Essays

Climate Change, Cutaneous Ageing, and Skin Cancer: Mechanistic Pathways, Epidemiological Evidence, and Public Health Implications

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ABSTRACT: Climate change is transforming the environmental exposures that shape human skin health. Rising ultraviolet (UV) radiation, heat extremes, humidity fluctuations, and escalating air pollution form a shifting cutaneous exposome. Together, these stressors accelerate extrinsic skin ageing and increase the burden of skin cancer. At the mechanistic level, pathways include oxidative stress, extracellular matrix degradation, mitochondrial dysfunction, immunosuppression, and pollutant– UV synergy. Epidemiological evidence supports growing risks across populations, though data gaps remain. Particularly vulnerable groups include outdoor workers, climate migrants, children, the elderly, and immunocompromised patients. This article synthesizes current knowledge, identifies mechanistic and epidemiological links, and emphasizes prevention, from personal photoprotection to systemic climate adaptation. Situating dermatology within planetary health underscores the urgency of integrating skin health into climate policy and research priorities.

KEYWORDS: Climate change; cutaneous ageing; public health; skin cancer; ultraviolet radiation

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tective Strategies – 7. Prevention and Protective Strategies – 7.1. Behavioral Measures – 7.2. Clinical and Technological Measures – 7.3. Policy and Structural Measures – 8. Research Gaps and Future Directions – 9. Conclusion.

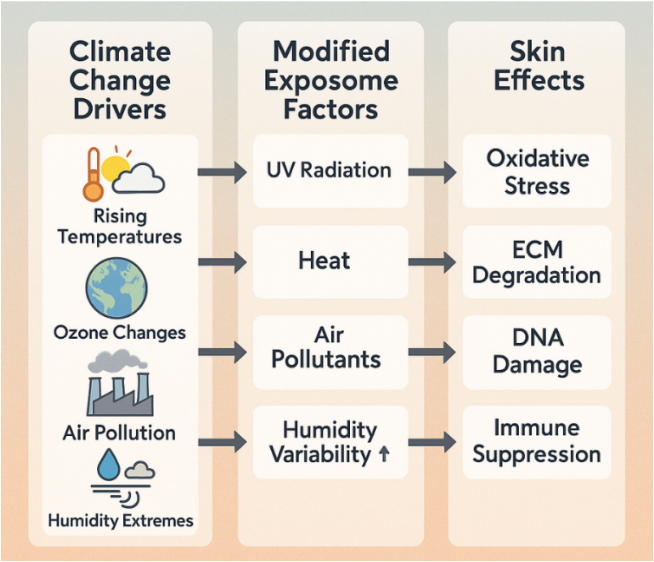
1. Introduction

Human skin is the body's largest organ and its primary environmental interface. It protects against ultraviolet radiation, toxins, pathogens, and fluctuating temperatures, while also serving as a visible marker of biological and environmental ageing. With accelerating climate change, this protective interface is under unprecedented strain.

The skin's health is governed by the cutaneous exposome, the cumulative external and internal factors influencing ageing, carcinogenesis, and disease.[1] Climate change acts as a multiplier across exposome domains, altering the intensity, duration, and interaction of exposures such as UV radiation, particulate matter, ozone, heat stress, and humidity extremes.[2] These exposures converge mechanistically via oxidative stress, chronic inflammation, impaired DNA repair, and immune dysregulation, producing two major clinical outcomes of concern: premature skin ageing and increased risk of skin cancer.[3]

Globally, skin cancer is the most common malignancy, and its incidence continues to rise (Parker, 2020). Meanwhile, extrinsic skin ageing contributes not only to aesthetic change but also to functional decline in barrier integrity, wound healing, and immune competence.[4] The intersection of climate change with these processes raises urgent questions for clinicians, researchers, and policymakers (Figure 1).

Figure 1. Climate change drivers and the cutaneous exposome.



[1] I. Khmaladze, M. Leonardi, S. Fabre, C. Messaraa, A. Mavon, *The Skin Interactome: A holistic genome–microbiome–exposome approach to skin health and ageing,* in *Clin Cosmet Investig Dermatol*, 13, 2020, 1021–1040.

[2] A. Anderson, F. Bruce, H.P. Soyer, C. Williams, R.B. Sauderson, *The impact of climate change on skin health*, in *Med J Aust*, 218, 9, 2023, 388–390.

[3] T.P.G. Watson, M. Tong, J. Bailie, K. Ekanayake, R.S. Bailie, *Relationship between climate change and skin cancer: a scoping review*, in *Public Health*, 227, 2024, 243–249.

