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Climate-Driven Xerosis: How Aridification is Reshaping Skin Barrier Function in Children and the Elderly

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Abstract

Background: *Xerosis, or dry skin, is a widespread dermatologic condition whose burden is intensifying with global aridification. Vulnerable populations, particularly children and the elderly, are disproportionately affected due to age-related impairments in skin barrier function.*

Objective: *To synthesize emerging evidence on how global climate-driven aridity disrupts skin physiology and contributes to xerosis across pediatric and geriatric populations.*

Methods: *A literature review was conducted using peer-reviewed articles published between 2011 and 2025, identified through PubMed and Scopus. Key search terms included "xerosis," "dry skin," "skin barrier," "transepidermal water loss," "arid climate," "low humidity," "pediatric skin," "infant skin barrier," "geriatric dermatology," "elderly skin," "ceramides," "climate change," and "environmental skin exposure."*

Results: *Climate stressors such as low humidity, extreme temperatures, wind exposure, and airborne pollutants were consistently associated with increased transepidermal water loss, disrupted lipid organization, and epidermal inflammation. Pediatric skin was uniquely susceptible due to a thin stratum corneum, underdeveloped lipid lamellae, and high surface-area-to-volume ratios. Geriatric skin exhibited reduced ceramide synthesis, elevated surface pH, and impaired desquamation. Institutional and indoor arid environments further worsened skin dryness. Multiple studies demonstrated that physiologic emollients (e.g., ceramide-dominant, urea-based) significantly improved hydration and barrier integrity, particularly in climate-vulnerable patients.*

Conclusion: *Xerosis is an underrecognized climate-sensitive dermatologic condition. Age-specific barrier vulnerabilities demand proactive, climate-adapted prevention and treatment strategies. Integrating dermatologic care with environmental health awareness is essential to reducing morbidity from xerosis in both pediatric and elderly populations.*

Keywords: Xerosis, Climate Change, Aridification, Pediatric Dermatology, Geriatric Skin, Environmental Dermatology

List of Abbreviations

- **AD**: Atopic Dermatitis
- **CCE:** Cornified Cell Envelope
- **FLG:** Filaggrin
- **IL:** Interleukin
- **NMFs:** Natural Moisturizing Factors
- **PM2.5:** Fine Particulate Matter (≤2.5 microns)
- **SC:** Stratum Corneum

- **SOC:** Skin of Color
- **SPEI:** Standardized Precipitation-Evapotranspiration Index
- **TEWL:** Transepidermal Water Loss
- **TRP:** Transient Receptor Potential
- **UV:** Ultraviolet

Introduction

Xerosis, commonly known as dry skin, is a prevalent dermatologic condition marked by scaling, roughness, and discomfort resulting from impaired skin barrier function. At the molecular level, xerosis is driven by the depletion of essential intercellular lipids, namely ceramides, cholesterol, and free fatty acids, which disrupts the lamellar bilayer structure of the stratum corneum, increases transepidermal water loss (TEWL), and compromises hydration retention mechanisms [1]. The stratum corneum's integrity relies on a delicate interplay between natural moisturizing factors (NMFs), lipid matrices, and proper desquamation, all of which are adversely affected by environmental stressors such as low humidity, temperature extremes, and particulate pollutants [2, 3]. In recent years, global climate change has introduced a new dimension to xerosis pathophysiology: progressive aridification. Defined as the long-term shift toward drier atmospheric and soil conditions, aridification is now affecting large swaths of the American Southwest and other global regions due to warming temperatures and altered precipitation patterns [4]. This environmental transformation accelerates skin barrier breakdown by increasing TEWL, reducing ambient humidity, and heightening exposure to airborne irritants such as dust and pollutants, many of which exacerbate oxidative stress and inflammation at the cutaneous level [5, 6]. Particularly vulnerable to these climate-related shifts are pediatric and geriatric populations. Infants and children possess a thinner stratum corneum, lower sebum production, and immature lipid synthesis pathways, making their skin more susceptible to dehydration and irritation under arid conditions [7, 8]. Older adults, on the other hand, experience age-related declines in ceramide content, epidermal turnover, and barrier repair capacity, which are further compounded by comorbidities, polypharmacy, and dry indoor environments [9, 10]. In both groups, the compounded effects of intrinsic vulnerability and extrinsic aridification create a potent risk environment for xerosis and its complications, including fissures, pruritus, secondary infection, and impaired quality of life.

