flow diagram (Fig. 1).

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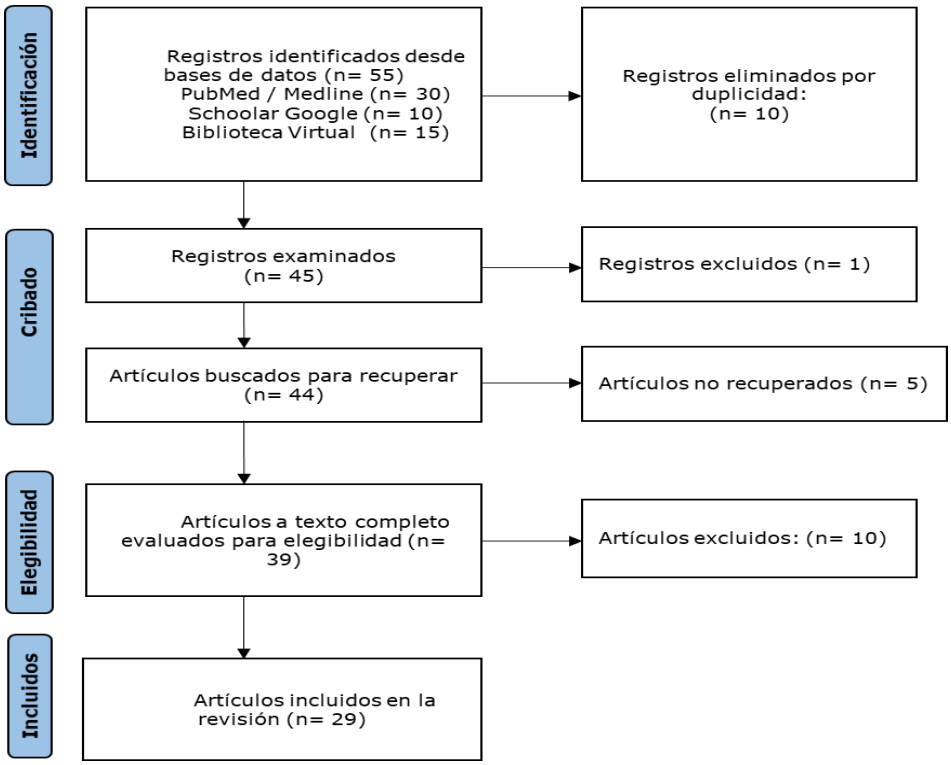

Fig. 1. PRISMA flow diagram for study selection.

Data extraction and analysis were carried out systematically using a standardized matrix. Information was integrated through a narrative qualitative synthesis, as methodological heterogeneity among the studies precluded quantitative meta-analysis. This approach enabled the identification of patterns, convergences, and relevant knowledge gaps for a comprehensive understanding of the studied phenomenon.

DEVELOPMENT

Any disease that disrupts the immune system is associated with adverse events that, one way or another, affect organs. This creates a vicious cycle, as these adverse events may interrupt treatment or necessitate systemic immunosuppressive therapy—posing a destructive factor for patients with such immune alterations. Therefore, it is vital to understand the severity of risk factors in vitiligo patients and mitigate the risk of major complications according to their disease stage.[8]

Another critical aspect is quality of life, which can be significantly impaired by psychosocial comorbidities in vitiligo. The most representative conditions are anxiety and depression—including major depressive disorder, bipolar disorder, and dysthymia—with a reported prevalence ranging up to 62,3 %. Additionally, anxiety disorders—such as generalized anxiety disorder, agoraphobia, social phobia, and panic disorder—show a prevalence of 67,8 %. Sleep disturbances, anger, emotional deterioration, cognitive impairment, behavioral alterations, and somatoform disorders are also commonly observed.[9]

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A study by Han et al.,[10] described that many complications or risk factors in vitiligo patients manifest early in childhood. Maternal autoimmunity is associated with a 31 % prevalence of autoimmune, allergic, and neuropsychiatric disorders in offspring. This indicates a strong association between vitiligo and autoimmune diseases—and their severity—particularly when autoimmune conditions appear in childhood. Among the findings were: alopecia areata (64 %), alopecia totalis (63 %), psoriasis (40 %), atopic dermatitis (20 %), allergic rhinitis (30 %), asthma (45 %), attention-deficit/hyperactivity disorder (90 %), and mood disorders (39 %).

When addressing dermatological diseases, it is essential to describe their associated risks. In vitiligo patients, there is a significant incidence of concurrent psoriasis and a high prevalence of preexisting psoriasis. A study by Kridin et al.,[11] found a strong bidirectional correlation: vitiligo patients have a 64 % higher risk of developing psoriasis. These percentages reveal that when an immune-mediated condition coexists with psoriasis, the clinical picture is exacerbated, often leading to additional comorbidities. There is a 95 % risk of developing pronounced scaling and even marked erythroderma.