[4] M. Aryan Kya, *Geospatial Patterns of Non-Melanoma Skin Cancer in Relation to Climate Changes in Iran,* in *Asian Pac J Cancer Prev*, 25, 3, 2024, 1053–1063.

This article addresses five aims:

1. To outline how climate change is reshaping the cutaneous exposome.
2. To detail mechanistic pathways linking these exposures to ageing and carcinogenesis.
3. To review epidemiological evidence for climate-related skin outcomes.
4. To identify vulnerable populations.
5. To propose preventive and adaptive strategies spanning individuals, clinical practice, and public health.

2. Climate Change and the Cutaneous Exposome

The cutaneous exposome encompasses ultraviolet radiation, air pollutants, meteorological conditions (temperature, humidity), lifestyle, and endogenous factors. A relevant addition to this list is high-energy visible light (HEVL), or blue light, from both solar and digital sources, which contributes to oxidative stress and pigmentary changes. Climate change alters these domains in complex, interactive ways.

2.1. Ultraviolet Radiation Shifts

Although the Montreal Protocol has facilitated partial ozone recovery, climate feedback loops continue to influence surface UV patterns.[5] Stratospheric cooling linked to greenhouse gases may slow ozone repair, while loss of reflective surfaces such as ice and snow amplifies ground-level UV.[6] Changes in cloud dynamics further alter UV intensity. Modeling suggests that by 2100, mid-latitude regions may experience a net increase in erythemally effective UV radiation, despite global emission reductions.
For dermatology, this means heightened exposure to DNA-damaging wavelengths, UVB driving mutagenesis and UVA driving oxidative stress and photoageing. Importantly, UV interacts with other exposome factors. For example, pollutants such as polycyclic aromatic hydrocarbons (PAHs) absorb UV and become more reactive, compounding oxidative stress.

2.2. Heatwaves and Extreme Temperatures

Heat is one of the most direct climate hazards. Global warming has increased both the frequency and severity of heatwaves, with profound consequences for skin physiology. Elevated temperatures disrupt barrier function by increasing trans-epidermal water loss (TEWL) and altering lipid organization[4]. Heat shock proteins are upregulated, modulating immune and inflammatory responses.

[5] S. MADRONICH, G.H. BERNHARD, P.J. NEALE, *et al*, *Continuing benefits of the Montreal Protocol and protection of the stratospheric ozone layer,* in *Photochem Photobiol Sci,* 23, 6, 2024, 1087–1115.

[6] N. SINGH, C. WIGMANN, P. VIHAY, *et al., Combined Effect of Ambient Temperature and Relative Humidity on Skin Aging Phenotypes in the Era of Climate Change: Results From an Indian Cohort Study,* in *Dermatitis*, 36, 1, 2025, 72-79.

Chronic or repeated heat stress may accelerate intrinsic ageing processes through mitochondrial dysfunction and epigenetic alterations.[7] Behavioral effects compound these risks: during heat events, individuals often increase outdoor exposure and reduce protective clothing, inadvertently raising UV dose.[8]

2.3. Air Pollution Intensification

Climate change exacerbates air pollution through stagnant weather patterns, wildfire smoke, and altered photochemistry. Key pollutants affecting the skin include:

- *Particulate matter* (PM2.5 and PM10): Penetrates follicular openings, inducing oxidative stress and inflammation.
- *Ground-level ozone* (O3): Damages lipids and antioxidants in the stratum corneum, compromising barrier integrity.[9]
- *Nitrogen oxides* (NOx) and *sulfur oxides* (SOx): Potentiate inflammatory cascades.
- *Polycyclic aromatic hydrocarbons* (PAHs): UV-activated mutagens forming DNA Adducts.[10]

Epidemiological studies link chronic pollution exposure to pigmentary disorders, lentigines, and wrinkle formation.[11] Pollutants also exacerbate UV-induced DNA damage, acting as co-carcinogens.

2.4. Humidity Extremes

Changing precipitation patterns produce alternating extremes of low and high humidity. Low humidity compromises stratum corneum hydration, leading to barrier fragility, xerosis, and accentuated wrinkling. High humidity promotes microbial dysbiosis, fungal infections, and irritant dermatitis.[12] These shifts modulate both skin ageing and susceptibility to neoplasia via immune perturbation.

