Lifestyle Medicine for Melasma Management: A Narrative Review of Modifiable Behavioral and Environmental Factors.

ABSTRACT:

Background: Melasma is a chronic acquired pigmentary disorder affecting an estimated 1% to 33% of the global population, with higher prevalence among individuals with darker skin phototypes in regions of intense ultraviolet exposure. Although pharmacological treatments including topical depigmenting agents and oral tranexamic acid remain the mainstay of therapy, high recurrence rates underscore the need for complementary strategies targeting modifiable risk factors. Lifestyle medicine, which emphasizes evidence-based behavioral interventions across six domains, offers a promising yet underexplored framework for melasma management.

Objective: This narrative review aims to synthesize current evidence on modifiable behavioral and environmental factors that influence melasma onset, severity, and recurrence, and to propose an integrated lifestyle medicine framework for clinical application.

Methods: A comprehensive literature search was conducted in PubMed/MEDLINE, Scopus, and Web of Science for English-language publications from January 2010 to February 2026. Search terms combined melasma-related terminology with lifestyle domains including photoprotection, nutrition, oxidative stress, gut microbiome, psychological stress, sleep, physical activity, alcohol consumption, and environmental exposures. Original research, systematic reviews, meta-analyses, and expert consensus statements addressing modifiable factors were included.

Results: Strong evidence supports ultraviolet and visible light photoprotection as the cornerstone of melasma prevention. Emerging evidence identifies dietary antioxidants, gut microbiome composition via the estrobolome, psychological stress through the cortisol–

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melanocortin axis, sleep quality, and alcohol consumption as modifiable contributors to melasma pathogenesis. Environmental factors including air pollution demonstrate plausible mechanistic links. Physical activity presents a dual relationship, conferring systemic anti-inflammatory benefits while potentially exacerbating melasma through heat-induced melanocyte activation.

Conclusions: An integrated lifestyle medicine approach addressing photoprotection, nutrition, stress management, sleep optimization, and substance avoidance may serve as a valuable adjunct to pharmacotherapy in melasma. We propose the Melasma Lifestyle Modification (MLM) Framework as a practical clinical tool. Prospective interventional studies are needed to validate these lifestyle-based strategies.

Keywords: *melasma; lifestyle medicine; modifiable risk factors; photoprotection; oxidative stress; gut–skin axis; antioxidants; narrative review*

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1. INTRODUCTION

Melasma is a common acquired disorder of hyperpigmentation characterized by symmetric brown-to-gray macules and patches on sun-exposed facial areas, most frequently presenting in centrofacial, malar, and mandibular distribution patterns [1,2]. The condition predominantly affects women, who comprise over 90% of diagnosed cases, particularly those of reproductive age with Fitzpatrick skin phototypes III through V [3,4]. Epidemiological studies report a global prevalence ranging from approximately 1% in general populations to as high as 50% in high-risk groups, with notably elevated rates among East Asian, South Asian, Hispanic, Middle Eastern, and African populations [2,5].

The pathogenesis of melasma is multifactorial and incompletely understood. At the molecular level, melanogenesis in melasma is regulated through several intracellular signaling pathways that converge on the microphthalmia-associated transcription factor (MITF), a master regulator of melanocyte differentiation and pigment production. These pathways include the cyclic adenosine monophosphate/protein kinase A (cAMP/PKA), stem cell factor/c-kit, Wnt/β-catenin, phosphatidylinositol-3-kinase/Akt, and nuclear factor kappa B pathways [6,7]. Ultraviolet radiation activates these cascades both directly and indirectly through paracrine signaling from keratinocytes, fibroblasts, and endothelial cells, leading to upregulation of tyrosinase, tyrosinase-related proteins, and ultimately increased melanin synthesis [6,8]. In addition to traditional epidermal melanocyte hyperactivity, melasma is now recognized as a disorder of the entire cutaneous microenvironment, involving dermal inflammation, solar elastosis, vascular proliferation, mast cell infiltration, and senescent fibroblasts [8,9].Beyond the biological complexity of the disease, melasma exerts a substantial psychosocial burden. A large international survey of over 5,000 melasma patients across 34 countries found that 34% reported Dermatology Life Quality Index scores exceeding 10, indicating a very large impact on quality of life [10]. More than half of

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respondents reported concealing affected skin, and nearly a quarter felt socially excluded by colleagues [10]. The emotional impact encompasses feelings of embarrassment, frustration, depression, and diminished self-esteem, with prevalence of anxiety and depression ranging from 8.7% to over 16% among affected individuals [4,11].

Despite the availability of effective treatments, including hydroquinone, triple combination cream, and oral tranexamic acid, melasma remains notoriously recalcitrant, with high relapse rates upon treatment discontinuation or continued exposure to triggering factors [12,13]. This therapeutic challenge has prompted growing interest in identifying and addressing modifiable risk factors that contribute to disease onset and recurrence.

Lifestyle medicine is an evidence-based medical specialty that uses behavioral interventions—encompassing nutrition, physical activity, sleep, stress management, avoidance of risky substances, and social connectedness—to prevent, treat, and reverse chronic disease [14]. While lifestyle medicine principles have been successfully applied to numerous chronic conditions, their systematic application to melasma management remains largely unexplored. This is surprising given that several well-established melasma triggers, including ultraviolet exposure, stress, hormonal fluctuations, and dietary factors, are inherently behavioral and modifiable.

