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CLIMATE CHANGE AND GLOBAL SKIN HEALTH: EMERGING DERMATOLOGIC CHALLENGES, MECHANISTIC INSIGHTS, AND FUTURE PREVENTIVE STRATEGIES

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Abstract

Climate change is having a far-reaching effect on skin health in terms of overload of UV rays, air pollution, and extreme weather conditions. This is an evidence-based review that comprises extensive literature reviews (1975-2026) regarding epidemiologic relationships, mechanistic processes, and evidence-based interventions. The results indicate that every 10-mg/m3 particulate matter increment is linked to 1.3% of an increased risk of atopic dermatitis exacerbation (RR 1.013; 95% CI 0.999-1.027), and sulfur dioxide exposure is associated with an increased risk by 2.9% . Ozone depletion is a contributor to 2 percent increment in melanoma incidence per 1 percent stratospheric ozone reduction. These are meditated through the mechanistic actions of oxidative stress, the activation of the aryl hydrocarbon receptor, the disruption of barriers, and the deregulation of immunity. New clinically proven interventions are antioxidant formulations, barrier-repair, AHR-modulating agents, and climate-adapted photoprotection. Children, outdoor workers, old people, and underprivileged communities are vulnerable groups that experience unequal levels of risk. The paradigm shift in the field of climatic change is practically transforming the work of dermatologists, including the need to incorporate climate-responsive practices in everyday care and to engage in policy-making efforts to reduce emissions and to conduct specific studies aiming to bridge significant gaps in the knowledge base on how interventions work and the issue of health disparities.

1. Introduction

The twenty-first century poses the greatest danger to the global population's health in the form of climate change. The average global temperatures have already increased by about 1.3degC on average since the pre-industrial days, and it is projected that the 1.5degC level will be exceeded between 2030-2035 at current emissions levels in the trajectory [1]. This has been categorically stated by the Lancet Countdown on Health and Climate Change as a public health emergency, which has stated that the year 2024 was registered as the hottest year in human history. This global warming has set in motion an increase in the rate of extreme weather events such as unprecedented heatwaves, disastrous wildfire, catastrophic floods and extended droughts [2]. This was noted by the World Meteorological Organization, which recorded that January 2026 alone saw high temperatures in Australia, devastating rainfall in South Africa, and at the same time some extreme cold conditions in North America and Europe- a

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sign of the instability that is being experienced by our changing climate [3]. The Anthropocene concept is acknowledging that human actions, especially burning fossil fuels, have taken the center stage in determining the planetary systems and with inseparable connections to the health outcomes in human beings. The skin is the main body contact with the outside world, which has structural, immunological, and microbiological protection against an infinite number of environmental influences. Being the biggest organ, it inherits all the historical environmental exposures- a principle that is gradually becoming known as the cutaneous "exposome" . This exposome is a combination of ultraviolet radiation, pollution in the air around us, extremes of temperatures and humidity and a myriad of chemical compounds all of which combine to form the structure, functionality and susceptibility to disease in our skin [4]. Extreme environmental research has shown that long term exposure to climate stress factors has direct effects on the skin, with studies of the Antarctic showing that long-term exposure to cold and UV radiation directly increases transepidermal water loss, erythema, and melanin synthesis- objective evidence of climate-induced barriers dysfunction, and inflammatory reactions [5]. The vulnerability of the skin, as the outer organ, to the proliferating environmental changes that define our time makes skin as well as uniquely exposed to environmental shifts but also makes dermatology uniquely placed at the forefront of climatically sensitive medical activity [6]. Although there is an increasing appreciation of climate change as a health crisis, dermatology has been slow to achieve systemic incorporation of climate into clinical practice, research priorities and training curricula. The review uniquely stands out among its predecessors by using a purposeful focus on clinically proven interventions and emerging interventions to move beyond the limitations of descriptive epidemiology to fill the translational gap that presently restricts the care of patients [7]. The particular outcomes are five-fold: First, to synthesize epidemiologic data on the association of climate change with dermatologic disease in populations around the world; Second, to clarify the mechanistic pathways by which environmental stressors have cutaneous effects; Third, to critically assess evidence-based interventions and emerging therapeutic approaches with proven clinical efficacy; Fourth, to identify critical research gaps that hinder progress; and Fifth, to suggest clinical and policy frameworks that allow dermatologists to provide climate-adaptive care [8]. Covering these objectives, the review should provide the clinicians, researchers, and policymakers with the evidence base to react to the dermatologic issues of a changing climate appropriately.

2.0 Methodology

2.1 Search Strategy and Databases

An extensive literature review was carried out in several electronic databases such as PubMed/MEDLINE, Scopus, Web of Science, EMBASE and Cochrane Library to find the studies of interest discussing the association between climate change and skin health. The search dates were between January 1975 and February 2026, which covered both early research and recent publications in the ever-changing area. The search strategy was using the key terms related to climate exposure and dermatologic outcome, such as (climate change" OR global warming" OR air pollution" OR particulate matter" OR UV radiation therapeutic effects) AND (skin toxematologic outcome) derived as (skin and dermatology) or (cutaneous and melanoma) or (skin cancer and atopic dermatitis) or (psoriasis and eczema) or (acne and ultraviolet radiation). This broad based strategy has captured literature on the interdisciplinary spectrum of environmental science to clinical dermatology [9].

2.2 Inclusion and Exclusion Criteria

Inclusion criteria required that the studies be peer-reviewed original research, systematic reviews, or meta-analyses; report dermatologic outcomes associated with climate or environmental factors; assess interventions with clinical outcome data where available; and human studies (epidemiologic or interventional), with appropriate supportive evidence in the form of mechanistic in vitro or in vivo data where possible, needed to demonstrate biological plausibility [10].

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The studies that were eliminated were non-English publications that could not be translated, case reports or case series that could not be generalized to the population, and studies that did not clearly define the exposure-outcome relationships, thus could not allow meaningful synthesis to be conducted.

2.3 Quality Assessment and Data Synthesis

AI tools could dramatically change how doctors spot and identify illnesses. Deep learning models examine medical scans with impressive precision. They frequently outperform results from experienced clinicians in many areas. These breakthroughs could cut down on wrong diagnoses and help patients get better care. Catching diseases early with the help of AI screening has shown real promise in hospital studies. Rolling out these tools in hospitals and clinics takes thoughtful preparation and clear guidelines. [11]

3.0 Climate Change Drivers Affecting Skin Health

3.1 Increased Ultraviolet Radiation

The ultraviolet radiations are one of the most widely researched exposures to climate that have direct dermatologic implications. The stratospheric ozone depletion was identified as a worldwide challenge in the 1970s and this caused the development of the Montreal Protocol that has been effective in eliminating ozone depleting gases. Nonetheless, the recovery paradox has become complicated where the climate change itself can slow down ozone recovery by cooling the stratosphere and changing distribution patterns and circulation of ozone-depleting substances that modify the distribution of ozone-depleting substances and their movement [12]. These interactions result in regional differences in UV-B and UV-A patterns, with certain regions having more UV-radiation at ground-level, despite the global efforts to recover the ozone. These exposures are further intensified by altitude effects with the UV intensity rising by about 10-12 percent per 1000 meters elevation with high-altitude populations at greatest risk. Cumulative UV exposure is further amplified by behavioral changes in response to the effect of prolonged outdoor play during warm weather conditions, which leads to increased rates of skin cancer incidence around the world.

3.2 Ambient Air Pollution

The ambient air pollution has turned out to be a significant determinant of skin conditions, including various pollutants that are generated by different sources and have different cutaneous outcomes [13]. Particulate matter 2.5 mm and 10 mm are combustion products, industrial emissions, traffic, and natural sources, the geographic distribution patterns of which are concentrated in the areas of the urban center and industrial corridors. Through the combustion of fossil fuels and photochemical processes, gaseous pollutants such as nitrogen dioxide (NO2), sulfur dioxide (SO2), and the ozone formed at ground level (O3) are produced, but also volatile organic compounds are produced by both anthropogenic and biogenic sources. Other microcontaminants of interest are microplastics and nano plastics, which have been found in atmospheric fallout and potentially entering cutaneous areas, and per- and polyfluoroalkyl substances (PFAS), which are recalcitrant to breakdown in the environment and build up in the body of the human being [14]. Complicated interactions between pollution and climate phenomena, such as temperature inversion that trap pollutants close to the floor, photochemical smog establishment that is enhanced by more heat, and wildfire smoke events that are becoming more frequent and severe all augment human exposure to these dermatotoxicants.