These age-stratified responses to environmental dryness fall under the broader scope of climate-sensitive dermatology, an emerging field that evaluates how ecological and atmospheric changes alter disease distribution, severity, and therapeutic needs [11]. As extreme temperatures, humidity variability, and airborne irritants become more common, dermatologists must adopt climate-informed strategies to identify, prevent, and manage xerosis in high-risk populations. This review synthesizes current literature on climate-driven xerosis, with a focus on pediatric and elderly populations, to guide both clinical care and public health interventions in a rapidly changing environmental landscape.

Materials and Methods

This review was conducted as a narrative literature review aimed at synthesizing evidence on how climate-driven aridification affects skin barrier function, particularly in pediatric and geriatric populations. To gather relevant studies, a comprehensive search was performed using three major databases: PubMed, Scopus, and Google Scholar. The search was conducted focusing on literature published between January 2011 and May 2025. A combination of keywords and Boolean operators was used, including "xerosis," "dry skin," "skin barrier," "transepidermal water loss," "arid climate," "low humidity," "pediatric skin," "infant skin barrier," "geriatric dermatology," "elderly skin," "ceramides," "climate change," and "environmental skin exposure." Inclusion criteria required that studies be published in English and directly address xerosis or skin barrier integrity within the context of environmental or climatic stressors such as low humidity, high temperatures, or airborne particulate exposure. Eligible studies included clinical research, experimental studies, epidemiologic investigations, and structured literature reviews that focused on children (ages 0–18), older adults (≥60 years), or experimental models representative of these populations. Articles were excluded if they centered on dermatologic conditions unrelated to xerosis (e.g., acne, psoriasis) or if the cause of xerosis was unrelated to climate factors, such as chemotherapy, dialysis, or genetic disorders. Titles and abstracts were initially screened for relevance, followed by full-text review of selected studies. Data extraction included study design, population demographics, environmental variables, key mechanistic findings, and outcomes related to skin hydration, lipid content, and transepidermal water loss. Studies were categorized by age group (pediatric vs. geriatric), environmental exposure (e.g., arid vs. temperate climates), and methodological focus (clinical, experimental, or epidemiologic).

Results

Mechanisms of Xerosis in Arid Climates

Xerosis, or dry skin, is primarily a consequence of disrupted skin barrier integrity, particularly in environments characterized by low humidity and high wind exposure.

Table 1: Climate-Related Environmental Triggers and Dermatologic Effects

Environmental Factor	Mechanism of Skin Barrier Disruption	Dermatologic Manifestation	Vulnerable Populations
Low relative humidity	↓ NMFs, ↑ TEWL, impaired desquamation	Xerosis, scaling, rough texture	Children, elderly
Heatwaves / high temperatures	TRPV3 activation, ↑ inflammatory cytokines, ↓ lipid synthesis	Pruritus, barrier breakdown, erythema	Elderly, eczema-prone individuals
Air pollution ($PM2.5$, NO_2)	Oxidative stress, lipid peroxidation, ↓ filaggrin and tight junction proteins	Eczematous flares, chronic xerosis	Children, urban elderly