The objective of this narrative review is to synthesize current evidence on modifiable behavioral and environmental factors relevant to melasma, organized through the lens of lifestyle medicine, and to propose an integrated clinical framework—the Melasma Lifestyle Modification (MLM) Framework (Figure 1)—for complementing existing pharmacological and procedural treatment approaches. Specifically, this review addresses the question: what is the current evidence for modifiable behavioral and environmental factors in the onset, severity, and recurrence of melasma, and how can these factors be organized into a practical lifestyle medicine framework for clinical application?

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2. METHODS

This narrative review was conducted in accordance with the Scale for the Assessment of Narrative Review Articles (SANRA) checklist [46]. A comprehensive literature search of PubMed/MEDLINE, Scopus, and Web of Science databases was performed for publications from January 2010 through February 2026. The primary search combined melasma-specific terms ("melasma" OR "chloasma" OR "facial hyperpigmentation") with lifestyle-related terms across seven domains: photoprotection and visible light, nutrition and antioxidants, gut microbiome, psychological stress, sleep, physical activity and heat exposure, and alcohol and environmental exposures. Domain-specific sub-searches were performed to ensure comprehensive coverage. The initial search retrieved approximately 1,400 records; after duplicate removal and title/abstract screening, 153 full-text articles were assessed, and 85 publications were included (18 RCTs, 32 observational studies, 15 systematic reviews/meta-analyses, and 20 consensus statements or mechanistic studies).

Inclusion criteria encompassed English-language original research (randomized controlled trials, cohort studies, case-control studies, and cross-sectional studies), systematic reviews, meta-analyses, and expert consensus statements that addressed modifiable behavioral or environmental factors in relation to melasma. Exclusion criteria included case reports with fewer than five subjects, conference abstracts without full-text availability, and studies focused exclusively on pharmacological or procedural interventions without a lifestyle component. Foundational mechanistic studies, including selected in vitro and animal studies, were included where they provided essential pathophysiological context for lifestyle factor relationships. Reference lists of included articles were hand-searched for additional relevant publications. The evidence was synthesized narratively and organized according to the six pillars of lifestyle medicine.

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3. MODIFIABLE LIFESTYLE AND ENVIRONMENTAL FACTORS

3.1 Ultraviolet Radiation, Visible Light, and Photoprotective Behavior

Chronic light exposure represents the most consistently identified and the most modifiable trigger for melasma, contributing to both disease onset and relapse [1,2]. Ultraviolet B radiation upregulates the expression of melanocyte-specific genes including tyrosinase and tyrosinase-related protein 1, while also stimulating keratinocyte release of pro-melanogenic factors such as alpha-melanocyte-stimulating hormone (α-MSH), endothelin-1, and stem cell factor [6,8]. UVA radiation contributes through generation of reactive oxygen species and downregulation of catalase activity [8]. The central role of UV protection is underscored by a study demonstrating that use of broad-spectrum sunscreen with a sun protection factor of 50+ during pregnancy reduced melasma development to only 2.7% among 200 Moroccan women [15].

Beyond ultraviolet radiation, visible light (400–700 nm), particularly the blue-violet spectrum (400–500 nm), has emerged as an important contributor to melasma. Melanocytes express the photoreceptor opsin-3, which detects blue light and activates calcium flux, subsequently triggering mitogen-activated protein kinase pathways and increasing MITF expression and melanin production [16]. Importantly, visible light has been shown to induce pigmentation that is darker and more persistent than that caused by UVA1 radiation, particularly in individuals with Fitzpatrick skin types III–VI [17].

A question of practical relevance is whether blue light from electronic device screens contributes meaningfully to melasma. Several studies have addressed this concern. Duteil et al. conducted a prospective, randomized, comparative study in 12 melasma patients, delivering blue light equivalent to 8 hours of high-intensity screen exposure daily for 5 consecutive days. No significant worsening of melasma lesions was observed by colorimetric

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analysis [18]. The explanation lies in irradiance: the blue light emitted by electronic screens is 100 to 1,000 times less intense than solar visible light at comparable wavelengths [18,19]. A dosimetry assessment confirmed that even prolonged device use generates cumulative doses far below the threshold required to induce pigmentation [19]. These findings suggest that while solar visible light is a meaningful trigger for melasma, current evidence does not support restricting screen exposure as a clinical recommendation for melasma patients.

Photoprotective behavior is therefore the cornerstone of melasma prevention and should encompass both UV and visible light protection. Tinted sunscreens containing iron oxides absorb visible light wavelengths and have demonstrated superior efficacy in preventing melasma relapse compared to UV-only sunscreens in a randomized controlled trial [15,20]. Clinical recommendations include daily application of broad-spectrum sunscreen with SPF 50+ and high UVA protection, use of iron oxide-containing tinted formulations, protective clothing including wide-brimmed hats, and behavioral sun avoidance during peak UV hours [1,20].

3.2 Nutrition, Antioxidant Status, and Dietary Factors

Oxidative stress plays an increasingly recognized role in melasma pathogenesis. Elevated levels of oxidative stress markers, including lipid peroxidation products and proinflammatory cytokines, have been detected in melasma-affected skin compared to adjacent healthy skin [21,22]. This oxidative imbalance may be both a consequence of ultraviolet exposure and a contributing factor to sustained melanogenesis through upregulation of tyrosinase activity and disruption of normal antioxidant defense mechanisms.

