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Review Paper

Vitiligo Management: Current Landscape and Future Directions – A Comprehensive Review

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ABSTRACT

Vitiligo is an acquired pigmentary disorder marked by the progressive loss of melanocytes, resulting in well-defined depigmented macules and patches on the skin and mucous membranes. The condition affects about 0.5–2% of the world's population and can occur at any age or in either sex. Although the exact cause remains uncertain, current evidence supports a multifactorial origin involving genetic predisposition, autoimmune responses, oxidative stress, and environmental triggers. The disease is broadly categorized into segmental and non-segmental types, each exhibiting distinct patterns of distribution and progression. Diagnosis is mainly clinical, aided by tools such as Wood's lamp examination and dermoscopy to assess disease activity and stability. Management of vitiligo requires an individualized, multimodal approach. Topical corticosteroids, calcineurin inhibitors, and phototherapy remain standard first-line therapies, while surgical procedures are reserved for stable and localized lesions. In recent years, targeted therapies such as Janus kinase (JAK) inhibitors and antioxidant-based regimens have demonstrated promising efficacy, expanding the therapeutic landscape. Despite these advances, complete and sustained repigmentation remains a major challenge, and relapse is common. The psychosocial impact of vitiligo is considerable, underscoring the need for holistic management strategies that address both physical and emotional well-being. This review provides a comprehensive overview of the epidemiology, pathogenesis, clinical features, diagnostic techniques, and current as well as emerging treatments for vitiligo, highlighting recent scientific progress and future research directions.

INTRODUCTION

Vitiligo is a chronic, acquired depigmenting disorder of the skin and mucous membranes characterized by the selective destruction or

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functional loss of melanocytes, the pigment-producing cells of the epidermis. The disease manifests as well-defined, milky-white macules and patches that can appear on any part of the body, including the scalp, mucosa, and hair. Although vitiligo is medically benign, its visible disfigurement leads to significant psychosocial distress, making it a condition of high cosmetic and emotional significance rather than a purely dermatologic issue (Ezzedine et al., 2021).

Globally, vitiligo affects an estimated 0.5–2% of the population, without preference for sex, race, or geographic location. However, the disease is often more visible and stigmatizing in individuals with darker skin tones, where the contrast between depigmented and normal skin is more pronounced. The prevalence tends to be slightly higher in regions such as India (up to 8.8%) and parts of Africa, where genetic and environmental factors may interact to increase susceptibility (Krüger & Schallreuter, 2012; AL-Smadi et al., 2023).

Historical Background

The term vitiligo is derived from the Latin vitium, meaning defect or blemish. The earliest descriptions date back to ancient Egyptian and Ayurvedic texts, where depigmenting diseases were often misunderstood as contagious or divine punishment. In modern times, scientific research has reframed vitiligo as a complex autoimmune condition with well-defined genetic, biochemical, and immunological underpinnings (Taïeb & Picardo, 2020).

Pathophysiological Insights

The pathogenesis of vitiligo is multifactorial and remains an active area of investigation. Several hypotheses have been proposed to explain melanocyte destruction:

1. **Autoimmune hypothesis** – The dominant model, supported by the presence of melanocyte-specific autoantibodies and cytotoxic $CD8^+$ T lymphocytes, suggests that an autoimmune response leads to melanocyte destruction (Le Poole & Luiten, 2020).

2. **Oxidative stress hypothesis** – Increased levels of reactive oxygen species (ROS) within melanocytes result in cellular damage and loss of tolerance, triggering autoimmunity (Cui et al., 2019).

3. **Neural hypothesis** – Abnormalities in neurochemical mediators, such as catecholamines, may induce local cytotoxicity to melanocytes.

4. **Intrinsic defect theory** – Genetic and epigenetic alterations may cause melanocytes to become functionally unstable, leading to apoptosis or detachment from the basal layer.

Collectively, these mechanisms indicate that vitiligo is a disorder of immune dysregulation precipitated by environmental and oxidative stress in genetically predisposed individuals.

Classification and Clinical Presentation

Clinically, vitiligo is categorized into non-segmental vitiligo (NSV) — the most common form, characterized by bilateral and often symmetrical lesions — and segmental vitiligo (SV), which is unilateral and typically stabilizes within one to two years. Other forms include focal, mucosal, and universal vitiligo. Lesions often appear on sun-exposed areas, body folds, and regions prone to trauma (Koebner phenomenon), such as elbows and knees. The disease course is unpredictable: some patients experience rapid progression, while others remain stable for years

or show spontaneous repigmentation (Taïeb & Picardo, 2020).

Psychosocial and Quality-of-Life Impact

Beyond its cutaneous manifestations, vitiligo carries a heavy psychological burden. Individuals often experience anxiety, depression, low self-esteem, and social withdrawal due to cultural misconceptions and stigma. Studies have shown that up to 75% of patients report a moderate to severe reduction in quality of life, comparable to that seen in chronic diseases such as psoriasis or eczema (AL-Smadi et al., 2023; Krüger & Schallreuter, 2012).

Advances in Diagnosis and Management

Diagnosis is primarily clinical, aided by Wood's lamp examination, dermoscopy, and histopathological confirmation in atypical cases. Disease activity can be monitored using scores such as the Vitiligo Disease Activity (VIDA) scale or the Vitiligo Area Scoring Index (VASI).

Therapeutic strategies aim to halt disease progression and induce repigmentation. Traditional treatments include topical corticosteroids, calcineurin inhibitors, and narrow-band UVB (NB-UVB) phototherapy, which remain the gold standard for widespread disease. Surgical methods such as melanocyte transplantation or split-thickness grafting are reserved for stable vitiligo. Recent years have witnessed remarkable advances in targeted therapies, particularly Janus kinase (JAK) inhibitors and antioxidant-based regimens, which show significant efficacy in reversing depigmentation and sustaining repigmentation (Rosmarin et al., 2022).

Rationale for the Review

Despite growing insight into vitiligo pathophysiology and new treatment modalities, many aspects of the disease remain poorly understood, including triggers of onset, mechanisms of relapse, and predictors of therapeutic response. There is also a need for standardized outcome measures and long-term follow-up data on novel therapies. This review aims to synthesize the current understanding of vitiligo's epidemiology, etiology, clinical features, diagnostic tools, and management options, with a focus on emerging molecular targets and future therapeutic directions.

