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SYSTEMIC REVIEW

Ultraviolet Radiation, Extreme Temperatures, and Skin Diseases: A Systematic Review

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Abstract

Introduction: Climate change is intensifying environmental stressors such as ultraviolet (UV) radiation and extreme ambient temperatures, both of which have critical dermatological implications. UV radiation is a known carcinogen and immunomodulator, while extreme heat and cold affect skin barrier function and inflammatory responses. Despite growing evidence on their individual effects, their combined impact on dermatological disease patterns remains underexplored.

Objective: To systematically evaluate and synthesize observational evidence on the individual and synergistic effects of UV radiation and extreme temperatures on the incidence and severity of skin cancers and inflammatory skin diseases, particularly within the context of climate change.

Methods: A systematic literature review was conducted across PubMed, Scopus, and Web of Science up to March 2025, including cohort and cross-sectional studies examining the relationship between UV radiation, temperature extremes, and skin conditions. Risk of bias was assessed using the Newcastle–Ottawa Scale. Studies focusing on non-human subjects, case reports, and randomized trials were excluded. Outcomes were analyzed qualitatively due to heterogeneity in exposure and measurement methods.

Results: Eight studies met inclusion criteria. Findings consistently showed that higher ambient temperatures and UV exposure independently increased skin cancer risk, with evidence suggesting biological synergy via suppression of apoptosis in UV-damaged keratinocytes. Behaviorally, higher temperatures were a stronger predictor of sunburn and skin cancer procedures than UV index alone. For inflammatory dermatoses, results were mixed: atopic dermatitis improved in warm-humid climates but worsened with heat or cold stress; UV exposure alleviated psoriasis but exacerbated lupus and pemphigus. It is projected that a 2°C global temperature increase could lead to an 11% rise in skin cancer incidence by 2050.

Discussion and conclusion: This review highlights a dual role of UV radiation and temperature as both environmental and behavioral risk modifiers of skin disease. The effects on neoplastic conditions are predominantly aggravating, while those on inflammatory diseases are variable. Climate change is expected to increase dermatological disease burden, particularly in underserved regions. Public health responses should integrate sun protection and thermal adaptation strategies into dermatologic care and climate resilience frameworks.

Keyword: *skin neoplasms; ultraviolet rays; climate change; dermatitis; atopic; temperature extremes (MeSH)*

Introduction

Ultraviolet (UV) radiation and ambient temperatures are among the most influential environmental variables impacting dermatological health. UV radiation, particularly UVB (280–315 nm), is a well-documented carcinogen and is directly implicated in the pathogenesis of skin cancers, including basal cell carcinoma (BCC), squamous cell carcinoma (SCC), and cutaneous malignant melanoma. The International Agency for Research on Cancer (IARC) has classified solar radiation as a Group 1 carcinogen due to the substantial epidemiological and experimental evidence linking UV exposure with skin neoplasms. Multiple large-scale epidemiological studies have shown a direct correlation between cumulative sun exposure and increased risk of BCC and SCC, whereas intermittent, intense sun exposure is more closely associated with melanoma [1].

In addition to its carcinogenic effects, UV radiation influences a spectrum of inflammatory and autoimmune dermatoses. For instance, moderate UV exposure has therapeutic benefits in diseases such as psoriasis and atopic dermatitis via immunomodulatory mechanisms, while excessive exposure exacerbates photosensitive conditions like lupus erythematosus and polymorphic light eruption [2].

Ambient temperature, particularly extreme heat, also plays a critical role in skin disease epidemiology. High temperatures increase the risk of UV-related damage indirectly by promoting behaviors such as wearing lighter clothing or spending more time outdoors during peak sunlight hours, especially in temperate climates [3]. Moreover, experimental studies have demonstrated that heat stress can act as a co-carcinogen: elevated temperatures impair DNA repair mechanisms (e.g., via inhibition of p53-mediated apoptosis), promoting survival of UV-damaged keratinocytes and accelerating tumor development. Conversely, cold and dry environments exacerbate xerosis and inflammatory skin diseases, such as atopic dermatitis and psoriasis, by impairing the skin barrier function [4].

The global climate crisis is now altering the intensity and distribution of both UV radiation and extreme weather events. Stratospheric ozone depletion and reduced cloud cover in some regions have increased ground-level UV irradiance. At the same time, global warming is producing more frequent and intense heatwaves, while seasonal boundaries are becoming increasingly blurred [5]. These changes are expected to reshape the epidemiology of skin diseases worldwide—both in terms of neoplastic burden and chronic inflammatory conditions.

While a robust body of literature confirms the independent impacts of UV radiation and temperature extremes on skin health, few studies have systematically assessed their **combined** or **synergistic effects**, particularly within the context of ongoing climate change [6]. For example, while it is well known that UV radiation causes DNA damage, recent laboratory evidence indicates that heat may enhance this damage by downregulating protective tumor suppressor responses (e.g., p53 and apoptotic pathways), thereby facilitating malignant transformation in the skin [6,7].These co-carcinogenic effects of heat and UV have been demonstrated in murine models as early as the 1980s and were later substantiated by cellular studies showing heat-induced production of heat shock proteins (HSPs) that interfere with UV-induced apoptosis [8].