The role of dietary and supplemental antioxidants in melasma management has been the subject of a recent systematic review encompassing 30 studies published over the past decade [22]. The review evaluated vitamin C, cysteamine, silymarin, Polypodium leucotomos extract (PLE), tomato extract/lycopene, zinc sulfate, and melatonin. Among these, vitamin C

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demonstrated the most consistent evidence, with multiple studies showing that combining vitamin C with physical therapies such as chemical peels and lasers yielded results superior to either modality alone [22]. Cysteamine, a naturally occurring aminothiol, showed efficacy comparable to hydroquinone with a more favorable tolerability profile in several randomized studies [22]. A detailed summary of oral antioxidant and photoprotective supplements is presented in Table 2.

Polypodium leucotomos extract has received particular attention as an oral photoprotective supplement. PLE is derived from a tropical fern native to Central and South America and contains phenolic compounds with antioxidant, anti-inflammatory, and photoprotective properties [23]. In a randomized, double-blind, placebo-controlled trial, 21 female patients with epidermal melasma receiving SPF 45 sunscreen were randomized to oral PLE 240 mg twice daily or placebo for 12 weeks. The PLE group showed a significant reduction in mean MASI scores from 5.7 to 3.3, while the placebo group showed no improvement [24]. However, a subsequent trial in 33 Hispanic women found no significant intergroup difference, although both groups improved [25]. A pilot study in 40 Asian patients found that PLE as adjunct to topical hydroquinone 4% and SPF 50+ sunscreen provided additional improvement in mMASI scores and melanin indices compared to placebo [26]. Overall, PLE appears to offer modest adjunctive benefit with an excellent safety profile, though results remain inconsistent across trials.

From a dietary perspective, a recent case-control study of 150 Chinese patients provided novel insights into nutritional factors associated with melasma risk [27]. Alcohol intake emerged as a strong independent risk factor (OR: 20.05, 95% CI: 1.17–343.17) in multivariate analysis, representing the first study to identify this association. The underlying mechanism may involve ethanol-induced skin hyperpigmentation through aldehyde

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dehydrogenase-dependent pathways, as well as alcohol-related hepatic dysfunction and impaired melanin metabolism [27,28].

Micronutrient deficiencies have also been implicated in melasma susceptibility. Vitamin B12 deficiency, iron deficiency anemia, and folate insufficiency have been associated with melasma in observational studies, suggesting that nutritional adequacy is a modifiable factor warranting clinical attention [29]. Conversely, copper, which is essential for tyrosinase catalytic activity, may promote melanogenesis when present in excess, highlighting the nuanced relationship between mineral balance and pigmentation [30].

Practical dietary recommendations for melasma patients should emphasize increased consumption of antioxidant-rich foods (fruits, vegetables, green tea, and foods rich in vitamins C and E), adequate intake of folate-rich foods, omega-3 fatty acids for their anti-inflammatory properties, and limited consumption of processed foods, refined sugars, and alcohol (Table 4). However, it must be acknowledged that no randomized controlled trial has directly evaluated the impact of a comprehensive dietary intervention on melasma outcomes, representing a significant gap in the literature.

3.3 Gut–Skin Axis and Microbiome

The concept of the gut–skin axis—whereby gut microbial composition influences cutaneous health through systemic immune and metabolic pathways—has gained significant traction in dermatology. While well-established for conditions such as psoriasis, atopic dermatitis, and acne, its relevance to melasma is an emerging area of investigation [31]. A summary of gut microbiome studies relevant to melasma is presented in Table 3.

A pivotal study by Liu et al. used 16S ribosomal RNA sequencing to compare the gut microbiota of melasma patients with healthy controls [32]. Significant differences were found in the abundance of several bacterial genera, with Collinsella spp. identified as a distinctive

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member of the melasma-associated microbiota. The proposed mechanism centers on the estrobolome—the collection of gut bacteria capable of metabolizing estrogens. Specifically, β-glucuronidase enzymes secreted by gut microbiota promote the intestinal reabsorption of deconjugated estrogens, which re-enter systemic circulation and may stimulate melanogenesis through estrogen receptor-β activation on melanocytes and the Wnt/β-catenin pathway in keratinocytes [32,33].

Investigation of the skin microbiome in melasma has also yielded relevant findings. A recent study characterizing microbial dysbiosis in 40 melasma subjects found significant differences in community structure between lesional and perilesional skin, with correlations between specific bacterial genera and clinical parameters including melanin index, erythema, and barrier function markers [34]. The authors noted that reduced antioxidant activity in melasma lesions may be partly linked to the observed microbial dysbiosis [34].

From a therapeutic perspective, a double-blind randomized controlled trial by Piyavatin et al. demonstrated that a synbiotic formulation containing six probiotic strains with fructooligosaccharides significantly reduced modified MASI scores in melasma patients, accompanied by reductions in skin erythema and melanin indices [35]. The proposed mechanism involves synbiotic-driven enhancement of farnesoid X receptor signaling, reduced systemic oxidative load, and consequent downregulation of tyrosinase activity [31,35].

While the gut–skin axis represents a compelling area of investigation, the evidence base remains limited. A systematic review of nutraceutical interventions across dermatological conditions noted that melasma, as a hormonally and metabolically influenced condition, shows more modest responses to probiotic interventions compared to immune-mediated conditions such as atopic dermatitis and psoriasis [31]. Nevertheless, the mechanistic plausibility of the estrobolome pathway and the positive results from the single available

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RCT warrant further investigation. Lifestyle recommendations that support gut health—including dietary fiber intake, fermented food consumption, and avoidance of antibiotic overuse—may offer indirect benefits for melasma management.