3.3 Temperature Extremes

Climate change has essentially changed the global temperature patterns where both the heats and the cold events have become more common and more severe. The frequency, duration and severity of heat waves have increased and record temperatures are now occurring over the period at higher rates than ever recorded in instrumental records [15]. Ironically, climate change has also caused instability in polar vortices resulting in greater cold extremes and winter storms in the

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middle latitudes although the overall trends are to become warmer. Such extremes of temperatures have direct physiologic impacts on skin in several ways. Exposure to heat puts stress on thermoregulatory capacity, causing cutaneous blood flow and sweat production to rise, and extreme cold causes a decrease in perfusion and worsening of the barrier properties directly related to the exposure effect on stratum corneum lipids and proteins [16]. Temperature-related pathology occurs as a result of skin and an intermediary in temperature homeostasis.

3.4 Altered Humidity and Precipitation

Climate change that is characterized by modifications in hydrologic cycles has far-reaching consequences on the health of skin in several ways. Drought and aridification, which cover a growing geographic area, lower the humidity of the ambient air and directly impair the epidermal barrier performance, by lowering the stratum corneum hydration and changing the normal composition of natural moisturizing factors [17]. On the contrary, greater humidity in other areas favors the growth of microbes on skin surfaces and retention of sweat, which is part of the cause of intertrigo, miliaria, and inflammatory dermatoses exacerbation [18]. Flooding and heavy rainfall that are getting more frequent and more severe with the rise in climate change subject populations to waterborne pathogens, promote growth of molds in damaged housing, and promote transmission of cutaneous infections via contaminated water contact.

3.5 Extreme Weather Events

The growing rate and intensity of severe weather phenomena is one of the most obvious examples of climate change that has direct dermatologic implications. Drought-enhanced and higher temperature wildfires produce immense exposure to particulate matter in the form of smoke columns, which extend thousands of kilometers in distance, and they led to direct burn injuries and respiratory and dermatologic conditions worsened by air toxics delivery [19]. Floods and hurricanes provide grounds to spread infectious diseases, make wound healing more complicated due to the exposure to contaminated water, and alter the provision of dermatologic care because of the damage to the healthcare infrastructure [20]. With desertification and land-use change, dust storms, which are becoming more common, place particulate matter on the skin surface and spread allergens that may induce or aggravate inflammatory dermatoses. The stress-exacerbated dermatologic conditions associated with the mental health sequelae of extreme weather events, such as post-traumatic stress disorder and depression, may also be seen to have complex biopsychosocial pathways between climate disasters and skin disease.

Table 1. *Major Climate Stressors and Dermatologic Effects.*

Stressor	Exposure Pathway	Primary Dermatology Associations	Mechanism	Populations at Highest Risk
Increased UV radiation	Direct solar exposure; stratospheric ozone depletion	Melanoma, BCC, SCC, photoaging, photodermatosis	DNA damage, (CPD, 6-4PP); oxidative stress; immunosuppression; MMP activation	Outdoor workers; children; fair-skinned population; high-altitude residents
Particulate matter (PM2.5, PM10)	Inhalation; dermal deposition	Atopic dermatitis flares; acne; pigmentation	AHR activation; ROS generation; inflammatory cytokine release; barrier disruption	Urban populations; near-roadway

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		disorders; skin aging		residents; infants
Nitrogen dioxide (NO2)	Traffic-related air pollution	AD exacerbation; lentigines; eczema	Oxidative stress; barrier dysfunction; melanocyte stimulation	Children; asthma-AD comorbid patients
Sulfur dioxide (SO2)	Industrial emissions; coal combustion	Contact dermatitis; eczema flares	Direct irritant; mucocutaneous inflammation	Industrial-adjacent communities
Ground-level ozone (O3)	Photochemical formation	Inflammatory dermatosis; skin aging; barrier disruption	Lipid peroxidation; antioxidant depletion; protein carbonylation	Urban populations; summer peaks
Extreme heat	Heat wave exposure	AD flares; miliaria; intertrigo; urticaria	TRPV3 activations; sweat composition changes; pruritus; barrier protein disruption	Elderly; infants; outdoor workers
Wildfire smoke	Inhalation; dermal contact	AD exacerbation; contact dermatitis; burn injuries	PM/PAH exposure; oxidative stress; thermal injury	Fire-prone regions; firefighters
Flooding/humidity	Water exposure; indoor dampness	Cutaneous infections; wound complications; AD flares	Microbial proliferation; barrier maceration; mold allergens	Disaster-affected; low-income housing
Microplastics/nanoplastics	Dermal contact; ingestion; inhalation	Barrier disruption; inflammation; contact dermatitis (emerging evidence)	ROS; IL-1/IL-6/TNF signaling; fibroblast senescence	General population (ubiquitous)

4.0 Epidemiologic Evidence: Climate Change and Dermatologic Diseases

4.1 Skin Cancer

4.1.1 Melanoma

The occurrence of melanoma has also shown impressive relationships with the climate change factors in various geographical locations. The trend of incidence across the globe depicts an average growth of 4 percent each year in the Scandinavian nations where there is an evident correspondence between the length of the summer and the escalating cases of melanoma. The association between melanoma and ultraviolet radiation is intermittent with a high exposure pattern, i.e. recreational sun exposure on a day with good weather would be more risky than cumulative exposure in the work environment [21]. This has been caused by ozone depletion and a 1 percent loss in stratospheric ozone is linked to a 2 percent rise in the incidence of melanoma and

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a 1 percent decrease in stratospheric ozone. These risks are amplified by behavioral amplification, which means outdoor recreational activities are promoted by warmer temperatures and the total sun exposure time is not accompanied by an equal increase in protective behavior, suggesting that people are not adopting protective measures correspondingly (Taylor et al., 2015).

4.1.2 Keratinocyte Carcinomas

Basal cell carcinoma and squamous cell carcinoma are both strongly related to cumulative sun exposure, and so highly vulnerable to alterations in UV patterns induced by climate changes. The occupational risk is also high, as the outdoor workers and the employees of the agricultural sector are at a very high risk because of the long exposure period during the peak hours of UV sunlight [22]. Geographic redistribution of keratinocyte carcinomas has been revealed, whereby increasing incidence is found in historically low-risk areas such as Scandinavia and Canada where climate change has increased exposure to UV during seasons and distorted sun exposure behaviours.

4.1.3 Behavioral and Demographic Paradoxes

The phenomenon of pleasant weather paradox explains that the better local weather, the more people tend to be active outside and, at the same time, less protective because people think that sunny warm days can be used as an opportunity to enjoy the moment and not to get cancer [23]. Efforts to overcome this paradox through migration have resulted in the fact that migrations between low-UV and high-UV areas often do not lead to proper adoption of sun protective practices by the populations despite the exposure of these populations changing dramatically with migration [24]. Both protection and detection have socioeconomic gradients, with more access to sun protection by high-income populations and more recreational sun exposure by low-income populations, and more occupational exposure by lower-income populations and less access to skin cancer screening and treatment.

4.2 Atopic Dermatitis and Eczema

4.2.1 Air Pollution Associations

The association between atopic dermatitis and air pollution has been measured in meticulously more precise manners through meta-analyses [25]. Every 10-mg /m3 rise in the PM10 level causes a relative risk of 1.008 AD-associated healthcare visits, which is good-certainty evidence. In the case of PM 2.5, the relative risk is 1.013 with respect to each 10-mg/m3 increase, but with moderate confidence because of the heterogeneity of studies. Sulfur dioxide shows the highest association with each 10-mg/m3 of AD exacerbations associated with a relative risk of 1.029, and this is high-certainty evidence [26]. The relative risk of nitrogen dioxide is 1.014 per 10-mg/m3 and the pooled estimate has moderate certainty.

4.2.2 Temperature and Weather Effects

High temperatures worsen atopic dermatitis by several goals such as the activation of TRPV3 channels in keratinocytes and changes in sweat constituents and retention as well as. Extreme cold on the other hand interferes with functioning of barriers, causes xerosis and pruritus via other mechanisms [27]. The changes in humidity result in two-sided risks, where the low humidity affects the barrier functioning by dehydrating the stratum corneum, and the high humidity enhances the growth of microbes and the sweat retention diseases [28]. There are associations between precipitation and more severe AD, but the evidence is of low certainty because of confounding factors [29].