2.5. Multiplicative Impacts

The *synergistic effects* of combined climate stressors are particularly concerning. UVA and ozone exposures synergistically increase oxidative burden, while UV–PAH interactions yield enhanced DNA adduct formation[1]. Urban heat islands exemplify convergence:
elevated local temperatures intensify ozone formation, while socioeconomic disparities limit access to protective resources.[13]

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[7] W. Ni, N. Nikolaou, C.K. Ward-Caviness, *et al.*, *Associations between medium- and long-term exposure to air temperature and epigenetic age acceleration,* in *Environ Int*, 2023, 178.
[8] W.L. Kenney, D.H. Craughead, L.M. Alexander, *Heat waves, aging, and human cardiovascular health,* in *Med Sci Sports Exerc.,* 46, 10, 2014,1891-9.
[9] J. Krutmann, W. Liu, L. Li, *et al.,* 2014. *Pollution and skin: from epidemiological and mechanistic studies to clinical implications,* in *J Dermatol Sci*, 76, 3, 2014, 163-8.
[10] G. Bocheva, R.M. Slominski, A.T. Slominski, *Environmental Air Pollutants Affecting Skin Functions with Systemic Implications,* in *Int J Mol Sci*, 24, 13, 2023, 10502.
[11] J.C. Fussell, F.J. Kelly, *Oxidative contribution of air pollution to extrinsic skin ageing,* in *Free Radic Biol Med,* 1, 151, 2020, 111-122.
[12] N. Singh, C. Wigmann, P. Vijay, *et al*., *Combined effect of ambient temperature and humidity on skin ageing phenotypes,* in *Dermatitis*, 36, 1, 2025, 72–79.
[13] N. Balato, F. Ayala, M. Megna, *et al.*, *Climate change and skin,* in *G Ital Dermatol Venereol*, 148, 1, 2013, 135–146.

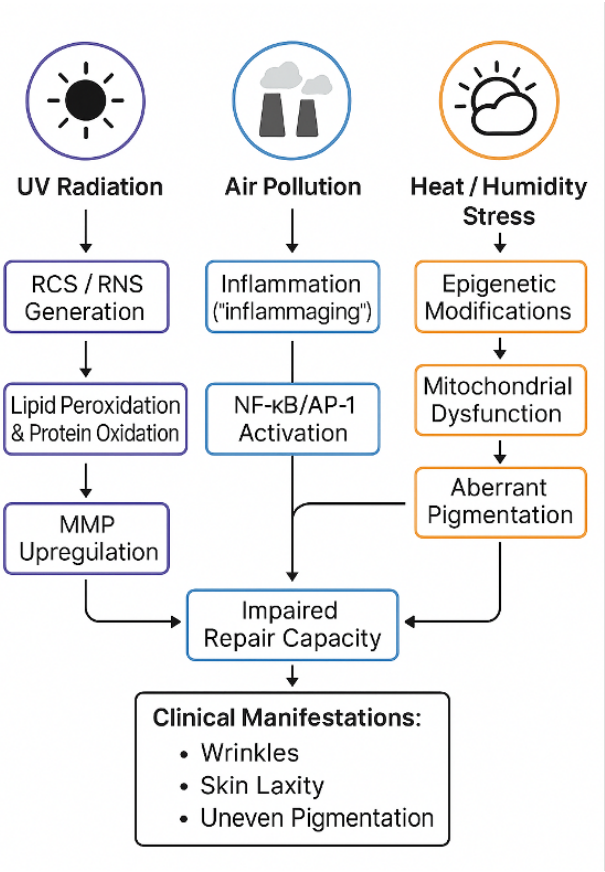
Thus, the exposome under climate change is not a linear addition of risks but a network of interactions, amplifying dermatological damage beyond single exposures.

3. Mechanistic Pathways Linking Climate Stressors to Skin Ageing

3.1. Extracellular Matrix (ECM) Degradation

One of the hallmarks of extrinsic skin ageing is the breakdown of collagen and elastin, the structural proteins that maintain dermal integrity. Both UV radiation and pollutants upregulate matrix metalloproteinases (MMP-1, MMP-3, MMP-9), which degrade ECM proteins.[14] UVA penetrates into the dermis, activating AP-1 and NF-κB signaling that stimulate MMP transcription, while ozone oxidizes skin lipids to bioactive mediators that further enhance MMP expression.[15] Clinically, these processes manifest as coarse wrinkles, loss of elasticity, and sagging (Figure 2).

Figure 2. Mechanistic pathways from climate stressors to skin ageing.