Additionally, behavioral patterns driven by environmental temperature play a significant role in modifying UV exposure risk. A large ecological study in the United States found that the best climatic predictor of skin cancer surgery rates among Medicare patients was not irradiance itself, but rather the number of days with temperatures exceeding 24°C. These findings suggest that human behavior more time spent outdoors, reduced clothing, and recreational exposure during warm periods can amplify UV-related risks even when the actual UV index is moderate [9,10].

Despite such critical interactions, most existing studies focus on singular exposures or specific skin conditions. Regional disparities in data further limit our understanding; most evidence comes from North America, Europe, and parts of Asia and Australia, leaving significant gaps in tropical and developing regions that may be disproportionately affected by climate shifts. This fragmentation impedes the formulation of globally relevant public health strategies and clinical guidelines [11].

Therefore, there is an urgent need for comprehensive syntheses and longitudinal studies that examine the **confluence of UV radiation and thermal stress**, incorporate behavioral variables, and project how climate-driven environmental shifts will shape the global burden of dermatological disease.

This review was conducted to answer the central question: How do UV radiation and extreme temperatures, individually and synergistically, influence the epidemiology of skin diseases in the context of ongoing climate change? We hypothesized that increasing UV radiation and rising global temperatures are not only independently associated with a higher burden of skin cancer and inflammatory dermatoses, but also interact biologically and behaviorally to amplify these effects.

Furthermore, we postulated that while UV radiation may exert both protective and aggravating roles depending on the type of inflammatory dermatosis (e.g., protective in psoriasis, aggravating in lupus), the net effect of climatic shifts is an overall increase in dermatological disease burden, particularly in vulnerable regions with limited access to adaptive resources.

The primary objective of this systematic review was to critically evaluate and synthesize the available scientific evidence on the relationship between UV radiation, ambient temperature extremes, and skin disease outcomes. Our aim was to integrate epidemiological, mechanistic, and behavioral findings to present a unified understanding of how these environmental stressors, in the context of climate change, affect both neoplastic and inflammatory skin conditions.

Through this approach, we intended not only to clarify the scientific knowledge available but also to identify gaps in the literature, highlight public health implications, and propose future directions for research and policy, particularly in anticipation of a more extreme and unpredictable climatic future.

Methods

Study design

This was a systematic review of the literature focused exclusively on observational studies (cohort and cross-sectional designs) that examined the association between ultraviolet (UV) radiation, extreme ambient temperatures, and skin diseases.

Eligibility criteria

TYPES OF STUDIES

We included peer-reviewed observational studies (prospective or retrospective cohorts and cross-sectional analyses). Randomized controlled trials, case reports, editorials, letters, and reviews were excluded.

TYPES OF PARTICIPANTS

Eligible studies assessed human populations of any age group, although the majority of included studies focused on either adults or elderly individuals. Studies conducted in both general and dermatological clinic-based populations were considered.

TYPES OF EXPOSURES

We included studies that evaluated the effects of:

- Ultraviolet (UV) radiation exposure (natural solar exposure measured via UV index or global horizontal irradiance),
- Extreme temperatures (both high and low ambient temperatures),
- Or the interaction between UV radiation and temperature.

TYPES OF OUTCOMES

PRIMARY OUTCOMES

- Incidence or prevalence of skin cancer, including basal cell carcinoma, squamous cell carcinoma, and cutaneous melanoma.

SECONDARY OUTCOMES

- Incidence, prevalence, or exacerbation of inflammatory skin diseases such as atopic dermatitis, psoriasis, chronic actinic dermatitis, lupus erythematosus, and pemphigus vulgaris.

Information sources and search strategy

ELECTRONIC SEARCHES

We conducted a structured search of scientific literature databases. The databases searched included PubMed, Scopus, and Web of Science. The search terms combined synonyms and medical subject headings (MeSH) related to:

- Skin diseases (e.g., "skin cancer," "atopic dermatitis," "psoriasis"),
- Environmental exposures (e.g., "ultraviolet rays," "heatwaves," "temperature"),
- Climate change and environmental modifiers.

The search was conducted up to March 2025. No language or geographic restrictions were applied during the initial search.

ADDITIONAL SOURCES

We also examined the reference lists of included studies and relevant reviews for additional eligible studies not identified through database searches.

Data collection and analysis

STUDY SELECTION

Titles and abstracts were screened independently by two reviewers. Full-text articles were then retrieved and assessed for eligibility according to the predefined inclusion criteria. Disagreements were resolved by consensus.

DATA EXTRACTION AND MANAGEMENT

Data were extracted using a standardized form that included the following variables: study design, population characteristics, exposure type and assessment method, outcome definitions, main findings, and region of study. Data were entered into an Excel spreadsheet and double-checked for accuracy.

Risk of bias assessment

Risk of bias for the included cohort studies was assessed using the Newcastle–Ottawa Scale (NOS). This tool evaluates studies based on three domains: selection of study groups, comparability of groups, and ascertainment of outcomes. Studies were categorized as low, moderate, or high risk of bias based on their total NOS scores. Only studies with a cohort design were appraised for risk of bias.

Bias Management

HANDLING OF MISSING DATA

When relevant information (e.g., UV measurement units, exposure thresholds, or population subgroups) was not reported, we attempted to contact study authors. In the absence of responses, studies were included if they met the core inclusion criteria, and potential limitations were noted in the qualitative synthesis.