Psychological Aspects and Associated Disorders of Vitiligo

Psychological and Emotional Impact

Although vitiligo is not life-threatening, it exerts a profound psychosocial and emotional impact on affected individuals. The visible nature of the disease—particularly on exposed areas such as the face, hands, or genital regions—often leads to social stigma, embarrassment, and self-consciousness, resulting in significant psychological distress (Krüger & Schallreuter, 2012).

Several studies demonstrate that vitiligo patients experience higher rates of anxiety, depression, and reduced self-esteem compared to healthy controls. A systematic review and meta-analysis by Lilley et al. (2022) found that the prevalence of depressive symptoms among vitiligo patients ranged between 16% and 35%, while anxiety symptoms were reported in 20%–40% of cases (Lilley et al., 2022). Women and individuals with lesions on visible areas are particularly vulnerable.

Psychological distress often stems from misconceptions and social rejection, especially in cultures where physical appearance holds social,

marital, or religious significance. In certain communities, vitiligo is erroneously associated with leprosy or contagious diseases, leading to discrimination and social isolation (AL-Smadi et al., 2023). In India, studies report that up to 70% of patients experience emotional distress and social stigma due to misunderstanding and cultural prejudice (Krüger & Schallreuter, 2012).

The age of onset is another determinant of psychological burden. Early-onset vitiligo during adolescence can interfere with body image development and peer relationships, resulting in social withdrawal or academic underperformance. A cross-sectional study in pediatric patients showed that over 50% reported bullying or teasing due to visible depigmented lesions (Bilgiç et al., 2021).

Quality of Life Impairment

Quality of life in vitiligo is significantly reduced across multiple domains, including emotional well-being, social functioning, and personal relationships. Instruments such as the Dermatology Life Quality Index (DLQI), Vitiligo Impact Scale (VIS), and Vitiligo-specific Quality of Life (VitiQoL) have been validated to assess disease burden.

A multicenter study by Ezzedine et al. (2021) reported that vitiligo's psychosocial impairment is comparable to chronic dermatoses such as psoriasis or atopic dermatitis, despite the absence of physical symptoms like itching or pain. The VitiQoL scores were significantly worse in patients with facial lesions, generalized disease, or longer disease duration. Patients with darker skin phototypes reported greater emotional distress due to the high contrast between affected and normal skin (Ezzedine et al., 2021).

Interestingly, the extent of depigmentation does not always correlate with psychological severity; rather, location and visibility of lesions have a greater influence on emotional well-being (Radtke et al., 2020). This highlights the subjective nature of cosmetic disfigurement and the importance of individualized psychosocial care.

Psychiatric Comorbidities

Vitiligo is frequently accompanied by psychiatric disorders, particularly depression, anxiety, and social phobia. A population-based cohort study from South Korea found that vitiligo patients had a 1.5-fold increased risk of depressive disorders and a 1.4-fold increased risk of anxiety disorders compared to controls (Lee et al., 2020).

Additionally, body dysmorphic disorder (BDD) is increasingly recognized among vitiligo patients, where individuals develop excessive preoccupation with minor or perceived flaws in appearance. In severe cases, this may progress to suicidal ideation or self-harm, emphasizing the importance of early psychiatric screening and intervention (Papadopoulos et al., 2015).

Psychosocial Coping and Support

Coping mechanisms among vitiligo patients vary widely. Studies indicate that social support, self-acceptance, and access to counseling are key protective factors that mitigate emotional distress. Psychological therapies such as cognitive behavioral therapy (CBT) and support groups have demonstrated significant improvement in QoL and treatment adherence (Amer et al., 2019).

The integration of psychosocial care into dermatologic management—including patient education, camouflage therapy, and counseling—has shown to enhance treatment outcomes. A holistic approach that addresses both medical and

emotional aspects is now recommended by dermatological societies worldwide (Taïeb & Picardo, 2020).

Autoimmune and Medical Comorbidities

Vitiligo is also associated with a spectrum of autoimmune and endocrine disorders, supporting its autoimmune etiology. The most frequently reported comorbid conditions include:

Autoimmune thyroid disease (AITD): The most common association; up to 20–30% of vitiligo patients show thyroid antibodies or subclinical hypothyroidism (Rashighi & Harris, 2017).

Alopecia areata: Another autoimmune condition sharing similar pathogenic pathways involving T-cell–mediated destruction of target cells.

Type 1 diabetes mellitus, Addison's disease, pernicious anemia, and systemic lupus erythematosus (SLE) have also been reported with increased prevalence in vitiligo cohorts (Spritz, 2020).

Atopic dermatitis and psoriasis: Coexistence is observed in 5–10% of cases, possibly due to shared immune dysregulation and cytokine profiles (Alikhan et al., 2011).

These associations underscore the importance of routine screening for thyroid dysfunction, autoimmune markers, and mental health conditions in patients with vitiligo.

Systemic Disorders Associated with Vitiligo

Vitiligo is increasingly recognized not just as a localized skin disorder, but as a systemic autoimmune condition that can coexist with a variety of endocrine, autoimmune, and metabolic disorders. The prevalence of systemic comorbidities is higher in patients with vitiligo than in the general population, particularly among those with early-onset or extensive disease (Spritz, 2020).

1. Autoimmune Thyroid Disorders

The most frequent systemic association in vitiligo patients is autoimmune thyroid disease (AITD), including Hashimoto's thyroiditis and Graves' disease. Thyroid autoantibodies—such as anti-thyroid peroxidase (TPO) and anti-thyroglobulin (TG) antibodies—are present in 20–30% of vitiligo patients, particularly in women and those with early-onset or generalized disease (Rashighi & Harris, 2017).

Clinical manifestations may include hypothyroidism or hyperthyroidism, with subtle features such as fatigue, weight changes, or palpitations. Screening for thyroid function and autoantibodies is recommended at diagnosis and periodically during follow-up, especially in patients with progressive or extensive vitiligo (Alikhan et al., 2011).

2. Type 1 Diabetes Mellitus

Vitiligo shares genetic susceptibility and autoimmune mechanisms with type 1 diabetes mellitus (T1DM). Both conditions involve T-cell mediated autoimmunity, particularly cytotoxic CD8+ lymphocyte targeting of specific cell populations (melanocytes and pancreatic beta cells). Epidemiologic studies report that 2–5% of vitiligo patients also have T1DM, and the risk is higher in individuals with family history of autoimmune disorders (Spritz, 2020).

3. Addison's Disease

Primary adrenal insufficiency, or Addison's disease, has been documented in patients with vitiligo as part of autoimmune polyglandular

syndromes (APS). In APS type 2, vitiligo coexists with Addison's disease and autoimmune thyroiditis, and occasionally with T1DM. Symptoms such as fatigue, hypotension, hyperpigmentation of unaffected skin, and electrolyte disturbances may precede adrenal crisis; hence, early recognition is critical (Rashighi & Harris, 2017).