4.2.3 Geographic and Temporal Patterns

Gradients of urban-rural areas always reveal greater burden of atopic dermatitis in urban areas with traffic and industrial pollution as the primary contributors [30]. Seasonal variation indicates the complicated interactions between pollution and temperature with the spring and fall periods frequently reporting the highest exacerbations as pollution and temperature differences are most intense in these periods [31]. Volatile weather changes in the short-term have also become a possible trigger of exacerbation that does not

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depend on the absolute temperature or pollution rates another factor is climate volatility [32].

4.3 Psoriasis and Inflammatory Dermatoses

The relationship between temperature and psoriasis exhibits a complicated distribution with the cold-weather exacerbation being the dominant phenomenon in the majority of cases and some patients having the exacerbations caused by heat exposure [33]. Such benefits and harms happen especially with UV exposure in the case of psoriasis where advantageous UV therapy can be applied to the patient, whereas untamed exposure by the environment can lead to flares and heighten the chances of skin cancer onset [34]. The effects of pollution act via activation of aryl hydrocarbon receptors in the keratinocytes, which cause inflammatory cascades that can cause or worsen psoriatic lesions [35]. An indirect mechanism is the stress-mediated flares, where climate-related psychological discomfort due to extreme weather conditions or relocation triggers inflammatory dermatoses via neuroimmunological mechanisms [36].

4.4 Acne Vulgaris

Acne has been linked to pollution, and studies have been accumulating into the direct effects of particulate matter on sebocyte activation and sebum production. Heat and humidity play a role in the pathogenesis of acne by maintaining sweat, blocking the ducts, and changing the cutaneous microbiome [37]. Lipid peroxidation in sebum has been found as one of the effects of ozone, which may elevate potential comedogenicity and inflammatory effects. There is growing evidence to implicate traffic-related pollution with prevalence of acne with people living around major roadways exhibiting increased rates of both inflammatory and comedonal acne [38].

4.5 Photoaging and Pigmentary Disorders

Photospecific UV-pollution aging is a pathological interaction, in which particulate matter and ultraviolet radiation result in faster matrix degradation than that induced by either exposure. Formation of Lentigines has certain connection to exposure to nitrogen dioxide, which involves the traffic pollution in the formation of hyperpigmented facial hair regardless of the presence of UV radiation [39]. In melasma, there are complicated interplay between UV radiation, heat and pollution, the factors each may have a role in the activation of melanocytes and deposition of pigments.

4.6 Infectious Dermatoses

4.6.1 Vector-Borne Diseases

Distribution Lyme disease has shown geographic expansion owing to the warming climate, with the Ixodes ticks expanding their range to latitudes and altitudes that used to be inhospitable to them [40]. The change in the transmission areas of leishmaniasis is occurring due to increasing the geographic range of the sandfly vectors where temperatures are rising [41]. Aedes mosquitoes transmit dengue and chikungunya viruses, which are on the rising range as the climate changes lower mortality in winter and increase periods of transmission.

4.6.2 Waterborne and Soil-Borne Infections

Infections caused by flooding are wound infections due to the exposures of water contamination and infections caused by dermatophytes due to prolonged wetness [42]. Recreational water contact is a risk factor due to the expansion of geographic range of Vibrio species and marine mycobacteria by warmer waters. Infections associated with displacement including scabies and cutaneous larva migrans are becoming more common among the population that is displaced due to a disaster caused by climate change and lives in a temporary shelter with a large number of people [43].

4.7 Contact Dermatitis and Irritant Dermatitis

The irritant contact dermatitis due to pollutants is the direct toxicity of stratum corneum and underlying epidermis to the particulate matter and gaseous pollutants. Climate alteration of allergens has been proven in the case of poison ivy, whereby an increase in CO2 in the atmosphere enhances the strength of urushiol and growth of the plant [44]. Such exposures arise in the changing climates, as workers in outdoor industries suffer

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prolonged exposure periods and new allergen or irritant profiles [45].

5.0 Mechanistic Insights: From Environment to Pathology

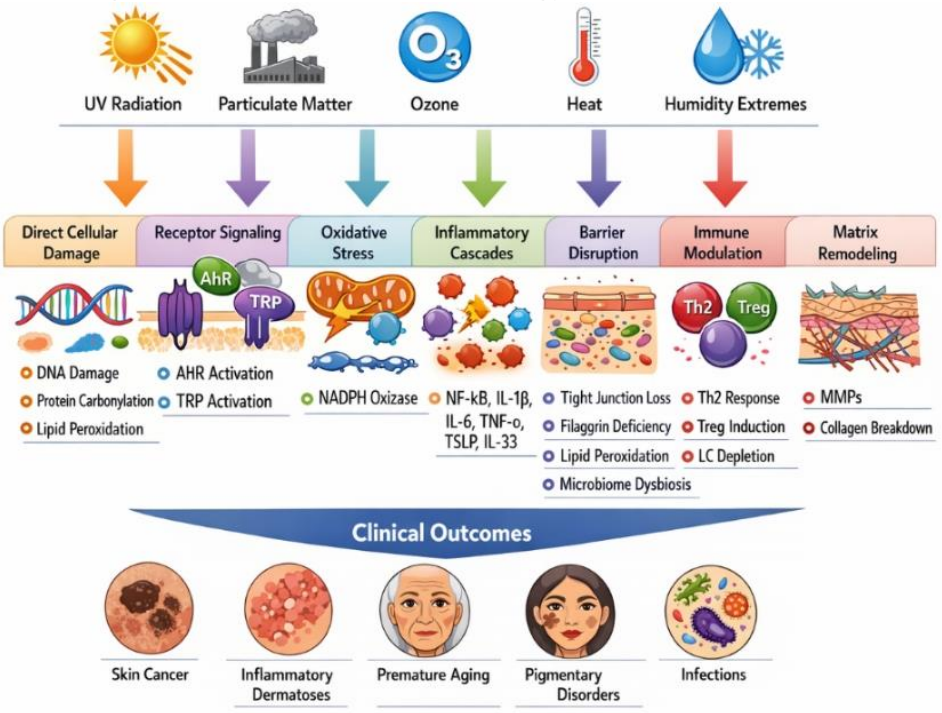

Figure 1. *Mechanistic Pathways of Climate-Induced Skin Damage. Integrated mechanistic model of climate-induced cutaneous pathology. Environmental stressors (UV radiation, particulate matter, ozone, heat, humidity extremes) activate convergent signaling pathways: (1) Direct cellular damage DNA photoproducts, protein carbonylation, lipid peroxidation; (2) Receptor-mediated signaling aryl hydrocarbon receptor (AhR) activation by PAHs, transient receptor potential (TRP) channel activation by heat; (3) Oxidative stress mitochondrial ROS, NADPH oxidase activation, antioxidant depletion; (4) Inflammatory cascades NF-κB, MAPK, inflammasome activation, cytokine release (IL-1β, IL-6, TNF-α, TSLP, IL-33); (5) Barrier disruption—tight junction protein downregulation, filaggrin deficiency, lipid barrier oxidation, microbiome dysbiosis; (6) Immune modulation Th2 polarization, Treg induction, LC depletion; (7) Matrix remodeling MMP activation, collagen degradation, elastosis. These pathways collectively drive clinical phenotypes: skin cancer, inflammatory dermatoses, premature aging, pigmentary disorders, and infections.*

5.1 Oxidative Stress Pathways

Oxidative stress is an archetypal constituent unifier by which various climate-related exposures produce cutaneous damage. Reactive oxygen species are generated by ultraviolet radiation, particulate matter, ground-level ozone and polycyclic aromatic hydrocarbons, both in a direct photochemical reaction and indirect cellular pathway [46]. These radicals of oxygen such as superoxide anion, hydrogen peroxide and hydroxyl radicals trigger lipid peroxidation that undermines membrane integrity and produces secondary reactive aldehydes that can alter proteins and DNA. Most susceptible to the action of antioxidants is the stratum corneum, the most external barrier, where lipophilic antioxidants, especially vitamin E, get used up in the neutralization of external oxidants [47]. Mitochondrial dysfunction is a downstream effect, where accumulating oxidative damage compromises electron transport chain activity and forms a vicious circle of more ROS production and cellular death.