Wind exposure	Microabrasions, corneocyte disruption, ↑ desiccation	Scaling, inflammation, xerotic eczema	Outdoor workers, infants
UV radiation	DNA damage, ↓ ceramides, ↑ TEWL	Photodamage, fissuring, xerosis in exposed sites	Elderly, fair-skinned children
Indoor heating / AC	Decreases ambient humidity, ↑ TEWL	Winter xerosis, dryness in care homes and schools	Institutionalized elderly, kids
Dust exposure (arid climates)	Induces inflammation, ↑ skin irritability and barrier stress	Irritant dermatitis, xerosis-related flares	Elderly, asthmatic children

In arid climates, the reduction in ambient humidity leads to a significant increase in TEWL, compromising the skin's ability to retain moisture [1, 2]. This water loss is exacerbated by a concurrent reduction in the lipid matrix, specifically ceramides, cholesterol, and free fatty acids, which normally form the lamellar structures essential for skin hydration and barrier function [7, 12]. Moreover, the degradation of filaggrin-derived NMFs, such as amino acids, urea, and lactic acid, further impairs the stratum corneum's capacity to bind water, especially under conditions of prolonged dryness [1]. Environmental factors such as wind and airborne pollutants also act as external aggravators, accelerating skin barrier breakdown and increasing inflammation. Fine particulate matter (PM2.5), commonly found in arid and dust-prone regions, has been shown to induce oxidative stress and cytokine release, contributing to further lipid depletion and impaired corneocyte cohesion [3, 5]. Seasonal changes, particularly during the dry winter months or in areas affected by desertification, also trigger xerotic flares by intensifying desquamation disorders and delaying barrier repair [10, 13]. Collectively, these mechanistic pathways underscore how aridification, compounded by climate change, profoundly impacts the stratum corneum, especially in pediatric and geriatric populations who already possess structurally or functionally immature skin barriers [7, 9].

Vulnerable Populations

Children and the elderly are particularly susceptible to climate-driven xerosis due to intrinsic structural and physiological vulnerabilities in the skin barrier.

Table 2: Age-Related Changes in Stratum Corneum and Their Relevance to Xerosis

Parameter	Pediatric Skin	Adult Skin	Elderly Skin
SC Thickness	Thin, underdeveloped	Mature	Thinner due to epidermal atrophy
Lipid Content (ceramides, etc.)	Low (immature synthesis)	Normal	Decreased synthesis and altered composition
NMF Production	Low (limited filaggrin breakdown)	Normal	Decreased due to aging and comorbidities
TEWL	High	Low–moderate	High (impaired barrier recovery)
Sebum/Sweat Production	Low	Normal	Decreased sebaceous and sweat gland activity
Barrier Recovery Time	Slow	Optimal	Prolonged

In children, incomplete development of the stratum corneum results in higher TEWL, reduced NMFs, and immature lipid processing systems [1, 7]. Pediatric skin, especially in dry climates such as the Southwestern United States and North Africa, has a thinner epidermis, higher surface area-to-volume ratio, and underdeveloped corneocyte cohesion, making it highly sensitive to arid environments and wind exposure [5, 13]. Moreover, seasonal birth patterns may further exacerbate risk; infants born in the fall and winter months demonstrate higher prevalence of atopic dermatitis and xerosis, likely due to decreased UV exposure and impaired skin maturation [13]. Among elderly populations, xerosis arises from a combination of epidermal thinning, sebum reduction, and a decline in ceramide and cholesterol synthesis, factors that diminish the skin's ability to retain moisture and repair barrier injury. Flattened rete ridges and reduced lipid compactness further impair hydration and immune surveillance, particularly in chronically exposed or poorly moisturized areas such as the lower legs and forearms [7, 14]. In institutional settings like nursing homes, the prevalence of xerosis can exceed 85%, yet regular emollient use remains inconsistently implemented, especially in semi-dependent or independent residents [15, 16]. Both children and older adults also experience climate-compounded flare-ups during periods of low humidity or dust exposure, which provoke oxidative stress and trigger inflammatory mediators that further disrupt barrier integrity [3, 5]. Thus, xerosis in these populations not only reflects intrinsic dermatologic fragility but also underscores the urgent need for climate-adapted skincare protocols in both pediatric and geriatric care.