[14] J. Krutmann, W. Liu, L. Li, X. Pan, M. Crawford, *et al., Pollution and skin: from epidemiological and mechanistic studies to clinical implications*, in *J Dermatol Sci*, 76, 3, 2014, 163-8.
[15] G. Bocheva, R.M. Slominski, A.T. Slominski, *Environmental Air Pollutants Affecting Skin Functions with Systemic Implications,* in *Int J Mol Sci*, 24, 13, 2023, 10502.

3.2. Oxidative Stress

Reactive oxygen species (ROS) are central mediators of environmentally induced ageing. UVA generates ROS such as singlet oxygen and superoxide, while ozone and PAHs contribute additional oxidative load.[16] Excess ROS overwhelms endogenous antioxidant defenses (e.g., catalase, superoxide dismutase, glutathione), leading to lipid peroxidation, protein oxidation, and DNA base modifications such as 8-oxo-deoxyguanosine.

Persistent oxidative damage depletes stem cell pools, accelerates telomere shortening, and drives cellular senescence in fibroblasts and keratinocytes.[17] The accumulation of senescent cells further amplifies tissue ageing via thesenescence-associated secretory phenotype (SASP), characterized by chronic inflammatory cytokine release.

3.3. Inflammaging

Environmental stressors provoke low-grade, persistent inflammation that contributes to inflammaging. UV irradiation activates pattern recognition receptors and inflammasomes, triggering secretion of IL-1β, TNF-α, and IL-6. Heat stress enhances NF-κB activation and cytokine expression.[18] Pollutants such as PM2.5 activate aryl hydrocarbon receptors, leading to pro-inflammatory gene transcription.

Over time, this chronic inflammation remodels dermal ECM, impairs barrier function, and establishes a pro-tumorigenic microenvironment.

3.4. Epigenetic Alterations

Epigenetic drift, accumulated changes in DNA methylation, histone modifications, and noncoding RNA profiles, represents another key link between climate stressors and skin ageing. Long-term exposure to elevated ambient temperature is associated with accelerated epigenetic ageing.[19] UV exposure alters methylation of tumor suppressor genes, impairing genomic stability. Heat and oxidative stress modify histone acetylation, altering transcription of genes regulating repair and antioxidant responses.[20]

These changes are not purely theoretical: miRNA signatures such as circulating miR-19a-3p and miR-19b-3p have been correlated with human ageing trajectories.[21] Epigenetic marks therefore represent both biomarkers of environmental ageing and potential therapeutic targets.

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[16] J.C. Fussell, F.J. Kelly, *Oxidative contribution of air pollution to extrinsic skin ageing,* in *Free Radic Biol Med,* 1, 151, 2020, 111-122.

[17] I. Khmaladze, M. Leonardi, S. Fabre, *The Skin Interactome: A Holistic "Genome-Microbiome-Exposome" Approach to Understand and Modulate Skin Health and Aging,* in *Clin Cosmet Investig Dermatol*, 13, 2020, 1021-1040.

[18] N. Singh, C. Wigmann, P. Vihay, *et al., Combined Effect of Ambient Temperature and Relative Humidity on Skin Aging Phenotypes in the Era of Climate Change: Results From an Indian Cohort Study,* in *Dermatitis*, 36, 1, 2025, 72-79.

[19] W. Ni, N. Nikolaou, C.K. Ward-Caviness, *et al.*, *Associations between medium- and long-term exposure to air temperature and epigenetic age acceleration,* in *Environ Int*, 2023, 178.

[20] A. Cardenas, R. Fadadu, S. Bunyavanich, *Climate change and epigenetic biomarkers in allergic and airway diseases,* in *J Allergy Clin Immunol*, 152, 5, 2023, 1060–1072.

[21] C. Morsiani, L. Terlecki-Zaniewicz, S. Skalicky, *et al*, *Circulating miR-19a-3p and miR- 19b-3p characterize the human aging process,* in *Aging Cell*, 20, 7, 2021, e13409.