ASSESSMENT OF REPORTING BIAS

No formal statistical assessment of publication bias was conducted due to the absence of a meta-analysis and the limited number of studies per outcome.

Data synthesis

Due to heterogeneity in study designs, populations, exposure measurements, and reported outcomes, we did not perform a meta-analysis. Instead, a **qualitative synthesis** was conducted. Results were summarized descriptively and grouped by outcome type (neoplastic vs inflammatory diseases) and exposure (UV, temperature, or both). Patterns and potential mechanisms were highlighted, and the consistency of findings across geographic and demographic contexts was discussed.

Results

A total of 14 records were identified. Of these, 4 records were excluded, leaving 10 full-text articles for detailed eligibility assessment. Two full-text articles were then excluded for the study design. Ultimately, 8 studies met inclusion criteria for the qualitative synthesis (Figure 1).

Skin cancer and climatic factors ultraviolet radiation and skin cancer

UV radiation is the most important environmental factor in the genesis of skin cancers. Numerous epidemiological studies have confirmed the strong association between sun exposure and the

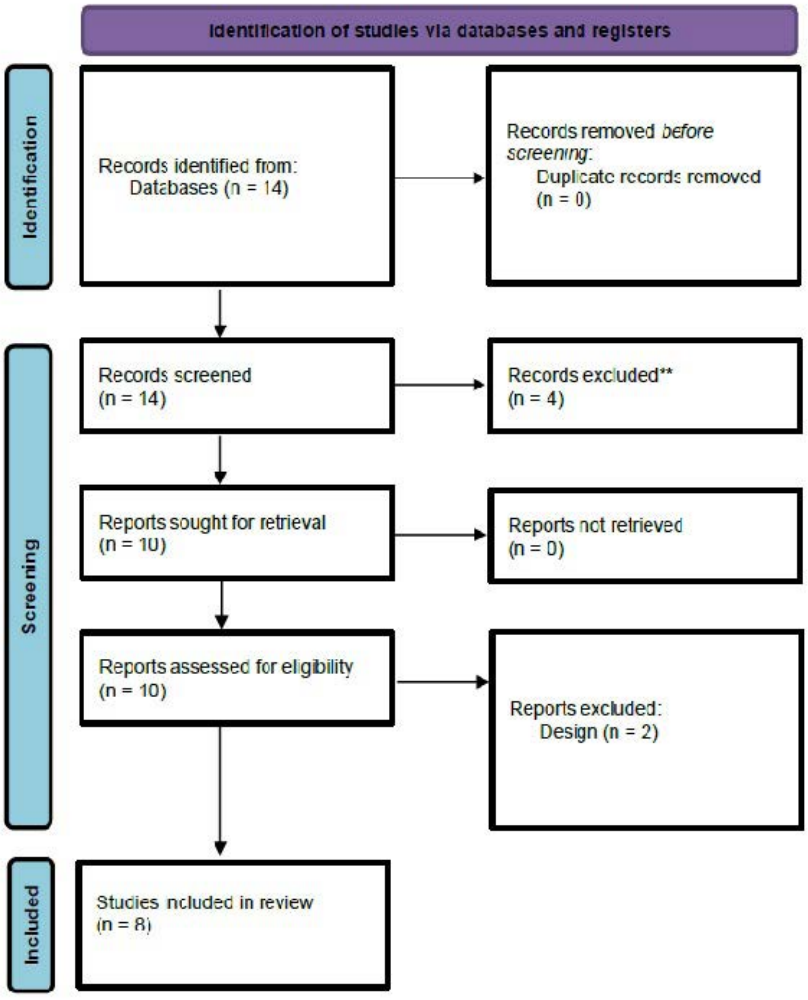

Figure 1: PRISMA Flow diagram.

Table 1: Key studies on the relationship between UV radiation, temperature, and skin cancer.

Authors (Year)	Population (Design)	Exposure Evaluated	Main Findings	Conclusions
Woodie et al. [15]	Older adults (Medicare) in the U.S. (ecological analysis by state)	Annual days with T° > 24 °C; global horizontal irradiance (GHI)	More warm days were associated with more skin cancer procedures across all regions; temperature predicted cases better than UV radiation.	Ambient heat was a stronger predictor of skin cancer incidence than UV intensity itself, suggesting that behavior on warm days increases risk.
Marion et al. [4]	947 adults in Ohio, U.S. (prospective cohort)	Ambient temperature and UV level during a beach day	15% of participants developed sunburns in the following week; high temperature was significantly correlated with sunburn (more than UV index).	Elevated ambient temperature increased sun exposure and was a critical determinant of sunburns, indicating higher long-term risk of actinic damage.
Dobbinson et al. [5]	Adolescents and adults in Australia (cross-sectional study)	Daily temperature and self-reported sunburn events	With T° > 22 °C, sunburn probability doubled or tripled; with T° > 28 °C, it increased more than 3-fold compared to mild days.	Heatwaves result in a higher incidence of acute sunburns in the population, implying increased cumulative skin cancer risk.
Van der Leun & de Gruijl [2]	Global population (modeled projection)	Global warming scenario (+2 °C) by 2050	Projected +11% increase in global skin cancer incidence compared to a scenario without warming.	Climate warming will likely significantly increase the global burden of skin cancer in the absence of additional preventive measures.

incidence of melanoma and non-melanoma skin cancers (basal cell carcinoma and squamous cell carcinoma) [12]. In the context of climate change, the concern lies in the possibility that certain trends could increase the UV dose received by populations. For instance, the thinning of the stratospheric ozone layer over recent decades allowed greater penetration of UVB, potentially contributing to the rise in skin cancer cases observed in the second half of the 20th century. It has been estimated that a 1% decrease in stratospheric ozone increases the incidence of skin cancer by a similar proportion (1–2% for melanoma, ~3–5% for squamous cell carcinoma) due to the resulting increase in UVB radiation at ground level [12].