Geographic and Environmental Evidence

The geographic distribution and environmental context of xerosis reveal strong correlations with arid climates, seasonal patterns, and built environments. Epidemiologic data from arid regions such as the Sahel, Middle East, and the American Southwest suggest that xerosis prevalence is significantly influenced by regional aridification trends, including rising dust levels, decreased precipitation, and hotter dry spells [4, 5]. In the American Southwest, aridification is intensifying, with projected dust increases of up to 57% by 2090, compounding skin barrier damage through wind-driven microabrasions, oxidative stress, and desiccation [5]. Indoor microclimates further exacerbate xerosis, particularly in institutional settings where air conditioning and indoor heating contribute to persistently low relative humidity [15, 16].

Table 3: Indoor vs. Outdoor Environmental Contributors to Xerosis

Environment	Trigger	Mechanism	Common Settings
Indoor	Heating/AC	Reduces ambient humidity, ↑ TEWL	Hospitals, nursing homes, classrooms
Indoor	Detergent or soap exposure	Alkaline pH strips NMF/lipids	Over-washing, institutional bathing
Outdoor	Wind, low RH	Corneocyte abrasion, ↑ desquamation	Deserts, rural/agricultural settings
Outdoor	UV exposure	DNA damage, ↓ ceramides	High-altitude, outdoor workers
Outdoor	Dust/pollutants	Oxidative stress, inflammatory cytokines	Urban or arid zones

Nursing homes and hospitals often maintain environments below 30% relative humidity, leading to increased TEWL and decreased stratum corneum hydration in residents with aging or compromised skin barriers [7, 14]. Seasonal xerosis flares are particularly common in temperate climates, with prevalence peaking during winter months when ambient humidity is low and indoor heating is widespread. These fluctuations in environmental humidity and temperature contribute to impaired desquamation, lipid degradation, and TEWL surges, particularly in children and the elderly, whose skin barrier systems are inherently weaker [1, 10]. Furthermore, reconstructed epidermal models exposed to even brief periods of low humidity show significant increases in TEWL and barrier protein loss, reinforcing the vulnerability of skin to rapidly changing moisture gradients [17]. Collectively, this evidence underscores the synergistic impact of geographic aridification and artificial indoor environments on the pathogenesis and severity of xerosis across both developing and industrialized regions.

Clinical Diagnosis and Presentations

Xerosis presents with a characteristic constellation of clinical findings, including fine scaling, fissures, roughness, and pruritus. These features often cluster in specific anatomical regions depending on age, environmental exposures, and comorbidities. In elderly patients, xerosis typically affects the lower legs, arms, and trunk, presenting as cracked, erythematous skin with a "craquelé" pattern and heightened itch intensity during low-humidity months [14,16]. In pediatric populations, cheeks, extensor surfaces, and flexural folds are most commonly involved due to immature stratum corneum development and increased surface-area-to-volume ratios, both of which amplify TEWL [7,8]. Distinguishing xerosis from related conditions such as atopic dermatitis, ichthyosis, and senile pruritus requires attention to lesion morphology, distribution, and associated symptoms. Unlike atopic dermatitis, which often presents with lichenified plaques and a relapsing inflammatory component, xerosis typically lacks persistent erythema and is more diffuse with fine white scaling [12,13]. Ichthyosis vulgaris, on the other hand, displays polygonal scaling and is frequently associated with filaggrin mutations, features not typically found in acquired xerosis [10]. Senile xerosis may resemble asteatotic eczema in chronic stages, but early presentations often show subtle roughness and decreased elasticity without active inflammation [18]. Pathophysiologically, xerotic skin demonstrates reduced lipid content (particularly ceramides and cholesterol), lower filaggrin-derived natural moisturizing factors, and increased skin surface pH, all contributing to impaired desquamation and water retention [1,12]. Climate-induced xerosis can be intensified by environmental factors such as wind, cold, or dust, which degrade corneocyte cohesion and further aggravate barrier dysfunction [5]. Diagnostic tools, including dermoscopy, confocal microscopy, and TEWL measurement, may aid in quantifying severity, particularly in vulnerable populations where symptoms may be underreported [9]. Ultimately, early recognition of xerosis and its exacerbating environmental triggers is essential. In elderly individuals, particularly those in institutional settings, symptoms may be mistakenly attributed to aging, resulting in under-treatment. In children, xerosis may be an early indicator of broader atopic diathesis, requiring both dermatologic and environmental intervention. These population-specific clinical patterns highlight the importance of integrating climate considerations into dermatologic assessment and management strategies for xerosis.

