American Journal of Clinical Dermatology (2023) 24:577–593
https://doi.org/10.1007/s40257-023-00770-y

REVIEW ARTICLE

# Climate Change, Skin Health, and Dermatologic Disease: A Guide for the Dermatologist

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Accepted: 12 March 2023 / Published online: 20 June 2023


## Abstract

Climate change has a pervasive impact on health and is of clinical relevance to every organ system. Climate change-related factors impact the skin's capacity to maintain homeostasis, leading to a variety of cutaneous diseases. Stratospheric ozone depletion has led to increased risk of melanoma and keratinocyte carcinomas due to ultraviolet radiation exposure. Atopic dermatitis, psoriasis, pemphigus, acne vulgaris, melasma, and photoaging are all associated with rising levels of air pollution. Elevated temperatures due to global warming induce disruption of the skin microbiome, thereby impacting atopic dermatitis, acne vulgaris, and psoriasis, and high temperatures are associated with exacerbation of skin disease and increased risk of heat stroke. Extreme weather events due to climate change, including floods and wildfires, are of relevance to the dermatologist as these events are implicated in cutaneous injuries, skin infections, and acute worsening of inflammatory skin disorders. The health consequences as well as the economic and social burden of climate change fall most heavily on vulnerable and marginalized populations due to structural disparities. As dermatologists, understanding the interaction of climate change and skin health is essential to appropriately manage dermatologic disease and advocate for our patients.

## Key Points

Climate change-related factors such as global warming and extreme weather events, along with air pollution, impact the skin's capacity to maintain homeostasis, playing a role in the pathogenesis of many cutaneous diseases including cutaneous malignancy, autoinflammatory disorders, heat-related illness, skin infections, and vector-borne diseases.

The health, social, and economic consequences of climate change fall most heavily on vulnerable and marginalized populations due to structural disparities.

Understanding the interactions between climate change and skin health is essential to appropriately manage dermatologic disease and advocate for patients.

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## 1 Introduction

The Sixth Assessment Report of the Intergovernmental Panel on Climate Change (IPCC) warned that global warming to 1.5 ˚C above pre-industrial temperatures would lead to increasingly hazardous climate conditions and place both humans and ecosystems at risk [1]. The consequences of climate change, including but not limited to stratospheric ozone depletion (SOD), air pollution, global warming, and extreme weather events, have widespread effects on human health (Fig. 1) [2]. This burden falls most heavily on vulnerable populations rather than on those who are most responsible for the current state in which we find ourselves [3]. Within the past decade, the association between the climate crisis and declining human health has become increasingly accepted, with multiple medical associations including the American Medical Association and the American Academy of Dermatology (AAD) committing to act as climate advocates [4, 5].

There is mounting evidence of the pervasive effects of climate change on skin health (Table 1). A 2019 survey of members of the International Society of Dermatology reported that 81% of respondents were concerned that climate change would directly impact their work as dermatologists and 88.6% were of the belief that climate change would impact the incidence of skin diseases [6]. A further

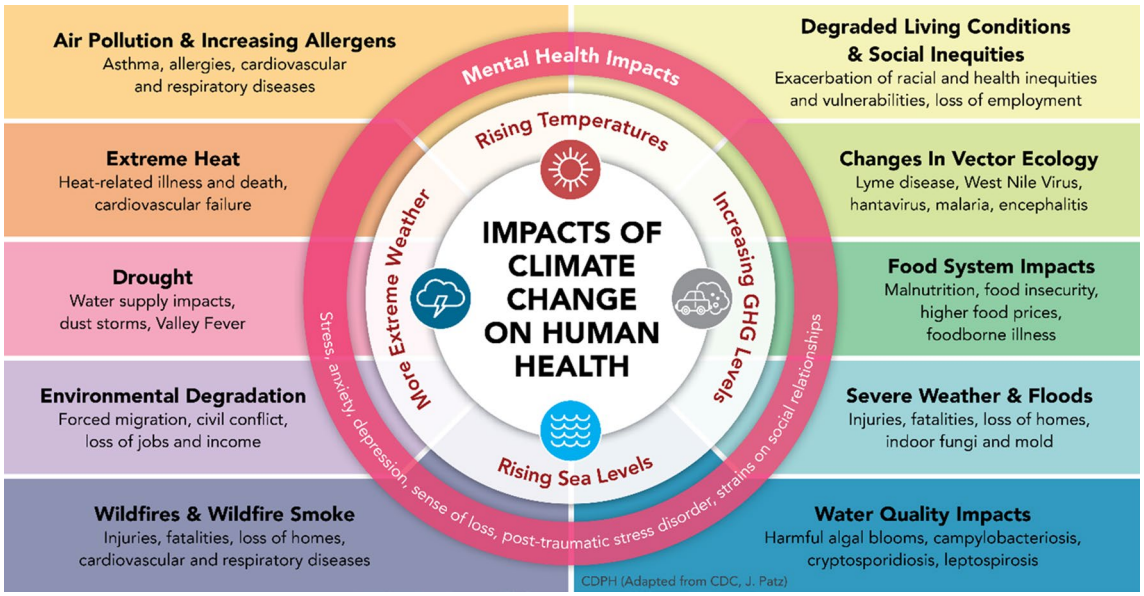


**Fig. 1** Multifactorial impacts of climate change on human health (Adapted from the U.S. Centers for Disease Control and Prevention and J. Patz, with permission). Available from: https://www.cdph.ca.gov/Programs/OHE/Pages/CCHEP.aspx

31% of dermatologists reported observing skin conditions in their communities that they believed were potentially related to climate change [6]. Not surprisingly, the skin is among the organ systems most affected by climate change as a consequence of its significant interface and exposure to the external environment (Fig. 2). Therefore, it is critical that dermatologists are not only aware of this crisis' impact on cutaneous disease, but also step forward as climate advocates for the benefit of both our patients and our planet.

## 2 Stratospheric Ozone Depletion

Ultraviolet radiation (UVR) is a known complete carcinogen that increases the risk of skin cancers including melanoma and keratinocyte carcinomas, as well as the incidence of photoaging, solar erythema, and photodermatoses (Table 1, Fig. 2) [7]. UVB radiation is particularly mutagenic due to precipitation of pyrimidine dimers that cause permanent DNA damage [8]. Melanocytes are critical to skin physiology, as the production of melanin provides UVR protection [9].

