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KEY TAKEAWAYS:

- Psoriasis confers a modest overall increase in malignancy risk, concentrated in NMSC and selected lymphoid cancers, and amplified by disease severity and cumulative PUVA/systemic exposure.
- Chronic systemic inflammation and, in some contexts, therapy-related immunomodulation jointly shape cancer risk in psoriasis, rather than biologic treatment being uniformly carcinogenic.
- Modern biologic and targeted therapies—including IL-17 and IL-23 inhibitors, as well as PDE4 and TYK2 inhibitors—generally show no clear increase in overall malignancy or recurrence risk, though ongoing pharmacovigilance and individualized oncology input remain crucial.
- Management should be multidisciplinary and risk-adapted, combining effective psoriasis control with tailored cancer surveillance and aggressive modification of lifestyle risk factors (smoking, alcohol, obesity, UV exposure).

Psoriasis and Malignancy

Ron Vender, MD, FRCPC

Introduction

Psoriasis is a chronic systemic inflammatory disease that confers a modest, but reproducible, increase in malignancy risk, particularly for non-melanoma skin cancer (NMSC) and lymphohematologic malignancies such as cutaneous T-cell lymphoma and Hodgkin lymphoma. This risk rises alongside disease severity and cumulative inflammatory burden.[1-4] Evidence from meta-analyses and large registries

show that once confounding factors are accounted for, the incidence of most solid tumours approximates that of the general population, while excess risk remains concentrated in NMSC and lymphoma.[2-6] Additionally, the high rates of smoking, alcohol use, obesity, and metabolic syndrome among individuals with psoriasis further amplifies the risk of lung, bladder, liver, breast, endometrial, and colorectal cancers, highlighting the synergistic impact of lifestyle factors and chronic inflammation.[1,2,7]

Inflammatory Pathways Linking Psoriasis and Cancer

Mechanistically, psoriasis exemplifies inflammation-driven carcinogenesis, with genome-wide and transcriptomic studies demonstrating overlap between psoriasis susceptibility loci and oncogenic pathways.[1,7] Persistent activation of innate and adaptive immunity sustains a cytokine-rich microenvironment characterized by reactive oxygen species, angiogenic factors, and impaired DNA repair, promoting tumour initiation and progression while undermining immunosurveillance.[1,7,8] The central tumour necrosis factor (TNF)–interleukin (IL)-23–IL-17 axis not only drives keratinocyte hyperproliferation and dermal inflammation but also recapitulates hallmarks of tumour-promoting inflammation, including nuclear factor kappa B (NF-κB) and signal transducer and activator of transcription 3 (STAT3) activation, vascular endothelial growth factor overexpression, recruitment of myeloid-derived suppressor cells, and alternatively activated macrophages.[1,7-9] Within this network, IL-12 predominantly supports antitumour immunity through Th1 polarization, interferon-γ production, and enhancement of cytotoxic lymphocyte and NK-cell function, whereas IL-23 and IL-17 sustain Th17 responses, angiogenesis, STAT3 activation, and immune evasion, and may contribute to resistance to immune checkpoint inhibitors.[8-10]

Context-Dependent Malignancy Risk in Psoriasis

Epidemiologic evidence from the past two decades consistently characterizes psoriasis as a condition with a moderate, context-dependent increase in cancer risk rather than a generalized carcinogenic state.[1-4] Systematic reviews and meta-analyses report overall cancer relative risks at approximately 1.14–1.21, with higher values observed for NMSC and lymphoma, and stronger signals among patients with moderate-to-severe disease or those receiving systemic or biologic therapies.[2-4] A large Danish study involving a cohort of over 60,000 patients demonstrated increased standardized incidence ratios for NMSC, lymphoma, and lung cancer, while UK and Australasian datasets corroborate elevated rates of cutaneous T-cell lymphoma and keratinocyte cancers, particularly in the setting of ultraviolet exposure, smoking, and historical immunosuppression.[2-4,6,11,12] Importantly, when these factors are rigorously controlled, the incidence of most solid tumours approximates that of the general population, underscoring that excess cancer risk in psoriasis is selective and heavily modulated by comorbidities and prior therapies.[2-4,6]

Historical Therapies and Malignancy Burden

Historical treatments account for much of the cutaneous and lymphoid malignancy burden observed in older cohorts.[2,3,11,13-15] Psoralen and ultraviolet A (PUVA) phototherapy is clearly carcinogenic, showing a dose-dependent increase in squamous-cell carcinoma and a probable increase in melanoma, especially after 200–250 sessions; this elevated risk persists for over a decade after exposure.[11,13,14] Cyclosporine, introduced before modern pharmacovigilance, suppresses T-cell-mediated tumour surveillance and facilitates oncogenic viral reactivation, leading to higher rates of NMSC and lymphoma, particularly with prolonged use or when administered sequentially with PUVA.[11,14,15] Methotrexate, while a long-standing systemic mainstay, has been associated with increased NMSC in observational cohorts and randomized cardiovascular trials, whereas acitretin appears chemopreventive for keratinocyte carcinoma by normalizing keratinocyte differentiation and promoting apoptosis in dysplastic clones.[11,13-16]

Malignancy Risk with Biologic and Targeted Therapies: Reassuring Long-Term Evidence

The introduction of biologics and newer targeted small molecules has substantially reshaped the malignancy risk-benefit calculus.[1,7,8,15] TNF inhibitors initially raised concern because TNF plays dual roles in tumour biology—chronic low-grade signalling can drive proliferation and angiogenesis, while TNF is also central for cytotoxic immune defence. However, contemporary meta-analyses and registry data show no significant increase in overall invasive malignancy in psoriasis or across other immune-mediated diseases, aside from a small but consistent excess of NMSC, particularly among patients with substantial prior phototherapy or actinic damage.[15,17-19] IL-12/23 and IL-23 inhibitors (e.g., ustekinumab, guselkumab, risankizumab, tildrakizumab) represent a more selective immunologic paradigm; long-term extension studies and pooled pharmacovigilance data have not demonstrated an increased rate of invasive malignancy or NMSC and meta-analyses in patients with prior cancer show no elevated risk of new or recurrent malignancy under these agents.[1,7,8,10,18,19]

Malignancy Considerations with IL-17 and IL-23-Targeted Therapy

IL-17 and IL-23 inhibitors are of particular interest given mechanistic evidence that IL-23/Th17 signalling is pro-tumorigenic.[1,7-9,20] Population-based and clinical data indicate that blocking IL-17 and IL-23 does not increase overall or site-specific malignancy rates and may, by dampening STAT3/NF-κB-driven inflammation while sparing cytotoxic T-cell function, theoretically reduce inflammation-driven carcinogenic potential, though this remains to be proven in prospective trials.[1,7-9,20,21] Large international datasets and focused series in patients with prior solid tumours report no signal of increased recurrence or new malignancy under IL-17A inhibitors, supporting their use in carefully selected patients with a history of cancer.[20,21] Apremilast, a phosphodiesterase 4 inhibitor, offers a non-immunosuppressive option that reduces pro-inflammatory cytokines and has not been associated with increased malignancy, even in patients with prior cancer or significant comorbid burdens.[8,15,22,23] Deucravacitinib, a highly selective tyrosine kinase 2 (TYK2) inhibitor, modulates IL-12, IL-23, and type I interferon signalling without broad Janus kinase inhibition and has shown malignancy rates comparable to placebo and background population levels; however, isolated cases of lymphoma highlight the need for ongoing pharmacovigilance as real-world data accumulate.[8,16,24,25]

Individualized Treatment Strategies in Psoriasis and Malignancy

Contemporary management of psoriasis in patients with current or previous malignancy emphasizes individualized, multidisciplinary decision-making rather than categorical avoidance of systemic therapy.[1-3,7,15,18,19,22,23,26] Initial assessment should document tumour type, stage, treatment course, prognosis, time since remission, and recurrence potential, recognizing that the traditional five-year "cancer-free" rule is increasingly challenged by emerging data showing that modern biologics—particularly IL-17 and IL-23 inhibitors—do not appear to increase new or recurrent malignancy compared with background rates in appropriately selected patients.[14,21,22,26] In the setting of active cancer, clinicians generally prioritize topical therapies, limited/targeted phototherapy (avoiding high cumulative ultraviolet exposure in those predisposed to skin cancer), and apremilast for mild to moderate disease, reserving biologics for severe or refractory psoriasis only after oncology consultation, especially when quality-of-life impairment is substantial.[8,14,15,22,23] For patients in remission, decisions to re-introduce or initiate systemic therapy should be guided by tumour biology, disease stability, and patient preferences. IL-17 and IL-23 inhibitors are often favoured over broadly immunosuppressive agents, while methotrexate and cyclosporine are reserved for exceptional indications, and acitretin remains suitable for those at high risk of NMSC.[11,14,21,22,23,26]