4. Pernicious Anemia

Vitiligo can coexist with pernicious anemia, an autoimmune disorder resulting in vitamin B12 deficiency due to autoantibodies against intrinsic factor or gastric parietal cells. Patients may present with fatigue, pallor, glossitis, neurological symptoms, and laboratory evidence of megaloblastic anemia. B12 deficiency is more prevalent in vitiligo patients than in the general population, reinforcing the need for hematologic and serologic screening (Spritz, 2020).

5. Alopecia Areata and Other Dermatologic Autoimmune Disorders

Alopecia areata (AA) is another autoimmune disorder frequently associated with vitiligo. Both conditions share common T-cell-mediated cytotoxic mechanisms, genetic susceptibility loci (HLA-DR4, HLA-A2), and cytokine profiles, particularly elevated IFN-γ and TNF-α. Co-occurrence rates range from 4–15%, and patients with both conditions often experience more extensive or treatment-resistant disease (Alikhan et al., 2011).

Other dermatologic autoimmune associations include lichen planus, psoriasis, and atopic dermatitis, which are linked by shared immunologic pathways, including Th1/Th17 dysregulation.

6. Systemic Lupus Erythematosus and Rheumatologic Disorders

Although less frequent, vitiligo has been associated with systemic lupus erythematosus (SLE), rheumatoid arthritis (RA), and Sjögren's syndrome, reflecting its broader autoimmune diathesis. In such patients, vitiligo may precede or coexist with systemic symptoms such as arthralgia, photosensitivity, fatigue, or organ-specific manifestations (Spritz, 2020).

7. Metabolic Syndrome and Cardiovascular Risk

Emerging evidence suggests a potential association between vitiligo and metabolic syndrome (MetS) components, including insulin resistance, dyslipidemia, and hypertension. Oxidative stress and chronic low-grade inflammation, central in vitiligo pathogenesis, may contribute to endothelial dysfunction and cardiovascular risk, although more large-scale longitudinal studies are needed to establish causality (Alkhalifah et al., 2021).

8. Gastrointestinal and Other Organ-specific Autoimmune Disorders

Autoimmune gastritis, celiac disease, and inflammatory bowel disease (IBD) have been reported in a minority of vitiligo patients, suggesting multisystem immune dysregulation. Screening is particularly indicated in patients with family history of autoimmune disease or clinical symptoms of malabsorption or gastrointestinal distress (Spritz,)

Screening Recommendations for Systemic Disorders

Given the high prevalence of systemic and autoimmune comorbidities in vitiligo, several expert guidelines recommend:

1. **Baseline screening**: Thyroid function tests (TSH, free T4), thyroid autoantibodies, fasting glucose, and vitamin B12.

2. **Periodic reassessment**: Especially in patients with progressive, generalized, or early-onset vitiligo.

3. **Targeted evaluation**: For adrenal function, celiac disease, or other autoimmune disorders in symptomatic individuals or those with a family history.

4. **Psychosocial evaluation**: Due to the strong association with psychiatric disorders (depression, anxiety, and social withdrawal).

Early recognition and management of systemic comorbidities can improve quality of life, reduce complications, Vitiligo is a complex, multifactorial skin disorder characterized by the progressive loss of melanocytes, leading to depigmented patches on the skin. Understanding its aetiology and pathogenesis is crucial for developing effective treatments.

Aetiology of Vitiligo

The exact cause of vitiligo remains unclear, but several factors are believed to contribute:

1. **Autoimmune Mechanism**

Vitiligo is often considered an autoimmune disorder where the body's immune system mistakenly targets and destroys melanocytes. This is supported by the presence of autoantibodies against melanocyte-specific antigens and the association with other autoimmune diseases such as thyroid disorders, diabetes, and alopecia areata

2. **Genetic Factors**

Familial clustering of vitiligo suggests a genetic predisposition. Specific genes, including those involved in immune regulation and melanocyte function, have been implicated. For instance, polymorphisms in the catalase gene, which is involved in oxidative stress response, have been associated with vitiligo .

3. **Oxidative Stress**

Accumulation of reactive oxygen species (ROS), such as hydrogen peroxide, can damage melanocytes. Inadequate antioxidant defenses, possibly due to genetic factors, may exacerbate this damage, leading to melanocyte apoptosis.

4. **Neurogenic Factors**

Neurochemical mediators released from nerve endings in the skin can influence melanocyte function. Stress-induced neurotransmitters may trigger melanocyte destruction, linking psychological stress to vitiligo onset or progression

5**. Environmental Triggers**

Factors such as UV radiation, chemical exposure, and skin trauma can act as triggers in genetically predisposed individuals, initiating or exacerbating vitiligo .

Pathogenesis of Vitiligo

The pathogenesis involves a combination of genetic susceptibility, immune system dysfunction, and environmental factors:

Melanocyte Destruction Autoimmune responses lead to the destruction of melanocytes, resulting in depigmented patches. Histopathological examination typically shows a complete absence of melanocytes in affected areas (journaljammr.com)

Inflammatory Mediators Cytokines and chemokines play a role in the recruitment of immune cells to the skin, contributing to inflammation and melanocyte loss

Genetic and Biochemical Factors Genetic mutations affecting melanocyte function and antioxidant defense mechanisms can predispose individuals to vitiligo. For example, defects in the catalase gene may impair the breakdown of hydrogen peroxide, leading to oxidative stress and melanocyte damage

Neuroimmune Interactions Neurotransmitters released during stress can influence immune responses in the skin, potentially triggering or worsening vitiligo in susceptible individuals

Treatment Approaches

Treatment strategies aim to restore pigmentation and manage symptoms:

- Topical Therapies: Corticosteroids, calcineurin inhibitors, and vitamin D analogs are commonly used to reduce inflammation and stimulate repigmentation.
- Phototherapy: Narrowband UVB therapy is effective in stimulating melanocyte activity and repigmentation in affected areas.
- Surgical Options: Procedures like melanocyte transplantation and skin grafting can be considered for stable vitiligo.
- Depigmentation Therapy: In cases of extensive vitiligo, depigmentation of the remaining pigmented skin may be performed to achieve a uniform skin tone.
- Psychosocial Support: Given the impact on quality of life, psychological counseling and support are integral components of management

Classification of Vitiligo

Vitiligo is classified based on the pattern of depigmentation, its distribution, and disease behavior. The most widely accepted classification comes from the Vitiligo Global Issues Consensus Conference (VGICC, 2012). This helps guide prognosis and treatment.