Ozone ($O_3$) is a component of the upper atmosphere, or stratosphere, and protects Earth's surface from UVR, primarily by absorbing all UVC and the majority of UVB radiation [10]. The negative impact of anthropogenic activities on upper atmospheric $O_3$ was demonstrated as early as the 1980s [10]. Many known ozone-depleting substances including chlorofluorocarbons and hydrochlorofluorocarbons are now controlled by the 1987 Montreal Protocol, an international treaty regulating substances that destroy $O_3$ [10, 11]. Despite restrictions on their production and use, ozone-depleting substances will remain problematic for decades as they are very long-lived in the atmosphere, delaying recovery of the ozone layer until mid-century or beyond, and serve as potent greenhouse gases (GHGs) [12]. Moreover, ozone-depleting substances such as nitrous oxide ($N_2O$) were not included within this treaty and therefore continue to contribute to stratospheric ozone depletion [10]. The consequences of such ozone destruction on skin health are compounded by elevated temperatures, which increase DNA damage due to UVR [13].

Skin cancer is the most common malignancy, with basal cell carcinoma (BCC) and cutaneous squamous cell carcinoma (cSCC) far more prevalent than melanoma [14]. Incident diagnoses of cutaneous melanoma within the United States increased from 44,499 in 2000 to 88,059 in 2019 [14]. Unfortunately, BCC and cSCC are not tracked by the CDC National Program of Cancer Registries, likely leading to significant underreporting of cases. However, research has demonstrated similar trends of increasing keratinocyte carcinoma incidence which may be due in part to increased exposure to UVR as a result of stratospheric ozone depletion [15–18]. For each 1% decrease in ozone layer thickness, the incidence of melanoma is expected to increase by 1–2% [19]. Similarly, the incidence of cSCC and BCC are projected to increase by 3–4.6% and 1.7–2.7%, respectively, for each 1% decrease of ozone layer thickness [19]. Of note, stratospheric ozone destruction does not have a uniform global impact, as the degree of both ozone loss and regeneration varies

**Table 1** Broad summary of climate-related factors affecting cutaneous disease

| INFECTIOUS DISEASES [23, 51, 74–76, 80, 81, 108] | | | |
|---|---|---|---|
| Pathogen Type | Disease Classification | Specific Pathogen(s) | Implicated Climate Factors |
| Bacterial | Tick-borne disease | *Borrelia burgdorferi* (Lyme disease)<br>*Rickettsia rickettsii* (Rocky Mountain spotted fever)<br>*Francisella tularensis* (Tularemia)<br>*Borrelia hermsii and Borrelia turicatae* (tick-borne relapsing fever) | Global warming<br>Expansion in geographic distribution of tick vectors, hosts, and reservoirs<br>Increased humidity<br>Extreme weather events<br>Drought |
| | Skin and soft tissue infections | *Staphylococcus aureus*<br>Group A and B *Streptococcus*<br>*Vibrio vulnificus*<br>*Vibrio parahaemolyticus*<br>*Aeromonas hydrophila*<br>*Burkholderia pseudomallei* (melioidosis)<br>*Chromobacterium violaceum*<br>Atypical mycobacteria including *Mycobacterium ulcerans* (Buruli ulcer) and *Mycobacterium abscessus*<br>*Clostridium tetani* (tetanus) | Extreme weather events<br>Flooding<br>Heavy precipitation<br>Tornadoes (tetanus)<br>Severe storms - hurricanes, typhoons, cyclones<br>Rising water temperatures (*Vibrio* spp.) |
| | Zoonotic | *Leptospira* spp. (Leptospirosis) | Extreme weather events<br>Hurricanes, typhoons<br>Heavy precipitation<br>Flooding |
| Viral | Mosquito-borne disease | Chikungunya virus<br>Dengue virus<br>Zika virus<br>Yellow fever virus<br>Rift Valley fever virus<br>West Nile virus | Global warming<br>Expansion in geographic distribution of mosquito vectors<br>Increased humidity<br>Extreme weather events<br>Flooding<br>Heavy precipitation<br>Habitat disruption<br>Urbanization |
| | Tick-borne disease | Crimean-Congo hemorrhagic fever virus<br>Bourbon virus<br>Heartland virus<br>Tick-born encephalitis virus | Global warming<br>Expansion in geographic distribution of vectors, hosts, and reservoirs<br>Increased humidity |
| | Other | *Coxsackie* spp. and *Enterovirus* spp. (hand, foot, and mouth disease) | Global warming<br>Increased humidity<br>Extreme weather events<br>Heatwaves |
| Fungal | Superficial | *Candida* spp.<br>*Malassezia furfur*<br>Dermatophytes *(Trichophyton* spp., *Microsporum* spp., *and Epidermophyton* spp.) | Global warming<br>Increased humidity<br>Extreme weather events<br>Heatwaves |
| | Deep | *Mucor* spp.<br>*Fusarium* spp.<br>Melanized fungi (chromoblastomycosis)<br>Dimorphic fungi (blastomycosis, coccidioidomycosis) | Global warming<br>Extreme weather events<br>Flooding<br>Heavy precipitation<br>Severe storms - hurricanes, typhoons, cyclones<br>Drought (coccidioidomycosis) |

Table 1 (continued)

| INFECTIOUS DISEASES [23, 51, 74–76, 80, 81, 108] | | | |
|---|---|---|---|
| Pathogen Type | Disease Classification | Specific Pathogen(s) | Implicated Climate Factors |
| Parasitic | Ectoparasite infestation | *Sarcoptes scabiei* (scabies)[a]<br>*Pediculus humanus humanus* (body lice)[b]<br>*Pediculus humanus capitis* (head lice) | Displacement, climate migration, and overcrowding in temporary shelters/camps |
| | Mosquito-borne disease | *Plasmodium* spp. (malaria)<br>*Wuchereria bancrofti* (lymphatic filariasis) | Global warming<br>Expansion in geographic distribution of mosquito vectors<br>Increased humidity<br>Extreme weather events<br>Flooding<br>Heavy precipitation<br>Habitat disruption |
| | Other vector-borne diseases | *Trypanosoma cruzi* (Chagas disease)<br>Vector: Triatome bug<br>*Leishmania* spp. (leishmaniasis)<br>Vector: sandfly | Global warming<br>Increased humidity |

| HEAT - RELATED ILLNESS [23, 62–65 ] | | |
|---|---|---|
| Disease Classification | Cutaneous Disease | Implicated Climate Factors |
| Heat-related dermatoses | Heat edema<br>Acute solar erythema<br>Miliaria<br>Transient acantholytic dermatosis<br>Intertrigo<br>Bacterial and superficial fungal infections of the skin | Global warming<br>Increased humidity<br>Extreme weather events<br>Heatwaves<br>Stratospheric ozone depletion leading to increased UV radiation |
| Impaired thermoregulation due to hypo/anhidrosis | Ectodermal dysplasias<br>Fabry disease<br>Hypermobile Ehlers-Danlos syndrome<br>Congenital insensitivity to pain with anhidrosis<br>Graft-versus-host disease<br>Atopic dermatitis and psoriasis with extensive BSA involvement | |

| AUTOIMMUNE AND INFLAMMATORY DERMATOSES | |
|---|---|
| Cutaneous Disease | Implicated Climate Factors |
| Atopic dermatitis [23, 24, 27, 29, 65, 83, 86, 109] | Global warming<br>Longer growing seasons for allergenic plants<br>Air pollution<br>Increased atmospheric $CO_2$ leading to increased plant biomass and larger quantities of pollen<br>Extreme weather events (direct impacts and secondary stress-related exacerbation)<br>Wildfires<br>Heatwaves<br>Flooding<br>Increased mold growth within domiciles<br>Displacement<br>Loss/destruction of medications |
| Psoriasis [23, 42, 43, 64, 87] | Global warming<br>Air pollution<br>Extreme weather events (direct impacts and secondary stress-related exacerbation)<br>Heatwaves<br>Wildfires<br>Displacement<br>Loss/destruction of medications |