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5.2 Aryl Hydrocarbon Receptor Signaling

The aryl hydrocarbon receptor is an important receptor to environmental toxicants, specifically, polycyclic aromatic hydrocarbons and dioxins found in air pollution and combustion products. When AhR binds the ligand, it relocates to the nucleus and activates the transcription of xenobiotic-metabolizing enzymes such as CYP1A1 and CYP1B1, which although intended to help neutralize them, in fact cause more oxidative stress during metabolism. AhR stimulation induces the release of inflammatory cytokines and impairs terminal differentiation in keratinocytes [48]. AHr signaling in melanocytes has the potential to induce melanogenesis, which leads to hyperpigmentation attributed to pollution. AhR stimulation in fibroblasts facilitates the expression of matrix metalloprotease and inhibits collagen production. The discovery of the central role of AhR has triggered the development of therapeutic agents, such as tapinarof, which also exhibits clinical effectiveness in inflammatory dermatoses and has potential in pollution-exposed groups.

5.3 Epithelial Barrier Disruption

5.3.1 Tight Junction and Cornified Envelope Integrity

Climate stressors have a direct negative effect on structural integrity of the epidermal barrier in a variety of ways. Temperature extremes release heat shock proteins which disorganize tight junction proteins such as occludin and zonula occludens-1 to raise paracellular permeability. Exposure to particulate matter suppresses the expression of major cornified envelope proteins such as filaggrin, loricrin, and involucrin lowering the mechanical integrity of the stratum corneum [49]. Ozone is a highly reactive gas and direct oxidation of stratum corneum lipids releases the organized lamellar structures that are critical to the barrier activity of these lipids. All these effects augment water loss via the trans-epidermal route, augment environmental allergen and irritant infiltration and provoke inflammatory activity that further impairs barrier integrity.

5.3.2 Microbiome Alterations

There are both direct and indirect impacts of climate change on skin microbial communities in the form of altered growth conditions of microbes and the host environment respectively. The changes in temperature and humidity cause the selection of certain bacteria or their rejection, which may result in the relocation of the community structure toward the pathogenic or pro-inflammatory ones [50]. In atopic dermatitis, dysbiosis, or depletion of microbial diversity and overgrowth of pathogenic microbes such as Staphylococcus aureus have been reported and can be flattened by climatic-induced environmental shifts. The disturbance of barrier-microbiome-immune crosstalk leads to self-reinforcing processes whereby barrier weakness is followed by microbial colonization which are then followed by immune response that further degrades barrier activity.

5.4 Immune Dysregulation

Environmental stressors regulate cutaneous immune responses by various ways. Th2 polarization that causes IL-4 and IL-13 are up-regulated by air pollutants, especially those that activate AhR, which is a mechanistic explanation of pollution-related atopic disease. Th17 role in psoriasis related to pollution has been shown whereby, particulate matter induces IL-23 and IL-17 in vulnerable individuals [51]. Ultraviolet radiation causes immunosuppression by activating the regulatory T cell and Langerhans cell depletion, which is also associated with skin cancer and may help to regulate the response to cutaneous infections. The NLRP3 inflammasome gets triggered by particulate matter in keratinocytes and immune cells, which release IL-1b and IL-18 that promote inflammatory dermatoses.

5.5 DNA Damage and Carcinogenesis

Ultraviolet radiation causes typical DNA damage in the form of cyclobutane pyrimidines dimers and 6-4 photoproducts, when left unrepaired, causes characteristic mutagenesis of tumor suppressor genes, such as TP53 in keratinocyte carcinomas and PTCH1 in basal cell carcinoma. Pollutants

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have an effect in carcinogenetics co-carcinogenic with polycyclic aromatic hydrocarbons that form DNA adducts which, although not equivalent to UV damage, can cause carcinogenesis and interact with UV to amplify tumor growth [52]. Oxidative stress can affect the DNA repair ability directly by making changes in the repair enzymes themselves and also by exhausting the cellular energy resources required in the repair processes. Chronic oxidative stress acceleration of telomere shortening could be the cause of cell senescence and age-associated cancer risk.

5.6 Extracellular Matrix Degradation

The up-regulation of matrix metalloproteinase is one of the most significant mechanisms between the exposure to the environment and skin aging. Both ultraviolet radiation and pollutants cause MMP-1, MMP-3 and MMP-9 expression via AP-1 and NF-kB signals, which result in collagen degradation and deposition of disorganized elastin substance, which is a characteristic feature of photoaging and pollution aging results [53]. The breakdown of collagen triggers a positive feedback process in that fragmentation of collagen via the signal transmitted by integrin receptor stimulates additional MMP expression and dysfunction in the fibroblasts. Photoaged skin fibroblasts acquire a senescent-associated secretory phenotype, producing inflammatory mediators that further extend and enhance matrix breakdown and impede tissue repair.

5.7 Neurogenic Inflammation and Pruritus

Direct heat and chemical irritant sensors and in particular the temperature sensitive ion channels include TRPV1, TRPV3 and TRPV4, which detect pollutants and other environmental hate [54]. Neuropathway activation of these channels on sensory nerve endings stimulates the release of neuropathway neuropeptides, such as substance P and calcitonin gene-related peptide, which cause vasodilation, mast cell degranulation, and inflammation cell recruitment. This electrogenic inflammation mediates pruritus via neural effects on nerve-fibres and via neural effects via inflammatory mediators via sensitisation of itch pathways. The pruritus-inflammation loops increase the activity of the disease, scratching leads to further damage to the barrier increasing the activity of the sensory nerves further.

6.0 Clinically Proven Treatments and Emerging Therapeutic Approaches

The increase in the load of climate-related skin disease has triggered the creation of interventions aimed at particular environmental stressors. This area is a critical evaluation of the clinically proven therapies and the new emerging therapies based on the supporting evidence provided by randomized controlled trials and high-quality clinical studies [55].

6.1 Photoprotection: Beyond Sunscreen

The current photoprotection practice has gone beyond UV filtration to help in managing the synergies between UV radiation and other climate stressors. Antioxidant-enhanced broad-spectrum sunscreens are also a breakthrough, with an RCT showing that a combination of UV filters with vitamins C, E and ferulic acid have a stronger effect on oxidative stress markers than UV filters. Adverse effects Clinical Clinical effects are lower erythema, lower pigmentation and better barrier properties but formulation factors such as photostability, substantivity and cosmetic acceptability are important to adherence. Complementary benefits are provided by oral photoprotection. Polypodium leucotomos extract has been shown to have RCT evidence of minimizing photodamage at minimal erythema dose and pigmentation. There is some evidence of photoprotection by some carotenoids such as lycopene and b-carotene, but the effects on cancer prevention are inconclusive [56]. Above all, the phase III ONTRAC trial established that nicotinamide 500 mg twice a day prevents new non-melanoma skin cancers by 23% in high-risk patients after 12 months (NCT01509911). There is emerging clinical evidence of photoprotective activity of polyphenols such as green tea extracts and resveratrol. Protection that is based on textiles is not utilized fully although its efficacy is proven to be effective. The UPF-rated clothing is the dependable protection that covers a broad spectrum in real-life situations, whereas the broad-brimmed hats and accessories provide the adjunctive protection [57]. Shade structures are

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significant urban planning interventions at the population level to protect people against UV at the community level.

6.2 Barrier-Repair Therapies for Pollution/Temperature-Associated Dermatitis

Intervention of climate-related barriers disruption necessitates specific treatment. Ceramide-containing preparation, specifically lipid-based barrier preparations have RCT evidence to support their use in the treatment and prevention of atopic dermatitis [58]. Filaggrin breakdown products (natural moisturizing factors) supplementation helps correct individual deficiencies in the polluted skin. Clinical evidence shows that there is less transepidermal water loss, enhanced stratum corneum integrity, and less flare frequency with regular use. Anti-pollution topicals is a novel category that is getting increasingly clinical supported. Physical resistance to the deposition of particulate matter is formed by film forming polymers and imaging experiments have shown less of the particle adhesion [59]. Industrial pollution includes metal ions, which are bound by the chelating agent with controlled exposure demonstrating less inflammatory markers. Temperature-adaptive skincare acknowledges that seasonal changes in formulation (warmer emolliens in winter, lighter in summer) enhance compliance and effect in AD patients.

6.3 AhR-Modulating Therapies

Ayl hydrocarbon receptor (AHR) pathway as an outcome of polycyclic aromatic hydrocarbon pollution-induced activation has become one of the central targets of treatment. Tapinarof, a first-in-class AhR agonist, exhibits paradoxical protective actions by use of antioxidant response element activation. Psoriasis (PSOARING 1/2) and atopic dermatitis (ADORING 1/2) Phase III RCTs have been associated with a substantial PASI score improvement with a rapid onset and positive safety profiles [60]. It is worth noting that there are no contraindications of climate-related triggers with tapinarof, which makes it especially useful with populations exposed to pollution. Potential avenues of preventative use of AhR modulators in high-risk setting are provided by additional modulators of the AhR.