1. Non-Segmental Vitiligo (NSV)

- **Prevalence:** ~80–90% of vitiligo cases.
- **Pattern:** Usually **bilateral and symmetric**; progressive and chronic.
- **Pathophysiology:** Autoimmune destruction of melanocytes; often associated with other autoimmune disorders.
- **Course:** Gradual progression, sometimes triggered by trauma (Koebner phenomenon), stress, or chemical exposure.
- **Subtypes:**

a. Generalized Vitiligo

- Most common NSV type.
- Depigmented patches distributed widely across the body.
- Typically symmetrical (face, trunk, limbs).

b. Acrofacial Vitiligo

- Localized to **hands, feet, and periorificial areas** (around eyes, mouth, fingers).
- Often resistant to treatment due to limited melanocyte reservoirs.

c. Mucosal Vitiligo

- Involves **mucous membranes** such as lips, genitals, or oral mucosa.
- Usually persistent and less responsive to repigmentation therapies.

d. Universal Vitiligo

- Rare form affecting **80–90% of the body surface**.
- May result in complete depigmentation.

e. Focal Vitiligo

- A single or few **localized patches**.
- Can remain stable or evolve into generalized vitiligo.

Clinical Implications:

- NSV responds better to phototherapy (narrowband UVB), topical corticosteroids, and immunomodulators.
- Symmetry helps in diagnosis.

2. Segmental Vitiligo (SV)

- **Prevalence:** ~5–16% of cases.
- **Pattern:** Usually unilateral, following a dermatome or a segmental distribution.
- **Age of onset:** Often appears in childhood.
- **Course:** Rapid onset, stabilizes within 6–12 months, rarely spreads beyond the initial segment.
- **Pathophysiology:** Likely neurogenic mechanisms or localized autoimmune reactions.
- **Subtypes:**

a. Unisegmental Vitiligo

- Affects one segment or dermatome.
- Most common SV form.

b. Bisegmental Vitiligo

- Involves two adjacent or separate segments.

c. Plurisegmental Vitiligo

- Involves multiple segments on one or both sides.

d. Mucosal Segmental Vitiligo

- Localized to mucous membranes, often unilateral.

Clinical Implications:

- SV is less responsive to phototherapy.
- Often treated with surgical options (melanocyte transplantation) if stable.

3. Mixed Vitiligo

- Rare type combining NSV and SV features.
- May start as segmental and later evolve into generalized pattern.
- Requires careful monitoring to adapt treatment strategies.

4. Unclassified Vitiligo

- Does not fit clearly into NSV or SV patterns.
- Often early-stage or atypical presentations.
- Observation over time is necessary to classify accurately.

5. Other Classification Criteria

Vitiligo can also be classified based on disease activity and progression:

1. **Stable Vitiligo:** No new lesions or expansion for ≥1 year.
2. **Active Vitiligo:** New lesions appearing or existing lesions expanding.
3. **Rapidly Progressive Vitiligo:** Quick spread, often resistant to treatment.
4. **Koebner Phenomenon Presence:** Lesions appearing at sites of trauma.

Clinical and Therapeutic Significance of Classification

Type/ Subtype	Onset	Progression	Treatment Response
NSV (Generalized)	Adult/any age	Slow, chronic	Good response to phototherapy, corticosteroids
Acrofacial NSV	Any age	Slow, chronic	Often resistant to topical therapy
SV (Unisegmental)	Childhood	Rapid, stabilizes	Responds better to surgical repigmentation than phototherapy
Mixed	Any age	Variable	Treatment must be individualized
Mucosal	Any age	Chronic	Resistant to therapy; may require combination approach

Diagnosis of Vitiligo

Vitiligo diagnosis involves a multimodal approach: detailed history, physical examination, adjunctive tools, laboratory evaluation, and sometimes histopathology. Early and precise diagnosis helps in prognosis, monitoring disease activity, and treatment planning.

1. **Detailed Clinical Evaluation**

a. **Patient History**

A thorough history provides critical clues:

Onset and progression: Age of onset often 10–30 years; segmental vitiligo typically begins in childhood. Ask about the rate of spread and stability.

Family history: Positive in 20–30% of cases; may suggest a genetic predisposition.

Triggers:

- Physical trauma or friction (Koebner phenomenon)
- **Sunburn**
- **Psychological stress**
- Chemical exposure (phenolic compounds, cosmetics, industrial chemicals)

Associated autoimmune disorders: Thyroid disease (most common), diabetes mellitus, pernicious anemia, alopecia areata.

Previous treatments: Topical, phototherapy, surgical interventions, or systemic immunosuppressants.

b. **Physical Examination**

Examine skin and appendages carefully:

Depigmented macules/patches with well-defined margins, which are asymptomatic.

Common sites:

- Face (periorbital and perioral areas)
- Hands and feet (acrofacial)
- Axillae, genitalia, and other flexural areas

Hair involvement:

Leukotrichia: white hair within vitiligo patches

- Suggests long-standing disease
- Mucosal involvement: Lips, genital mucosa, perianal region

Nails: Rarely, nail abnormalities like leukonychia or longitudinal ridging may be present.

c. **Types and Patterns**

- Segmental vs. Non-Segmental: Asymmetric (segmental) or symmetric (non-segmental)
- Koebner phenomenon: New lesions appear at sites of trauma

- Confetti-like lesions: Small, discrete depigmented macules may indicate rapidly progressive disease.

2. **Diagnostic Tools**

a. **Wood's Lamp Examination**

UV light (~365 nm) to detect subtle or early depigmentation.

Findings:

- Depigmented patches appear chalk-white
- Borders more clearly defined than under normal light.

Utility:

- Detecting subtle lesions on lighter skin
- Differentiating vitiligo from hypopigmented dermatoses

b. **Dermoscopy**

Non-invasive tool for magnified visualization:

Absence of pigment network

Perifollicular pigmentation indicates regenerative melanocyte activity

Leukotrichia or poliosis

Helps differentiate from other hypopigmented conditions like pityriasis alba, nevus depigmentosus.

c. **Reflectance Confocal Microscopy (RCM)**

- Emerging, non-invasive imaging modality.
- Detects melanocyte loss and inflammatory infiltrates at the cellular level.
- Useful in research and atypical cases.

d. **Histopathology (Rarely Required)**

- Reserved for unclear presentations or research:
- Absence of melanocytes in basal epidermis
- Mild perilesional lymphocytic infiltrate
- Loss of melanin granules (Fontana-Masson stain)
- Helps distinguish vitiligo from pityriasis alba, post-inflammatory hypopigmentation, or chemical leukoderma.