Table 1 (continued)

| AUTOIMMUNE AND INFLAMMATORY DERMATOSES | |
|---|---|
| Cutaneous Disease | Implicated Climate Factors |
| Acne vulgaris [23, 41, 48] | Global warming<br>Air pollution<br>Increased humidity<br>Extreme weather events (direct impacts and secondary stress-related exacerbation)<br>Heatwaves<br>Displacement<br>Loss/destruction of medications |
| Hidradenitis suppurativa [23] | Global warming<br>Increased humidity<br>Extreme weather events (direct impacts and secondary stress-related exacerbation)<br>Heat waves<br>Displacement<br>Loss/destruction of medications |
| Folliculitis [23] | Global warming<br>Increased humidity<br>Extreme weather events<br>Heat waves |
| Pemphigus vulgaris [42] | Air pollution<br>Global warming<br>Extreme weather events (direct impacts as well-as secondary stress-related exacerbation)<br>Extreme heat<br>Displacement<br>Loss/destruction of medications<br>Stratospheric ozone depletion leading to increased UV radiation |
| Hailey-Hailey disease [23] | Global warming<br>Extreme weather events<br>Extreme heat<br>Increased humidity |
| CONTACT DERMATOSES | |
| Cutaneous Disease | Implicated Climate Factors |
| Rhus dermatitis [23, 76, 109] | Global warming<br>Longer growing seasons<br>Increased atmospheric $CO_2$ leading to increased plant biomass<br>Extreme weather events leading in increased exposure to culprit plants<br>Severe storms<br>Flooding |
| Phytophotodermatitis [74, 109] | Global warming<br>Longer growing seasons<br>Increased atmospheric $CO_2$ leading to increased plant biomass<br>Stratospheric ozone depletion leading to increased UV radiation<br>Extreme weather events leading in increased exposure to culprit plants<br>Severe storms<br>Flooding |
| Irritant contact dermatitis [23, 74, 76, 82] | Extreme weather events<br>Flooding<br>Exposure to contaminants, pollutants, toxic waste, sewage |
| MARINE DERMATOSES | |
| Jellyfish envenomation [110] | Global warming<br>Rising ocean temperatures<br>Ocean acidification |
| Seabather's eruption (Cnidarian larvae) [108] | Global warming<br>Rising ocean temperatures |
| Cercarial dermatitis (*Shistosoma* spp.) [74, 108, 111] | Global warming<br>Rising water temperatures<br>Heavy precipitation<br>Anthropogenic nitrogen and phosphorus runoff |

Table 1 (continued)

| | |
|---|---|
| CUTANEOUS MALIGNANCY [12] | |
| Melanoma | Global warming<br>Air pollution<br>Stratospheric ozone depletion leading to increased UV radiation |
| Keratinocyte carcinomas<br>Basal cell carcinoma<br>Squamous cell carcinoma | Global warming<br>Extreme heat<br>Air pollution<br>Stratospheric ozone depletion leading to increased UV radiation |
| PSYCHODERMATOSES [23, 74] | |
| Trichotillomania and dermatillomania<br>Neurotic excoriations<br>Prurigo nodularis<br>Onychophagia | Extreme weather events (secondary stress-, grief-, and anxiety-related induction)<br>Displacement<br>Loss of property, possessions, livelihood, and/or loved ones<br>Ecoanxiety |
| Delusions of parasitosis | Extreme weather events (secondary stress-, grief-, and anxiety-related induction)<br>Displacement<br>Loss of property, possessions, livelihood, and/or loved ones |
| TRAUMA AND EXPOSURE-RELATED [23, 74, 76, 82] | |
| Immersion foot | Flooding |
| Chilblains (Pernio) | Flooding |
| Abrasions, lacerations, puncture wounds | Extreme weather events<br>Flooding<br>Severe storms - hurricanes, typhoons, cyclones<br>Tornadoes |
| Animal bites (dogs, snakes) and stings (fire ant, *Hymenoptera* spp., rove beetles, centipedes, moths) | Extreme weather events<br>Flooding<br>Severe storms - hurricanes, typhoons, cyclones |

NUTRITIONAL DEFICIENCIES [112–114]

| Nutrient | Cutaneous Manifestations | Implicated Climate Factors |
|---|---|---|
| Protein malnutrition | Flaky paint dermatosis<br>Depigmentation in areas of friction<br>Edema<br>Sparse, dry, brittle hair with a reddish tinge<br>Thin, soft nails<br>Cheilitis and glossitis | Global warming<br>Decreased vernalization for plants<br>Ocean warming[c]<br>Algal blooms[c]<br>Extreme weather events leading to crop destruction |
| Zinc deficiency | Acquired acrodermatitis enteropathica<br>Immune dysfunction<br>Candidiasis<br>Secondary bacterial infection | Heatwaves<br>Drought<br>Heavy precipitation<br>Flooding<br>Wildfires |
| Iron deficiency | Pallor<br>Angular cheilitis<br>Atrophic glossitis<br>Koilonychia<br>Pruritus and xerosis cutis<br>Dry and brittle hair, telogen effluvium | Stratospheric ozone depletion leading to increased UV radiation<br>Lower crop yields<br>Algal blooms[c]<br>Increase atmospheric $CO_2$ |
| Vitamin A deficiency | Phrynoderma<br>Diffuse xeroderma | Nutrient depletion of staple crops<br>Ocean acidification[c] |
| B-complex vitamin deficiency | Pellagra (B3 deficiency)<br>Seborrheic dermatitis-like eruption of face<br>Angular cheilitis<br>Glossitis and stomatitis<br>Peripheral edema in beriberi (B1 deficiency) | Air pollution decreases crop yields<br>Anthropogenic nitrogen and phosphorus runoff during storms<br>Algal blooms[c]<br>Sea-level rise<br>Coastal inundation of agricultural land<br>Salt intrusion into soil and fresh water<br>Habitat destruction<br>Pollinators<br>Marine fisheries[c] |

Table 1 (continued)

[a]Scabies infestation leads to increased risk of secondary *Staphylococcal* or *Streptococcal* infections due to disruption of the skin barrier. These secondary infections are implicated in pediatric cases of post-streptococcal glomerulonephritis and rheumatic heart disease, with scabies serving as an important risk factor for these diseases globally

[b]*Pediculus humanus humanus* is also a vector of *Rickettsia prowazekii* (epidemic typhus), *Bartonella quintana* (trench fever), and *Borrelia recurrentis* (louse-borne relapsing fever)

[c]These factors negatively impact the viability of fisheries and marine-based food sources globally

by latitude and recovery is likely to be affected by climate change [12, 20].