Management and Prevention

Effective xerosis management in climate-stressed pediatric and geriatric populations demands a multifaceted approach that targets both structural barrier repair and environmental adaptation. Central to treatment is the consistent use of emollients, which act through three primary mechanisms: occlusion, humectancy, and barrier lipid restoration.

Table 4: Biochemical Emollient Components and Their Mechanisms in Xerosis Management

Ingredient	Class	Mechanism of Action	Notes
Urea (5–10%)	Humectant/Keratolytic	Attracts water, exfoliates corneocytes, regulates filaggrin	Preferred for elderly xerosis
Ceramides	Lipid barrier repair	Replenishes SC lipid matrix, ↓ TEWL	Best in 3:1:1 ratio w/ cholesterol & FFAs
Petrolatum	Occlusive	Blocks water loss; ↓ TEWL by up to 98%	Effective but greasy; ideal for elderly
Lactic acid	Humectant & lipid synthesis booster	↑ ceramide synthesis, mild exfoliation	Useful in aged skin

Niacinamide	Anti-inflammatory/lipid booster	↑ ceramide production, barrier thickness	Useful in pediatric and geriatric skin
Glycerol	Humectant	Pulls moisture into SC	Must combine with occlusive to prevent TEWL increase
Sunflower oil	Emollient (natural)	High in linoleic acid; restores skin barrier	Preferred in children

Occlusives such as petrolatum reduce TEWL by up to 98%, forming a hydrophobic seal over the stratum corneum. Humectants like glycerin and hyaluronic acid draw moisture into the skin but can paradoxically increase TEWL if not paired with occlusives. Ceramide-dominant formulations restore epidermal lipid architecture and promote lamellar organization, key for long-term barrier function [12,19]. Among elderly populations, lipid-deficient skin with elevated surface pH and reduced desquamation renders xerosis nearly ubiquitous in institutional settings. Yet, studies reveal that moisturizer use is inconsistent or absent in nearly half of those affected, even when care staff are present [15]. Care protocols should therefore include standardized twice-daily emollient application, ideally following bathing with warm water and mild, fragrance-free cleansers. The "soak and smear" technique, brief soaking followed by thick emollient application, has proven especially effective for fissured or inflamed xerotic skin [16, 18]. In children, whose stratum corneum is thinner and more vulnerable to environmental fluctuations, urea-based and ceramide-containing moisturizers offer dual benefits of hydration and barrier repair. Urea concentrations between 5% and 10% improve keratinocyte differentiation and filaggrin expression, reducing TEWL and improving pruritus in as little as 7 days [18]. Pediatric skin care regimens should emphasize emollient use after each wash and education of caregivers in humidification strategies and skin-safe cleansing practices [20]. Prevention extends beyond topical agents. In long-term care and school environments, climate-adaptive skin care protocols should include humidity monitoring, use of emollients with optimized physiologic pH (~4.5–5.5), and scheduled assessments for xerosis severity. Seasonal transitions, particularly into winter or during regional droughts, require anticipatory adjustments such as increased moisturizer frequency and substitution of lotions with ointments [10]. Incorporating emollient use into routine institutional care, combined with caregiver training and patient education, can drastically reduce xerosis incidence and associated complications like pruritus, infection, and chronic eczema.