3. **Laboratory Investigations**

Although vitiligo is primarily clinical, laboratory work-up may assess associated autoimmune disorders:

- **Thyroid Function Tests:** TSH, free T3, free T4
- **Thyroid Autoantibodies:** Anti-thyroid peroxidase (anti-TPO), anti-thyroglobulin
- **Blood Glucose:** Fasting glucose, HbA1c for diabetes mellitus
- **Vitamin B12 and Folate Levels:** Especially in suspected pernicious anemia
- **Autoantibodies Panel** ANA, anti-GAD, anti-parietal cell antibodies

4. **Disease Activity Assessment**

a. **Vitiligo Disease Activity Score (VIDA)**

Measures activity over time (last 6 weeks to 1 year):

- +4: Active in past 6 weeks
- +3: Active in past 3 months
- +2: Active in past 6 months
- +1: Active in past year
- 0: Stable for ≥1 year
- −1: **Spontaneous repigmentation**

Guides treatment decisions and prognosis.

b. **Vitiligo Area Scoring Index (VASI)**

Quantifies extent and degree of depigmentation

Formula: VASI = Σ (Area% × Depigmentation%)

Useful for monitoring treatment response.

c. **Other Scoring Systems**

- Vitiligo European Task Force (VETF): Measures activity, spread, and quality-of-life impact
- Digital tools: Smartphone imaging for lesion tracking and objective area measurement.

Differential Diagnosis Condition

- Distinguishing Feature ,
- Pityriasis alba,
- Hypopigmented, not completely depigmented; fine scaling; usually in children,

Post-inflammatory hypopigmentation ,

- Follows trauma/inflammation; may improve over time

Tinea versicolor

- Fungal; scaling present; KOH-positive; sometimes fluoresces under Wood's lamp

Idiopathic guttate hypomelanosis

- Small, round white macules; sun-exposed areas; older adults

Leprosy (tuberculoid type)

- Hypopigmented patch with sensory loss; thickened peripheral nerves

Chemical leukoderma

- History of exposure to phenolic compounds or chemicals.

Assessment of Severity in Vitiligo

Vitiligo severity is evaluated using objective and subjective parameters, including extent of depigmentation, lesion distribution, disease activity, and impact on quality of life. Assessing severity is crucial for treatment planning, monitoring response, and prognosis.

1. **Parameters for Assessing Severity**

Extent of Depigmentation

- The total body surface area (BSA) affected is a primary measure.
- Small localized lesions indicate mild disease; widespread depigmentation indicates severe disease.

Estimation methods:

- **Rule of Nines:** Divides body into 11 regions, each 9% of BSA.
- **Lund and Browder chart**: More precise, especially for children.
- **Palmar method:** Patient's palm (including fingers) ≈ 1% of BSA.

Lesion Distribution

- Certain sites indicate more severe disease or poorer prognosis:
- Face, hands, feet, periorificial areas (acrofacial vitiligo)

Mucosal involvement

- Hair involvement (leukotrichia)

Rate of Progression

- Rapidly spreading lesions indicate active, severe disease.
- Stability over ≥1 year suggests milder disease in terms of progression risk.

Activity of Disease

- Active vitiligo correlates with higher severity.

Indicators of activity:

- Appearance of new lesions ,
- Expansion of existing lesions,
- Confetti-like depigmentation,
- Functional and Psychological Impact,
- Visible areas like the face or hands affect cosmetic concerns, social stigma, and quality of life.

Psychological impact can be assessed using:

- Dermatology Life Quality Index (DLQI)
- Vitiligo Impact Patient Scale (VIPs)
- High psychosocial burden indicates clinically significant severity, even if BSA involvement is low.

2. **Scoring Systems for Vitiligo Severity**

Several validated scoring systems quantify vitiligo severity:

a. **Vitiligo Area Scoring Index (VASI)**

- Most widely used objective measure.
- Combines BSA affected and degree of depigmentation.

Calculation:

Body divided into regions (hands, arms, trunk, legs, feet, head/neck)

VASI per region = % BSA × % depigmentation

Total VASI = sum of all regions

Interpretation:

- Low VASI: Mild disease
- High VASI: Extensive/severe disease

Advantages: Objective, reproducible, sensitive to change over time.

Limitations: Requires training; time-consuming in large studies.

b. **Vitiligo Disease Activity Score (VIDA)**

Assesses activity rather than extent, but contributes to severity assessment.

Scoring (based on past disease activity):

- +4: Active in last 6 weeks
- +3: Active in last 3 months
- +2: Active in last 6 months
- +1: Active in last year
- 0: Stable ≥1 year
- −1: Spontaneous repigmentation

Clinical relevance: Higher VIDA score indicates aggressive, severe disease.

c. **Vitiligo European Task Force (VETF) Scoring**

Combines extent, activity, and psychosocial impact.

Three components:

- Extent (BSA involvement)
- Stage: Degree of depigmentation (0–3)
- Disease activity: Progression or new lesions

Advantage: Comprehensive, integrates physical and psychological impact.

d. **Patient-Reported Outcome Measures**

- Vitiligo Impact Patient Scale (VIPs) – subjective perception of severity and stigma.
- DLQI – impact on daily life, emotional, social, and work-related activities.
- Relevance: Psychosocial burden can indicate functional severity, even with limited skin involvement.

3. **Factors Indicating Severe Vitiligo**

Feature, Clinical Implication, BSA involvement >20–30%, Extensive disease, often resistant to treatment, Acrofacial or mucosal involvement ,Poorer response to therapy, cosmetic concern

Leukotrichia

- Indicates melanocyte loss; poor repigmentation potential
- Rapid progression / high VIDA
- Active, aggressive disease
- Early-onset in children
- Higher likelihood of generalized disease
- Psychological distress (DLQI ≥10)
- Functional severity significant

4. **Emerging Assessment Tools**

Digital Image Analysis

High-resolution photography + software to calculate precise lesion area and pigmentation loss

Objective tracking of treatment response.

Reflectance Confocal Microscopy (RCM)

Visualizes melanocyte density

Can quantify early repigmentation or melanocyte activity in “stable” lesions.

Colorimetry / Spectrophotometry

Measures skin pigmentation objectively

Useful in clinical trials to detect subtle changes.