## 3 Air Pollution

A primary role of the skin is protection from the external environment to allow for internal homeostasis. Along with providing a physical barrier, the skin is an immunologic organ with both innate and adaptive immune function [9]. The exposome encompasses an individual's internal and external environmental exposures over a lifetime and, when considered in the context of genetic influences, provides a new paradigm by which we interpret disease etiology [21].

The epithelial barrier hypothesis suggests that exposure to the urban exposome, including air pollution, causes damage to the epithelium of the skin and mucosal surfaces and increases the incidence of allergic and inflammatory diseases (Table 1) [22]. Chemical air pollutants, including $O_3$, particulate matter ($PM_{2.5}$: ≤2.5 μm; $PM_{10}$: ≤10 μm), volatile organic compounds, and GHGs such as nitrogen dioxide ($NO_2$), are components of the external exposome that are known to increase the risk of both the development and exacerbation of allergic diseases including atopic dermatitis (AD) [21]. These effects are a consequence of air pollution's lipophilicity which facilitates direct penetration of the stratum corneum, absorption through eccrine and follicular ostia, and dermal translocation from the systemic circulation [23]. Such exposure to air pollution damages the epithelium of the skin through oxidative stress which dually results in immune dysregulation and barrier dysfunction [24]. $PM_{2.5}$, $PM_{10}$, and $O_3$ have the ability to bind the aryl hydrocarbon receptor, a ligand-activated transcription factor that is found on the surface of keratinocytes and melanocytes and plays a role in the skin's adaptive response to environmental stimuli [12, 23]. This leads to the formation of reactive oxygen species (ROS), lipid peroxidation, upregulation of metalloproteases, inflammatory cytokine production, mitochondrial dysfunction, DNA damage, and apoptosis [23]. Other effects include downregulation of filaggrin and loricrin by $PM_{2.5}$, $NO_2$, and volatile organic compounds, leading to barrier dysfunction and increased transepidermal water loss [23].

Although the pathogenesis of AD remains a topic of some debate, hypotheses include inflammatory responses to environmental allergens and barrier dysfunction leading to host vulnerability [25]. The evidence of an association between air pollutants including GHGs and development of AD has been thoroughly documented within the literature [26–31]. This includes natural outdoor pollutants such as that from wildfires, anthropogenic outdoor pollutants such as carbon monoxide (CO), sulfur dioxide ($SO_2$), $NO_2$, and PM, and anthropogenic indoor pollutants [25]. A proposed mechanism underlying the association between air pollution and AD is stratum corneum damage due to increased ROS [25]. This is supported by a study of skin biopsies from 75 patients with AD that demonstrated an association between markers of damage due to ROS and AD severity [32].

With respect to maternal-fetal exposure, evidence suggests that air pollution alters DNA methylation patterns which disrupts placental function, resulting in fetal reprogramming. Specifically, prenatal exposure to traffic-related air pollution triggers epigenetic modification, tipping the adaptive immune response towards a Th2 phenotype, predisposing to AD postnatally [27]. This phenomenon is demonstrated by a cohort study of 3390 newborns enrolled between 1995 and 1999 showing an increased incidence of AD in regions with higher traffic-related air pollution [33]. A larger retrospective study of 16,686 infant–mother pairs enrolled in the 2005 Taiwan Birth Cohort Study demonstrated that AD was significantly associated with CO exposure during the gestational period, but a similar association was not found with the other air pollutants studied, including $NO_2$, $O_3$, $SO_2$, and $PM_{10}$ [34]. However, a study of 209,168 individuals aged 0 to 75 years did identify a significant association between long-term average concentration of $PM_{2.5}$, $PM_{10}$, $SO_2$, $NO_2$, and CO and the incidence of AD [28]. Within the United States (US), a study of 91,642 children demonstrated a significant association of eczema with elevated mean annual $NO_2$, $SO_2$, sulfur trioxide ($SO_3$), arsenic, nickel, lead, vanadium, and zinc. Moreover, moderate to severe eczema was significantly associated with higher nitrogen trioxide ($NO_3$), organic carbon, $PM_{2.5}$, copper, lead, and zinc [35]. Air pollution is associated with flares of AD as well, with a time-series study of 513,870 healthcare visits demonstrating that exposure to higher levels of $SO_2$, $O_3$, and $PM_{10}$ were associated with same-day visits for AD [36]. Ultrafine particles (< 0.1 μm) may also exacerbate pruritus in patients with AD [37].