Fortunately, the implementation of the Montreal Protocol has initiated the recovery of the ozone layer; however, the effects of that period of high UV exposure may still be reflected in skin cancer rates for years due to the prolonged latency of cutaneous carcinogenesis [13]. Beyond the ozone layer, climate change can influence UV radiation through changes in cloud cover, air pollution, and the length of seasons. In high latitudes, exceptionally sunny and dry summers have been documented (such as in Northern Europe in 2018), resulting in unusually high UV indices [12]. Studies in Eastern Europe show that between 1979 and 2015, reduced cloudiness increased the daily erythemal UV dose by 5–8% per decade [12]. Likewise, global warming tends to shorten winter durations in temperate regions; combined with higher UV levels during traditionally less sunny months, this could lead to more consistent sun exposure throughout the year [14]. In sum, although ambient UV radiation may both increase or decrease locally depending on changes in clouds and aerosols, concern persists that populations will be exposed to more effective UV on average as the climate warms, which could increase the risk of skin cancer.

Epidemiological evidence supports this expectation. A recent ecological study in the United States analyzed data from the Medicare population (older adults) correlating skin cancer excision rates with regional climate metrics [15]. The authors found that the best climatic predictor of the number of skin cancers removed was the number of warm days in the year: in each state, the number of days above 24 °C was significantly associated with more skin cancer surgeries, even more than measured solar irradiance (GHI) [15]. In other words, regions with warmer climates (more hot days) presented a higher burden of skin cancer, suggesting that heat-induced behaviors (more outdoor activities with minimal clothing, etc.) increase population UV exposure. The study concluded that due to global warming, skin cancer incidence could continue to rise because of behavioral changes related to high temperatures and proposed focusing prevention efforts on those habits [15].

Similar observations come from other settings. In Australia, a country with extreme UV indices, it was reported that the risk of sunburn in adolescents and adults multiplies on very hot days: when the temperature exceeds ~22 °C, the likelihood of sunburn doubles or triples, and above 28 °C the risk is more than triple compared to cooler days [5]. This, despite UV intensity possibly being the same, indicates that heat causes people to stay longer in the sun or use less protection. In fact, a prospective study of 947 adults visiting a beach in Ohio (USA) found that ambient temperature was a stronger predictor than the UV index of who would develop sunburn in the week following sun exposure [4]. Researchers measured both UV dose and temperature during the beach stay and found that higher temperatures significantly increased sunburn rates, regardless of UV levels [4]. They concluded that increasing ambient temperatures likely lead to riskier behaviors (prolonged sunbathing, less clothing, more swimming that reduces photoprotection, etc.), thereby raising the incidence of sunburns and, in the long term, skin cancer [4].

It is worth noting that heat not only increases exposure: experimental studies suggest that temperature itself may have a co-carcinogenic effect on the skin. Since the 1940s, it has been known that mice exposed to UV develop tumors more quickly if simultaneously subjected to high heat. Modern research has identified a possible mechanism: heat exposure induces the production of heat shock proteins (HSPs), which can inhibit apoptosis of UV-damaged cells, allowing the survival of mutated keratinocytes [14]. In cell models, heat was shown to reduce p53 activation after UVB radiation, decreasing cell cycle arrest and programmed cell death in DNA-damaged cells [14]. This reinforces the idea of a biological synergy between UV and temperature in skin carcinogenesis. Overall, the evidence suggests that climate change by intensifying effective UV exposure and raising temperatures will likely increase skin cancer incidence in the coming decades, exacerbating an already concerning trend. Indeed, it has been projected that a 2 °C increase in global temperature could raise global skin cancer incidence by ~11% by the year 2050 [14]. Table 1 summarizes key studies linking climatic factors to skin cancer risk.

Extreme temperatures and skin cancer

Extreme temperatures, particularly excessive heat, influence the risk of skin cancer both indirectly and directly. Indirectly, heat alters human behavior: very hot days often lead people to engage in more outdoor activities while wearing less clothing, thereby increasing the dose of UV radiation received [5]. Moreover, it has been documented that above certain temperature thresholds, sunburn rates spike, as discussed previously [5]. This behavioral effect is especially notable in temperate regions, where longer and more intense summers due to global warming extend the season of high sun exposure [5].

On the other hand, directly, heat exposure can enhance the harmful effects of UV radiation on the skin. Studies in animals and human cells indicate that elevated temperatures impair the repair of actinic damage by interfering with tumor suppression mechanisms (e.g., the p53 pathway) [14]. Thus, skin exposed simultaneously to

UV and heat suffers deeper damage than with UV alone, facilitating the long-term development of skin tumors.