3.4 Psychological Stress and the Cortisol–Melanocortin Axis

The relationship between psychological stress and melasma, while mechanistically plausible, remains incompletely established. Stress activates the hypothalamic-pituitary-adrenal axis, leading to increased production of adrenocorticotropic hormone (ACTH) and cortisol [36]. ACTH is derived from the same precursor peptide, proopiomelanocortin, as α-MSH, providing a direct neuroendocrine link between stress and melanogenesis [6,36]. Furthermore, elevated cortisol can create an imbalance in estrogen levels, and this estrogen excess upregulates MSH levels, which in turn increases melanin production [37].

Epidemiological support for the stress–melasma association comes from a recent case-control study in which mental pressure was positively associated with melasma incidence (OR: 1.99, 95% CI: 1.25–3.17) in univariate analysis [27]. In a cross-sectional study of psychiatric morbidity, Deshpande et al. found that 54% of melasma patients identified stress as a precipitating factor, and 42% had depressive disorders, supporting the bidirectional relationship between psychological stress and melasma through cortisol-mediated pathways [36].

A critical and often overlooked dimension is the bidirectional relationship between melasma and psychological distress. Melasma itself causes significant emotional burden, with patients reporting feelings of unattractiveness, social withdrawal, embarrassment, and depression [10,11]. This creates a potential vicious cycle: psychological distress worsens hormonal dysregulation, which may exacerbate melasma, which in turn deepens emotional distress. Breaking this cycle through stress management may therefore address both a potential trigger and its consequence.

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Although no randomized trial has specifically evaluated stress reduction interventions for melasma outcomes, well-established stress management techniques—including mindfulness meditation, cognitive behavioral therapy, and regular relaxation practices—offer plausible benefits through cortisol reduction and hormonal re-equilibration. Integration of psychological support and stress management counseling into melasma treatment plans represents a pragmatic, low-risk addition to standard care.

3.5 Sleep Quality and Circadian Rhythm

The relationship between sleep and melasma is a largely unexplored but biologically compelling area. Insomnia or habitual late sleeping was identified as a risk factor for melasma (OR: 1.88, 95% CI: 1.18–2.99) in the same Chinese case-control study previously discussed [27]. While this represents a single observational study, the underlying biology supports further investigation.

Sleep deprivation is a well-known inducer of systemic inflammation. In the context of melasma, this is significant because lesional skin demonstrates elevated levels of CD4 T cells, mast cells, macrophages, interleukin-17, and cyclooxygenase-2 compared to healthy skin, indicating an active inflammatory component [38]. Sleep deprivation may potentiate this inflammatory milieu, thereby sustaining the melanogenic stimulus. Additionally, the release of α-MSH from the epidermal melanin unit, which plays a role in the hypermelanogenesis of melasma, may be influenced by circadian and sleep-related hormonal fluctuations [39].

Melatonin itself warrants special attention in this context. Beyond its role as a circadian regulator, melatonin possesses antioxidant properties, including free radical scavenging and antioxidant enzyme activation. Notably, melatonin directly inhibits tyrosinase and inducible nitric oxide synthase production, both of which are involved in melanogenesis [22]. A prospective, randomized, double-blind, placebo-controlled multicenter trial evaluated

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melatonin in 50 adult women with moderate melasma and showed some clinical improvement, though further details on this trial remain limited in available literature [22].

A letter to the editor published in the Anais Brasileiros de Dermatologia specifically highlighted the underexplored interaction between sleep and melasma, calling for the routine application of sleep questionnaires in melasma populations [39]. The authors proposed that if evidence for this interaction is confirmed, strategies aimed at improving sleep quality might enhance the efficacy of melasma treatment and improve patients' quality of life. This remains an important research gap.

Practical sleep-related recommendations for melasma patients should include maintenance of consistent sleep schedules (7–8 hours nightly), limiting blue light exposure from screens before bedtime (which may also support circadian alignment), practicing good sleep hygiene, and addressing underlying sleep disorders when present. While these recommendations are supported primarily by biological plausibility rather than melasma-specific trials, they carry negligible risk and offer potential benefits for both skin health and overall wellbeing.

3.6 Physical Activity, Exercise, and Heat Exposure

Physical activity presents a nuanced and somewhat paradoxical relationship with melasma. On one hand, regular exercise confers well-established systemic benefits relevant to melasma pathophysiology, including reduction of cortisol levels, anti-inflammatory effects, improved sleep quality, and metabolic regulation [40]. On the other hand, heat is recognized as an independent trigger for melanocyte activation, and exercise-associated heat generation may exacerbate melasma even in the absence of UV exposure [41].

Heat increases the activity of melanocytes through mechanisms that are distinct from UV-mediated pathways. Patients who exercise regularly, including those who train indoors, may

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find their melasma difficult to control because they are generating sustained facial heat [41]. This effect is particularly relevant for vigorous or prolonged exercise in warm environments.

Complicating this further, the use of certain skincare products during exercise may trigger irritation and subsequent post-inflammatory hyperpigmentation. Products containing active ingredients, essential oils, or fragrances may provoke an exaggerated response when combined with the increased blood flow, skin permeability, and heat associated with exercise [41]. Given that melasma-affected skin demonstrates delayed barrier recovery, this sensitivity may be heightened in affected individuals.

Practical recommendations should therefore not discourage exercise but rather guide patients to mitigate heat exposure during physical activity. Strategies include exercising during cooler hours or in air-conditioned environments, using cooling accessories such as damp towels or cooling bandanas, minimizing the duration of high-intensity activity in hot conditions, using gentle and fragrance-free skincare products during workouts, and applying sunscreen before outdoor exercise. The net benefit of regular physical activity for melasma patients likely remains positive when appropriate precautions are taken.