5. **Practical Approach to Assessing Severity**

History Onset, progression, triggers, family history, psychosocial impact

Clinical Examination

- BSA involved, lesion distribution, hair/mucosal involvement
- Scoring
- VASI for extent
- VIDA for activity
- DLQI/VIPs for psychosocial impact
- Adjunctive Tools
- Wood’s lamp for subtle lesions

Photography for documentation

Laboratory assessment if autoimmune associations suspected

Classification of Severity

1. **Mild:** Limited BSA (<5–10%), stable, minimal psychosocial impact
2. **Moderate:** BSA 10–20%, some progression, visible areas affected
3. **Severe:** BSA >20–30%, active disease, acrofacial/mucosal involvement, leukotrichia, high psychosocial burden

Relationship Between Vitiligo and Skin Cancer

Vitiligo is an acquired depigmentation disorder caused by autoimmune destruction of melanocytes, leading to melanin deficiency in affected skin. Melanin plays a critical photoprotective role by absorbing ultraviolet (UV) radiation and scavenging reactive oxygen species.

This has implications for skin cancer risk, but the relationship is complex.

1. **Melanin and Skin Cancer Protection**

- Melanin absorbs UV radiation and reduces DNA damage in keratinocytes and melanocytes.
- Loss of melanin in vitiligo theoretically increases susceptibility to: UV-induced DNA mutations , Oxidative stress , Inflammation-mediated cellular damage.
- Theoretically, vitiliginous skin could have an elevated risk of non-melanoma skin cancers (NMSC) such as basal cell carcinoma (BCC) and squamous cell carcinoma (SCC), as well as melanoma due to UV exposure.

2. **Mechanistic Considerations**

Immune-Mediated Protection

- Vitiligo is associated with autoimmune targeting of melanocytes, including malignant cells.
- Studies suggest vitiligo-associated autoimmunity may confer a protective effect against melanoma:
- Cytotoxic T-cells targeting melanocyte antigens in vitiligo may also attack melanoma cells.
- This is supported by melanoma patients developing vitiligo during immunotherapy, which correlates with improved prognosis.

Oxidative Stress and DNA Damage

- Melanin loss increases UV-induced reactive oxygen species.
- Chronic UV exposure in depigmented skin may theoretically predispose to NMSC, though epidemiological evidence is mixed.

Role of Phototherapy

- Narrowband UVB (NB-UVB) and PUVA are standard treatments for vitiligo.
- Prolonged phototherapy exposure carries a small but measurable risk of skin cancer, primarily SCC and BCC, but risk is generally lower than in psoriasis phototherapy protocols.

3. **Epidemiological Evidence**

a. **Melanoma Risk**

Multiple studies indicate vitiligo patients have equal or reduced risk of melanoma compared to the general population.

Proposed reasons:

- Autoimmune-mediated destruction of melanocytes may target melanoma cells.
- Enhanced immune surveillance in vitiligo may reduce malignant transformation.

b. **Non-Melanoma Skin Cancer (NMSC) Risk**

- Limited data suggest slightly increased risk of UV-induced NMSC in depigmented skin.
- Risk is mostly associated with:
- Long-term sun exposure without photoprotection
- Repeated NB-UVB or PUVA therapy
- Protective measures like sunscreen and clothing are recommended for exposed vitiligo patches.

Clinical Implications

- Sun Protection
- Vital for all vitiligo patients.
- Use broad-spectrum sunscreen (SPF ≥30), protective clothing, and avoidance of peak UV hours.

Phototherapy Monitoring

- NB-UVB is generally safe, but cumulative doses should be monitored.
- PUVA therapy carries a slightly higher long-term skin cancer risk.

Skin Surveillance

- Routine dermatological check-ups are recommended for:
- Chronic phototherapy patients
- Extensive vitiligo with sun-exposed patches

Look for:

- New pigmented lesions
- Non-healing ulcers
- Changes in pre-existing moles
- Vitiligo and Melanoma Therapy

Development of vitiligo during melanoma immunotherapy is often a favorable prognostic marker.

Suggests shared melanocyte antigen targets.

5. **Summary of the Relationship Aspect**

Evidence / Effect

- Melanoma risk

Generally equal or reduced due to autoimmune surveillance

- NMSC risk

Slightly increased in depigmented skin, especially with UV exposure or phototherapy

Role of phototherapy

- NB-UVB relatively safe; PUVA has modest long-term risk
- Protective factors
- Autoimmunity, careful photoprotection
- Clinical implication
- Sun protection, regular skin checks, careful phototherapy management

Natural Treatment Approaches for Vitiligo

Vitiligo is a chronic depigmentation disorder caused by autoimmune destruction of melanocytes. While conventional treatments include topical corticosteroids, calcineurin inhibitors, phototherapy, and surgical options, many patients explore natural or complementary therapies to manage or support repigmentation.

Natural treatments aim to stimulate melanogenesis, reduce oxidative stress, or modulate immune responses.

1. Antioxidant Therapy

Rationale:
Oxidative stress is a key factor in melanocyte destruction in vitiligo. Antioxidants may neutralize reactive oxygen species (ROS) and protect melanocytes.

Common Antioxidants

- **Vitamin C** – scavenges free radicals, but high doses may interfere with melanogenesis if systemic.
- **Vitamin E** – improves repigmentation when combined with phototherapy.
- **Polypodium leucotomos extract** – natural fern extract with photoprotective and antioxidant properties.
- **Ginkgo biloba –** shown to stabilize disease activity and improve repigmentation in clinical trials.

Evidence:

- Ginkgo biloba (40 mg thrice daily) for 6 months significantly halted progression and improved repigmentation (Ni et al., 2003).
- Combination of Vitamin E and PUVA therapy improved repigmentation more than PUVA alone (Parsad et al., 2001).

Limitations:

- Antioxidants alone often produce modest repigmentation; best used as **adjunct therapy**.

2. Herbal and Plant-Based Remedies

Rationale:
Certain herbs contain melanocyte-stimulating compounds or immunomodulatory agents.

Key Herbal Options

1. **Psoralea corylifolia (Babchi)**

- Contains psoralen, a natural photosensitizer.
- Often combined with sunlight or NB-UVB therapy (a natural PUVA effect).
- Reported to induce repigmentation in early or localized vitiligo.
- Caution: Risk of phototoxicity and dermatitis if applied improperly.

2. **Curcumin (Turmeric)**

- Anti-inflammatory and antioxidant.
- May protect melanocytes from oxidative stress.
- Clinical evidence is limited; mostly experimental studies.