6.4 Antioxidant Therapies

Topical antioxidants have direct counteractions of UV and pollution-induced oxidative stress. The RCT evidence of vitamin C (L-ascorbic acid) suggests protection against ultraviolet radiation and collagen. There are synergistic effects of vitamin E with vitamin C, and the UV protection is supported by the RCT data. Ferulic acid preserves and increases vitamins C and E, and RCT has shown better photoprotection. The clinical evidence of anti-aging effects of coenzyme Q10 and idebenone on environmentally exposed skin has been established. The polyphenol-based formulations are promising and green tea polyphenols (epigallocatechin-3-gallate) have been shown to reduce UV-induced erythema and DNA damage by reducing damage of DNA in clinical trials. Resveratrol has potential clinical data whose limits have to be identified, whereas curcumin is limited by bioavailability factors that require nanoparticle preparations in studies. Activators of Nrf2 are a new approach and sulforaphane of broccoli sprout extract has already passed Phase II trials to protect against UV radiation. There is an active development of topical formulations [61].

6.5 Anti-Pollution Skincare: Clinical Evidence Base

Formulations that contain particulate matter-removing have proven to be effective in clinical trials, with dedicated cleansers having been shown to decrease the amount of cutaneous particles. Film-forming component leave-on protectants have clinical support of sustained protection. Anti-inflammatory topicals such as niacinamide, have been shown through RCT evidence to reduce inflammation, improve barrier functioning and reduce pigmentation of pollution exposed skin [62]. Panthenol demonstrates clinical effect in barrier repair whereas the Centella asiatica extracts have recent clinical evidence on pollution-exposed skin.

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6.6 Climate-Adapted Management of Inflammatory Dermatoses

The dynamic treatment algorithms are necessary in the treatment of atopic dermatitis under climate triggers conditions. Proactive and reactive therapy modification according to the forecasts of pollution and weather, the treatment of seasons with more intensity, as well as incorporation of environmental control in the management strategy are evidence-based. In the case of psoriasis, UV-based treatment needs a balanced attention towards therapy and carcinogenic risk of the environment, which is escalated by climate, and heat-related flare prevention measures are in the spotlight [63].

6.7 Emerging Targeted Therapies

TRP channel modulators treat heat induced dermatoses. TRPV1 antagonists are currently being trialled in pruritus and inflammatory skin dermatoses, and TRPV3 inhibitors demonstrate preclinical efficacy in heat-induced flares of AD. The Mitochondrial-targeted antioxidants such as MitoQ and SkQ1 are under clinical trials in aging and inflammatory diseases and are of direct relevance to climate-related oxidative stress [64]. Cellular senescence accelerating with climate is the focus of senolytics and senomorphics applied in early clinical trials in age-related disease.

6.8 Systemic Therapies and Climate Considerations

Systemic therapies especially the biologics bring climate considerations other than efficacy. The stability of the temperature throughout shipping and storage, cold chain, and the environmental impact of the packaging waste and transportation emissions should be considered in the choice of the treatment. The development of the formulation should be grounded in sustainability so that the carbon footprint in dermatologic care development is minimized without harm to therapeutic efficacy [65].

Table 2. *Clinically Proven and Emerging Interventions for Climate-Related Skin Conditions.*

Intervention Category	Specific Agent/Approach	Mechanism of Action	Clinical Evidence Level	Key Outcomes	Target Climate Stressor
Oral photoprotection	Nicotinamide	Enhancement of DNA repair; ATP production	Phase III RCT (ONTRAC)	23% reduction in new NMSC at 12 months; NNT = 11	UV radiation
	Polypodium leucotomos	Antioxidant; photoprotective	Multiple RCTSs	Reduce MIN-induced erythema; decreased pigmentation action	UV radiation
	Carotenoids (lycopene, β-carotene)	Free radical scavenging	RCTs (mixed evidence)	Protection against UV-induced erythema; limited prevention on data	UV radaiation

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Topical photoprotection	Broad-spectrum UV filters + antioxidants	UV absorption/reflection + ROS neutralization	RCTs	Superior reduction in oxidative stress makers vs. UV filters alone; decreased pigmentation	UV + pollution
AHR modulation	Tapinarof	AHR agonist (with antioxidant response)	Phase III RCTs (PSOARING, ADORING)	PASI 75: 35-40%; EASI 75: 45-50%; rapid onset	Pollution (PAH-mediated effects)
Barrier repair	Ceramide-dominant formulations	Lipid supplementation; barrier reinforcement	RCTs (multiple)	Reduced TEWL; decreased AD flare frequence; improve SC integrity	Temperature extremes; low humidity; pollution
	Filaggrin breakdown product formulations	NMF supplementation	RCTs	Improved hydration; reduced TEWL; decreased pruritus	Low humidity temperature extremes
Anti-pollution topicals	Film-forming polymers	Physical barrier against particle deposition	Controlled exposure studies	Reduced particle adhesion; decreased inflammation markers	PM-PAHs; urban pollution
	Chelating agents	Metal ion binding	In vitro + limited clinical	Reduced metal-induced oxidative stress	Industrial pollution
Antioxidants	Vitamin C (L-ascorbic acid) +Vitamin E + Ferulic acid	ROS scavenging; UV protection synergy	RCTs	Reduced UV-induced erythema; decreased MMP expression; collagen stimulation	UV; pollution

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	Green tea polyphenols (EGCG)	Antioxidant; anti-inflammatory	Clinical studies	Reduced UV-induced erythema; decreased DNA damage	UV; pollution
	Niacinamide	Anti-inflammatory; barrier improvement	RCTs	Reduced inflammatory lesions; improved barrier function; decreased pigment action	Pollution; UV
TRP channel modulators	TRPV1 antagonists	Inhibition of heat/itch signaling	Phase II/III trials	Reduced pruritus in AD, psoriasis	Heat extremes
Climate-adapted management	Environmental monitoring-based treatment adjustment	Personalized prophylaxis based on triggers	Observational studies	Reduced exacerbation; improved QoL	All climate stressors
Sustainable biologics	Temperature-stable formulations	Reduced cold chain requirements	Pharmacokinetics	Maintained efficacy with reduced environmental footprint	General (practice sustainability)

7.0 Vulnerable Populations and Health Disparities

Not all populations are impacted equally by climate change with some groups being more at risk of contracting dermatologic disease because of their physiological state, work practices, socioeconomic status, and lack of access to protective resources.

7.1 Children

Children are one of the most vulnerable groups in terms of skin disease related to climate. They have immature barrier action of their developing skin, and have an increased ratio of body surface area to weight that enhances percutaneous absorption of environmental pollutants. The immune system is especially vulnerable to allergic sensitisation in the developing stages and behavioural attributions such as prolonged outdoor activities and total reliance on caregivers to protect against the sun increase vulnerability to these dangers. These weaknesses are reflected in disease patterns in children, where atopic dermatitis predominates and predisposition to cutaneous infections is high in extreme weather conditions. More worryingly, studies that focus on guiding climate changes among children are few, posing significant evidence gaps that can be used in prevention and treatment of such groups [66].

7.2 Elderly

Age-related physiological changes present unique dermatologic issues to the elderly population due to the effects of climate. Decreased lipid production, thinning of epidermis and decreased antioxidant activity interfere with the barrier function and repair processes. Dysfunctional thermoregulation raises the risk of morbidity due to heat, such as miliaria and heat stroke, and a lifetime accumulating environmental exposure is

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observed as skin cancer. Polypharmacy also contributes to vulnerability with many drugs producing photosensitivity reactions which get worse with more UV radiation [67].

7.3 Outdoor Workers

Outdoor workers represent a group with a high risk of occupational UV exposure two to three times greater than indoor workers. Farmers also have other risks such as exposure to pesticides and zoonotic diseases, whereas those working in construction and in the streets experience combined heat stress and particulate air pollution without proper work protection measures at the workplace. Outdoor workers have a reduced productivity in extreme heat conditions, but the people in informal sector such as construction workers, street vendors are the most exposed people and often have no formal social protection or insurance. Evident climate effects can be seen in Kenya, with tea plantation workers and motorcycle taxi operators developing hyperpigmentation, melasma and photocontact dermatitis but there is no financial means to access sunscreen, which is many times the day pay [68].