Risk-Adapted Surveillance and Prevention

Risk-adapted surveillance and lifestyle modification are crucial components for mitigating malignancy risk in psoriasis.[1-3,7,11,13,14,17,27] Before starting systemic or biologic therapy, a

comprehensive baseline evaluation—including full-skin examination, dermatoscopy of suspicious lesions, lymph-node palpation, and review of prior phototherapy and immunosuppressant exposure—should be combined with age-appropriate screening (e.g., breast, colorectal, cervical, prostate) according to national guidelines, with earlier or intensified screening for patients with long-standing severe disease or significant carcinogenic exposures.[1-3,7,9,11,13-15,22] During treatment, registry data do not justify uniformly intensified cancer screening solely because of biologic use; rather, clinicians should maintain standard guideline-based screening, complemented by annual skin examinations for most patients and six-monthly skin checks for those with prior PUVA exposure, NMSC, or extensive immunosuppression, with synchronization of dermatologic and oncologic follow-up in patients with a cancer history.[2,3,11,13,14,17,22,23] Lifestyle counselling should focus on smoking cessation, alcohol moderation, weight management, and photoprotective behaviours, which collectively reduce independent cancer risks, decrease systemic inflammatory burden, and help reframe malignancy prevention as a shared, modifiable target in psoriasis care.[1,2,7,11,13,14,27]

Conclusion

Psoriasis is best understood as a chronic inflammatory disease that confers a modest, selective increase in malignancy risk—principally for NMSC and lymphoid cancers—driven by systemic inflammation, lifestyle factors, and historical exposures such as PUVA, methotrexate, and cyclosporine, and that when these confounders are addressed, the incidence of most solid tumours approaches that of the general population.[1-4,6,7,11,13-15,25,27] The TNF–IL-23–IL-17 axis mechanistically links psoriatic inflammation to carcinogenesis while providing a rationale for pathway-specific targeting. Current evidence indicates that IL-17 and IL-23 inhibitors, apremilast, and selective TYK2 inhibitors do not significantly increase overall invasive malignancy or cancer recurrence, apart from a persistent NMSC signal associated with TNF inhibitors.[1,5,7-9,10,14,15,17,19-26,28] Consequently, management should shift from categorical avoidance of systemic therapy in patients with current or prior malignancy to an individualized, multidisciplinary decision-making approach that integrates tumour biology, prior carcinogenic exposures, and patient values, within a framework of guideline-based screening and proactive lifestyle modification, as growing registry and biomarker data increasingly allow malignancy risk in psoriasis to be quantified and actively managed as part of personalized care.[1-3,7,11,13-15,18,19,22,23,25,27]

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Immune-Mediated Inflammatory Skin Disease in Middle Eastern and North African Populations: Atopic Dermatitis at the Intersection of Biology, Culture, and Structural Inequity

Sameh Hanna, MD, DABD

Abstract

Atopic dermatitis (AD), a prototypical immune-mediated inflammatory disease, represents a growing and underrecognized public health burden among Middle Eastern and North African (MENA) populations. Global estimates suggest that AD affects more than 170 million individuals worldwide, with increasing prevalence in low- and middle-income regions undergoing rapid urbanization, including much of the MENA region. Despite this growing impact, MENA populations remain underrepresented in epidemiologic datasets and in the development of clinical trial guidelines. This narrative review examines current evidence on AD in MENA populations, integrating insights on epidemiology, immunopathogenesis, clinical phenotype, socio-cultural context, health system capacity, and geopolitical determinants. In addition to clinical and immunopathological considerations, particular emphasis is placed on unmet needs, quality-of-

life impact, and structural barriers to care. Gaining an understanding of these region-specific factors is essential for dermatologists caring for patients of MENA ancestry and for the development of equitable, context-sensitive management strategies.

Defining the Middle Eastern and North African Population

The term "Middle Eastern" is frequently used imprecisely in dermatologic literature, often conflating ethnicity, geography, religion, and culture. The Middle East and North Africa (MENA) region spans North Africa, the Levant, the Arabian Peninsula, and parts of Western Asia **(Figure 1)**, encompassing populations with substantial heterogeneity in genetic ancestry, skin phototype, climate exposure, and healthcare access **(Figure 2)**. In medical literature, individuals from this region are commonly misclassified as "White" or, more egregiously, "Caucasian," masking meaningful differences in disease burden and presentation. Similarly, "Arab" is a culturo-linguistic designation and should not be conflated with race, ancestry, or specific ethnicity.

Global dermatology research demonstrates that atopic dermatitis (AD) prevalence varies widely among populations sharing ancestry who live in differing environmental and socioeconomic contexts. This underscores the dominant influence of urbanization, environmental exposures, healthcare access, and ethnicity-context over ethnicity alone.[1-3] These findings highlight the limitations of extrapolating data derived from Western cohorts to MENA populations.

Epidemiology of Atopic Dermatitis in the MENA Region

AD prevalence is increasing across much of the MENA region, particularly within urban centres. Global Burden of Disease analyses indicate that approximately 171 million individuals worldwide were affected by AD in 2019, corresponding to just over 2% of the global population.[2] While

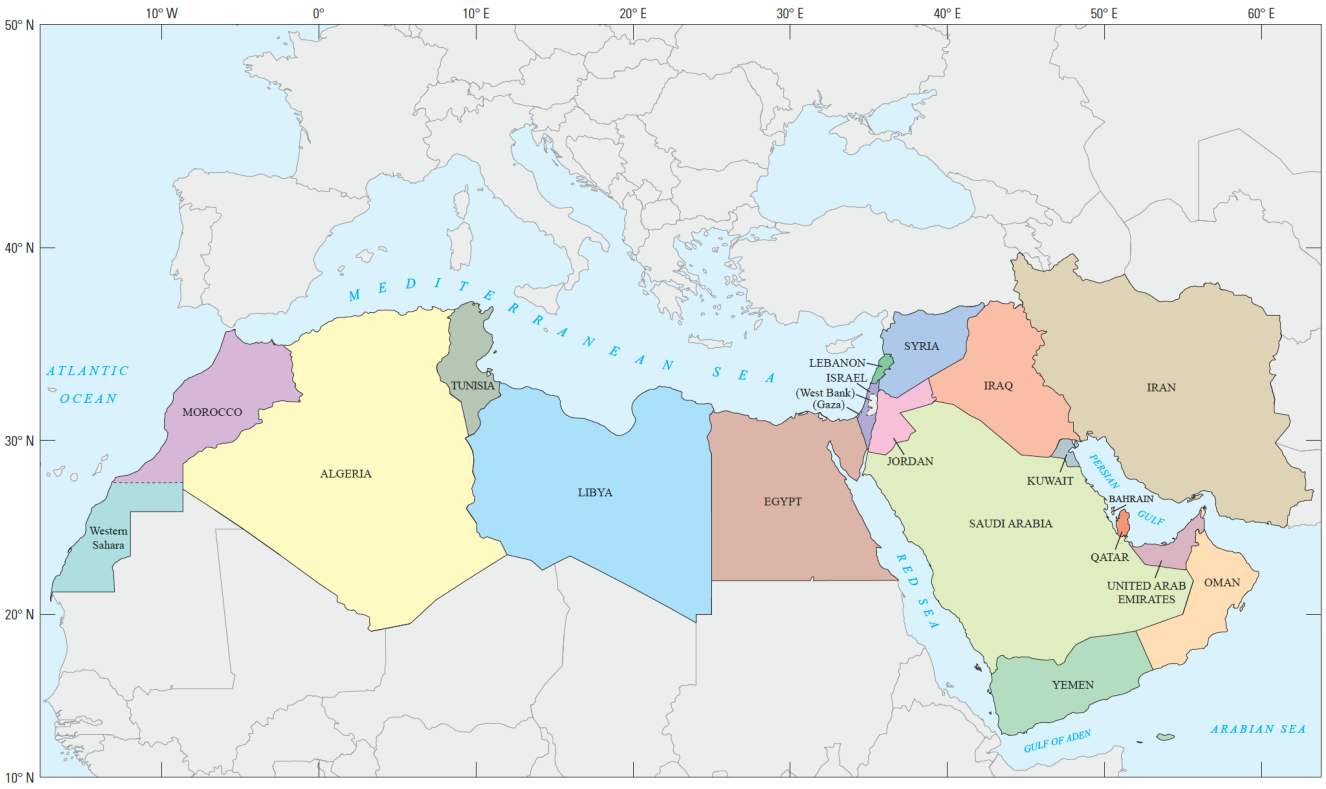


Figure 1. Map of the Middle East and North Africa region. 2015 Minerals Yearbook; *adapted from Taib M. 2015 Minerals Yearbook The Middle East and North Africa, 2019.*

age-standardized prevalence rates in the MENA region have remained relatively stable from 1990 to 2019, absolute case numbers and years lived with disability remain high, reflecting substantial population growth and demographic shifts.[6]