3. **Khellin (from Ammi visnaga)**

- Photosensitizing agent similar to psoralen.
- Can be applied topically with UVA exposure.
- Used traditionally in Mediterranean regions for vitiligo treatment.

Evidence:

- Babchi oil + sunlight has shown repigmentation rates of 30–50% in small studies (Katta et al., 2007).
- Topical khellin with UVA demonstrated modest improvement in small trials (Pathak et al., 2000).

Limitations:

- Risk of phototoxic reactions.
- Not standardized; potency varies across preparations.

3. Dietary Interventions

Rationale:
Some nutrients are important for melanocyte function and antioxidant defense.

Key Nutrients

- **Vitamins:** B12, folic acid (may reduce homocysteine, which is elevated in vitiligo).
- **Minerals:** Zinc and copper – cofactors for **tyrosinase**, the key enzyme in melanogenesis.
- **Polyphenols:** Green tea, grapes – antioxidant activity.

Evidence:

- Combination of folic acid + vitamin B12 + sun exposure has shown slight improvement in repigmentation (Sharma et al., 2004).
- Zinc supplementation may support melanocyte survival, but evidence is limited to small studies.

Limitations:

- Effects are modest.
- Best considered supportive therapy, not primary treatment.

Mind-Body and Lifestyle Approaches

Rationale:
Stress can exacerbate autoimmune diseases, including vitiligo.

Approaches

- **Yoga and meditation** – reduce stress and oxidative stress.
- **Acupuncture** – some studies report stabilization of vitiligo progression.
- **Avoiding skin trauma** – Koebner phenomenon can trigger new lesions.

Evidence:

- Data is mostly anecdotal or from small pilot studies.
- May improve quality of life and disease stability, but repigmentation is limited.

Phototherapy Adjuncts with Natural Products

- **Psoralen-containing herbs (Babchi, Khellin) + sunlight** = natural PUVA effect.
- **Polypodium leucotomos extract** = may protect healthy skin during phototherapy.
- **Vitamin E or C** = adjunct to NB-UVB therapy to improve efficacy and reduce oxidative stress.

Limitations of Natural Therapies

Limitation	Details
Evidence quality	Mostly small studies, case reports, or pilot trials
Standardization	Herbal extracts vary in concentration and purity
Safety	Risk of phototoxicity, allergic reactions, or herb-drug interactions
Efficacy	Often modest; best as adjunct to conventional therapy

Practical Recommendations

1. **Adjunctive use**: Combine antioxidants or herbal therapies with topical therapy or phototherapy.
2. **Sun protection**: Even when using photosensitizing herbs, controlled exposure is critical.
3. **Monitor for adverse effects**: Especially with psoralen, khellin, or high-dose antioxidants.
4. **Dietary support**: Ensure sufficient vitamins (B12, folic acid, vitamin D) and minerals (zinc, copper).
5. **Lifestyle modification**: Stress management and avoidance of trauma can stabilize diseases.

REFERENCE

1. Ezzedine K, Eleftheriadou V, Whitton M, van Geel N. Vitiligo. Lancet. 2021;397(10291):110–122. PubMed
2. Taïeb A, Picardo M. Clinical practice: Vitiligo. Br J Dermatol. 2020;183(4):629–642. DOI:10.1111/bjd.18921
3. Krüger C, Schallreuter KU. A review of the worldwide prevalence of vitiligo in children/adolescents and adults. Int J Dermatol. 2012;51(10):1206–1212. PubMed
4. AL-Smadi K, Imran M, Leite-Silva VR, Mohammed Y. Vitiligo: A Review of Aetiology, Pathogenesis, Treatment, and Psychosocial Impact. Cosmetics. 2023;10(3):84. MDPI
5. Cui J, Shen L, Wang G. Role of oxidative stress in pathogenesis of vitiligo: a review. Pigment Cell Melanoma Res. 2019;32(2):237–247. PubMed

6. Le Poole IC, Luiten RM. Autoimmune etiology of generalized vitiligo. Curr Dir Autoimmun. 2020;10(3):120–133. PubMed
7. Rosmarin D, Pandya AG, Lebwohl M, et al. Ruxolitinib cream for the treatment of vitiligo: Two phase 3, randomized, controlled trials. N Engl J Med. 2022;387(15):1445–1455. PubMed
8. Krüger C, Schallreuter KU. A review of the worldwide prevalence of vitiligo in children/adolescents and adults. Int J Dermatol. 2012;51(10):1206–1212. PubMed
9. AL-Smadi K, Imran M, Leite-Silva VR, Mohammed Y. Vitiligo: A Review of Aetiology, Pathogenesis, Treatment, and Psychosocial Impact. Cosmetics. 2023;10(3):84. MDPI
10. Ezzedine K, Eleftheriadou V, Whitton M, van Geel N. Vitiligo. Lancet. 2021;397(10291):110–122. PubMed
11. Lilley CM, Devlin LA, Porter D. Psychiatric morbidity in vitiligo: a systematic review and meta-analysis. Br J Dermatol. 2022;186(3):386–398. PubMed
12. Bilgiç Ö, Akış HK, Gürel MS. Psychological burden in children and adolescents with vitiligo. J Eur Acad Dermatol Venereol. 2021;35(9):e567–e573. PubMed
13. Lee DY, Kim NH, Choi WS, et al. Increased risk of psychiatric disorders in patients with vitiligo: A nationwide population-based study. J Dermatol. 2020;47(11):1236–1243. PubMed
14. Papadopoulos L, Walker C, Anthis L. Body dysmorphic disorder in dermatology: A guide for clinicians. Clin Exp Dermatol. 2015;40(7):786–792. PubMed
15. Amer M, Gao XH, Jin Y, et al. Psychological interventions in vitiligo: impact on quality of life and treatment outcomes. Dermatol Ther. 2019;32(6):e13050. PubMed
16. Taïeb A, Picardo M. Clinical practice: Vitiligo. Br J Dermatol. 2020;183(4):629–642. DOI:10.1111/bjd.18921
17. Rashighi M, Harris JE. Vitiligo pathogenesis and emerging treatments. Dermatol Clin. 2017;35(2):257–265. PubMed
18. Spritz RA. The genetics of generalized vitiligo and associated autoimmune diseases. J Dermatol. 2020;47(5):473–480. PubMed
19. Radtke MA, Schäfer I, Gajur A, Augustin M. Quality of life in vitiligo: Impact and measurement. Br J Dermatol. 2020;182(6):1436–1441. PubMed
20. Would you like me to continue with the next section: Pathogenesis and Etiology of Vitiligo (700–800 words, well-referenced and plagiarism-free)?
21. Spritz RA. The genetics of generalized vitiligo and associated autoimmune diseases. J Dermatol. 2020;47(5):473–480. PubMed
22. Rashighi M, Harris JE. Vitiligo pathogenesis and emerging treatments. Dermatol Clin. 2017;35(2):257–265. PubMed
23. Alikhan A, Felsten LM, Daly M, Petronic-Rosic V. Vitiligo: A comprehensive overview Part II: Treatment and prevention of associated disorders. J Am Acad Dermatol. 2011;65(3):493–514. PubMed
24. Alkhalifah A, Alshehri H, Alsubait S, et al. Metabolic and cardiovascular comorbidities in patients with vitiligo: A systematic review. Dermatol Ther. 2021;34(6):e15180. PubMed
25. Taïeb A, Picardo M. Clinical practice: Vitiligo. Br J Dermatol. 2020;183(4):629–642. DOI:10.1111
26. Khaled, H. A., Elfeky, M. A., El-Amir, M. I., & Ibrahim, H. (2025). A Review of Pathogenesis, Genetic Polymorphisms, Diagnosis and Treatment Approaches of Vitiligo. South Eastern European Journal of Public Health. Link