7.4 Pregnant Women and Fetuses

The pregnant women and fetus are a uniquely vulnerable dyad, which needs special attention. Pollutants may have transplacental effects on the development of fetal skin and immune programming, which may predispose them to future allergic disease. Due to climate stress, pregnancy dermatoses can face a different manifestation and severity, and the post-partum consequences on the skin of newborns have not been studied in depth yet [69].

7.5 Immunocompromised Populations

Immunocompromised patients show a high susceptibility to UV-induced carcinogenesis, and both organ transplantation recipients and their risk of UV-induced skin cancer are very high and synergistic with an increasing ambient UV radiation. This population is most at risk of infection by the climate-sensitive pathogens, as well as, many immunosuppressive drugs cause photosensitivity, resulting in dangerous interactions with the growing sun exposure [70].

7.6 Migrants and Displaced Populations

Migration of the climate presents unique ermatologic susceptibilities because people relocate between low-UV and high-UV areas without sun-protective behaviours of adaptation or sufficient acclimatisation. Multiple factors contribute to overcrowding of refugee populations, which makes it easier to transmit scabies and cutaneous infections, in addition to having minimal healthcare access and poor hygiene conditions. These are environmental and occupational dermatologic exposures, which are latent factors of migrant dermatoses, the need to be monitored and intervened to address them [71].

7.7 Low-Income and Marginalized Communities

Environmental justice models demonstrate that the poor communities experience an unequal burden of skin diseases associated with climate changes. Such groups are more exposed to pollution and urban heat islands and do not have access to other protective measures, such as sunscreen, air conditioning, and health services. Deficiency of housing quality in the form of mold, dampness, insufficiency of insulation of houses and other features further increases these risks, providing conditions that worsen inflammatory dermatoses [72]. The UN Environment Programme points out that access to cooling is not and not supposed to be a luxury of the rich since it all comes down to health and dignity.

7.8 Global South Disparities

Populations in Global South have the worst climate related dermatologic problems and the least resources to mitigate them. The exposure to UV in the equatorial areas is the highest in the world, however, dermatologic infrastructure is severely wanting. Climate change increases the number of neglected tropical diseases such as cutaneous leishmaniasis, mycetoma and scabies, as the shifting ecosystems increase the range of transmission. Individuals with albinism have a high risk of skin cancer extending to 1,000 times

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that of the general population and lack adequate access to sun protection. In Kenya, where temperatures increased as high as 2.1o C since record keeping started, outdoor laborers develop photodamage that is visible to the eye but do not use sunscreen due to cost reasons [73]. Health records are incomplete with no classification systems that can be used in monitoring climate diseases, and African populations are also underrepresented in worldwide dermatological studies. To resolve these inequalities, there is an urgent need to take interdisciplinary action to develop climate-resilient health systems and safeguard skin health equity in an ever-warming world.

Table 3. *Vulnerable Populations, Risk Factors, and Targeted Interventions.*

Population	Physiologic/Social Vulnerability factors	Climate Stressors of Greatest Concern	Associated Dermatologic Conditions	Targeted Interventions (Evidence-based)	Policy/Research priorities
Children	Immature barrier function; higher BSA: weight; developing immune system; dependence on caregivers	UV radiation; air pollution; extreme heat	Atopic dermatitis; infections; photodermatoses	Pediatric-appropriate sunscreens; barrier-repair formulations; school-based sun safety programs	Pediatric-specific RCTs; child-safe anti-pollution formulations; school climate curricula
Elderly	Thinner epidermis; reduced lipids; impaired thermoregulation; cumulative exposure	Heat extremes; UV; pollution	Skin cancer; xerosis; heat-related dermatoses	Regular skin cancer screening; emollients; heat wave response plans	Geriatric dermatology climate research; age-appropriate photoprotection guidelines
Outdoor workers	Occupational exposure; inadequate protection; limited control over environment	UV; heat; pollution; wildfires	Skin cancer; actinic keratoses; contact dermatitis	Workplace sun protection programs; UPF clothing; shaded rest areas; health surveillance	Occupational exposure limits; workers compensation for climate-related disease; enforcement of protections
Low income communities	Housing quality (mold, poor insulation); limited AC; pollution proximity; healthcare access barriers	Heat; flooding; pollution; indoor dampness	AD exacerbations; infections; infestations	Subsidized cooling; housing remediation; community health worker programs	Environmental justice polices; climate-resilient housing standards; healthcare access expansion

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Global South populations	Equatorial UV; limited dermatologic infrastructure; NTD burden; albinism prevalence	Extreme UV; heat; changing vector zones	Skin cancer (albinism); NTDS; infections	Sunscreen distribution (albinism programs); teledermatology; task-shifting training	International climate health financing; essential medicines access; health system strengthening
Climate migrants	Acclimatization gap; protection behavior absence; healthcare disruption	UV exposure changing; new disease vectors	Sunburn; skin cancer; unfamiliar infections	Targeted education at migration corridors; culturally adapted sun safety messaging	Migration-aware health system; surveillance of migrant skin health
Immunocompromised	Enhanced carcinogensis; infection susceptibility; medication photosensitivity	UV; emerging pathogens	Skin cancer; opportunistic infections	Rigorous photoprotection; infection prevention; adjusted medication timing	Drug-environment interaction studies; immunosuppression-specific guidelines

8.0 Future Preventive Strategies and Public Health Approaches

8.1 Individual-Level Prevention

Individualized environmental risk assessment is a new area of climate-adapted dermatology, which combines the local air quality and UV indices with clinical decisions that offer patients real-time and place-specific protection recommendations. Personal exposure sensors Wearable sensors are being developed that may be used to track UV dose, deposition of particulate matter, and temperature exposures in real-time which may be used to make a personalised recommendation on protection measures. The use of smartphone applications that provide real-time protection recommendations based on the prevailing environmental factors has been promising in enhancing sun protection practices although their application with clinical care is still in the periphery of their use. Seasonal adaptive algorithms of climate-appropriate skincare programs necessitate different environmental difficulties over the course of the year. Pollution-specific product choice, which involves film forming barrier protectants and antioxidant laden formulations, deals with geographical and temporal differences in air quality. Temperature-responsive formulations are a new technology, and the development of materials science allows topical products, in which the properties change in response to ambient conditions [74]. The basic interventions include behavioral interventions to prevent exposure to climate conditions at an individual level, and shade-seeking behavior, avoiding outdoor activities during the hours with the highest UV levels, and wearing protective clothing are the primary defenses against climate-induced skin damage. Community-based programs have shown efficacy in enhancing sun protection behaviors, and multi-component programs have been successful in ensuring long-term behavioral change in various groups of people.

8.2 Clinical Practice Integration

Environmental health screening must become a routine practice in dermatology, which includes systematic evaluation of occupational and

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residential exposures, record of occupational and residential hazards, and monitoring of disease remittance with regard to environmental antecedents. The information allows climate-informed planning of treatment, such as choice of therapies depending on the environmental setting, treatment pre-emptive deepening during high-risk seasons like pollution outbreaks or heat waves, and teledermatology use in case of extreme weather conditions that can limit access to healthcare services. Education in dermatology workforce needs to incorporate climate health competencies in training programs and through continuing medical education programs that deal with climate-dermatology interconnections. The close interdisciplinary cooperation with environmental health professionals, allergists and occupational medicine specialists will be necessary to provide the complete patient care [75].

8.3 Community and Population-Level Strategies

Skin health benefits at the population level provided by urban planning interventions can be discussed in a variety of ways. The green spaces also help reduce pollution and cooling thereby reducing heat island effects as well as air quality issues. Shaded parks minimize UV rays within outdoor communities whereas cool roofs and sidewalks minimize ambient temperatures within urban settings. The programs of tree planting can solve the two issues at the same time: the quality of the air is getting better, and the shades are provided. Behavioral change evidence has been presented by school-based programs such as sun safety education, and curriculum-based programs have resulted in prolonged changes in sun protection behaviors of children. Playground shade structures have been shown to offer controlled UV protection, and policies to get access to sunscreen to prevent outdoor activities. Occupational exposure limits, which acknowledge the risk particularities of outdoor worker climate-related skin disease, are necessary in the workplace of outdoor workers. The programs on prevention of heat stresses and the supply of protective gears such as UPF-rated clothing and sunscreen are significant employer requirements [76].