Country-level studies reveal marked heterogeneity in AD prevalence estimates. International Study of Asthma and Allergies in Childhood (ISAAC)-based surveys and multinational analyses report pediatric AD prevalence ranging from approximately 3–4% in Egypt to more than 15–20% in Saudi Arabia and the United Arab Emirates, depending on the diagnostic criteria applied **(Table 1)**.[7-9] Symptom-based prevalence estimates consistently exceed physician-diagnosed rates, reflecting underdiagnosis, limited access to dermatology specialists, and inconsistent diagnostic frameworks. Migration patterns and geopolitical instability add further complexity to epidemiologic interpretation, particularly in Gulf states where expatriate populations constitute the majority of residents.[9]

Clinical Presentation and Diagnostic Challenges

AD in MENA patients may present with features that exhibit subtle differences from classic descriptions based primarily on lighter skin phototypes. **(Figures 3, 4, and 5)** Erythema may be less apparent, presenting as violaceous or red-brown in patients with constitutively darker skin phototypes, leading to underestimation of disease severity. Features such as pronounced lichenification, follicular eczema, prurigo-like nodules, and extensive post-inflammatory hyperpigmentation may be more commonly

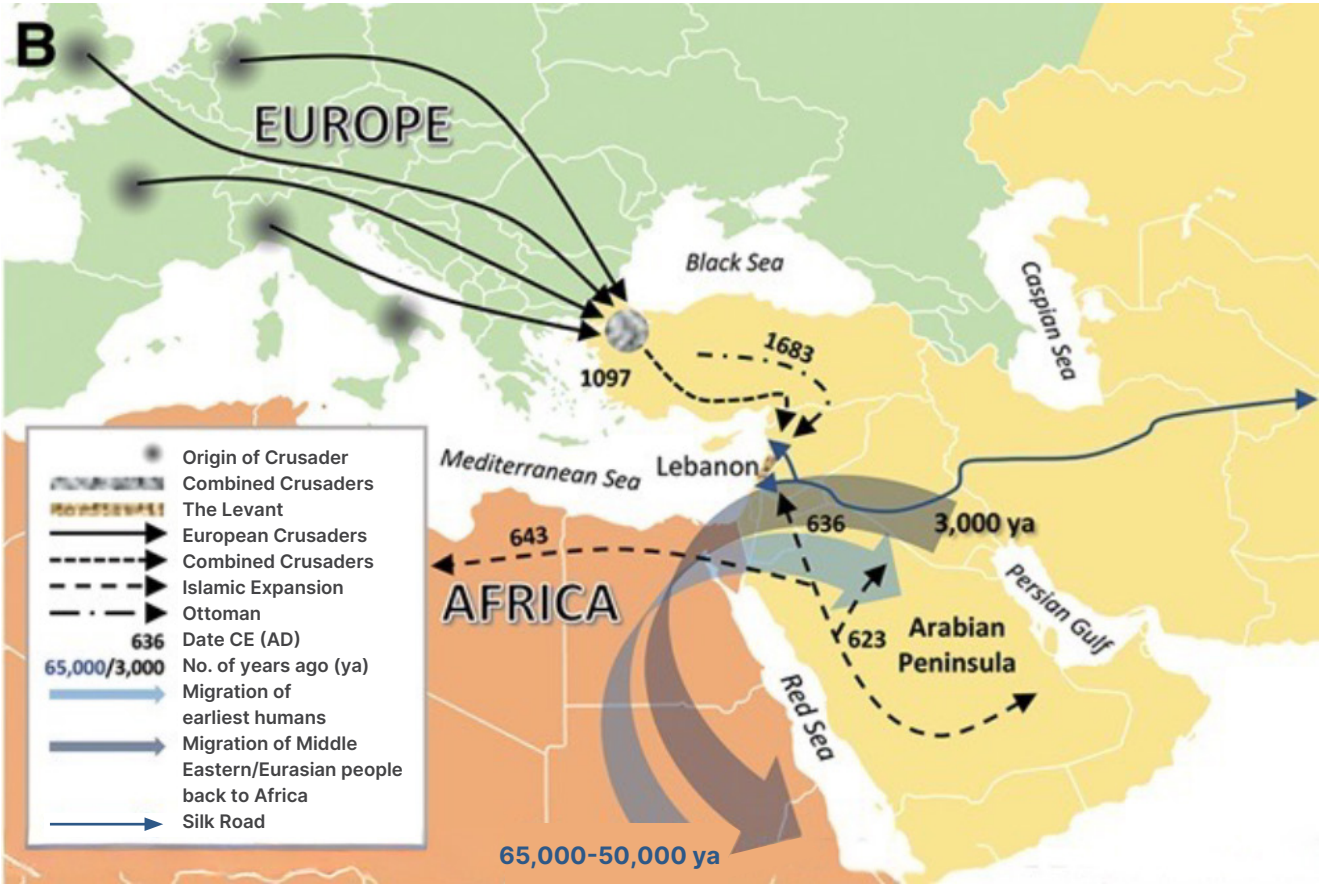


Figure 2. An overview of selected migration patterns that contributed to the ethnic origins of people in the Middle East; *adapted from Kashmar M, et al. Consensus Opinions on Facial Beauty and Implications for Aesthetic Treatment in Middle Eastern Women, 2019.*



Figure 3. Hot, humid weather leading to exacerbation of AD with follicular or eczematized miliaria phenotypes; *from Mahmoud O, et al. Burden of Disease and Unmet Needs in the Diagnosis and Management of Atopic Dermatitis in the Arabic Population of the Middle East. Journal of Clinical Medicine. 2023*
Figure 4. Vaccination is not mandatory in much of the region (incl Egypt) so presentations like eczema herpeticum are more common; *from Mahmoud O, et al. Burden of Disease and Unmet Needs in the Diagnosis and Management of Atopic Dermatitis in the Arabic Population of the Middle East. Journal of Clinical Medicine. 2023*
Figure 5. Patients who are more melanized, tend to have more extensor than flexor involvement and may have more profound itch; *from Mahmoud O, et al. Burden of Disease and Unmet Needs in the Diagnosis and Management of Atopic Dermatitis in the Arabic Population of the Middle East. Journal of Clinical Medicine. 2023*

observed. Palmar hyperlinearity and ichthyosis vulgaris may be less prominent, emphasizing the importance of objective scoring.[9,10] These considerations may influence topical treatment selection, emphasizing early anti-inflammatory therapy paired with barrier repair to prevent flares.

Post-inflammatory hyperpigmentation is a major driver of patient distress and perceived disease burden despite "objective" improvement in clinical outcome measures such as Eczema Area and Severity Index (EASI) or SCORing Atopic Dermatitis (SCORAD). Post-inflammatory hyperpigmentation often persists long after inflammatory activity has resolved. Secondary infection remains common, particularly in settings of delayed diagnosis, limited access to topical therapies, or environmental heat.[11,12]

Immunopathogenesis: Genetics, Environment and the Microbiome

The immunologic underpinnings of AD in MENA populations are generally aligned with global patterns, characterized by Th2-skewed inflammation, epidermal barrier dysfunction, and neuroimmune dysregulation. However, as region-specific modifiers are elucidated, their influence appears increasingly important.

In MENA patients, AD shows genetic heterogeneity that may shape phenotype and complicate diagnostic evaluation. Loss-of-function (LoF) variants in the filaggrin gene (*FLG*), the strongest known single-gene risk factor for AD, are common in northern European cohorts but are rare in Middle Eastern populations and absent in most African populations, according to a large systematic review and meta-analysis.[13] Consequently, in MENA patients, barrier impairment may more commonly reflect inflammation-mediated downregulation of *FLG* rather than inherited *FLG* LoF mutations. Additional variation in type 2 signalling pathways (eg, interleukin -4 receptor alpha [IL4RA] and signal transducer and activator of transcription 6 [STAT6] polymorphisms described in Egyptian

children) may also contribute to disease susceptibility.[9]

When *FLG* LoF variants are present, they are associated with earlier disease onset and more persistent clinical course, supporting a lower threshold for early, proactive barrier repair efforts, infection prevention, and anti-inflammatory control.[10,14] Genotype-informed counselling may also aid in explaining and managing fluctuations in xerosis and recurrent flares despite treatment adherence.[10] From a therapeutic standpoint, clinical data indicates that dupilumab improves skin barrier function irrespective of *FLG* genotype, suggesting that response to targeted biologic therapy is not contingent on *FLG* status.[15] Overall, integrating MENA-specific genetic context with careful visual assessment and objective severity scoring can improve diagnostic accuracy and optimize treat-to-target management.