While extremely high temperatures may eventually deter sun exposure (beyond a certain point, people seek shelter in cool indoor environments), in practice, data suggest that the net effect of heat is to increase the incidence of skin cancer. In warm subtropical areas such as Queensland (Australia) or the southern United States, where the population is accustomed to prolonged summers, records show a very high incidence of skin cancer attributable to the combination of intense sun and warm climate [16]. An analysis in the U.S. found that the number of days >24 °C was linked to cancers in typically exposed areas (head, neck, hands) as well as in usually covered areas (trunk, limbs), while average solar irradiation did not show as strong an association [15]. In other words, heat appeared to better explain the occurrence of cancers even in body areas that would normally receive less sun, which underscores the behavioral influence (example: on very hot days, people may swim or sunbathe with their torsos exposed, increasing exposure of the trunk and legs).

Extremly low temperatures are not a risk factor for skin cancer per se; rather, cold climates limit UV exposure (due to more clothing and fewer outdoor activities), which correlates with lower skin cancer incidence in high latitudes. However, climate change is projected to bring milder winters, which may reduce that "seasonal protection" and extend the annual window of UV risk [15].

Inflammatory skin diseases and climatic factors

Climate and climate change-related phenomena also affect the epidemiology of various inflammatory skin diseases, including atopic dermatitis (eczema), psoriasis, autoimmune blistering diseases, and other chronic inflammatory dermatoses. Unlike cancer, where the deleterious effect of UV radiation is direct, in inflammatory diseases the climatic impact can be bidirectional: certain extreme conditions exacerbate inflammation or trigger flare-ups, while others may alleviate symptoms or reduce incidence, depending on the specific disease. Below is a review of the evidence by primary type of exposure.

UV radiation and inflammatory dermatoses

Solar UV radiation has varying effects on inflammatory skin diseases. On one hand, moderate sun exposure can be beneficial for some conditions: for instance, UV light is known to induce local immunosuppression and is used therapeutically (phototherapy) to treat psoriasis, chronic eczema, and even vitiligo. Epidemiological studies suggest that regions with higher sunlight exposure tend to have lower prevalence of atopic dermatitis in children, possibly due to greater vitamin D synthesis or the immunomodulatory effect of sunlight [17]. In an analysis of more than 91,000 children in the U.S., Silverberg et al. [7] found that eczema prevalence was significantly lower in areas with the highest annual UV index and higher average temperatures, compared to colder, less sunny regions [17]. In fact, warm, humid, and sunny climates (such as the southeastern U.S.) showed lower rates of atopic dermatitis than colder and drier areas, highlighting the possible protective role of ambient UV radiation in this disease [17].

On the other hand, intense UV exposure can worsen or trigger certain inflammatory dermatoses, particularly those that are photosensitive. A clear example is cutaneous lupus erythematosus, where UV radiation can trigger lesion flare-ups and exacerbate systemic disease; similarly, conditions like cutaneous dermatomyositis or polymorphic light eruption are triggered or worsened by excessive sun exposure. Although our focus is on adults, it is worth noting that in susceptible populations such as lupus patients, heat waves and high UV episodes may result in a higher frequency of cutaneous flare-ups) [4]. Another related inflammatory entity is chronic actinic dermatitis, a chronic eczema-like hypersensitivity to UV light. A study in Korea observed an increase in chronic actinic dermatitis incidence over the past 15 years, temporally correlated with climate changes and increased air pollution [18]. The authors suggested that rising UV intensity and possibly environmental photosensitizers could be behind this increase, although further data are needed to directly attribute it to climate change.

Regarding psoriasis, the relationship with UV radiation is opposite to that of cancer: sunlight often improves psoriatic plaques in many patients, and UVB phototherapy is a standard treatment. Studies on seasonal variation show that psoriasis tends to worsen in winter (when UV levels are lower) and improve in summer with more hours of sunlight [19]. Therefore, an increase in UV radiation is not expected to worsen psoriasis; in fact, some patients may benefit from sunnier climates. However, it should be noted that excessive sun (sunburns) can induce the Koebner phenomenon in psoriasis (appearance of lesions in previously unaffected skin), and extreme heat may cause skin dehydration, which are indirect factors not favorable to patients. Overall, there is no indication that the incidence of psoriasis directly increases due to more UV radiation; on the contrary, some ecological analyses suggest that the global burden of psoriasis may not grow due to climate change in terms of UV, and could even be slightly mitigated in formerly cold regions that now have milder climates.

Finally, some autoimmune blistering diseases, such as bullous pemphigoid and pemphigus vulgaris, have been studied in relation to climatic factors. A study in the U.S. [20], investigated thousands of hospitalizations for pemphigus vulgaris in relation to weather and pollution data [20]. It was found that patients hospitalized for pemphigus disproportionately came from regions with higher UV radiation and lower humidity compared to the general hospitalized population [20]. Months or locations with high UV indices showed increased risk of pemphigus admissions, suggesting that sunlight may trigger or worsen this autoimmune disease (possibly through oxidative stress induction or exposure of cutaneous autoantigens) [20]. Although pemphigus is not a "chronic inflammatory" disorder in the traditional sense, it is an autoimmune skin disease that may be sensitive to environmental factors, and these findings support the notion that climate change, by intensifying UV exposure, may influence the epidemiology of various immunologic dermatoses.