8.4 Health System Adaptation

Extreme weather conditions demand disaster preparedness of climate resilient dermatology services to maintain continuity of care during floods, wildfires and hurricanes. The supply chain resilience of dermatologic drugs, especially biologic drugs that are temperature sensitive, need to deal with the weaknesses revealed by climate-related disruptions. The infrastructure of telemedicine is a back-up during the time when the face-to-face care is not possible because of the extreme weather conditions. Sustainable dermatology practice targets the climate contribution of the healthcare sector by minimizing waste production by using biodegradable materials and minimal packaging, optimizing biologic therapy to reduce the use of cold chains, and creating low-carbon methods of procedural dermatology [77]. The surveillance systems such as climate sensitive disease registries can be used to early warn about a disease outbreak and also detect developing dermatologic threats and have the information used to respond to the public health.

8.5 Policy and Advocacy

Evidence that PM2.5, NO 2, and SO 2 are associated with skin disease should guide air quality standards toward more stringent limits that ensure the health of the dermatology. Health impact assessment should incorporate dermatologic outcomes in order to capture all the advantages of pollution reduction. Climate mitigation policies are the skin health protection, where emission abatement generates both short- and long-term dermatologic results. The Montreal Protocol forms an example of an insight on how international environmental agreements can provide quantifiable skin cancer prevention by means of protection of the ozone layer [78]. The priorities in the funding of research need to have climate-dermatology programs that are oriented towards longitudinal studies that allocate standardized exposure assessment and intervention trial of climate-adaptive strategies. In the absence of this investment, evidence base required to guide clinical practice will not be sufficient enough.

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9.0 Critical Research Gaps and Future Directions

9.1 Epidemiologic Gaps

Although the effects of climate change on skin health are becoming well known, there is still a lot of epidemiologic gaps that reduce our understanding of such associations. Longitudinal studies that follow individual level exposures and outcomes over decades have not been done thus causing lack of causal association and identification of critical exposure windows. Certainly, the lack of pediatrics and old-age population representation in climate-dermatology studies implies that developmental and age-associated vulnerabilities are not well defined yet. The limited information that exists in the Global South where the effects of climate are the most serious and where the dermatologic infrastructure is the poorest, presents an ironic circumstance in which the most affected individuals are the least researched [79]. The lack of standardized measures of climate volatility and health impacts and inconsistent exposure measures and outcome definitions between studies (e.g. of how rapid weather changes impact skin disease) make meta-analyses and evidence synthesis difficult, whereas standardizing measures of climate volatility and health impacts cannot be quantified at all.

9.2 Mechanistic Gaps

There are various areas in which we have not fully grasped our mechanistic understanding of climate-related skin disease. The current lack of knowledge on the mechanisms of nitrosative stress of pollution-related skin disease makes the relative role of reactive nitrogen species compared to reactive oxygen species not yet answered. The unfinished explanation of the microplastic and nanoplastic effects on the skin is an emerging issue considering the prevalence of such environmental pollutants [80]. There has been inadequate focus on gene-environment interaction such as the susceptibility polymorphisms of skin disease related to climate, which can determine at-risk groups to direct their prevention. The effects of epigenetic changes surprisingly caused by exposure to chronic climate stressors could mediate long-term disease possibility, but longitudinal epigenetic research is absent. Microbiomeclimate disease tripartite interactions are a very complicated system in which environmental fluctuations to microbial communities are subsequently amplified by host susceptibility, but this field has not been thoroughly elucidated yet [81].

9.3 Therapeutic Gaps: The Need for Clinically Proven Interventions

The most significant disparity in climate-dermatology studies lies in the striking lack of connection between mechanistic knowledge and interventions that have been proven clinically. The majority of anti-pollution skincare products that are promoted to consumers are not rigorously clinically validated, thus claims are made on the basis of in vitro test data or small, uncontrolled studies instead of randomized controlled trials with clinically meaningful endpoints. There are not many RCTs that were developed in relation to climate-exposed groups, and thus even well-known therapies have not been tested in the areas where they are needed most of all. There are no FDA-approved signs of pollution-related skin disorders, heat exacerbated dermatoses or disease aggravated by climate, which constitutes a regulatory void inhibiting development of therapy. There are no head to head comparisons of interventions in climate affected patients to select the best treatment and there is not enough information on children formulations of climate adjusted skincare to have an age suitable options. Poorly known biomarkers to predict the personal response to climate-adaptive interventions lack the ability to create personalized interventions and interfere with the effectiveness of clinical trials [82].

9.4 Implementation Gaps

Even where evidence is present, gaps of implementation cannot be translated into practice. There has been slow translation of research findings into clinical guidelines and most dermatology guidelines have not considered climatic factors. Clinical decision support and population surveillance would be possible through the integration of climate variables within electronic health records, although this is

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uncommon. There is a need to develop clinically viable environmental exposure assessment instruments that can be practiced in an everyday practice that do not require special equipment. Assessment of the practical efficacy of preventive interventions in various environments is necessary to comprehend the translation of efficacy undertaken in controlled studies to efficacy of community practice [83].

9.5 Equity Gaps

Marginalized populations are severely disadvantaged by climate change; thus, research is urgently required to help discover health disparities. Intervention studies conducted in low-resource environments should consider the feasibility constraint such as availability of products, cost and limited healthcare infrastructure. The development of culturally sensitive prevention policies to target different populations necessitates community-based research methodologies that allow local knowledge and practice to be respected and at the same time work towards addressing the impacts that climate change bears to the community [84].

9.6 The Translational Gap: From Mechanism to Clinic

Although there have been advanced mechanistic insights into the manner in which climate stressors cause skin damage, there has been a lack of therapies that have specifically targeted climate-induced skin damage. This translational gap is indicative of the lack of regulatory pathways that would appreciate climate-related dermatologic conditions as a legitimate therapeutic target and would provide incentive to the industry to invest. Industry incentives encouraging the creation of climate-adaptive dermatologic products, similar to incentives in orphan drugs or in treating tropical diseases, would quickly stimulate the creation of therapeutics to address the health of more and more patients whose skin condition is becoming progressively influenced by our warming climate [85].

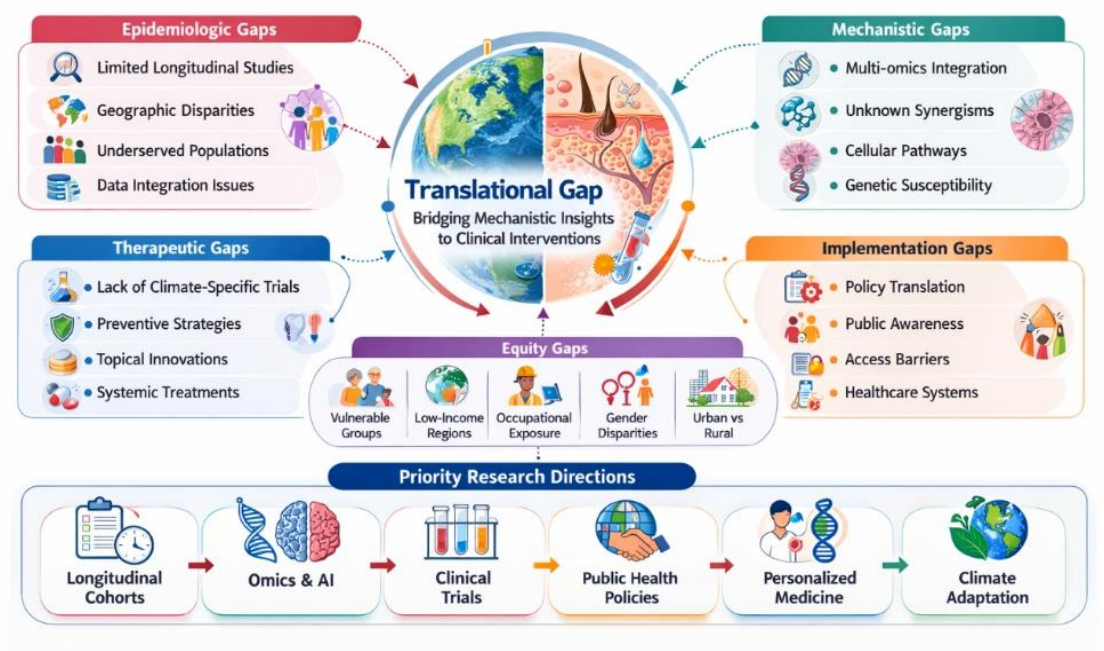

Figure 2. ***Research Gaps and Future Directions Framework.***

Conceptual framework identifying critical research gaps in climate change and skin health, organized by domain. The framework illustrates the interconnections between epidemiologic, mechanistic, therapeutic, implementation, and equity gaps, and proposes priority research directions to address each gap. Central to the

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framework is the translational gap—the need to convert mechanistic understanding into clinically proven interventions for affected populations.