Environmental factors likely play a prominent role on disease expression **(Table 2)**. High temperatures, low humidity, air pollution, dust exposure, and urban crowding exacerbate transepidermal water loss and promote cutaneous inflammation.[2,11] Microbiome studies conducted in Egypt demonstrate altered skin microbial diversity in patients with AD, with correlations between dysbiosis and elevated serum IgE levels, supporting environment–immune–microbiome interactions in disease expression.[16]

Country	Prevalence Range	Prevalence	Study	Study Information
United Arab Emirates (UAE)	5.5-39.1%	16.7% 39.1% 11.6% 5.5% 12.9% 14.6% 9% 12%	Silverberg et al. Silverberg et al. Maspero et al. Al Hammadi et al. Ibrahim et al. Ibrahim et al. Al Hammadi et al. Behbehani et al.	Diagnosed AD, large population study. Only ISAAC criteria, large population study. Large population study. Dubai patients covered by private insurance. 1,944 children aged 6-7 years. 1,793 children aged 13-14 years.
Saudi Arabia	12.5-45.4%	19.8% 37.1% 15.3% 45.4% 12.5% 43.5% 32.6% 31.9% 14%	Silverberg et al. Silverberg et al. Maspero et al. Alakeel et al. Al-Frayh et al. Al-Frayh et al. Al-Frayh et al. Al-Frayh et al. Nahhas et al.	Diagnosed AD, large population study. Only ISAAC criteria, large population study. Large population study. 854 Taif citizens. Overall prevalence of physician-diagnosed AD. City of Hofuf. City of Riyadh. City of Jeddah. Children aged 6-12 years.
Egypt	3.6-12.01%	3.6% 12.01%	Maspero et al. Al Dhduh et al.	Large population study. 308 students aged 11-14 years.
Kuwait	19.5%	19.5%	Ziyab et al.	3,775 adolescents aged 11-14 years.
Syria	3.9%	3.9%	Reda et al.[5]	ISAAC
Lebanon	11.5-11.8%	11.5% 11.8%	Hallit et al. Waked et al. Waked et al.	 5,544 children/adolescents aged 5-14 years. 3,909 children/adolescents aged 5-12 years.

Table 1. Epidemiology of atopic dermatitis in the Middle East and North Africa region; *adapted from Mahmoud O, et al. Burden of disease and unmet needs in the diagnosis and management of atopic dermatitis in the Arabic population of the Middle East, 2023*

Abbreviation: ISAAC: International Study of Asthma and Allergies in Childhood

Socio-Cultural Determinants of Disease Expression

Cultural practices have a significant influence on how AD presents and is managed in the MENA region. Clothing customs, including occlusive garments and head coverings, alter local skin microclimates. Studies examining the scalp microbiome of women wearing hijab demonstrate differences in the microbiome compared with uncovered scalps, with potential implications for inflammatory dermatoses.[17]

Traditional remedies and culturally-rooted topical preparations are commonly used, yet they may exacerbate dermatitis or delay patients from seeking medical care.[18] Additionally, stigma surrounding visible skin disease, particularly among women and adolescents, may further discourage timely help-seeking, highlighting the importance of culturally informed counselling.

Healthcare Resources, Geopolitical Context, and Access to Care

Healthcare infrastructure varies widely across the MENA region. While high-income Gulf states may offer access to biologics and other advanced systemic therapies, countries affected by conflict or constrained by limited resources often struggle to provide basic emollients and topical corticosteroids.[2,11] Dermatologist density remains low in many countries, and AD care is frequently delivered by non-specialists.

Global treatment guidelines, largely developed in high-income Western contexts, are often ill-suited to these environments. In response, international health organizations advocate for

Factor	Associated Effect on AD Risk
Urban versus rural dwelling	↑ risk for AD with urban dwelling
Socioeconomic status	↑ risk for allergic sensitization in kids with higher parental socioeconomic status/goods ownership
Education level of parents	↑ risk for allergic sensitization and AD in children with increasing parental education level
Climate	↑ risk for AD in colder climates; ↓ risk for AD with UV light exposure
Pollution	↑ risk for AD with exposure to pollution, maternal exposure to cigarette smoking in prenatal period
Family size	↑ risk for AD with smaller family size
Personal hygiene, sanitation	↑ risk for AD with better personal hygiene in early childhood; ↑ risk for allergic sensitization/AD in children with access to sanitation (modern toilets, piped drinking water)
Antibiotic use	↑ risk for AD with antibiotic exposure in prenatal period and during the first year of life; ↓ risk with antibiotic exposure after the first year of life
Breastfeeding	↓ risk for AD in infants with familial history of AD
Farm and animal exposure	↓ risk for AD with frequent prenatal exposure to farm animals; most protective when compounded with direct exposure; potential ↓ risk with postnatal exposure to furry pets, particularly dogs
Intestinal microflora	↓ risk for AD with greater diversity of gut microflora in infancy; ↓ risk for allergic sensitization/AD with colonization favoring lactobacilli and bifidobacteria in infancy or childhood

Table 2. Environmental risk factors for atopic dermatitis; *adapted from Al-Afif KAM, et al.. Understanding the burden of atopic dermatitis in Africa and the Middle East., 2019*

integrating common inflammatory skin diseases such as AD into primary care frameworks, including teledermatology and resource-stratified treatment algorithms.

Disease Burden and Quality-of-Life

AD imposes a substantial humanistic and economic burden on populations across the MENA region. Multi-country analyses demonstrate that adults and adolescents with AD lose an average of approximately 0.19 quality-adjusted life years annually, with indirect costs accounting for nearly two-thirds of total disease-related expenditures.[19] In several countries, AD-related costs represent up to 0.04–0.08% of national gross domestic product.[19]

Sleep disturbance, reduced school and work productivity, heightened anxiety and depression, and caregiver burden are consistently reported, often rivalling or exceeding those associated with other chronic inflammatory diseases.[12,19]

Patient Education Awareness and Misinformation

Surveys from Egypt and Lebanon identify substantial gaps in patient education, structured support programs, and access to digital health resources.[20] While social media platforms provide opportunities for engagement and peer support, they also facilitate the dissemination of misinformation, including the promotion of ineffective or harmful treatments. Addressing these gaps is critical for improving long-term outcomes.

Implications for Management and Future Directions

Optimizing AD management in MENA populations requires a multilevel approach that incorporates phenotype-specific assessment, cultural competence, and resource awareness. Priorities include earlier diagnosis in primary care, individualized education addressing chronicity and pigmentary sequelae, and improved inclusion of MENA populations in clinical trials. Expanded research into genetic, microbiome, and environmental modifiers of disease is essential.

Conclusion

AD in MENA populations exemplifies the intersection of immune dysregulation, environmental influences, cultural practices, and structural inequities. Dermatologists must look beyond simplistic ethnic classifications to recognize the diverse, context-dependent realities of these patients. Advancing equitable dermatologic care will require better representation of MENA populations in research, adapting guidelines to resource-constrained settings, and addressing socio-cultural barriers to dermatologic care.

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Dermatologic Manifestations of Perimenopause and Menopause: Structural, Functional, and Therapeutic Considerations

Malika Ladha, MD, FRCPC, FAAD

Introduction

Perimenopause and menopause represent major endocrine transitions across a female's life course. In Canada, over 10 million women are aged 40 years or older, meaning a substantial proportion of the population is within the perimenopausal, menopausal, or post-menopausal stages.[1] The average age of natural menopause in Canadian women is approximately 51 years, although the menopausal transition may begin several years earlier.[1]

Perimenopause is defined as the interval preceding menopause during which ovarian function becomes irregular, leading to fluctuating estrogen and progesterone levels, irregular menstrual cycles, and the onset of vasomotor and somatic symptoms.[2] Menopause is diagnosed retrospectively after 12 consecutive months of amenorrhea and reflects permanent cessation of ovarian follicular activity.[3] The hypoestrogenic state that follows menopause has systemic effects on multiple organ systems, including the cardiovascular system, skeletal system, and the skin.

Skin and hair are estrogen-responsive tissues, with estrogen receptors expressed in keratinocytes, fibroblasts, sebocytes, melanocytes, endothelial cells, and hair follicles.[4] As a result, declining estrogen levels during perimenopause and menopause contribute to structural, functional, and aesthetic changes. These changes may prompt consultation requests to Dermatology services and as such, our specialty should be prepared.