3.5. Mitochondrial Dysfunction

Mitochondria are particularly susceptible to oxidative insults due to limited DNA repair capacity. UVA and pollutants induce mitochondrial DNA (mtDNA) deletions, impairing oxidative phosphorylation and ATP production. Dysfunctional mitochondria release further ROS, creating a feed-forward loop of damage.[22]

This decline in bioenergetic capacity compromises fibroblast collagen synthesis and keratinocyte renewal, accelerating visible ageing. Moreover, mtDNA mutations are increasingly recognized in actinic keratoses and early carcinogenesis.[23]

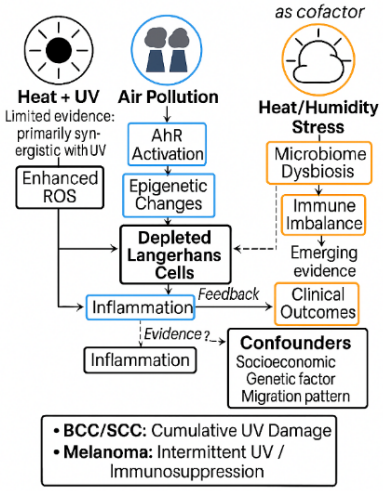
4. Mechanistic Pathways Linking Climate Stressors to Skin Carcinogenesis

While ageing and cancer share overlapping mechanisms, several distinct carcinogenic pathways emerge under climate change exposures.

4.1. Direct DNA Damage

UVB is the principal inducer of direct mutagenesis, forming cyclobutane pyrimidine dimers (CPDs) and 6-4 photoproducts. These lesions, if unrepaired, cause hallmark C→T transitions in p53 and other tumor suppressor genes.[24] UVA contributes indirectly via ROS, producing oxidative lesions such as 8-oxoguanine that mispair during replication (Figure 3).

Figure 3. Potential mechanistic pathways from climate stressors to the development of skin carcinogenesis.



[22] I. KHMALADZE, M. LEONARDI, S. FABRE, *The Skin Interactome: A Holistic "Genome-Microbiome-Exposome" Approach to Understand and Modulate Skin Health and Aging,* in *Clin Cosmet Investig Dermatol*, 13, 2020, 1021-1040.

[23] T.P.G. WATSON, M. TONG, J. BAILLIE, *et al., Relationship between climate change and skin cancer: a scoping review,* in *Public Health,* 227, 2024, 243–249.

[24] R.M. LUCAS, S. YAZAR, A.R. YOUNG, *et al., Human health in relation to exposure to solar ultraviolet radiation under changing stratospheric ozone and climate,* in *Photochem Photobiol Sci,* 18, 3, 2019, 641–680.

4.2. Pollutant-Driven Mutagenesis

Airborne PAHs (e.g., benzo[a]pyrene) penetrate skin, undergo metabolic activation by cytochrome P450 enzymes, and form bulky DNA adducts.[25] When combined with UV, DNA repair capacity becomes overwhelmed, escalating mutation rates. Heavy metals in PM2.5 also interfere with repair pathways, compounding mutagenesis.

4.3. Immunosuppression

UV radiation reduces cutaneous antigen-presenting cell function, particularly by depleting Langerhans cells and skewing T-cell responses toward tolerance. IL-10 and regulatory T-cell expansion create an immunosuppressive milieu.[26] Pollution exacerbates this by promoting oxidative stress and regulatory cytokine expression.[27] This immune dampening undermines tumor surveillance and facilitates malignant progression.

4.4. Microbiome Disruption

The skin microbiome is increasingly recognized as a modulator of carcinogenesis. Heat, humidity, and pollutants shift microbial communities, sometimes increasing pro-inflammatory taxa or reducing protective commensals.[28] Dysbiosis may promote carcinogenesis indirectly by altering immune tone and epithelial barrier integrity.

4.5. Synergistic Hazards

In practice, climate-linked stressors rarely occur in isolation; instead, the skin is exposed to clustered insults that interact non-linearly, producing damage greater than the sum of their parts Synergistic interactions magnify cancer risk:

- UV + PAHs: Mutagenic synergy through DNA adducts and ROS.
- UV + Ozone**:** Additive oxidative stress and lipid peroxidation.
- Heat + UV: Behavioral (less protective clothing) and biological (enhanced ROS) Amplification.[29]

Thus, climate change creates carcinogenic exposome clusters, rather than isolated risks.

[25] E.R. Parker, *The influence of climate change on skin cancer incidence*, in *Int J Womens Dermatol*, 7, 1, 2020, 17–27.
[26] R.M. Lucas, S. Yazar, A.R. Young, *et al.*, *Human health in relation to exposure to solar ultraviolet radiation under changing stratospheric ozone and climate,* in *Photochem Photobiol Sci,* 18, 3, 2019, 641–680
[27] F.M. Isler, S.J. Coates, M.D. Boos, *Climate change, the cutaneous microbiome and skin disease,* in *Int J Dermatol,* 62, 3, 2023, 337–345.
[28] *Ibidem.*
[29] A. Anderson, F. Bruce, H.P. Soyer, C. Williams, R.B. Sauderson, *The impact of climate change on skin health*, in *Med J Aust*, 218, 9, 2023, 388–390.