10.0 Clinical Practice Recommendations

10.1 Environmental History Taking

The practice of climate-adapted dermatology can be based on systematic environmental history taking. The clinicians are advised to regularly measure the level of occupation and outdoor exposure time to measure the risk of UV and pollution and the proximity of the residence to traffic and industry as possible signs that indicate a high risk of pollutants. The investigation of patient knowledge about air quality indices and related protection measures has shown that there is a chance to educate patients. Observation of seasonal disease flare patterns aids in determining climate precipitants, and evaluation of cooling, shade, and sun protection availability assists in determining resource constraints that may need to be addressed. The history of migration and the acclimatization may be of special interest to patients who travel between the climate zones since their deficiency can make them more susceptible to the adverse effect of the new environment due to the deficit of adaptive behavioral patterns [86].

10.2 Diagnostic Considerations

Climate-sensitive skin disorders need to be diagnosed by identification of patterns that are indicative of environmental factors. Air pollution, extreme temperatures and UV exposure should also be included as contributing or aggravating factors in the differential diagnosis. The interpretation of the use of validated instruments such as Eczema Area and Severity Index, Psoriasis Area and Severity Index, and Dermatology Life Quality Index should be considered in the context of the environment and should refer to the possibility of the current scores to be the reflection of the disease baseline or climate-increased flares [87].

10.3 Treatment Algorithms Incorporating Climate Factors

Climate factors should be incorporated in the treatment planning in step-wise approaches that are based on exposure assessment. Highly polluted patients should receive barrier-repair therapy and antioxidant supplements earlier, whereas those in extreme temperature should obtain seasonal treatment regimen guidelines. Intervention approaches combining conventional treatment with climate-adaptive interventions, including the introduction of film-forming protectants into the standard AD programs at high-pollution times, can be the most effective way to go [88].

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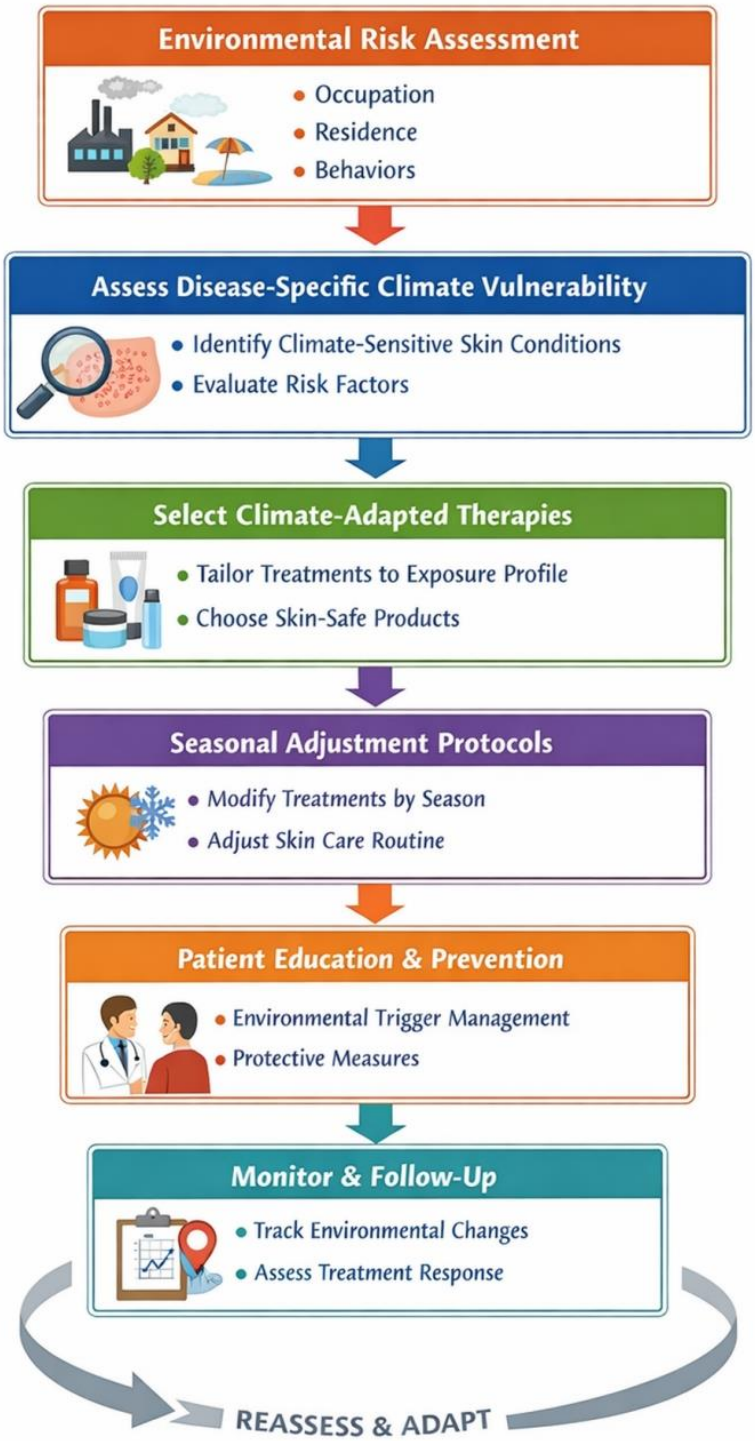

Figure 3. Clinical Algorithm for Climate-Adapted Dermatology Care.

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Proposed clinical algorithm for integrating climate considerations into dermatology practice. The algorithm guides clinicians through: (1) Environmental risk assessment (occupation, residence, behaviors); (2) Disease-specific climate vulnerability evaluation; (3) Selection of climate-adapted therapies based on exposure profile; (4) Seasonal treatment adjustment protocols; (5) Patient education on environmental trigger management; (6) Monitoring and follow-up with environmental tracking. The algorithm emphasizes the iterative nature of climate-adapted care and the need for personalized approaches based on individual risk profiles.

10.4 Patient Education and Counseling

Practical instructions on how to go about climate risks should be part of patient education. Patient education about air quality index interpretation helps them to alter outdoor activities and increase the level of protection in case the pollution is high. Awareness of the UV index would assist the patient to schedule outdoor activities and wear sufficient protection. Heat-related cutaneous morbidity in at-risk patients such as those with impaired thermoregulation or taking drugs that impact heat tolerance can be prevented through heat stress prevention. Sustainable skincare practices such as the choice of the products that have the lowest impact on the environment aligns dermatologic care with the larger climate goals [89].

10.5 Referral and Multidisciplinary Care

Multidisciplinary collaboration can be effective in the management of complex climate-related skin disease. Occupational medicine referrals make the workplace accommodations and documentation of occupational disease claims easier. Staff specializing in environmental health may help in finer exposure analysis and remediation guidelines of complicated instances that comprise various environmental triggers. Teledermatology can be important to the populations which are vulnerable to climate change since when extreme weather conditions occur, people can still receive care and those specialists can be extended to other areas that lack adequate specialists [90].

Conclusion

The relationship between climate change and augmented load of skin cancer, inflammatory dermatoses, infections, and aging-related conditions continues to be clearly founded on more and more thoroughly characterized mechanistic courses of action encompassing oxidative stress, activation of aryl hydrocarbon receptors, impairment of the barrier, and dysregulation of immunity. It has been shown that epidemiologic evidence has measurable risks, especially against atopic dermatitis and skin cancer, although people who are at risk, such as children, the elderly, outdoor workers, and marginalized populations, experience disproportionate and compounding risks . The call to action is obvious: climate change is not a threat of the future but a fact of the present that predetermines dermatologic practice. The dermatology community needs to stop being aware and start taking action as it is possible and even compulsory to incorporate climate adaptation strategies into regular care. The call to action must be coordinated, involving clinicians to factor environmental health into practice and advise patients about climate-adaptive health measures; researchers to focus on critical gaps, especially intervention trials; policymakers to raise the quality of the air and invest in climate-resilient health systems; educators to consider climate health in dermatology training programs; and industry to develop and test climate-adaptive dermatologic products. Climate change and skin health intersect is a challenge and an opportunity, an opportunity to reconsider dermatologic care to a changing world, to identify new ways of preventing and treating health, and to support the environmental policy that will eventually define the skin health of the generations to come. It is obvious; the necessity is extreme; the way to go is through togetherness.

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