Cutaneous Changes

Cutaneous aging during perimenopause and menopause reflects the combined effects of intrinsic aging, cumulative environmental exposures, and estrogen deficiency. Declining estrogen levels accelerate changes across multiple anatomical layers of the skin **(Table 1)**.

Epidermis

Estrogen plays a critical role in maintaining epidermal thickness, keratinocyte proliferation, and barrier function. Hypoestrogenism is associated with reduced mitotic activity in basal keratinocytes, leading to epidermal thinning and impaired barrier integrity.[5,6] These changes contribute to increased transepidermal water loss, xerosis, and heightened skin sensitivity. In addition, reduced estrogen levels are associated with diminished synthesis of epidermal lipids and natural moisturizing factors, further compromising stratum corneum cohesion.[7] Clinically, these alterations manifest as dryness, pruritus, irritation, and skin sensitivity.

Dermis

The dermis is particularly sensitive to estrogen withdrawal. Dermal fibroblasts express estrogen receptors and respond to estrogen by increasing collagen synthesis and elastin

Layer	Change	Pathophysiology	Symptom
Epidermis	Xerosis	Reduced sebum and lipid production	Rough, itchy, flaky skin
	Increased sensitivity	Weakened skin barrier	Increased reactivity to topical products and environmental factors
	Slower cell turnover	Decreased keratinocyte proliferation	Dull complexion, delayed wound healing
	Pigmentary changes	Dysregulated melanocyte activity	Uneven tone
Dermis	Collagen loss	Reduced fibroblast activity	Sagging skin, fine and deeper rhytids
	Reduced elastin	Decreased synthesis and increased degradation	Loss of skin resilience and elasticity
	Thinner dermis	Loss of extracellular matrix proteins and reduced hydration	Fragile skin prone to injury
	Reduced vascularization	Decreased capillary density	Pallor, reduced nutrient delivery, slower wound healing
Hypodermis	Fat redistribution	Hormonal shifts	Loss of facial volume
	Volume loss	Decreased adipocyte activity in the face	Hollowing in the peri-ocular area, temples, and cheeks
	Impaired thermoregulation	Reduced sweat gland activity	Temperature sensitivity, increased dryness
Muscle	Muscle atrophy	Age related sarcopenia, worsened by hormonal decline	Loss of facial tone and firmness
	Decreased muscle tone	Reduced neuromuscular stimulation	Drooping of facial features, especially around jawline and eyes
Bone	Bone resorption	Decreased bone density of the maxillary and mandibular bone	Flattening of cheeks, receding jawline, deepening nasolabial fold

Table 1. Cutaneous changes in menopause; *courtesy of Malika Ladha, MD, FRCPC, FAAD*

maintenance.[4] Following menopause, dermal collagen content declines rapidly, with studies demonstrating a reduction of up to 30% within the first 5 years.[8-10] Loss of collagen types I and III, degradation of elastic fibers, and reduced hyaluronic acid result in decreased tensile strength, elasticity, and hydration. Clinically, these changes present as fine lines, wrinkles, and laxity.[11]

Fat, Muscle, and Bone

Estrogen influences adipocyte distribution and subcutaneous fat volume. Menopause is associated with redistribution and loss of subcutaneous fat, particularly in the face, contributing to volume depletion and accentuation of skeletal contours.[12] Although muscle and bone changes are not cutaneous, loss of facial bone density and muscle tone further exacerbate soft tissue descent and alters facial architecture, further compounding perceived skin aging.

Hair Changes

Hair follicles are sensitive to hormone changes. Estrogen prolongs the anagen (growth) phase of the hair cycle, while estrogen withdrawal shortens anagen duration and increases the proportion of follicles in telogen.[13] During menopause, there is relative androgen excess due to declining estrogen levels, rather than increased androgen production, which can lead to androgen-sensitive hair changes. In addition, reduced estrogen levels are associated with decreased sebaceous gland activity, leading to a drier scalp and hair shaft. Hair may become finer, more brittle, and less pigmented due to reduced melanocyte activity within the hair follicle.[18,19]

Hair Loss

The most common clinical presentation is female pattern hair loss (FPHL), characterized by diffuse thinning over the crown and widening of the midline part. In genetically predisposed women, the relative increase in androgens promotes follicular miniaturization.[14-16] Some may experience telogen effluvium, particularly during perimenopause, driven by hormonal fluctuations rather than sustained hypoestrogenism. Frontal fibrosing alopecia disproportionately affects post-menopausal females, potentially reflecting interactions between hormonal, immune, and environmental factors.[17]

Hirsutism

Hirsutism, defined as excessive terminal hair growth in androgen-sensitive areas (e.g.,

Treatment Route	Medication	Considerations
Topical	Minoxidil 2% or 5%; **preference for 5%**	Can cause temporary paradoxical shedding Toxicity to pets
Systemic	Dihydrotestosterone (DHT) blockers (finasteride 2.5 mg po OD; dutasteride 0.5 mg po OD)	Concurrent contraception uses for perimenopausal women
	Spironolactone	Potassium monitoring in patients over 45 years
	Minoxidil (start with 0.625 mg po OD; increase to 2.5 mg po OD as tolerated).	Caution in patients with cardiac history
Procedural	Platelet-rich plasma	Further research for standardization and outcomes
	Hair transplant	Requires adequate hair density at donor site
At-home	Low-level laser helmet	Further research for standardization and outcomes
	Wigs, camouflage	

Table 2. Treatment Options for Female Pattern Hair Loss (FPHL); *adapted from Kamp E, et al. Menopause, skin and common dermatoses. Part 1: hair disorders, 2022*

upper lip, chin, chest), can develop or worsen during perimenopause and menopause.[18-20] The relative androgen excess leads to increased hair follicle sensitivity. Management strategies include topical eflornithine, laser-based hair reduction (if the hair colour has not lightened), and systemic antiandrogens, such as spironolactone or finasteride.[19,21,22]

Genitourinary Syndrome of Menopause

Genitourinary syndrome of menopause (GSM) encompasses vulvovaginal, urethral, and peri-anal changes related to estrogen deficiency during perimenopause and menopause.[23] The syndrome is characterized by vulvovaginal atrophy, dryness, irritation, pruritus, burning, dyspareunia, urinary urgency, and recurrent urinary tract infections, which reflect the hypoestrogenic impact on the skin and mucosal tissues.[24]

Dermatologists may encounter GSM through presentations such as pruritus, irritation, erythema, or lichenification of the vulvar or peri-anal skin. GSM can also exacerbate dermatologic conditions such as eczema, lichen simplex chronicus, lichen sclerosus, or candidiasis, highlighting the importance of recognizing the underlying hormonal etiology.[23]

Early recognition of GSM is critical for preserving quality of life and minimizing sequelae, such as sepsis secondary to recurrent urinary tract infections. Our colleagues in Family Medicine or Gynecology will manage local and systemic hormonal treatments.[24] Dermatologists can offer guidance on general genital care, including, but not limited to, washing with water only, avoiding pH-altering soaps, use of moisturizers and lubricants, use of bleach-free toilet paper, and avoidance of irritating pads.

Treatment Considerations

Management of menopausal skin and hair changes requires a multimodal, individualized approach. It will also necessitate collaboration with our Family Medicine and Gynecology colleagues who are trained to prescribe systemic hormone replacement therapies.

Hormone Therapies

Systemic hormone therapy has been shown to increase skin thickness, collagen content, hydration, and elasticity when initiated near menopause onset.[20,21,25] However, its use must be based on a patient-specific risk-benefit assessment. Topical estrogen applied to non-facial skin has demonstrated cutaneous benefits, though facial use remains controversial due to limited formulations and supporting data. In addition, it may also trigger or worsen other dermatological conditions, such as rosacea or melasma.

Topical Therapies

Topical retinoids improve epidermal turnover and stimulate dermal collagen synthesis. However, topical retinoid therapy may require alterations in application to minimize dryness and irritation, which can be further compounded by decreased estrogen levels in the skin. Barrier repair with ceramides, humectants, and occlusives is essential for managing xerosis, and photoprotection remains foundational. Energy-based devices, biostimulatory injectables, and hyaluronic acid filler gels may address skin laxity and volume loss.

Hair Therapies

Topical minoxidil (5% formulation) remains first-line therapy for FPHL. Antiandrogen therapies may be considered in select patients. Low-level laser helmet therapy and platelet-rich plasma have shown promise, though require further high-quality evidence **(Table 2)**.[16]

Future Directions

Despite strong biological plausibility, the role of topical estrogen for managing facial skin aging remains incompletely defined. Future research priorities include pharmacokinetic studies evaluating systemic absorption, randomized controlled trials assessing long-term safety and efficacy, and exploration of estrogen receptor-selective compounds that preferentially activate cutaneous pathways. Development of standardized formulations and interdisciplinary clinical guidelines will be critical to the safe integration of hormonal strategies into dermatologic practice.