3.7 Alcohol Consumption and Risky Substance Use

A recent case-control study identified alcohol consumption as a potential novel risk factor for melasma. In the multivariate analysis of 150 Chinese patients, alcohol intake showed an odds ratio of 20.05 (95% CI: 1.17–343.17) [27]. However, this finding must be interpreted with considerable caution: the extremely wide confidence interval, which nearly crosses the null value of 1, reflects the small sample size and limited number of alcohol-consuming participants. This observation is best regarded as hypothesis-generating, requiring replication in larger, diverse cohorts before any causal inference can be drawn. Nevertheless, plausible biological mechanisms support further investigation.

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Matsumoto et al. demonstrated that ethanol intake can induce skin hyperpigmentation in a dose-dependent manner through an aldehyde dehydrogenase 2 activity-dependent mechanism [28]. Other studies have reported that ethanol consumption exacerbates UV-induced hyperpigmentation, and patients with alcohol-related liver disease exhibit pigmentation disorders characterized by excess melanin in giant melanosomes with a normal number of melanocytes—a pattern with pathogenic similarities to melasma [27,42]. Additionally, alcohol may contribute to oxidative stress, hepatic dysfunction affecting estrogen metabolism, and nutritional deficiencies that could collectively promote melanogenesis.

Regarding tobacco smoking, direct evidence linking smoking to melasma is limited. However, smoking is a well-established contributor to oxidative stress, premature skin aging, and impaired cutaneous microcirculation, all of which may theoretically aggravate the melasma phenotype [43]. Given the overall health implications, advising melasma patients to limit alcohol consumption and avoid smoking is consistent with both general health recommendations and the emerging melasma-specific evidence.

3.8 Environmental and Occupational Factors

Environmental exposures beyond UV radiation contribute to oxidative stress and may influence melasma development. Air pollution, particularly particulate matter and polycyclic aromatic hydrocarbons, penetrates the skin and generates reactive oxygen species, potentially exacerbating the oxidative stress environment that sustains melanogenesis in melasma-affected skin [27,44].

Interestingly, the Chinese case-control study found that change of residence and house renovation were protective factors against melasma (OR: 0.03, 95% CI: 0.00–0.30 and OR: 0.13, 95% CI: 0.03–0.58, respectively) [27]. The authors hypothesized that relocation from more polluted areas to cities with cleaner environments could account for this protective effect, reflecting the broader impact of environmental quality on skin health.

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Occupational exposures also deserve consideration. Workers with significant outdoor sun and heat exposure, such as agricultural laborers, may face compounded risk. The prevalence of melasma among paddy field workers in India has been reported to reach 41%, highlighting the interaction between occupational UV exposure, heat, and skin phototype [5]. Similarly, individuals working near heat sources such as ovens or stoves may experience localized heat-triggered melanocyte activation [41].

While individual modification of ambient air quality may be limited, clinical recommendations can include awareness of pollution exposure, use of antioxidant-rich skincare to counteract pollution-related oxidative stress, and occupational counseling regarding UV and heat protection for at-risk workers.

4. DISCUSSION

4.1 Synthesis of Evidence and the MLM Framework

The evidence reviewed above demonstrates that melasma, while fundamentally a disorder of pigmentary regulation, is substantially influenced by a constellation of modifiable behavioral and environmental factors. These factors can be organized into a proposed Melasma Lifestyle Modification (MLM) Framework comprising three tiers (Figure 1):

Tier 1 – Direct melanogenic triggers: UV radiation, solar visible light, and heat exposure. These factors act directly on melanocytes and the cutaneous microenvironment. The evidence for photoprotection is robust and unambiguous, representing the highest-priority modifiable factor.

Tier 2 – Indirect hormonal and inflammatory modulators: Psychological stress (via the cortisol–melanocortin axis), sleep deprivation (via inflammation and α-MSH dysregulation), alcohol consumption (via hepatic and enzymatic pathways), gut microbiome dysbiosis (via the estrobolome and systemic inflammation), and air pollution (via oxidative stress). These

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factors act through intermediate systemic pathways and have moderate to emerging levels of evidence.

Tier 3 – Protective and buffering factors: Antioxidant-rich nutrition, supplementation (PLE, vitamin C, synbiotics), regular physical activity (with heat precautions), and adequate sleep. These factors modulate the underlying susceptibility and may reduce the likelihood and severity of melasma flares.

4.2 Evidence Levels and Research Gaps

The strength of evidence varies considerably across the lifestyle domains reviewed (Table 1). Photoprotection against UV and visible light is supported by multiple randomized controlled trials and consensus guidelines. The evidence for dietary antioxidants, particularly oral supplements such as PLE, is supported by a small number of RCTs with mixed results (Table 2). The gut microbiome–melasma connection rests on a single RCT and several observational studies (Table 3). For psychological stress, sleep quality, and alcohol consumption, the evidence is primarily observational, derived largely from the single large case-control study by Shi et al., which requires replication in diverse populations [27].

Several critical research gaps warrant attention. First, no prospective interventional study has evaluated a comprehensive lifestyle medicine intervention program for melasma. Such a study, incorporating photoprotection counseling, dietary modification, stress management, and sleep optimization, would provide the strongest test of the lifestyle medicine approach. Second, the gut–skin axis pathway needs validation in larger, multi-ethnic populations with longitudinal follow-up. Third, the contribution of individual dietary patterns versus supplementation to melasma outcomes remains unclear. Fourth, the observed association between alcohol and melasma, while striking, requires confirmation given the wide confidence interval and potential confounding factors.