5. Epidemiological Evidence

5.1. UV Radiation and Skin Cancer

Epidemiological links between UV exposure and skin cancer are long-established. Lifetime cumulative UV dose strongly predicts non-melanoma skin cancer (NMSC) incidence, with latitude-dependent gradients.[30] Recent modeling studies suggest climate-driven increases in UV could further elevate global skin cancer incidence by mid-century, particularly in regions with ozone thinning.[31]

Outdoor workers remain disproportionately affected. The WHO/ILO Joint Estimates project over 180,000 new NMSC cases annually worldwide attributable to occupational solar UV exposure.[32] However, the claim that 'nearly 30% of global NMSC between 2000– 2019 were attributable to occupational UV' is inaccurate; the WHO/ILO data indicates ~30% of NMSC deaths, not cases, though case estimates are similarly high.

5.2. Heat and Temperature Extremes

Heat itself is not traditionally classified as a carcinogen, but epidemiological signals are emerging. Analyses in Texas found higher melanoma incidence in regions with prolonged high temperatures, independent of UV dose, Global studies suggest female cancer mortality rises disproportionately with increasing mean annual temperature[17]. Moreover, heat influences behavioral risk factors, greater sun exposure during heatwaves, and may act biologically to enhance UV carcinogenicity.[33]

5.3. Air Pollution and Ageing

Cohort studies in Europe and Asia have linked chronic exposure to PM2.5 and NO2 with extrinsic ageing markers, including pigment spots, lentigines, and coarse wrinkles.[34] One German cohort showed significant associations between traffic-related pollution and facial lentigines in women >50 years.[35]

5.4. Air Pollution and Skin Cancer

Evidence linking pollution to cancer is less consistent. Mendelian randomization studies in European populations found no causal association between pollution and melanoma[36]. However, experimental da-

[30] F. Pega, N.C. Momen, K.N. Streicher, *et al.*, *Global burden of NMSC attributable to occupational UV exposure,* in *Environ Int,* 181, 2023, 108226.

[31] S. Madronich, G.H. Bernhard, P.J. Neale, *et al.*, *Continuing benefits of the Montreal Protocol and protection of the stratospheric ozone layer*, in *Photochem Photobiol Sci*, 23, 6, 2024, 1087–1115.

[32] F. Pega, N.C. Momen, K.N. Streicher, *et al.*, *Global burden of NMSC attributable to occupational UV exposure,* in *Environ Int.,* 181, 2023, 108226.

[33] W.L. Kenney, D.H. Craughead, L.M. Alexander, *Heat waves, aging, and human cardiovascular health,* in *Med Sci Sports Exerc.,* 46, 10, 2014,1891-9.

[34] J.C. Fussell, F.J. Kelly, *Oxidative contribution of air pollution to extrinsic skin ageing,* in *Free Radic Biol Med,* 1, 151, 2020, 111-122.

[35] J. Krutmann, W. Liu, L. Li, X. Pan, M. Crawford, G. Sore, S. Seite, *Pollution and skin: from epidemiological and mechanistic studies to clinical implications*, in *J Dermatol Sci*, 2014, 76, 3, 163-8.

[36] M. Zhang, J. Wang, R. Huo, *et al.*, *Association between air pollution and skin cutaneous melanoma: A Mendelian randomization study,* in *Medicine* (Baltimore)*, 3,* 103, 18, 2024, e38050.

ta support plausible mechanisms, and observational studies in heavily polluted regions report higher NMSC incidence.[37]

5.5. Multiplicative Evidence

A 2024 scoping review synthesized climate-related influences on skin cancer, identifying UV, occupation, air pollution, and temperature as the strongest evidence-based factors, though interactions remain underexplored (Table 1). [38]

6. Vulnerable Populations

Climate change does not distribute risk equally. Specific groups face disproportionate vulnerability to skin ageing and cancer due to biology, occupation, or socioeconomic context.