Regarding other dermatological alterations, Monseley et al.,[12] warned that alopecia areata (AA)—the most common immune-mediated form of hair loss—is present in 64 % of vitiligo patients. A personal history of any immune-mediated disease plays a fundamental role in AA risk (95 %), as it is not only linked to vitiligo but also to multiple sclerosis, systemic lupus erythematosus, psoriasis, hypothyroidism, and rheumatoid arthritis. In contrast, inflammatory bowel disease and Graves' disease/hyperthyroidism show no significant association with AA risk. Wu et al.,[13] reported a modest association between vitiligo and cardiovascular events—a 3 % risk—which, although seemingly minor, must be considered depending on the patient's overall pathology. Taneja et al.,[14] emphasized the importance of routine screening in vitiligo patients, highlighting abnormal cholesterol and homocysteine levels in lipid profiles. They recommend routine assessment of homocysteine and lipid profiles in vitiligo patients, as these should be considered significant and independent contributors to cardiometabolic risk.

Vallerand et al.,[15] detailed how skin involvement in vitiligo negatively impacts mental health. Their findings indicate that patients with major depressive disorder (MDD) have a 64 % higher risk of worsening or developing vitiligo. Corroborating this, patients using antidepressants showed a 17 % improvement with reduced depressive symptoms. Chang et al.,[16] focused on the link between autoimmune disorders and subsequent dementia risk, noting that psychiatrists and neurologists diagnosed dementia in vitiligo patients. The risk was 52% higher compared to controls.

Additionally, vitiligo in adults is associated with hepatitis C virus (HCV) infection, with a reported incidence of 34,4 %. Fawzy et al.,[17] suggested that HCV may trigger adult-onset vitiligo. Thus, adults with new-onset vitiligo—unlike childhood-onset cases—should be screened for HCV in endemic regions. Ma et al.,[18] noted that HCV infection is linked to several cutaneous manifestations, including lichen planus and psoriasis. Compared to controls, HCV-infected patients had a 63,4 % higher risk of developing lichen planus, psoriasis, vitiligo, alopecia areata, and cutaneous lupus erythematosus.

Regarding autoimmune comorbidities like alopecia areata, Abd El-Raheem et al.,[19] found no association with mutant genotypes versus wild-type, nor with allele A versus allele G. However, vitiligo risk was significantly higher with *TNF-α* G/A and A/A genotypes (11% baseline), indicating that *TNF-α* mutant genotypes confer susceptibility to vitiligo in Egyptian patients. Arousse,[20] observed that family history influences 22,1 % of cases, with severe forms showing earlier onset and more persistent disease duration.

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Metabolic syndrome is another key concern. Tanacán et al.,[21] reported metabolic syndrome rates of 37,4–40 % related to cholesterol levels, with a 26,5 % incidence of disease severity in vitiligo patients. Taneja et al.,[14] linked vitiligo to metabolic abnormalities—including glucose and lipid dysregulation—as well as vitamin B12 and folate deficiencies, which elevate circulating homocysteine levels, reflecting ongoing abnormal metabolic processes.

Environmental factors also play a role. Enomoto et al,[22] identified smoking as a significant risk factor for hand vitiligo. While non-smokers showed widespread distribution—including fingertips and joints—smokers exhibited lesions predominantly on fingertips. Lee et al.,[23] reported a 69 % increased vitiligo risk in smokers after adjusting for age, sex, exercise, alcohol use, BMI, diabetes, hypertension, dyslipidemia, stroke, and ischemic heart disease history. Paradoxically, these results suggest smoking may exert suppressive effects on vitiligo development.

Several authors also note an altered skin cancer risk profile in vitiligo. Kim et al.,[24] found increased skin cancer risk in the Korean vitiligo population. However, white vitiligo patients show lower incidence of both melanoma and non-melanoma skin cancers compared to controls. Bae et al.,[25] explained that vitiligo-associated autoimmunity affects cells beyond melanocytes, conferring reduced skin cancer risk. Notably, vitiligo patients showed a 95 % significantly reduced risk of malignant neoplasms in organs such as the colon and rectum.

The aforementioned risk factors are clinically significant in vitiligo management. As multiple researchers emphasize, disease severity depends on the interplay of these factors, underscoring the need for thorough patient anamnesis to prevent adverse outcomes. Bae et al.,[25] stress minimizing potential complications, while Sachar et al.,[26] advocate comprehensive clinical histories to monitor the condition and prevent concurrent immune disorders.

Finally, certain medications must be considered. Sachar et al.,[267] reported that regular paracetamol use is associated with a 9% higher vitiligo risk in women. Conversely, Bae et al.,[27] highlighted levodopa's potential protective effect on melanocytes, possibly preventing vitiligo—supported by reports of hair repigmentation following levodopa treatment. Passeron,[28] affirmed the utility of phototherapy, topical steroids, topical calcineurin inhibitors, and ruxolitinib for facial repigmentation, calling for further research to better address risk factors in vitiligo patients.

CONCLUSIONS

Vitiligo is associated with multiple adverse processes whose clinical expression varies according to concomitant risk factors and individual comorbidities, resulting in a highly personalized disease course. Identifying associated dermatological conditions—such as lichen planus, psoriasis, alopecia areata, and other autoimmune diseases—is essential for optimizing diagnosis and enabling holistic management. Certain medications, like paracetamol, may increase vitiligo risk or progression, whereas therapeutic alternatives—including levodopa, phototherapy, topical steroids, and calcineurin inhibitors—promote repigmentation and disease control. In this context, timely recognition of triggering factors is crucial to improving clinical management and guiding more effective therapeutic strategies.

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