Table 5: Emollient Therapy Strategies: Pediatric vs. Elderly Xerosis

Parameter	Pediatric Skin	Elderly Skin
Barrier Characteristics	Immature SC, high surface-area-to-volume ratio	Thinned SC, ↓ ceramides, lipid synthesis, epidermal turnover
Recommended Emollients	Urea (5–10%), ceramide-based, niacinamide, sunflower oil	Urea (10%), lactic acid, petrolatum-based, MVE ceramide cleansers
Preferred Vehicles	Lotions/creams for ease; avoid strong fragrances	Ointments preferred in low humidity; fragrance-free essential due to sensitivities
Application Technique	Post-bath, damp skin; parent-assisted "soak and smear" for severe cases	Twice daily post-bath; caregiver-administered in institutional settings
Key Challenges	Product adherence, caregiver education, allergic potential	Mobility/cognitive decline, polypharmacy interactions, underdiagnosis
Add-ons for Severe Cases	Topical corticosteroids (low potency), wet wraps	TRPM8 agonists for pruritus, barrier pH correction (pH 4.0 emulsions)

Lastly, public health guidance must account for aridification trends and indoor dryness. Educational campaigns should advocate for emollient literacy, avoidance of harsh soaps, and proper product selection, especially in underserved communities and hot, low-humidity regions [21]. With the increasing burden of climate-driven xerosis, management must shift from reactive treatment to proactive barrier protection.

Discussion

Climate-driven xerosis represents a growing dermatologic challenge, particularly among pediatric and geriatric populations, two groups with inherently compromised skin barrier function and limited adaptive capacity. As aridification intensifies across global regions due to rising temperatures, decreased humidity, and increased particulate exposure, the burden of xerosis is expected to rise in both clinical prevalence and severity [4, 5]. This review highlights the urgent need for tailored emollient therapies, preventive infrastructure in institutional settings, and widespread skin health literacy campaigns. The vulnerability of the pediatric and elderly skin barrier, compounded by structural differences, immunologic immaturity or decline, and environmental exposures, demands that xerosis management evolve beyond symptom relief to embrace proactive, climate-responsive strategies. These include ceramide-dominant emollients, seasonal skin care protocols, and caregiver education in schools, daycares, and long-term care facilities [9, 15]. Moreover, standardized xerosis

screening tools and digital hydration metrics may become necessary components of dermatologic care in high-risk populations.

Looking ahead, predictive modeling of xerosis "outbreaks" based on environmental conditions, similar to pollen or UV forecasts, could allow dermatologists and public health officials to anticipate flare periods and deploy early interventions. Integration of climate metrics such as the Standardized Precipitation-Evapotranspiration Index (SPEI), ambient PM2.5 levels, and seasonal wind trends could inform targeted skin health alerts and emollient distribution efforts in vulnerable regions [5, 12]. Dermatologists also have a critical role to play in shaping climate policy. As frontline witnesses to environmental damage on skin health, they are uniquely positioned to advocate for sustainable urban planning, clean air legislation, and equitable access to hydration infrastructure and skin barrier repair products. Incorporating skin barrier vulnerability into climate resilience frameworks could reduce morbidity, particularly among under-resourced rural and institutionalized populations.

Finally, substantial research gaps persist. Few studies disaggregate xerosis outcomes by age, ethnicity, socioeconomic status, or geography, limiting our understanding of how aridification interacts with social determinants of health. Longitudinal studies in pediatric and geriatric cohorts, particularly those living in arid zones or high-pollution regions, are urgently needed. Emphasis should also be placed on inclusive trials that evaluate emollient efficacy across Fitzpatrick skin types and age groups. In conclusion, xerosis cutis is no longer a benign dermatologic nuisance, it is a sentinel indicator of climate vulnerability. Integrating environmental dermatology into routine practice, public health, and global policy is essential to mitigate its expanding burden.

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