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4.3 Integration with Existing Treatment Paradigms

The lifestyle medicine approach proposed here is not intended to replace pharmacological or procedural treatments but rather to complement them. Treating melasma is unlikely to be effective if underlying triggers are not addressed [12,45]. This principle underscores the importance of identifying and modifying behavioral risk factors alongside prescribing depigmenting agents or performing procedures.

A practical clinical workflow might incorporate lifestyle assessment at the initial melasma consultation, with targeted counseling based on identified modifiable risk factors. For example, a patient presenting with moderate melasma who reports inadequate sunscreen use, high work-related stress, irregular sleep, and regular alcohol consumption would benefit from an integrated treatment plan addressing all four domains in addition to standard pharmacotherapy. Detailed practical recommendations are summarized in Table 4.

4.4 Special Populations

Certain populations warrant specific consideration within the lifestyle medicine framework. Pregnant women, who are particularly susceptible to melasma due to hormonal changes, face limitations in pharmacological treatment but may benefit substantially from lifestyle modifications including rigorous photoprotection and nutritional optimization [2,15]. Perimenopausal and menopausal women experiencing hormonal fluctuations and potentially new-onset melasma may similarly benefit from stress management and sleep optimization strategies alongside hormone-related counseling [37]. Individuals with darker skin phototypes, who bear a disproportionate burden of melasma globally, may have different thresholds for visible light-induced pigmentation and should receive appropriately tailored photoprotection advice including iron oxide-containing sunscreens [17,20].

4.5 Digital Health Opportunities

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The lifestyle medicine approach to melasma management may benefit from digital health technologies. Wearable UV monitors can provide real-time feedback on cumulative UV exposure, prompting timely sunscreen reapplication or behavioral avoidance. Mobile health applications for dietary tracking, sleep monitoring, and stress management offer accessible tools for patient engagement. Teledermatology platforms can facilitate ongoing lifestyle counseling and treatment monitoring, particularly in regions with limited access to dermatological care. While these digital tools have not been specifically validated in melasma populations, their integration into lifestyle medicine programs represents a promising direction.

4.6 Strengths and Limitations

This review provides, to our knowledge, the first comprehensive synthesis of modifiable lifestyle factors in melasma through the lens of lifestyle medicine. While previous reviews have extensively addressed pharmacological and procedural treatments [12,13], and some have touched upon individual lifestyle factors such as photoprotection or antioxidants, none have systematically integrated behavioral and environmental modifiers across all six lifestyle medicine domains into a unified clinical framework. However, several limitations should be acknowledged. The narrative review methodology, while appropriate for synthesizing a heterogeneous evidence base, does not provide the same level of systematic rigor as a systematic review with meta-analysis. The quality of available evidence is variable, with much of the lifestyle-specific data coming from observational studies with inherent risks of confounding and bias. Notably, the pivotal case-control study by Shi et al. [27], which provides key data on alcohol, sleep, and stress as risk factors, was conducted in a single-center Chinese population; whether these findings generalize to diverse ethnic groups remains unknown and represents an important limitation. Future multi-ethnic, multicenter studies are needed to validate these associations across different populations. Additionally,

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while oral tranexamic acid has become an important adjunctive therapy in melasma management [12,13], this review focused specifically on behavioral and environmental modifications rather than pharmacological agents; the role of tranexamic acid and other systemic therapies is well-covered in existing treatment-focused reviews. The proposed MLM Framework, while grounded in current evidence, remains theoretical and requires prospective validation.

5. CONCLUSION

Melasma is a multifactorial disorder in which modifiable behavioral and environmental factors play a substantial but often underappreciated role. This narrative review demonstrates that beyond the well-established primacy of photoprotection, emerging evidence supports the relevance of nutrition and antioxidant status, gut microbiome composition, psychological stress, sleep quality, alcohol consumption, and environmental exposures to melasma pathogenesis and clinical outcomes.

The proposed Melasma Lifestyle Modification Framework (Figure 1) organizes these factors into a three-tiered model of direct triggers, indirect modulators, and protective factors, providing a practical structure for clinical counseling. While the evidence base for some domains remains preliminary, the low-risk nature of lifestyle interventions, combined with their potential benefits for overall health, supports their integration into melasma management strategies.

We call for prospective interventional studies evaluating comprehensive lifestyle medicine programs in melasma populations, large-scale multi-ethnic validation of the gut–melasma connection, and development of evidence-based clinical guidelines that incorporate lifestyle counseling alongside pharmacological treatment. Bridging the gap between dermatological

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care and lifestyle medicine holds promise for reducing the burden of this challenging condition and improving quality of life for the millions of individuals affected worldwide.