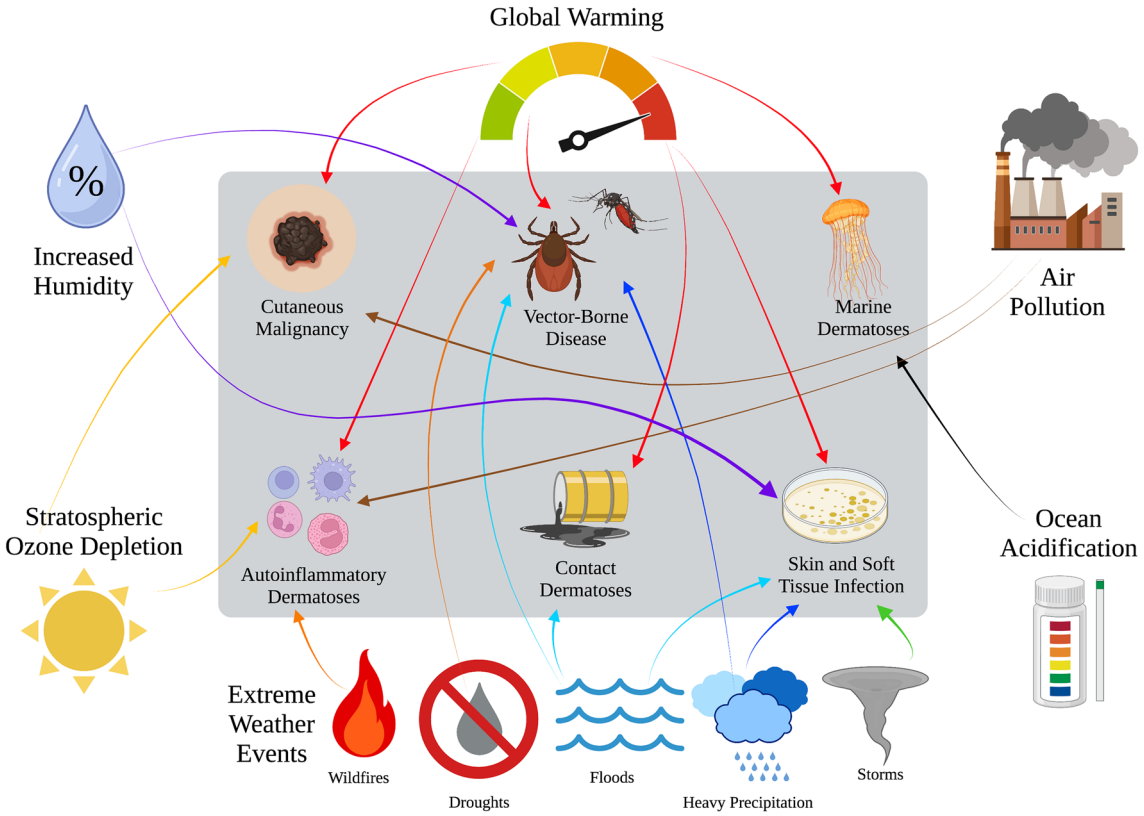


**Fig. 2** Pictorial illustrating the complex interlinkages between climate change impacts and cutaneous disease including autoinflammatory diseases, infections, contact dermatoses, malignancy, and marine insults. Figure created using Biorender.com software.

Air pollution's relevance to the dermatologist is not just in the setting of AD. PM is associated with photoaging including senile lentigines and rhytides [38, 39]. PM and polycyclic aromatic hydrocarbons (PAH) may also increase the risk of melasma and act synergistically with UV to increase cutaneous carcinogenesis [12, 38, 40]. Air pollution triggers oxidative stress and the release of inflammatory mediators within the skin, exacerbating autoinflammatory diseases. Air pollution exposure was associated with acne vulgaris in a time-series study in Beijing showing a significant association between outpatient visits for acne vulgaris and concentrations of $PM_{2.5}$, $PM_{10}$, and $NO_2$ using two-pollutant modelling [41]. Additionally, concentrations of $PM_{2.5}$ and $PM_{10}$ are associated with higher rates of hospitalization for pemphigus. Greater concentrations of air pollution may cause psoriasis to flare [42, 43]. Cadmium, a heavy metal found in batteries and used in television manufacturing and the aircraft industry, is an important environmental pollutant. Exposure is also associated with the occurrence of psoriasis [38, 44, 45]. Specifically, blood cadmium levels were found to be significantly higher in a sample of patients with a diagnosis of psoriasis, with a positive correlation between serum level and severity of disease [44].

## 4 Global Warming

The skin has a diverse microbiome that functions as a component of the immune system and provides protection from pathogenic microbes [46]. The skin microbiome typically remains stable over time despite exposure to the external environment [46, 47]. However, climate change has the potential to perturb the microbiome in the setting of global warming [48]. GHG emissions have caused a 1.2 ˚C increase in global temperatures since pre-industrial times [49]. If radical steps are not taken to combat the climate crisis, this is expected to increase to 1.5 ˚C between 2030 and 2052 [49]. With a 1.5 ˚C rise in average global temperature, the risks to human health, food and water security, and ecosystem viability, will increase significantly [49].

Increased temperatures due to global warming have the potential to disrupt the skin microbiome, thereby impacting the incidence of dermatologic disease [48]. Increased temperature, as well as humidity, have been associated with overall growth of bacteria on the skin (Table 1, Fig. 3) [48, 50]. In a population-based study of monthly skin and soft tissue infection (SSTI) incidence, temporal variation in SSTI was significantly associated with both mean

temperature and specific humidity [51]. In a retrospective analysis of SSTI within the US ($n = 616{,}375$), the prevalence of community acquired methicillin-resistant *Staphylococcus aureus* was greatest in the South, illustrating a possible association between higher temperatures and *S. aureus* growth [52]. In contrast, preferential growth of *Staphylococcus epidermidis* over *S. aureus* at lower temperatures has been demonstrated [48, 53]. Production of sebum was reported to increase by 10% per 1 ˚C increase in temperature, which may in turn increase growth of microbes including *Cutibacterium* spp. and *Malassezia* spp. [48, 54]. Other results of anthropogenic activities, including air pollution and elevated UVR, also influence the cutaneous microbiome [48]. The air pollutant $N_2O$ disturbs commensal microbes with potential for dysbiosis in the setting of greater negative impact on *Staphylococcus capitis* and *Corynebacterium tuberculostearicum* than *S. aureus* [53, 55]. Research has further demonstrated UVR-induced disturbance of the skin microbiome, including increased *Cyanobacteria* spp. and decreased *Lactobacillaceae* spp. and *Pseudomonadaceae* spp. with varying doses of both UVA and UVB radiation, resistance of *Malassezia* spp. to UVR due to the presence of a UV-filtering compound, and decreased superantigen production by *S. aureus* when exposed to UVB and psoralen plus UVA [48, 56–58].

Dermatologic diseases known to be associated with disturbance of the skin microbiome include AD, acne vulgaris, and psoriasis. AD severity, specifically, is associated with *S. aureus* colonization [48, 59]. Bacterial gene sequencing of 16s ribosomal RNA from the skin of children with AD demonstrated increased *S. aureus* sequences during flares compared with both baseline and post-treatment skin [59]. Increasing temperatures may therefore lead to increased severity and more frequent flares of AD due to elevated proportions of *S. aureus* and decreased proportions of commensal bacteria that inhibit *S. aureus* including *S. epidermidis* [48]. Incidence of acne vulgaris may also increase due to elevated temperatures as a result of the known association with *Cutibacterium acnes*, which would be expected to increase with elevated sebum production [48]. The potential influence on psoriasis is less clear. Although associated with increased levels of *S. aureus,* psoriasis is associated with lower levels of *Cutibacterium* spp. [48, 60].

## 5 Extreme Weather Events

Climate change has led to increasing frequency of extreme weather events, including heatwaves, droughts, wildfires, heavy rainfall, flooding, and hurricanes [1]. Along with the direct impacts on cutaneous disease, additional repercussions of extreme weather events include conflict exacerbation, forced migration, deterioration of mental health, and greater transmission of infectious diseases, all of which further enhance the risk of cutaneous disease (Table 1, Figs. 2 and 3) [1, 23].