Conclusion

Perimenopause and menopause are associated with profound changes in skin, hair, and genitourinary tissues, driven by estrogen deficiency and relative increases in androgen activity. These hormonal changes affect multiple anatomical layers and significantly influence the health and function of the skin and hair. Dermatologists are uniquely positioned to recognize these patterns, counsel patients, and implement evidence-based therapies. Ongoing research is essential to optimize management strategies and clarify the role of hormonal and non-hormonal interventions in this patient population.

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From Dressing to Scar Maturation: A Practical Guide to Post-Procedure Care in Dermatologic Surgery

Jorge R. Georgakopoulos, MD, FRCPC

At-A-Glance Principles

- Prioritize a clean, moist, protected wound environment (e.g., petrolatum with a non-adherent dressing) to reduce crusting and support re-epithelialization.
- Avoid routine use of topical antibiotics on clean dermatologic wounds; randomized studies show similar infection rates versus petrolatum, with a higher risk of allergic contact dermatitis in antibiotic-treated groups.[1]
- Minimize wound tension early (first 6–12 weeks) through activity modification, appropriate timing of suture removal, and consideration of prolonged taping/Steri-strips at high-tension sites to reduce scar widening and hypertrophy.[2,3]
- Start silicone therapy (gel or sheets) once epidermal integrity is restored, which often occurs 2 weeks after suture removal, and consider early use in patients at higher risk for hypertrophic or keloid scarring.[4,5]
- Treat the 'inflammatory' scar early, as persistent erythema/telangiectasia and early hypertrophy often respond better to early intervention (e.g., vascular laser, intralesional corticosteroid) than that for late-stage scars.[6]

Timeframe	Goals	What to do	Escalate if…
Day 0–2	Achieve hemostasis; protect the repair; pain/swelling control	• Set expectations with patients. • Maintain the pressure dressing for 48 hours. • Elevate the area for swelling control. • Over-the-counter pain control (acetaminophen ± NSAID). • Avoid stretching, exercise, and heavy lifting.	• Bleeding is not controlled despite 20 minutes of firm pressure; rapidly expanding swelling/ hematoma develops; or pain is severe or disproportionate. • Infection is unlikely within the first 48 hours.
Day 2–7	Maintain a clean and moist environment; monitor for early issues	• Perform daily wound care: gentle cleansing (saline or water), pat dry, apply a layer of petrolatum, and cover with a non-adherent dressing. Change daily or when saturated. • Showering is usually permitted after 48 hours but avoid direct water pressure and soap. • Avoid stretching, exercise, and heavy lifting.	• This period carries the highest risk for infection. Escalate for increasing pain or warmth, purulence, fever, spreading erythema, malodor, or wound edge separation.
Week 1–6	Tension control; initiate early scar optimization	• Remove sutures at an appropriate timeline based on skin tension (see Section 2.4). • Continue petrolatum until fully epithelialized. Consider paper tape/Steri-strips across linear scars to offload tension (often 6–12 weeks on high-tension sites). • Start silicone gel or sheets once the epidermis is intact. • Begin gentle scar massage when fully epithelialized (often 2–4 weeks after suture removal). • Emphasize sun protection (keeping scars covered with a bandage when in direct sunlight). • Re-introduce stretching, exercise, and heavy lifting slowly after suture removal.	• Early intervention for scar management is warranted for raised, firm, or pruritic scars developing early, persistent erosions, exuberant granulation, or functional limitation.
Month 2–12	Scar remodelling (colour, thickness, pliability); monitor for tumour recurrence	• Maintain sun protection (SPF 50+) with re-application every few hours. • Continue silicone therapy for at least 12 weeks for ≥12 hours per day. • Consider vascular laser for persistent erythema/ telangiectasia. • Use intralesional corticosteroids ± 5-FU for hypertrophic scars.	• Late intervention for scar management is warranted for progressive thickening/ contracture, significant symptoms, patient distress, or concern for tumour recurrence at the scar site.

Table 1. Postoperative care timeline; *courtesy of Jorge R. Georgakopoulos, MD, FRCPC*

Abbreviations: FU: fluorouracil; **NSAID:** nonsteroidal anti-inflammatory drug

Section 1. Procedure-Specific Postoperative Care

Across dermatologic surgery, most outcomes are driven by a few modifiable variables: hemostasis, moisture balance, tension, and early inflammation control. The following notes offer a pragmatic framework for tailoring care without overcomplication.

1.1 Linear Closures (Excision, Elliptical Biopsy, Layered Repair, Mohs Surgery)

Pressure dressings are most useful for repairs with dead space, in patients taking antithrombotic medications, and at highly vascular sites such as the scalp and face. Patients should be counselled to restrict activity and avoid stretching across the repair for at least 1–2 weeks, with longer periods recommended for high-tension sites such as the back, shoulders, and joints. Sutures should be kept clean and lightly lubricated with a thin layer of petrolatum. After suture removal, consider prolonged support with paper tape/Steri-strips at high-tension sites to reduce the risk of scar widening.[2,3]

1.2 Punch Biopsies

Closed punch sites should be treated similarly to small linear closures, with daily application of petrolatum plus non-adherent dressing; consider pressure dressings for sites on the scalp or face. If punch biopsy sites are left open (selected scalp or small truncal sites), wound care should be managed as secondary intention healing **(see Section 1.4)**.

1.3 Shave Biopsies

For shave biopsies, the goal of postoperative care is rapid re-epithelialization with a moist, non-traumatized wound bed. Daily care should consist of gentle cleansing followed by application of petrolatum and coverage with a non-adherent dressing. Patients should be advised to avoid picking at crusts, as crust formation is a marker of excess dryness/trauma. The risk of bleeding is greatest in the first 24 hours; therefore, pressure instructions should be reinforced, and aspirin/nonsteroidal anti-inflammatory drugs (NSAIDs) should be avoided if clinically appropriate. For larger shave biopsies or sites exposed to friction, consider hydrocolloid/foam dressings to reduce adherence/trauma.

1.4 Electrodessication and Curettage (ED&C) and Secondary-intention Wounds Including Shave Excisions and Post-Mohs Surgery

Secondary intention can provide acceptable cosmetic and functional outcomes for select defects when patients are counselled and wound care is optimized.[7]

During the Initial 24–48 hours, maintain a pressure dressing and bleeding protocol should be reinforced, emphasizing the importance of firm uninterrupted pressure. After 24–48 hours, daily wound care should include cleansing followed by application of petrolatum and coverage with a non-adherent dressing. For highly exudative wounds, consider absorbent dressings (foam/alginate) with petrolatum applied to the wound edges.

Patients should be counselled to expect a longer healing curve (often several weeks, depending on wound size and site), and advised about the likelihood of temporary erythema and contour changes. Troubleshoot common issues, such as hypergranulation tissue, (which may be managed with silver nitrate or topical corticosteroids to exuberant tissue), maceration (by reducing occlusion/exudate), and pain, which should prompt assessment for infection or overly dry dressing.

Section 2. Phase-based Care

2.1 Bandaging and Dressings: Choosing the Right Level of Occlusion

A simple dressing strategy prevents most downstream issues by aiming for a wound environment that is moist but not macerated. Contemporary wound-healing principles emphasize that “Skin cells swim, they don’t crawl.” Older theories about drying out wounds lead to excess crusting and should be avoided.

2.2 Immediate Postoperative Period (0–48 hours)

Patients should be instructed on bleeding protocol: remove the outer wrap if it becomes saturated, apply firm, continuous pressure with clean gauze for 20 minutes (no peeking). Repeat once if needed; seek medical attention if bleeding is persistent. Pain and swelling can be managed with elevation and/or compression. Acetaminophen is preferred for pain, with NSAIDs used only when appropriate. Keep dressing dry, and patients should avoid soaking, hot tubs, and heavy exertion during this period.

2.3 Days 2–7: Cleanse, Moisturize, and Protect

Daily cleansing of the wound with saline water, then pat dry, avoid rubbing. Apply a thin layer of petrolatum and cover with a non-adherent dressing that is changed daily or sooner if saturated. Showering is usually acceptable after 48 hours; however, patients should avoid direct water pressure, soaps applied to the site, and soaking in baths until the wound is epithelialized. Patients should be counselled on expected normal findings, including mild oozing/serous crusting, localized tenderness, and mild erythema at the wound edges.