FIGURE 1

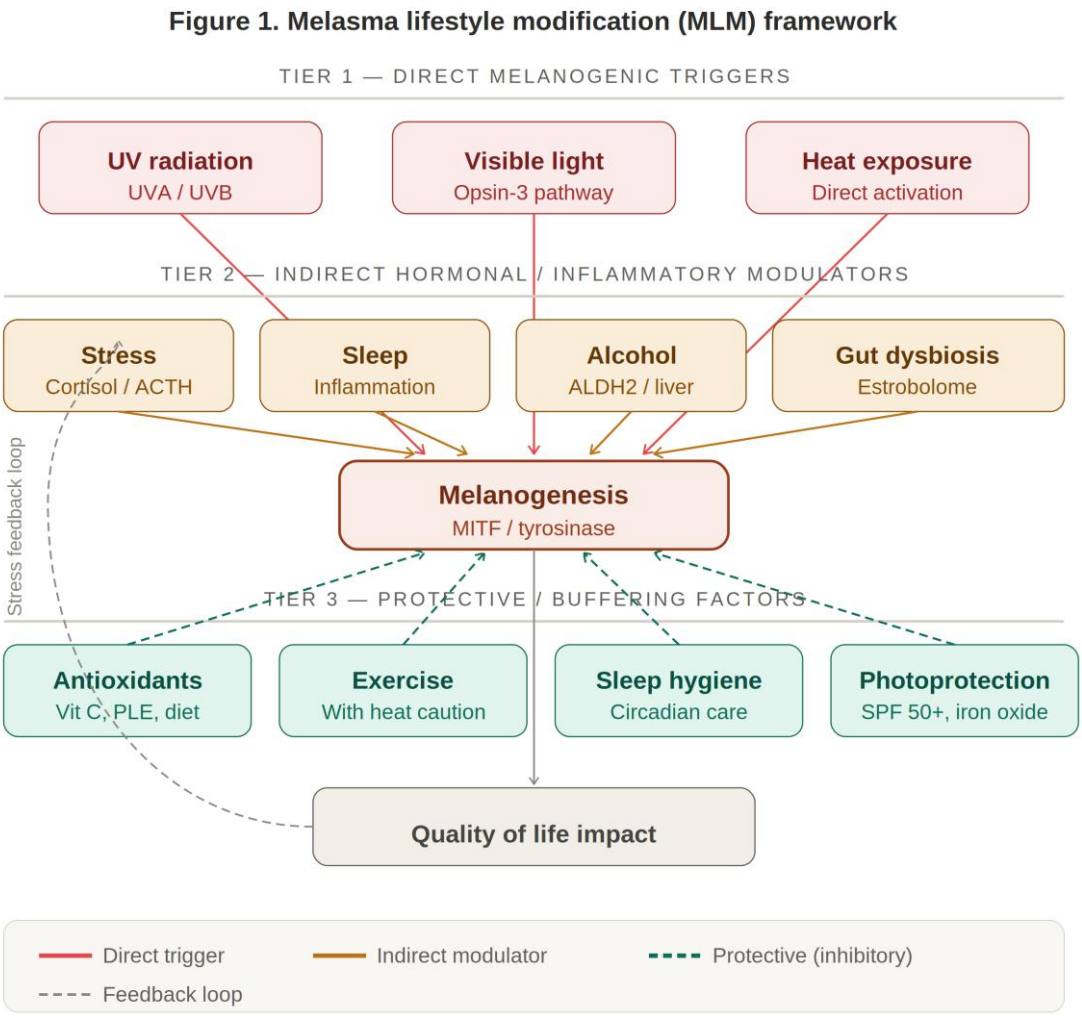

Figure 1. Melasma Lifestyle Modification (MLM) Framework. *The framework organizes modifiable factors into three tiers: Tier 1 (red, solid arrows) represents direct melanogenic triggers acting on melanocytes (UV radiation, solar visible light, and heat); Tier 2 (amber, solid arrows) represents indirect hormonal and inflammatory modulators acting through systemic pathways (psychological stress, sleep deprivation, alcohol consumption, and gut microbiome dysbiosis); Tier 3 (teal, dashed arrows indicating inhibitory/protective action)*

represents protective and buffering lifestyle factors (dietary antioxidants, physical activity with heat precautions, sleep hygiene, and photoprotective behaviors). All pathways converge on the central node of melanogenesis (MITF/tyrosinase activation). A bidirectional feedback loop connects melanogenesis to quality-of-life impact, which may perpetuate the stress–melasma cycle. Abbreviations: ACTH, adrenocorticotropic hormone; ALDH2, aldehyde dehydrogenase 2; MITF, microphthalmia-associated transcription factor; PLE, Polypodium leucotomos extract; POMC, proopiomelanocortin; SPF, sun protection factor.

TABLES

Table 1. Summary of evidence for modifiable lifestyle factors in melasma

Factor	Evidence level	Mechanism	Key evidence	Recommendation
UV/VL exposure	High (RCTs)	Direct melanogenesis via tyrosinase, Opsin-3	Multiple RCTs; Lakhdar et al.; Boukari et al.	SPF 50+, iron oxide, behavioral avoidance
Antioxidants/PLE	Moderate (RCTs, mixed)	ROS scavenging, tyrosinase inhibition	Ahmed et al. 2013; Goh et al. 2018; SR 2025	Oral PLE, vitamin C, dietary antioxidants
Gut microbiome	Emerging (1 RCT)	Estrobolome, β-glucuronidase, SCFA	Liu et al. 2022; Piyavatin et al. 2021	Fiber, fermented foods, synbiotics
Stress	Low–Moderate	HPA axis, ACTH/α-MSH, cortisol–estrogen	Shi et al. 2025; Deshpande et al. 2018 [36]	Stress management, CBT, mindfulness
Sleep	Low	Inflammation, α-MSH, melatonin	Shi et al. 2025 (OR 1.88); Sampaio Xerfan et al. 2020 [39]	7–8 h sleep, sleep hygiene
Exercise/Heat	Low	Heat-melanocyte activation; anti-inflammatory	Passeron & Picardo 2018 [41]; Warburton et al. 2006 [40]	Cool exercise, timing, gentle skincare
Alcohol	Emerging	ALDH2, liver dysfunction, estrogen	Shi et al. 2025 (OR 20.05)	Limit alcohol consumption
Air pollution	Low (mechanistic)	PM, PAHs, ROS generation	Roberts et al.; Shi et al.	Antioxidant skincare, awareness