### 5.1 Heatwaves

The skin plays a critical role in thermoregulation through vasoreaction as well as sweat production from eccrine glands [61]. In the setting of overheating, vasodilation facilitates radiation of heat from the surface of the skin, and sweating allows for evaporative cooling [9]. Global warming due to GHGs, particularly methane ($CH_4$) and $CO_2$, has led to an increased incidence of heatwaves and associated risk of heat-related illness (HRI) (Table 1) [62]. HRI range from mild manifestations including miliaria and heat edema to potentially fatal heat stroke [23, 62]. Those at greatest risk include infants, the elderly, individuals with poor aerobic conditioning, those with comorbidities (obesity, cardiovascular disease, renal disease), and individuals who perform strenuous activity outdoors (often occupation-related) [62].

Dermatologic conditions that cause either hypohidrosis or anhidrosis increase the risk of HRI. These include ectodermal dysplasias, as well as Fabry disease, hypermobile Ehlers-Danlos syndrome, and congenital insensitivity to pain with anhidrosis [9, 62]. The various congenital ichthyoses are also commonly associated with anhidrosis or hypohidrosis in the setting of dysfunctional sweating due to obstruction of eccrine ducts [9, 62]. In acute graft-versus-host disease, immune attack of the eccrine glands may lead to loss of physiologic sweating in the chronic phase [62, 63]. Patients with inflammatory dermatoses such as AD or psoriasis covering significant body surface area are also thought to be at risk of HRI [62]. Potential mechanisms include obstruction of eccrine ducts, autonomic dysfunction, and inhibition of sweat response by histamine [62, 64–66]. Risk of HRI due to abnormal thermoregulation is also observed in non-dermatologic diseases such as Parkinson's disease (dyshidrosis, hyperhidrosis, hypohidrosis), multiple system atrophy (hypohidrosis), multiple sclerosis (hypohidrosis), and Sjögren syndrome (hypohidrosis) [62, 67–69]. Rarely, hypohidrosis or anhidrosis can be acquired due to acute solar erythema, miliaria profunda, or thermal burns involving the dermis and covering significant body surface area [62].

Medications, including many commonly prescribed by dermatologists, also have the potential to interfere with appropriate thermoregulation in the setting of extreme heat, particularly in the elderly [23, 62]. An extensive list of drugs have anticholinergic properties, which can lead to inhibition of nerve signaling necessary for eccrine sweating [9, 23]. The major drug classes with anticholinergic effects frequently utilized by dermatologists are antihistamines,

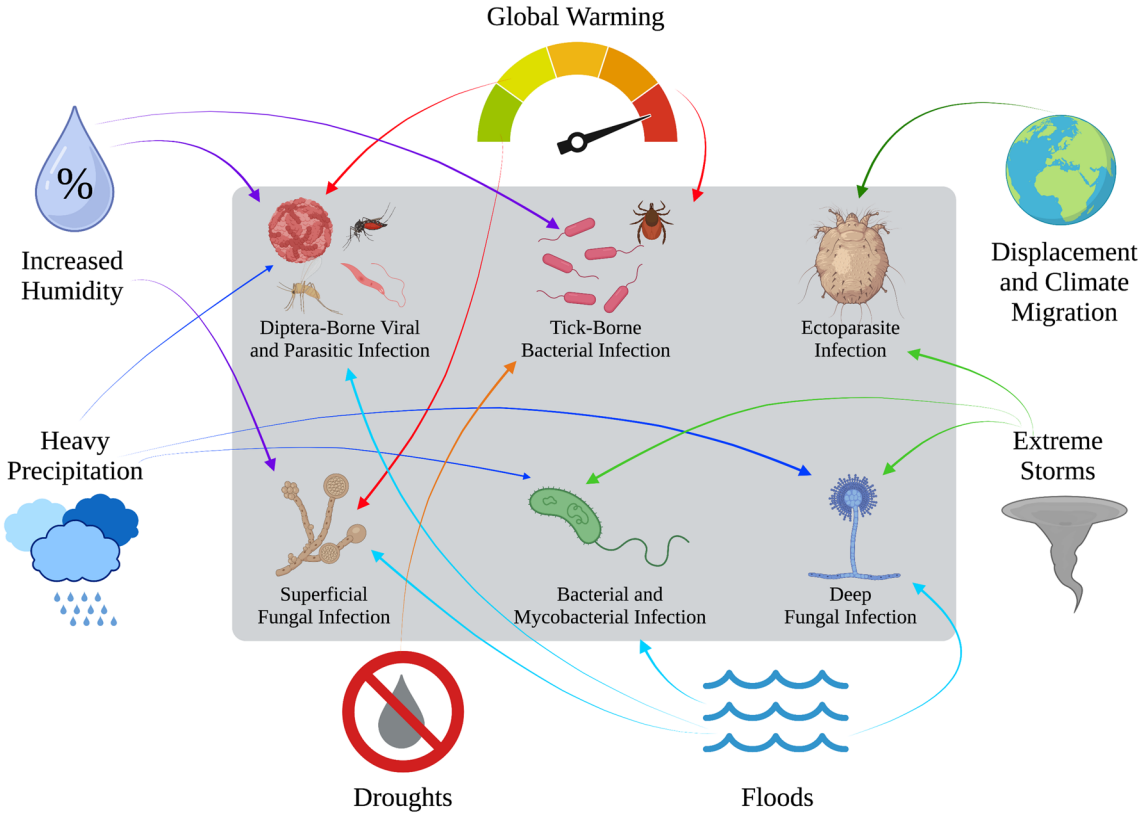


**Fig. 3** Pictorial illustrating the complex interlinkages between climate change impacts and infectious diseases of dermatologic significance including bacterial, mycobacterial, fungal, vector-borne, and ectoparasitic infections. Figure created using Biorender.com software

tricyclic antidepressants like doxepin and amitriptyline, glycopyrrolate, and botulinum toxin [62]. Other commonly prescribed medications that increase risk of HRI include antihypertensives and diuretics due to decreased vascular responsiveness and dehydration [62]. In a case-control study of 78 patients admitted for heat stroke, the length of hospital stay was longer in those on a medication known to alter thermoregulation, most commonly anticholinergics, diuretics, or sympathomimetics [70]. In a larger study using prescription event symmetry analysis of 6700 patients admitted for dehydration or HRI, patients on anticholinergic agents and cardiovascular medications were at significantly higher risk of incident hospital admission [71]. Interestingly, a significant association was not observed for antihistamines [71].

Numerous cutaneous diseases are triggered or exacerbated by heat, humidity, and sweating (Table 1, Fig. 2). These include miliaria, transient acantholytic dermatosis, intertrigo, tinea versicolor, dermatophyte infections, folliculitis, hand, foot, and mouth disease, mastocystosis, Hailey-Hailey disease, and hidradenitis suppurativa, among others [23]. With the number of both high-heat and high-humidity days steadily increasing, higher incidences and more severe flares of many heat-sensitive dermatologic diseases could be observed [23].