In summary, UV radiation associated with climate change has a dual impact on inflammatory skin diseases: it may reduce incidence or severity of some (e.g., less eczema in sunny climates, improvement of psoriasis), but worsen others (lupus or pemphigus flare-ups, increase in photodermatoses). This means that the effects are not uniform and depend on the specific pathology and individual factors (e.g., skin phototype, genetic predisposition). **Table 2** presents illustrative studies showing how climatic factors (UV and temperature) affect inflammatory skin diseases.

Extreme temperatures and inflammatory dermatoses

Thermal stress whether due to intense cold or extreme heat can disrupt skin homeostasis and trigger inflammatory responses. Low temperatures are often associated with drier skin, impaired skin barrier function, and increased susceptibility to irritants, which in predisposed individuals can lead to eczema flare-ups. For example, the study by Chen et al. in China (Table 2) showed a clear increase in visits for atopic dermatitis during cold periods [21]. In fact, in that analysis, the proportion of consultations attributable to cold was seven times greater than that attributable to heat, suggesting that in subtropical climates like Chengdu, winter poses a greater challenge for patients with eczema than summer [21]. This aligns with clinical experience, as many atopic patients tend to worsen in winter due to dry air and indoor heating. Climate change could, in theory, mitigate some of this effect in colder regions by making winters milder;

Table 2: Key studies on extreme temperatures, UV radiation, and inflammatory dermatoses.

Authors (Year)	Population (Design)	Exposure Evaluated	Main Findings	Conclusions
Chen et al. [21]	Patients with atopic dermatitis in Chengdu, China (time-series 2015–2020)	Daily ambient temperature (low < 19.6 °C vs high > 25.3 °C)	Both intense cold and extreme heat were associated with increased visits for atopic dermatitis flares. Cold had a stronger effect (22.4% of visits vs 3% for heat).	Extreme temperatures destabilize atopic dermatitis, especially cold/dry conditions. Global warming may reduce cold-induced flares but increase heat-related ones, depending on local adaptation.
Ren et al. [20]	Adults hospitalized for pemphigus vulgaris in the U.S. (retrospective analysis 2002–2012)	Regional climate averages (temperature, humidity, UV index, pollution)	Pemphigus hospitalizations were more frequent in regions with significantly higher temperatures and UV, and lower humidity (adjusted OR for high UV ~1.78).	Pemphigus vulgaris was associated with warm climates and high solar exposure. Findings suggest that UV exposure (and possibly dry heat) may trigger or worsen autoimmune skin diseases such as pemphigus.
Langan et al. [22]	Children with atopic dermatitis in the UK (prospective cohort, N=25)	Exposure events (hot weather, sweating, humidity) recorded in diaries	In episode analysis, hot weather was significantly associated with increased scratching and pruritus on the same day. Humidity and sweat linked to flares 2–4 days later.	Preliminary evidence that heat and sweating can trigger flares of childhood atopic eczema. Thermal stress and excessive humidity act as inflammation triggers in vulnerable skin.
Abuabara et al. [24]	Patients with atopic dermatitis (synthesis of 18 international studies)	Climate-related events: global warming, droughts, wildfires, etc.	High temperatures linked to proinflammatory cytokine production and increased pruritus. Of 4 studies on warming and eczema, 1 found worse control with heat/sun, while 3 reported symptom improvement in hot, humid climates.	The relationship between global warming and atopic dermatitis is complex: heat may aggravate symptoms (pruritus, inflammation) especially in dry climates, but in humid climates, higher temperatures may relieve dry skin. Flares also reported after climate disasters (wildfires, storms) due to stress and irritant exposure.

however, it may also bring episodic extreme cold events (e.g., atypical winter storms) that cause acute flare-ups in non-acclimated populations. Additionally, shorter winters could be offset by longer springs with high pollen loads, which in allergic atopic patients may also exacerbate dermatitis.

As for high temperatures, heat and humidity can be a double-edged sword for inflamed skin. On one hand, high humidity improves skin hydration and may help conditions like atopic dermatitis by reducing dryness. This could explain why in humid tropical climates, some atopic patients report fewer flare-ups. However, excessive heat leads to profuse sweating, and sweat is a well-known irritant/itch trigger for eczematous skin. Langan et al. [22], documented that "hot weather" days immediately increased pruritus in children with atopic dermatitis, and that heavy perspiration was associated with worsening symptoms in the days following [22]. The mechanism may involve both itch induced by sweat (subclinical miliaria, irritation from sweat salts) [22], and mast cell activation with the release of pruritogenic cytokines as skin temperature rises [22]. Moreover, extreme heat waves can cause physical and psychological stress factors known to trigger flare-ups of inflammatory conditions (stress and heat were two major triggers highlighted in Langan's study) [22]. It is therefore not surprising that following extreme events such as heatwaves, wildfires, or hurricanes, increased visits for eczema and pruritus have been reported in affected areas. For instance, during the 2020 wildfires in California (USA), clinics located 280 km from the fires reported an uptick in patients with atopic dermatitis and intense pruritus, attributed to heat stress, smoke, and poor air quality [22].

