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

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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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