## 5.2 Flooding

Flooding increases agricultural and road-runoff, churns the soil, damages industrial facilities, and may overwhelm sewer systems, leading to contamination with many pathogens and chemicals [23]. Moreover, trauma from submerged objects as well as maceration from prolonged exposure to flood water have the potential to compromise the skin's natural barrier, creating portals of entry for both irritants as well as typical and atypical pathogens (Table 1) [23]. In the setting of climate change, the incidence of floods has increased due to melting polar ice, rising sea levels, and more frequent and severe extreme weather events including heavy precipitation, monsoons, tsunamis, and hurricanes [72]. Within the first half of 2022, floods were the most common natural disaster globally, causing more deaths and economic damage than extreme temperatures, drought, earthquakes, storms, or wildfires [73].

During the initial impact phase of a flood, trauma with associated risk of secondary wound infection is often observed [74]. Of 777 patients with traumatic wounds following a 2004 tsunami in Bangkok, 515 (66.3%) developed SSTI. Of the 305 patients with positive cultures, 71.8% were polymicrobial, with Gram negative bacilli being most

common [75]. *Aeromonas hydrophila,* a pathogen associated with fresh-water aquatic environments and isolated most frequently in warmer months, was the primary culprit [75]. Other atypical pathogens implicated in SSTI in the setting of flooding include *Vibrio vulnificus*, *Vibrio parahaemolyticus, Burkholderia pseudomallei* (cutaneous melioidosis), *Mycobacterium ulcerans* (Buruli ulcer), *Mycobacterium abscessus* and other atypical mycobacteria, *Mucor* spp., *Fusarium* spp., *Blastomyces dermatitidis,* and other fungi including those associated with chromoblastomycosis [74, 76]. Tetanus due to wound contamination is also reported [77, 78]. In the month following a 2004 tsunami in Indonesia, 106 hospital admissions for tetanus were reported in the surrounding region [77]. Zoonotic infections such as leptospirosis are increasingly reported after heavy rainfall and flooding [23]. Importantly, many of these pathogens carry a high risk of mortality, often secondary to necrotizing fasciitis or sepsis, if not recognized and treated promptly.

Destruction of infrastructure and lack of access to potable water and sanitary living conditions in the setting of flooding are associated with a second wave of dermatologic conditions. This includes arthropod bites and stings in the setting of heavy rainfall, standing water, and/or increased humidity [79]. Proliferation of mosquitoes increases the risk of infectious diseases including arboviruses and malaria. Following flooding in Pakistan in 2022, the World Health Organization (WHO) reported a surge in cases of dengue fever, which is transmitted by the *Aedes aegypti* mosquito [80]. Spread of phlebotomine sand flies, the vector of *Leishmania* spp., is also associated with flooding and poor sanitation [74, 81]. Noninfectious manifestations include immersion foot, miliaria, and irritant and allergic contact dermatitis [74, 76, 82]. In a study of 235 patients in Indonesia following a 2004 tsunami, the second most common skin problem diagnosed was 'eczema,' with contact dermatitis comprising approximately three quarters of these cases [82]. In large part, contact dermatitis is secondary to exposure to pollutants and chemicals that frequently contaminate flood waters [23]. Overcrowded temporary living conditions due to forced displacement are associated with outbreaks of measles and ectoparasitic infestations including scabies and lice [74, 78].

### 5.3 Wildfires

As described in Sect. 3, air pollutants including $PM_{2.5}$ can damage the epithelial barrier of the skin, leading to increased incidence of dermatologic disease. Along with anthropogenic sources of air pollution, wildfires lead to elevated levels of $PM_{2.5}$ [83]. The incidence and severity of wildfires are increasing, with 19 events in 2021 in the US, compared with an average of 11 events annually from 2001 to 2020 [84]. California has seen a sharp rise in wildfires, with a 5-fold increase in annual burned area from 1972 to 2018 [85]. The primary contributor to this phenomenon is increased forest-fire extent due to warmer, dryer summer months as a consequence of climate change [85].

Although chronic elevations of $PM_{2.5}$ due to wildfires likely increase the burden of dermatologic disease, an acute association between wildfires and AD has also been demonstrated [83]. Following the 2018 Camp Fire in California, a cross-sectional time-series study assessing air pollution and dermatology clinic visits ($n = 8049$) was performed [83]. Air pollution was evaluated using fire status (a binary variable of whether a fire occurred within that week), concentration of $PM_{2.5}$, and satellite-based smoke plume density scores [83]. The rate of weekly dermatology clinic visits for AD was significantly greater during the Camp Fire versus during non-fire weeks in both pediatric and adult patient populations [83]. Within the adult population, the weekly number of systemic medications prescribed was significantly associated with air pollution exposure [83]. The adjusted rate ratio for weekly dermatology clinic visits for pruritus among pediatric patients was 1.82 for fire status and 1.55 for satellite-based smoke plume density scores, and each 10 μg/$m^3$ increase in $PM_{2.5}$ concentration was associated with a 7.7% increase in pediatric clinic appointments for pruritus [83]. Interestingly, older adults aged ≥ 65 years demonstrated statistically significant higher rates of clinic visits for both AD and pruritus acutely during the same wildfire, compared with younger adults. This finding suggests greater vulnerability to air pollution among elderly individuals and may be attributable to age-related compromise of skin barrier function [86]. Conversely, while wildfires in California also contributed to psoriasis flares, increased healthcare utilization for adults with psoriasis was delayed, beginning 5 weeks after the wildfire and peaking 8–9 weeks post-fire [87]. These findings highlight that dermatologists must be prepared to manage increased flares of inflammatory diseases not only during actively burning wildfires, but also in the months following.

### 5.4 Impacts on Healthcare Delivery

Beyond the direct resultant health harms already discussed, extreme weather events deeply affect the ability to provide care to the community. Damage to critical infrastructure including hospitals, power grids, and water sanitation facilities is often incurred, and emergency services are disrupted when roads are impassible [23]. Power outages and lack of access to potable water compromises sanitation and hygiene. Destruction of domiciles results in displacement, during which medications may be lost or damaged and patients may have no means of transportation to seek care [23]. These factors combine to impose severe emotional stress on community members, upending access to medical care during

disasters and exacerbating underlying chronic conditions including dermatologic disease.

## 6 Climate Justice and Migration

In July of 2022, the United Nations General Assembly unanimously passed a resolution recognizing "a clean, healthy and sustainable environment as a human right" with the goal of reducing environmental injustice, closing protection gaps, and empowering people, especially those marginalized and most vulnerable [88]. The spectrum of health consequences, as well as economic and social repercussions, of climate change disproportionately affect individuals living in low- and middle-income countries (LMICs), despite lesser contributions to the climate crisis compared with high-income countries [89–91]. The US, the European Union, China, and the Russian Federation hold significant responsibility for global warming, each accounting for over 100 billion metric tons of cumulative $CO_2$ emissions [90]. In contrast, the African continent is responsible for <4% of GHG emissions but is expected to have 500 times greater loss of healthy life-years due to climate change than European nations [92–94]. Eight of the ten countries most affected by extreme weather events from 2000 to 2019 were LMICs [95].