2.4 Suture Removal Timing

Adjust based on tension, location, patient factors (age, smoking, steroids), and closure type.

Face/neck	5–7 days
Trunk	12–14 days
Arms/legs	12–14 days
Back/shoulders (high tension)	14 days
Scalp	12–21 days
Hands/feet over joints (consider staged removal with taping)	14–21 days

2.5 Weeks 1–6: Tension Management and Early Scar Care

Once epidermal integrity is restored, start silicone gel or sheeting **(see Section 3)**. To further reduce tension across linear scars, especially those on the back, shoulders, or chest, consider applying paper tape; many clinicians continue taping for 6–12 weeks.[2,3] Begin scar massage once the wound is fully healed, typically 2–4 weeks after suture removal, using light pressure initially (see Section 3).[8,9] Sun avoidance is essential during this period to reduce the risk of post-inflammatory dyspigmentation. Scars should be protected with bandaging during sun exposure for the first 4 weeks, with sunscreen (SPF 50+) use thereafter.

Dressing	Best for	How to use	Pitfalls / pearls
Pressure dressing (gauze with tape)	First 24–48 hours for vascular sites, dead space, and for patients on anticoagulation.	Apply firm compression, keep dry, and remove per instructions.	Too tight can cause pain/ischemia, replace if dressing becomes saturated.
Non-adherent contact layer (e.g., Telfa, Adaptic) or a thick petrolatum layer with gauze and tape (lower cost)	Most wounds after day 1–2.	Apply petrolatum to the wound, then a non-adherent layer with gauze.	If adherence occurs increase petrolatum or switch to a silicone contact layer.
Hydrocolloid	Shave biopsies/secondary intention wounds with low-moderate exudate; areas of friction.	Leave 2–5 days if intact; change if leaking/odour develops.	Over-occlusion or heavy exudate may cause maceration; counsel patients regarding expected gel-like residue.
Foam / alginate	Secondary intention wounds with moderate to high exudate.	Use an absorbent layer; protect the periwound skin with a petrolatum or barrier ointment.	May adhere to dry wounds; reassess exudate frequently.

Table 2. Simplified guide to common dressing options; *courtesy of Jorge R. Georgakopoulos, MD, FRCPC*

2.6 Months 2–12: Remodelling and Targeted Interventions

Continue silicone gel for at least 12 weeks **(see Section 3)**.[4,5] For scars with persistent erythema/telangiectasia, consider vascular laser therapy, such as pulsed dye laser (PDL), once initial healing is complete; randomized trials support early PDL for improving scar appearance.[6] For early hypertrophic change characterized by a firm, raised or pruritic scar, consider using intralesional triamcinolone ± 5-fluorouracil (FU), alongside continued use of taping and silicone therapy.

Section 3. Evidence-informed Scar Management Toolbox

Silicone gel and silicone sheeting represent the best supported topical option for hypertrophic/keloid prevention and treatment. Start therapy following re-epithelialization, typically 2 weeks after suture removal. Silicone gel may be applied as a thin film twice daily or as silicone sheets (ideally ≥12 hours per day). Treatment should continue for at least 12 weeks and may be extended for high-risk scars or until the patient is satisfied, in some cases up to 1 year.

The evidence base supporting silicone therapy is robust. A Cochrane review including 20 trials and 873 participants demonstrated reduced hypertrophic scarring in prevention studies, and improvements in scar thickness and colour in treatment studies.[4] Similarly, a systematic review of over-the-counter scar products identified silicone gel and sheets as the only therapy supported by multiple higher-quality randomized controlled trials; evidence for alternatives such as onion extract, vitamin E, and trolamine was weak or inconsistent.[5]

Patient counselling is essential for managing expectations. Improvements with silicone therapy are gradual and are most pronounced for scar thickness, pliability, and erythema.

3.2 PaperTape or Steri-strips for Tension Offloading

These are applied after suture removal or once the wound has sealed, particularly for incisions located in high-tension areas or those crossing relaxed skin tension lines. The evidence base is robust for using paper tape. Randomized trials have demonstrated reduced hypertrophic scar formation and stretched scars,[2] and reviews of non-stretch taping techniques have reported improvements in height, colour, and pruritis.[3] To apply, tape should be placed along the scar line with gentle approximation of the wound edges and changed every 3–7 days or as needed. In high-tension regions, continue for 6–12 weeks.

3.3 Scar Massage

Scar massage can be started once the wound is fully epithelialized and non-tender (often 2–4 weeks post-suture removal). Massage should be performed for 1–2 minutes, 2–3 times/day using moisturizer. Patients should start gently and increase pressure as tolerated. While the strongest evidence for scar massage comes from the burn/hypertrophic scar literature,[8,9] clinical experience supports its subjective benefits.

3.4 Photoprotection and Dyspigmentation

To minimize irritation, cover the scar line with bandaging for the first 4 weeks, to avoid irritation with sunscreen use. After 4 weeks, patients should use daily broad-spectrum sunscreen with SPF 50 or higher, along with physical sun protection (hat/clothing) for 6–12 months to reduce the risk of post-inflammatory hyperpigmentation and persistent erythema. If hyperpigmentation persists after complete healing, consider topical depigmenting agents (e.g., azelaic acid, hydroquinone, when appropriate). When erythema/telangiectasia is present, treat the underlying vascular component, with options including PDL.

3.5 Hypertrophic Scars and Keloids: Early Escalation

Early identification of high-risk patients, including those with prior hypertrophic/keloid scars, darker skin phototypes, wounds located at high-tension sites (sternum, shoulders, back), and wounds crossing tension lines. First-line management consists of silicone therapy combined with tension offloading (tape) along with symptom control using emollients and antipruritics. For scars demonstrating early progression, treatment can be escalated to intralesional triamcinolone (e.g., 2.5–10 mg/mL for facial

sites and 10–40 mg/mL for truncal sites) ± 5-FU. Consider early referral for laser/fractional resurfacing or combination regimens.

Conclusions

This article provides a quick-reference guide for dermatologists and other clinicians performing dermatologic surgery, outlining stepwise post-procedure wound care, scar optimization, and complication avoidance. The included timelines, tables, and figures are designed to support efficient point-of-care review and can be used to refine standardized clinic post-care instruction sheets for patients. A patient centred flow diagram summarizing postoperative care and scar management timelines is presented in **Table 1**.



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Beyond Depigmentation: Understanding the Psychosocial Burden of Vitiligo in a Canadian Context

Vijay Sandhu, MD, FRCPC, DABD

Introduction

Vitiligo is a common cutaneous autoimmune disorder presenting with depigmented patches of skin. It is often described in textbooks as a cosmetic disease, with disease manifestations confined to the skin. However, for many patients living with vitiligo, this characterization could not be further from the truth.

In recent years, the approval of ruxolitinib cream (Opzelura), a topical Janus kinase (JAK)1/2 inhibitor, has renewed clinical and research interest in vitiligo. Prior to this advancement, treatment options for the disease had remained limited. Coupled with this shift in management, emerging evidence highlights that the impact of vitiligo goes well beyond the skin. As new therapies emerge, a nuanced understanding of the disease's psychosocial impact is paramount to informing dermatologic care, guiding equitable access to treatment, and shaping dermatologic policy within the Canadian context.

A Misunderstood Disease

Vitiligo affects approximately 1–2% of the world's population.[1] Historical reports of vitiligo date back more than 3,500 years.[2] However, despite its long-recognized existence, vitiligo remains misunderstood in some parts of the world, and is still mistaken for communicable diseases such as leprosy.[3] Studies examining beliefs about vitiligo consistently identify common misconceptions, including the belief that vitiligo is contagious, results from a lack of hygiene, or is caused by external superstitious forces (e.g., the "evil eye", or sorcery). In some countries,

women with this disease can be prohibited from marriage, speaking to the substantial stigma that can surround this disease.[3] In a country as culturally diverse as Canada, understanding how vitiligo is perceived across different communities, and the resulting social implications, is essential to delivering culturally competent dermatologic care.

A Highly Visible Disease

The presentation of vitiligo can be variable; however, two main patterns exist: segmental and non-segmental vitiligo.[2] Segmental vitiligo typically presents earlier in life, most commonly involving the face, and is often resistant to conventional therapies. In contrast, non-segmental vitiligo can manifest in both localized and generalized patterns. One notable variant, acrofacial vitiligo, predominantly affects highly visible and socially significant areas such as the hands and face. Disease involvement at these highly visible and socially significant sites further contributes to the burden of vitiligo. Facial involvement carries disproportionate social consequences, as the face is central to identity, communication, and social interaction. Multiple studies have shown that highly visible lesions are associated with a greater psychosocial burden and impaired quality-of-life.[4] The development of the Facial Vitiligo Area Scoring Index (F-VASI) reflects the growing recognition of the importance of facial vitiligo as an important entity distinct from vitiligo affecting other body sites.