6.1. Outdoor and Rural Workers

Agricultural, construction, and fisheries workers endure prolonged unprotected UV exposure. According to the WHO/ILO joint estimates, 30% of global non-melanoma skin cancer (NMSC) deaths between 2000–2019 were attributable to occupational UV exposure [19]. Rural populations also face limited access to dermatological services, compounding risk.[39]

6.2. Climate Migrants and Displaced Populations

Migration driven by desertification, flooding, and political instability can abruptly increase UV and heat exposure in populations unaccustomed to such climates. Limited healthcare access in refugee camps further exacerbates vulnerability.[40]

6.3. Immunocompromised Individuals

Patients with organ transplants or chronic immunosuppression experience amplified risk for UV- induced carcinogenesis. Climate-driven stressors may accelerate malignant progression by reducing immune surveillance.[41]

6.4. Children and the Elderly

Children's thinner skin and incomplete repair mechanisms make them more sensitive to cumulative photodamage, while elderly populations face reduced DNA repair and antioxidant capacity[10]. Moreover, heatwaves disproportionately affect elderly individuals with impaired thermoregulation[6].

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[37] M. Aryan Kya, *Geospatial Patterns of Non-Melanoma Skin Cancer in Relation to Climate Changes in Iran*, in *Asian Pac J Cancer Prev*, 25, 3, 2024, 1053–1063.

[38] T.P.G. Watson, M. Tong, J. Baillie*, et al., Relationship between climate change and skin cancer: a scoping review,* in *Public Health,* 227, 2024, 243–249.

[39] M.H. Fitzhugh, J. Wang, J.G. Powers, *Climate change and rural populations in dermatology,* in *Int J Womens Dermatol*, 11, 2, 2025, e214.

[40] G.S. Silva, M. Rosenbach, *Climate change and dermatology: introduction to a special issue, cit.*, 7, 1, 2021, 3–7.

[41] A. Anderson, F. Bruce, H.P. Soyer, C. Williams, R.B. Sauderson, *The impact of climate change on skin health*, in *Med J Aust*, 218, 9, 2023, 388–390.

6.5. Low-Income Communities

Populations in low-income countries often experience "double exposure": high UV/pollution levels and lack of access to sunscreen, shade, or dermatological care. Socioeconomic inequities create structural barriers to prevention.[42]

7. Prevention and Protective Strategies

Given the convergence of climate stressors on the skin, prevention requires an integrated, multi-level approach. Strategies must not only reduce acute exposures but also build resilience into the skin's barrier, immune, and repair systems. Crucially, these measures should be contextualized within climate adaptation frameworks to ensure equity and accessibility for high-risk groups. The following subsections outline these synergistic strategies across behavioral, clinical, and structural levels to mitigate the dermatological burden of climate change.

7.1. Behavioral Measures

Behavioral prevention remains the first and most scalable line of defense. Because climate change amplifies both environmental dose (UV, heat) and behavioral drivers (time outdoors, lighter clothing), everyday habits can meaningfully bend risk curves when applied consistently.

- *Sun protection*: Consistent use of broad-spectrum SPF 30+ sunscreen, protective clothing, and hats.[43] This includes an emphasis on mineral sunscreens (zinc oxide, titanium dioxide), particularly tinted formulations which offer enhanced protection against high-energy visible light (HEVL), and the synergistic use of topical antioxidants like Vitamin C to neutralize free radicals that bypass sunscreen filters.
- *Behavioral campaigns*: Public health interventions like SunSmart in Australia have reduced melanoma incidence in younger cohorts.[44]

7.2. Clinical and Technological Measures

Clinic-based strategies complement individual behaviors by shifting prevention and detection 'upstream.' Advances in imaging, machine learning, and barrier-repair approaches can reduce diagnostic delays, blunt extrinsic ageing pathways, and selectively target high-risk patients.

• *Early detection*: AI-enhanced dermoscopy and segmentation tools improve diagnostic accuracy and can be deployed at scale.[45]

[42] C.Y. Wright, D.J. Du Preez, D.A. Millar, *et al.*, *Epidemiology of skin cancer in Southern Africa,* in *Int J Environ Res Public Health*, 17, 3, 2020, 1017*;* R. Purcell, J. Mcgirr, *Rural health service managers' perspectives on preparing for climate change,* in *Aust J Rural Health, 26, 1, 2018, 20–25.*

[43] B. Diffey, Climate change, ozone depletion and the impact on ultraviolet exposure of human skin. *Phys Med Biol*, 49, 1, 2004, R1–R11.

[44] J. Makin, *Implications of climate change for skin cancer prevention in Australia,* in *Health Promot J Austr,* 22, 2011, S39–S41.