Vulnerable and marginalized populations in both LMICs and high-income countries shoulder disproportionate impacts from the climate crisis. These groups include children, the elderly, women, pregnant individuals, Blacks, Indigenous Peoples, People of Color, people facing poverty, unhoused individuals, those with a chronic disease or disability, as well as those with occupational exposure to extreme climate [89, 90, 96, 97]. Infants, children, and the elderly are at high risk of HRI, as are individuals carrying a pregnancy and with comorbidities such as diabetes mellitus, hypertension, renal disease, and cardiac insufficiency [62]. In the setting of structural racism and historic discriminatory housing policies in the US, such as redlining, communities of color are often exposed to hazardous conditions including more extreme temperatures resulting from the urban heat island effect, and significantly higher concentrations of air and water pollution as highways, toxic waste sites, and industrial facilities are more frequently sited near low-income communities and previously redlined neighborhoods which have remained minoritized [23, 97]. Redlining is a form of racial discrimination that began with the New Deal's National Housing Act of 1934, which promoted the exclusion of communities of color, specifically Black communities, from mortgage loans by deeming their neighborhoods as "risky investments" and thus outlining these neighborhoods in red on urban maps [98, 99]. Today, neighborhoods that were formerly redlined are more likely to house low-income and minority communities and disproportionately experience greater health impacts from heat and pollution [100]. Faulty and decaying infrastructure along with a lack of investment in these neighborhoods also leaves low-income communities and communities of Color vulnerable in the setting of extreme weather events [97]. Structural disparities affect access to medical care and the capacity of vulnerable and marginalized communities to adapt to climate change and respond to extreme weather events. As such, climate change is frequently referred to as a 'force multiplier' because the pressures from climate-related impacts accentuate existing disparities and widen the gaps in social determinants of health and access to care for marginalized groups such as Black, Hispanic, Indigenous, and low-income individuals.

Secondary or indirect repercussions of climate change similarly impact vulnerable communities to a greater extent. Resource scarcity including food and water insecurity due to drought and heatwaves, crop and infrastructure loss during floods and severe storms, and expansion of crop-destroying pests due to global warming all contribute to an increased risk of malnutrition, particularly in low-income countries [92]. Conflict is an additional trigger for displacement and migration and may stem from sociopolitical stressors amplified by climate events. Specifically, the association between higher temperatures and violence has been well documented [101]. An event coincidence analysis using data on armed-conflict outbreaks and climate-related natural disasters reported that 23% of armed-conflict outbreaks in ethnically highly-fractionalized countries between 1980 and 2010 coincided with climate-related extreme weather events [102].

Consequently, direct effects of climate change, such as widespread destruction from extreme weather events as well as ongoing indirect impacts on food and water insecurity and conflict, are capable of triggering mass population displacement [103]. These factors, along with expansion and shifting patterns of infectious and vector-borne diseases as a result of climate change, serve as drivers of migration [23]. A recent report projects that 143 million individuals living in Sub-Saharan Africa, South Asia, and Latin America could be displaced due to climate change by 2050, with the greatest impact on poor and climate-vulnerable communities in these LMICs [104]. Due to either acute displacement from extreme weather events or climate migration, overcrowding and a lack of sanitation in temporary shelters and refugee camps, are associated with proliferation of ectoparasitic infestations, including scabies, body lice, and head lice (Table 1, Fig. 3) [103, 105]. Importantly, scabies infection is a well-documented risk factor for bacterial superinfection of the skin and a leading driver of pediatric streptococcal sequalae, including glomerulonephritis and rheumatic fever, globally [23, 101]. Crowded living conditions also increase

the risk of serious louse-borne diseases including epidemic typhus, trench fever, and relapsing fever [103].

## 7 Discussion

The AAD's Position Statement on Climate and Health declares a commitment to "raise awareness about the effects of climate change on skin health and skin disorders, work with other medical societies to educate the public and mitigate the effects of climate change on global health, educate our patients about the effects of climate change on the health of their skin, and support and facilitate efforts of AAD members to decrease the carbon footprint of their dermatology practices and medical organizations" [4]. Dermatologists should be aware of the myriad ways in which climate change, skin health, and the practice of dermatology intersect (Table 1, Figs. 2 and 3).

The healthcare sector is carbon-intensive and attention to reducing the GHG emissions of our own care delivery is essential. This can be accomplished through measures such as implementation of programable thermostats, LED lighting, power-down policies for computers and equipment at night and on weekends, HVAC (heating, ventilation, and air conditioning) commissioning, reprocessing of single-use equipment when appropriate, sustainable procurement of medical supplies, procedural waste audits, proper waste segregation, greater reliance on virtual media for patient care, interviews, and education to reduce unnecessary travel, and use of carbon offsets when professional travel is required [106, 107]. In addition, climate literacy among dermatologists ensures that we as a profession are committed to providing quality medical care along with appropriate counseling to our patients. Such patient advice may include discussions of climate-related risk factors for skin disease, targeted risk communication prior to extreme weather events (evacuation plans, go-bags containing needed supplies and medications), appropriate preventative measures including donning UV-protective clothing and seeking shade to dually provide heat and sun protection, and consultation with web- and app-based air quality, UV, and heat indices prior to outdoor exposures. Patients with chronic dermatoses should be counseled on potential climate-related triggers, and those with comorbid diseases or who are prescribed certain classes of medications must be advised on the increased risk of acute HRI, including potentially fatal heat stroke.

Beyond the importance of climate change to the provision of dermatologic care, dermatologists are poised to act as climate advocates, individually in their communities and collectively through their health systems and medical societies. Dermatologists are uniquely trained to consider the interaction of multiple medications and organ systems and are particularly adept at solving complex medical problems. By nature, we are curious, intelligent, resourceful, and innovative. Now more than ever, we must draw upon those traits and skills to fully lean into leadership on climate change and strive for depoliticization by focusing the lens of health on this topic. We, as dermatologists, must not neglect the tremendous opportunity before us to advocate for the wellbeing of patients, educate the public and policy makers on the cutaneous manifestations of climate change, and create innovative solutions for mitigation that reduce health harms.

### Declarations

**Funding sources** The authors have no relevant funding source.

**Conflicts of Interest** The authors have no relevant conflicts of interest.

**Ethics approval** Not applicable.

**Patient consent to participate/publish** Not applicable.

**Availability of data and material** Not applicable.

**Code availability** Not applicable.

**Author contributions** Annika Belzer—conceptualization, investigation, writing, figure recreation. Eva Rawlings Parker—conceptualization, writing, critical review, editing, and supervision.

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