Other inflammatory diseases also respond to heat: rosacea can worsen significantly with high temperatures or consumption of hot beverages (due to vasodilation and flushing), so warmer summers may bring more difficult-to-control symptoms in rosacea patients. Cholinergic urticaria (a pruritic wheal-inducing rash triggered by heat or sweating) will logically become more prevalent or more active in settings with frequent heat waves. Although no specific population-based studies were found linking rosacea or urticaria to climate, these associations are noted in the literature on dermatological impacts of climate change [23].

Finally, it is important to note that extreme temperatures can pose systemic risks for patients with chronic skin diseases. Individuals with conditions that impair normal sweating—such as extensive psoriasis, severe atopic dermatitis, or congenital ichthyoses—have difficulty regulating body temperature and are more prone to heatstroke during heatwaves [23]. While this does not increase the *incidence* of the skin disease per se, it does raise associated morbidity. Similarly, patients with scleroderma or Raynaud's phenomenon may experience more skin ulcerations during extreme cold events. These examples fall outside the strict definition of incidence but illustrate the broader implications of thermal extremes for dermatological health under a changing climate.

General observed patterns

Integrating the reviewed evidence, several consistent patterns emerge:

- **Sustained increase in UV/heat-related skin cancer**: The incidence of skin cancer (melanoma and non-melanoma) shows a global upward trend that coincides temporally with environmental changes (increased sun exposure due to behavioral shifts, possible UV peaks from ozone depletion). Studies strongly suggest that climate change will amplify this increase by intensifying effective UV exposure (more sunny days, shorter winters) and promoting risk behaviors through warmer temperatures [15].

- **Biphasic effects on inflammatory skin diseases**: For inflammatory dermatoses, there is no uniform response to climate. Warm-humid climates appear protective for dry skin conditions (eczema) [17], whereas warm-dry climates or extreme heat events tend to worsen these same conditions. Dry cold clearly exacerbates diseases like atopic dermatitis (and possibly certain cases of psoriasis during winter), while moderate warmth may improve some of them. This pattern suggests that the net effect of climate change on inflammatory diseases will depend on local characteristics: regions that become warmer but retain sufficient humidity could see a reduction in certain dermatoses, while areas that become more arid or experience extreme events (droughts, wildfires) may see an overall worsening [17].

- **Exacerbations linked to extreme events**: Multiple studies report abrupt increases in skin problems following acute climate events (heatwaves, wildfires, floods). These situations combine various factors (trauma, stress, overcrowding, poor hygiene, exposure to allergens/dust) that trigger outbreaks of scabies, dermatitis, infections, etc., in affected populations [24]. If climate change increases the frequency of extreme events, transient epidemic peaks of certain dermatoses may be expected after each disaster. Although these peaks do not represent "chronic incidence," they do impose an

Table 3: Risk-of-bias assessment (Newcastle-Ottawa).

Study (Stars)	Aubara [24]	Chen [21]	Dobbinson [26]	Langan [22]	Marion [4]	Ren [20]	Van der Leun [2]	Woodie [15]
Selection	1	1	2	2	3	3	1	3
Comparability	2	2	2	1	2	2	2	2
Outcome	2	2	0	1	2	3	0	1
Global risk	Moderate	Moderate	High	Moderate	Low	Low	High	Moderate

additional burden on healthcare systems and reflect vulnerabilities in dermatologic patients.

- **Importance of adaptation and human behavior**: A cross-cutting theme is that the climate's impact on skin health is largely mediated by human adaptation. For instance, the harmful effect of heat on skin cancer risk can be nullified if populations adopt proper sun protection practices even on hot days (strict use of sunscreen, lightweight but long-sleeved clothing, avoiding peak sun hours) [15]. Similarly, many adverse effects on inflammatory dermatoses can be mitigated with simple measures: humidifying indoor environments during cold spells, climate control (air conditioning) during heatwaves, and protection from allergens during windy conditions, etc. Several studies implicitly indicate that climate alone does not determine outcomes; rather, interactions with behavior, infrastructure, and healthcare ultimately determine the magnitude of dermatological issues.

The methodological quality of the eight included cohort studies, as assessed by the Newcastle–Ottawa Scale, is summarized in Table 3.

- **Selection**: Scores ranged from 1 to 3 stars. Two studies [4,20], achieved the maximum of 3 stars for selection, three studies [5,15,22], scored 2 stars, and the remaining studies [1,21,24], scored 1 star.
- **Comparability**: Most studies (seven of eight) scored 2 stars, indicating that they adequately controlled for the most important confounders; Langan 2006 scored 1 star.
- **Outcome**: Ratings were more heterogeneous, ranging from 0 to 3 stars. Ren [20] scored highest with 3 stars; Aubara [24], Chen [21], Marion [4], and Langan [22], each scored 2 or 1 star; and Dobbinson 2008 and Van der Leun 2002 received 0 stars for outcome ascertainment.

On the basis of their total Newcastle–Ottawa scores, two studies were judged to be at low risk of bias [4,20], two at high risk [2,26], and the remaining four at moderate risk [15,21,22,24]. Overall, comparability across studies was generally strong, whereas outcome assessment contributed most to between-study variation in risk of bias.