Not a One Size Fits All Disease

The psychosocial burden of vitiligo is not experienced uniformly; rather, it is intricately intertwined with existing cultural and social disparities. Several studies have consistently shown that individuals with higher Fitzpatrick skin types experience a disproportionately greater psychosocial impact from vitiligo.[5] In individuals with more richly pigmented skin, disease visibility is accentuated further by the sharp contrast between depigmented lesions and surrounding skin, making lesions more visible and often more socially salient. This heightened visibility may contribute to the increased psychosocial burden reported by those with higher Fitzpatrick skin types.[4]

In addition to the increased visibility of disease, pre-existing cultural stigmatization of vitiligo can further intensify the stigma of the disease. As reviewed above, many communities share beliefs about vitiligo that contribute to social and marital exclusion.[3] For individuals with skin of colour, who may already face systemic social inequities, the added visibility of vitiligo can increase feelings of marginalization.

Stigmatization in Vitiligo

Stigma refers to social implications and discrimination that arise due to negative beliefs associated with a disease.[6] Stigmatization is an important driver of the psychosocial impact experienced by individuals with vitiligo. In one study, 90% of patients reported being questioned about their disease or being subjected to unwanted approaches by strangers due to their disease.[7] Although patients themselves understood that their disease is not contagious, this mistaken belief was held by friends and family, reinforcing social isolation.[3] In some cultural contexts, vitiligo is perceived as a “serious disease” due to its potential impacts on marriage prospects or employment opportunities.[3]

Qualitative research among South Asian women with vitiligo living in Britain further illustrates how existing cultural norms can shape lived experience.[8] In this study, all participants reported experiencing intrusive reactions and, in some cases, overt discrimination. Notably, some women with extensive depigmentation reported increased social acceptance when they were perceived as being of European descent, reflecting ongoing colourism and the sociocultural value placed on lighter skin in some communities. Conversely, other participants reported a loss of ethnic identity following complete depigmentation. These varied experiences to extensive depigmentation highlight the complexity of factors at play for patients living with severe disease or undergoing iatrogenic depigmentation. Such findings highlight the importance of dermatologists carefully assessing the possible motivations and

potential mental health outcomes for patients requesting depigmentation therapies.

Importantly, stigmatization is not limited to patients with darker skin types. In a separate study of White participants living with vitiligo, similar challenges in feeling "different" and stigmatization were reported, highlighting that these challenges span the spectrum of skin tones.[9]

In another study, those with higher levels of perceived stigmatization were independently associated with lower subjective well-being, reinforcing the central role of social stigma in shaping mental health outcomes.[10] Over time, the external stigma may become internalized, leading individuals anticipate negative judgment and alter their behaviour accordingly. This internalization can manifest as avoidance of intimate relationships, social withdrawal, and occupational limitations.

The Mental Health Burden of Vitiligo

Studies have shown an association between vitiligo and adverse mental health outcomes, such as anxiety and depression. Meta-analyses have demonstrated that the odds of developing depression are 4.96–5.05 times higher in those living with vitiligo compared to those without the condition.[11] Clinical depression rates have been reported as high as 25%,[11] with similar proportions reporting suicidal ideation.[12]

Anxiety disorders are likewise more prevalent among those with vitiligo, with meta-analyses demonstrating rates of anxiety up to 6.14 times higher than those observed in control populations.[13] Additional psychiatric disorders commonly reported in vitiligo populations include adjustment disorder, agoraphobia, and social anxiety.[14] In a systemic review, psychosocial comorbidities were more prevalent in vitiligo compared to acne, alopecia areata, atopic dermatitis, and urticaria.[4] Moreover, when compared to non-dermatologic disease, the mental health burden of vitiligo has been reported to be comparable to diseases such as chronic lung disease, arthritis, cancer, and congestive heart failure.[15]

Not surprisingly, disparities in mental health burden are also evident among those with vitiligo. Higher rates of depression and anxiety have been reported among individuals from non-Caucasian backgrounds, particularly those with darker skin types and more visible disease.[5]

The global VALIANT study, which included 3,541 individuals with vitiligo, further reinforces the impact of vitiligo on mental health.[5] In this study, 58.7% of participants reported anxiety and depression. Screening with the Patient Health Questionnaire (PHQ-9), a commonly used tool to screen and measure depression severity, revealed that 55% of participants had moderate to severe depressive symptoms. Higher prevalence rates were observed among participants in India, those with darker skin types, facial and hand involvement, and in those with body surface area involvement greater than 5%.

Additional factors associated with decreased quality-of-life included young age (particularly age less than 30 years), unmarried/single relationship status and longer disease duration.[4] Collectively, these findings reinforce that although vitiligo is medically benign, its psychological burden can rival that of serious systemic illnesses.

Impact on Daily Functioning and Employment

The consequences of stigmatization and psychiatric comorbidity extend into functional domains, including employment and career advancement. In one study of adults with vitiligo, employed participants reported a mean work impairment of 35.6%.[16] Facial involvement was associated with a greater impact on work-related decisions, further highlighting the burden of visible disease.

Data from the VALIANT study further highlighted the impact of vitiligo on employment, with 41.9% of respondents reporting that they believed they would have progressed further in their careers if they did not have vitiligo.[5] A study from Brazil described workplace exclusion and reduced job opportunities among individuals with vitiligo,[17] illustrating how visible skin disease may function as a social determinant of health.

Beyond employment, vitiligo frequently affects daily activities. In the VALIANT study, between 42.9% and 55.2% of participants reported that vitiligo affected their everyday lives.[5]

These effects included altered clothing choices, reluctance to shake hands, challenges with intimacy, and avoidance of social gatherings.

Gaps in Treatment and Resultant Coping Strategies

Despite the challenges faced by those living with vitiligo, treatment options have historically been limited until relatively recently. In a 2020 study, 94% of participants reported a need for new treatment options, and 49% of patients felt current therapies to be ineffective.[18] These findings show substantial unmet treatment expectations among patients.

Moreover, a considerable proportion of patients with vitiligo do not receive ongoing care. One study showed that within the first year after diagnosis, 60.8% of newly diagnosed patients did not receive vitiligo-related treatment.[19] This proportion increased to 82% in the subsequent year, suggesting progressive disengagement from active treatment. Whether this reflects a lack of treatment options, decreased patient confidence in treatment options, inadequate access to ongoing care, or insufficient follow-up remains unclear. Regardless, these findings demonstrate that many individuals with vitiligo navigate their disease without ongoing medical follow-up.

In the absence of consistently effective or accessible therapies, many individuals with vitiligo adopt coping strategies. Common approaches include concealing affected areas with clothing, cosmetic camouflage, altered body positioning, and avoidance of certain situations.[7] While these strategies may provide short-term psychosocial relief, they may also reinforce internalized stigma and social withdrawal in those with vitiligo.

Collectively, these findings illustrate a critical point: when treatments options are perceived as inaccessible or inadequate, the burden of disease shifts from medical management to social adaptation. Therefore, addressing vitiligo requires dermatologists to not only expand and optimize our therapeutical arsenal, but also to promote sustained patient engagement and acknowledge the psychosocial dimensions of care inherent to this disease.

Tools to Score Vitiligo Severity: Measuring What Matters

The largely "asymptomatic" nature of vitiligo presents challenges in accurately quantifying its impact on quality-of-life. The Dermatology Quality of Life Index (DLQI) is a commonly used tool to ascertain quality-of-life data in dermatologic diseases. However, it includes items related to physical symptoms such as pruritis, which are not relevant in vitiligo. Using such questionnaires may underestimate the psychosocial burden of vitiligo in comparison to other diseases such as eczema or psoriasis. The development of vitiligo-specific scales can help to bridge this gap.[20] These tools include the Vitiligo Impact Scale (VIS)−22 and the Vitiligo Life Quality Index (VLQI). More recently, the Vitiligo Impact Patient Scale (VIPs) was developed to quantify vitiligo impairment according to skin phototypes, acknowledging that disease visibility and social impact may vary according to pigmentation.

Conclusion

Although vitiligo is often labelled as an "asymptomatic" condition, its impact is far from benign. Across diverse cultural contexts and skin types, the condition is associated with significant stigma, mental health burden, and impairments in daily life and employment. For dermatologists, providing comprehensive care must therefore extend beyond achieving repigmentation. Meaningful vitiligo management requires recognition of cultural context, assessing psychosocial impact, and proactively addressing mental health.

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