• *Barrier restoration*: Antioxidant and anti-inflammatory skincare formulations can mitigate oxidative stress and extrinsic ageing.[46]
• *Chemoprevention*: Compounds such as nicotinamide show promise in reducing actinic keratoses and NMSC risk.[47]

7.3. Policy and Structural Measures

Because exposure is strongly shaped by the built and policy environment, durable gains require structural solutions. Urban form, workplace standards, and international treaties can lower population-level dose—especially for those least able to protect themselves.

- *Occupational protections*: Mandated provision of shade, clothing, and breaks for outdoor workers.[48]
- *Urban planning*: Increasing residential greenness and shaded environments reduces heat and UV burden.[49]
- *Global treaties*: The Montreal Protocol remains a landmark in reducing ozone-depleting substances and mitigating UV-related cancers.[50] (Table 2)

8. Research Gaps and Future Directions

Despite rapid progress, several high-leverage research gaps remain. Addressing them would clarify causality, quantify synergy, and accelerate the translation of findings into equitable prevention strategies:
1. *Synergistic exposures*: Few studies quantify how UV, heat, and pollution interact to accelerate ageing or carcinogenesis.
2. *Longitudinal cohorts*: Most evidence is cross-sectional; long-term cohort data stratified by climate zone are essential.[51]
3. *Equity lens*: Vulnerable populations, including climate migrants and immunocompromised groups, remain underrepresented.
4. *Translational research*: Development of low-cost, scalable interventions (e.g., antioxidant-enriched sunscreens, portable shade infrastructure) is critical for low-resource settings.

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[45] P. Thapar, M. Rakhra, D. Prashar, *et al*., *Skin cancer segmentation and classification with hybrid ML,* in *PLoS One, 20, 6,* 2025, *e0322659;* J.L. Thomas, A.H.M. Heagerty, P. Goldberg Oppenheimer, *Emerging diagnostics for skin cancer,* in *Glob Chall*, 9, 5, 2025, 2400274.
[46] I. Khmaladze, M. Leonardi, S. Fabre, C. Messaraa, A. Mavon, *The Skin Interactome: A holistic genome–microbiome–exposome approach to skin health and ageing*, in *Clin Cosmet Investig Dermatol*, 13, 2020, 1021–1040.
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9. Conclusion

Climate change is reshaping the cutaneous exposome by amplifying UV radiation, temperature extremes, and pollutant exposure. Together, these factors accelerate skin ageing and increase skin cancer risk through mechanisms of oxidative stress, DNA damage, epigenetic drift, and immune dysregulation. Epidemiological evidence confirms rising NMSC and melanoma burden, particularly in outdoor workers and vulnerable populations.

Prevention requires a multilevel response: personal sun-safe behaviors, clinical innovation in detection and chemoprevention, and policy frameworks that protect high-risk populations. Equity must remain central, as climate change disproportionately harms the most exposed and least resourced.

Dermatology thus stands at a climate-health frontier, where integrating mechanistic insight with prevention strategies can mitigate the skin-related toll of a warming world.

Public Health and Regulatory Implications

Climate change poses a complex challenge at the intersection of environmental governance, public health, and the protection of fundamental human rights, including the right to health. The increasing burden of climate-related cutaneous aging and skin cancer represents not only a medical concern but also a regulatory and ethical issue, as preventable environmental exposures continue to disproportionately affect vulnerable populations.

Rising ultraviolet radiation, air pollution, and extreme heat events amplify cumulative skin damage and carcinogenic risk, raising questions of institutional responsibility in environmental protection and health prevention. From a biolaw perspective, the failure to integrate skin cancer prevention and dermatologic protection into climate adaptation policies may be viewed as a shortcoming in the implementation of preventive health obligations. Public authorities have a duty to ensure that climate-related health risks are adequately recognized, monitored, and mitigated through evidence-based regulation and public health interventions.

Moreover, climate-driven inequalities in exposure and access to dermatologic care highlight ethical concerns related to distributive justice and health equity. Outdoor workers, children, the elderly, and socioeconomically disadvantaged groups are often more exposed to environmental risk factors while simultaneously facing barriers to preventive services and early diagnosis. Strengthening regulatory frameworks that promote equitable access to sun-protective measures, occupational safeguards, and skin cancer screening programs is therefore essential to uphold principles of fairness and social responsibility.

Future Directions: Legal, Ethical, and Policy Perspectives

Future directions should emphasize the integration of skin health into climate-related legal and policy frameworks at both national and international levels. Regulatory strategies should explicitly acknowledge skin aging and skin cancer as climate-sensitive conditions and incorporate preventive dermatology into environmental health legislation, occupational safety standards, and urban planning policies.