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Table 2. Oral antioxidant and photoprotective supplements evaluated in melasma

Supplement	Study	Design / N	Key findings	Adverse effects	Evidence quality
PLE (oral)	Ahmed et al. JAMA Dermatol 2013	RCT; N=21; 12 wk; PLE 240 mg BID + SPF 45	MASI 5.7→3.3 ($p<0.05$); placebo no change	None reported	Moderate
PLE (oral)	Goh et al. J Clin Aesthet Dermatol 2018	RCT; N=40; 12 wk; PLE + HQ 4% + SPF 50+	Additional mMASI and melanin index improvement vs placebo	None significant	Moderate (pilot)
Vitamin C (topical + physical)	Sarkar et al. Indian J Dermatol 2025 (SR)	SR of RCTs; split-face with microneedling, laser, peels	Combining vit C with physical therapies superior to monotherapy	Mild skin irritation	Moderate
Cysteamine (topical)	Sarkar et al. 2025; Niazi et al. 2022	SR; multiple RCTs vs HQ 4%	Efficacy comparable to HQ with fewer side effects	Odor, transient irritation	Moderate-High
Silymarin (topical)	Sarkar et al. 2025	RCTs; topical cream	Effective in reducing melasma severity	Minimal	Moderate
Lycopene (oral)	Sarkar et al. 2025	RCTs; oral adjuvant	Significant improvement as adjuvant therapy	None reported	Low-Moderate
Melatonin (topical)	Sarkar et al. 2025	RCT; N=50; multicenter, double-blind	Some clinical improvement; inhibits tyrosinase and iNOS	Not specified	Low-Moderate

Abbreviations: HQ = hydroquinone; MASI = Melasma Area Severity Index; mMASI = modified MASI; PLE = Polypodium leucotomos extract; RCT = randomized controlled trial; SPF = sun protection factor; SR = systematic review.

Table 3. Gut microbiome studies relevant to melasma

Study	Design	Key findings	Proposed mechanism	Relevance
Liu et al. Front Microbiol 2022	Case-control; 16S rRNA	Collinsella spp. distinctive in melasma; differential Actinobacteria, Bacteroidetes, Firmicutes	Estrobolome; β-glucuronidase promotes estrogen reabsorption	First gut microbiota characterization in melasma
Piyavatin et al. 2021	RCT; double-blind	Synbiotic (6 strains + FOS) reduced mMASI, erythema, melanin indices	FXR signaling; reduced systemic oxidative load	Only RCT of gut-directed intervention for melasma
Reddy et al. Front Microbiomes 2025	Observational; skin 16S rRNA; N=40	Skin dysbiosis in lesional vs perilesional; reduced antioxidant activity	Disrupted skin microbiome reduces antioxidant defense	Skin (not gut) microbiome in melasma
Microorganisms 2025 (SR)	SR; 60 RCTs across 5 skin conditions	Melasma shows modest probiotic response vs immune-mediated conditions	SCFA; bile acid-estrogen recycling via FXR	Contextualizes melasma in gut-skin axis literature

Abbreviations: FOS = fructooligosaccharides; FXR = farnesoid X receptor; mMASI = modified MASI; SCFA = short-chain fatty acids; SR = systematic review.

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Table 4. Practical lifestyle recommendations for melasma management

Domain	Recommendation	Evidence	Rationale
Photoprotection	Broad-spectrum SPF 50+ daily, year-round; tinted sunscreen with iron oxides; wide-brimmed hats; behavioral sun avoidance	High	UV is primary modifiable trigger; iron oxides protect against VL; 50% reduction with strict SPF in pregnancy
Nutrition	Antioxidant-rich diet (fruits, vegetables, green tea, vitamins C and E); consider oral PLE 240 mg BID; ensure adequate folate, B12, iron	Moderate	Oxidative stress elevated in melasma skin; PLE shows modest benefit in RCTs; iron deficiency associated with melasma
Gut health	Dietary fiber; fermented foods; consider synbiotic supplementation; avoid unnecessary antibiotics	Emerging	Estrobolome pathway; synbiotic RCT showed mMASI reduction; gut dysbiosis linked to melasma
Stress management	Mindfulness, CBT, relaxation techniques; address psychological impact of melasma; consider referral	Low-Mod	Cortisol-POMC-MSH axis; stress OR 1.99; bidirectional melasma-distress cycle; 42% psychiatric comorbidity
Sleep	7-8 hours nightly; consistent sleep schedule; limit pre-bedtime blue light for circadian benefit; sleep hygiene	Low	Insomnia OR 1.88; sleep deprivation induces inflammation; melatonin inhibits tyrosinase
Physical activity	Regular moderate exercise in cool environments; cooling accessories; gentle skincare during workouts	Low	Exercise reduces cortisol (beneficial) but heat activates melanocytes (harmful); balance needed
Substance avoidance	Limit alcohol consumption; avoid tobacco smoking	Emerging	Alcohol OR 20.05 (wide CI, requires replication); ALDH2 pathway; smoking increases oxidative stress

Evidence grading: High = multiple RCTs/consensus guidelines; Moderate = limited RCTs or strong observational; Low = single studies or mechanistic; Emerging = hypothesis-generating, requires replication. Abbreviations: CBT = cognitive behavioral therapy; ALDH2 = aldehyde dehydrogenase 2; POMC = proopiomelanocortin; VL = visible light.

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