Discussion

This systematic review reveals a consistent and multifaceted association between ultraviolet (UV) radiation, extreme ambient temperatures, and the incidence and severity of both neoplastic and inflammatory skin diseases. The review identified strong epidemiological evidence linking increased UV exposure and warmer temperatures with a higher risk of skin cancer—particularly basal cell carcinoma (BCC), squamous cell carcinoma (SCC), and melanoma. Importantly, behavioral factors and the co-carcinogenic role of heat appear to significantly enhance UV-related risks. Conversely, the relationship between climatic variables and inflammatory dermatoses is complex and bidirectional: while moderate sun and warmth may alleviate conditions like psoriasis and atopic dermatitis, excessive heat or UV can exacerbate diseases such as lupus erythematosus and pemphigus vulgaris. These findings suggest that climate change, by altering UV radiation patterns and increasing temperature extremes, could significantly shift the global dermatological disease burden.

Our findings align with previous ecological and epidemiological studies that underscore the significant role of ambient temperature in modulating skin cancer risk, often surpassing the influence of ultraviolet (UV) irradiance alone. For instance, Marion et al. [4] conducted a cohort study at an inland U.S. beach, revealing that sunburn incidence was associated with both temperature (odds ratio [OR] = 1.2; 95% confidence interval [CI]: 1.1–1.4) and UV index (OR = 1.6; 95% CI: 1.0–2.5), with evidence suggesting an interaction between temperature and UV radiation. This indicates that higher temperatures may amplify UV-related skin damage, potentially due to increased outdoor activity and behavioral factors during warmer conditions [24].

Similarly, Dobbinson et al. [5], observed that in Australia, higher ambient temperatures correlated with increased sunburn incidence among adolescents and adults, independent of UV index levels. These studies collectively suggest that temperature not only influences UV exposure behavior but may also have a direct biological effect on skin, enhancing susceptibility to UV-induced damage [25].

In contrast, the relationship between climatic factors and inflammatory dermatoses such as atopic dermatitis (AD) and eczema is more complex and heterogeneous. Silverberg et al. [17], found that the prevalence of eczema was significantly lower in U.S. states with higher temperature, outdoor humidity, and UV exposure, suggesting a protective effect of these climatic factors. However, other studies have reported exacerbation of AD symptoms under extreme temperatures. For example, a study by Langan et al. [22], indicated that both heat and dampness were associated with eczema flares, although their hypothesis-testing study did not find significant associations between eczema flares and climatic factors [7]. Furthermore, Ren et al. [20], demonstrated that higher temperatures, increased UV exposure, and lower humidity were associated with increased hospitalization rates for pemphigus vulgaris, an autoimmune blistering disease. This suggests that certain autoimmune dermatoses may be particularly sensitive to climatic variations, with potential implications for disease management and prevention strategies [20]. Collectively, these studies corroborate our findings, emphasizing the multifaceted impact of climatic factors on skin health. While warmer temperatures may have protective effects against certain inflammatory skin conditions, they concurrently pose increased risks for UV-induced skin damage and autoimmune dermatoses. This underscores the necessity for nuanced public health strategies that consider the diverse effects of climate on various skin diseases.

A major strength of this review lies in its integrative approach, synthesizing data from eight observational studies across diverse geographic settings and encompassing both neoplastic and inflammatory conditions. The inclusion criteria ensured methodological rigor by focusing on cohort and cross-sectional studies with defined exposure and outcome measures. Additionally, the risk of bias was systematically evaluated using the Newcastle–Ottawa Scale, with two studies rated low risk and most demonstrating adequate confounder control.

However, limitations exist. First, the small number of eligible studies (n=8) limits generalizability, especially for underrepresented regions such as Africa and South America. Second, the heterogeneity in exposure metrics (e.g., UV index, global horizontal irradiance, temperature thresholds) and outcome definitions precluded meta-analysis and may obscure true effect sizes. Third, behavioral variables were often inferred rather than directly measured, reducing precision in quantifying how heat-induced behavior modulates UV risk. Finally,

publication bias and language bias may have influenced the selection of studies, although no formal assessment was feasible.

These findings underscore the urgent need for public health strategies that integrate dermatological risks into climate adaptation frameworks. Health education campaigns should emphasize sun protection not only during high UV index periods but also during warm days when behavioral exposure surges. Dermatologists should consider environmental histories in managing chronic inflammatory dermatoses, particularly in patients living in climates with increasing variability. Future research should prioritize prospective longitudinal studies that examine the combined and interactive effects of UV and temperature on skin health across different population subgroups, including those with genetic or immunological vulnerabilities. It is also critical to expand the geographic scope of research to tropical and low-income regions where climate vulnerability is highest.

Conclusions

This systematic review confirms that UV radiation and ambient temperature extremes especially in the context of global climate change play a significant and synergistic role in the pathogenesis and exacerbation of a wide range of skin diseases. Skin cancer incidence is projected to rise due to both direct UV effects and behaviorally mediated exposure during warmer weather. Inflammatory dermatoses respond variably, with potential relief or worsening depending on disease type and local climatic conditions. These findings highlight the necessity of incorporating dermatological perspectives into climate change mitigation and adaptation policies and call for more geographically diverse and behaviorally nuanced research to guide evidence-based interventions.

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