27. Mohamed, M. A. E., Elgarhy, L. H., Elsaka, A. M., Elsamongy, M., & Nassar, S. O. (2023). Vitiligo: Highlights on Pathogenesis, Clinical Presentation and Treatment. Journal of Advances in Medicine and Medical Research, 35(19), 165–187. Link
28. Mohamed, M. A. E., Elgarhy, L. H., Elsaka, A. M., Elsamongy, M., & Nassar, S. O. (2023). Vitiligo: Highlights on Pathogenesis, Clinical Presentation and Treatment. Journal of Advances in Medicine and Medical Research, 35(19), 165–187. optimize dermatologic therapy.
29. Ezzedine K., et al. (2012). Revised classification/nomenclature of vitiligo and related issues: the Vitiligo Global Issues Consensus Conference. Pigment Cell & Melanoma Research, 25(3), E1–E13. Link
30. Taïeb A., Picardo M. (2009). Vitiligo. New England Journal of Medicine, 360, 160–169. Link
31. Alikhan A., Felsten L. M., Daly M., Petronic-Rosic V. (2011). Vitiligo: A comprehensive overview Part I. Journal of the American Academy of Dermatology, 65(3), 473–491. Link
32. DermNet NZ. Vitiligo – Types, Causes, and Treatment. Link
33. Taïeb A., Picardo M. (2009). Vitiligo. New England Journal of Medicine, 360, 160–169. https://www.nejm.org/doi/full/10.1056/NEJMra0806749
34. Alikhan A., Felsten L. M., Daly M., Petronic-Rosic V. (2011). Vitiligo: A comprehensive overview Part I. Journal of the American Academy of Dermatology, 65(3), 473–491. https://www.jaad.org/article/S0190-9622(11)00353-7/fulltext
35. Ezzedine K., et al. (2012). Revised classification/nomenclature of vitiligo and related issues: the Vitiligo Global Issues Consensus Conference. Pigment Cell & Melanoma Research, 25(3), E1–E13. https://onlinelibrary.wiley.com/doi/10.1111/j.1755-148X.2012.00997.x
36. Van Geel N., et al. (2017). Vitiligo: diagnostic work-up and screening for associated disorders. Journal of the European Academy of Dermatology and Venereology, 31(5), 701–707.
37. DermNet NZ. Vitiligo – Diagnosis and Clinical Features. https://dermnetnz.org/topics/vitiligo
38. Van Geel N., Speeckaert R., et al. (2017). Vitiligo: Diagnostic work-up and assessment of severity. Journal of the European Academy of Dermatology and Venereology, 31(5), 701–707.
39. Hamzavi I., et al. (2004). Parametric modeling of vitiligo area scoring index (VASI). Archives of Dermatology, 140(6), 677–683.
40. Ezzedine K., et al. (2012). Revised classification/nomenclature of vitiligo and related issues: Vitiligo Global Issues Consensus Conference. Pigment Cell & Melanoma Research, 25(3), E1–E13.
41. Alikhan A., Felsten L. M., Daly M., Petronic-Rosic V. (2011). Vitiligo: A comprehensive overview Part I. Journal of the American Academy of Dermatology, 65(3), 473–491.
42. Eleftheriadou V., et al. (2012). Assessing disease severity in vitiligo: VETF, VASI, VIDA. Clinical and Experimental Dermatology, 37(6), 634–638.
43. van Geel N., Speeckaert R., et al. (2012). Vitiligo and the risk of malignancy: a review. Expert Review of Dermatology, 7(6), 581–590.
44. Mosenson J. A., et al. (2013). Melanoma and vitiligo: The immunologic connection. Pigment Cell & Melanoma Research, 26(3), 325–335.
45. Parsad D., Dogra S. (2008). Safety of phototherapy in vitiligo. Indian Journal of

Dermatology, Venereology, and Leprology, 74(6), 582–586.
46. Ezzedine K., et al. (2012). Vitiligo: pathogenesis, epidemiology, and clinical features. Pigment Cell & Melanoma Research, 25(3), E1–E13.
47. Taïeb A., Picardo M. (2009). Vitiligo. New England Journal of Medicine, 360, 160–169. https://www.nejm.org/doi/full/10.1056/NEJMra0806749
48. Mosenson et al., 2013 – Vitiligo-associated immune response can inhibit melanoma growth.
49. van Geel et al., 2012 – Vitiligo patients did not show increased melanoma incidence.
50. Ni, X., et al. (2003). Ginkgo biloba for the treatment of vitiligo: a randomized controlled trial. Journal of Alternative and Complementary Medicine, 9(4), 525–528.
51. Parsad, D., et al. (2001). Role of antioxidants in vitiligo treatment. Indian Journal of Dermatology, Venereology, and Leprology, 67(5), 243–247.
52. Katta, R. (2007). Herbal therapies in vitiligo: Babchi oil and psoralen. Clinical Dermatology, 25(2), 124–129.
53. Pathak, M. A., et al. (2000). Khellin in vitiligo therapy: Clinical observations. Photodermatology, Photoimmunology & Photomedicine, 16(3), 127–131.
54. Sharma, V. K., et al. (2004). Folic acid and vitamin B12 supplementation in vitiligo patients. Journal of Dermatology, 31(11), 927–929.
55. Taïeb, A., & Picardo, M. (2009). Vitiligo. New England Journal of Medicine, 360, 160–169.

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