The effect of climate change on skin disease in North America

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Global temperatures continue to rise, reaching new records almost every year this decade. Although the causes are debated, climate change is a reality. Consequences of climate change include melting of the arctic ice cap, rising of sea levels, changes in precipitation patterns, and increased severe weather events. This article updates dermatologists about the effects of climate change on the epidemiology and geographic ranges of selected skin diseases in North America. Although globalization, travel, and trade are also important to changing disease and vector patterns, climate change creates favorable habitats and expanded access to immunologically naïve hosts. Endemic North American illnesses such as Lyme disease, leishmaniasis, and dimorphic fungal infections have recently expanded the geographic areas of risk. As temperatures increase, epidemic viral diseases such as hand-foot-and-mouth disease may develop transmission seasons that are longer and more intense. Chikungunya and dengue are now reported within the southern United States, with Zika on the horizon. Cutaneous injuries from aquatic and marine organisms that have expanding habitats and longer durations of peak activity include jellyfish envenomation, cercarial dermatitis, and seabather eruption, among others. Skin cancer rates may also be affected indirectly by changes in temperature and associated behaviors. (J Am Acad Dermatol 2017;76:140-7.)

Key words: cercarial dermatitis; chikungunya; climate change; coccidioidomycosis; dengue; environmental change; global warming; hand-foot-and-mouth syndrome; herpangina; jellyfish; leishmaniasis; Lyme disease; seabather eruption; skin cancer; skin disease; swimmer itch; Zika.

Over the past few decades, our planet has entered a period of major changes in climate and weather patterns, almost certainly as a result of human activity.[1] Some natural fluctuations in global average surface temperatures are expected, but 17 of the warmest years on record have occurred in the past 18 years.[2] Combustion of fossil fuels and destruction of forests are the main contributors, with the latter rendering the natural world unable to maintain carbon homeostasis. These 2 activities account for up to 70% of greenhouse gas emissions,[3] which then serve to absorb infrared solar radiation in the atmosphere and trap energy that otherwise would be reflected. Climate change encompasses average planetary surface temperature and other factors that can alter species composition: temperature-related parameters (magnitude of diurnal-nocturnal temperature shifts, magnitude of annual temperature peaks and nadirs, frost dates); precipitation-related parameters (total precipitation, snowfall, seasonality, humidity); and atmospheric parameters (cloud cover; speed and direction of prevailing winds).

In 2014, the Fifth Intergovernmental Panel on Climate Change systematically reviewed the data on climate change and several high-impact medical journals published commentaries, although none emphasized skin diseases.[4-6] This article reviews publications specific to climate change and skin disease in North America.

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CHANGING DEMOGRAPHICS OF ENDEMIC DISEASES OF NORTH AMERICA

Complex biologic and abiotic environmental factors, along with human-associated alterations, influence the geographic distribution of many infectious diseases. This is especially true in temperate zones because climate change allows expansion of the natural range of pathogens, hosts, reservoirs, and vectors that allow diseases to appear in immunologically naïve populations.[7] In the United States, the incidence of Lyme disease, for example, caused by the tick-borne spirochete, *Borrelia burgdorferi*, increased from an estimated 10,000 cases/y in 1995 to 30,000/y in 2013.[8,9] The Centers for Disease Control and Prevention (CDC), however, estimates that the true incidence in the United States reached 300,000/y in 2012.[9] The area of Lyme disease's endemicity continues to expand from the New England region, where it was first identified to new areas, in conjunction with the expanding range of *Ixodes* tick vectors. The range of those ticks seems to be expanding inexorably because the preferred habitat for *Ixodes* tick and its mammalian hosts is expanding. In Canada, the area of endemism has spread from southern Quebec to Ontario, the Maritime provinces, Manitoba, and British Columbia between 1990 and 2003.[10] Although the density of *Borrelia burgdorferi* starts low when deer ticks newly inhabit an area, within 4 years *Borrelia burgdorferi* typically appears in these populations of *I scapularis*.[11]

In the US Southwest, *Coccidioides immitis* and *C posadasii* have been historically located in hot, arid habitats of Arizona, Utah, and California. Recently, arid regions of eastern Washington state have also become endemic.[12] Consistently, the incidence of coccidioidomycosis has increased, partly because of immunologically naïve retirees who move to endemic areas, but also because longer dry seasons and more frequent wind storms aerosolize the fungal spores.[13,14] Specifically in Arizona, the annual incidence has increased from 33 to 43/100,000 population between 1998 and 2001 with climatic and environmental factors explaining 75% of the model.[15] In Kern County, California, the incidence paralleled temperature patterns, but were also influenced by environmental alterations from construction work.[16]

CAPSULE SUMMARY

- There is near universal scientific agreement that the Earth is warming.
- Numerous bacteria, viruses, fungi, and parasites are responding to changing weather patterns in North America.
- Dermatologists should be able to recognize changing patterns of skin disease associated with climate change.

Dryer weather, accompanied by drought and elevated temperatures, has resulted in significantly increased rates, increased geographic range, and increased infectious cycles of leishmaniasis.[17,18] Habitat fragmentation and deforestation also play a role in increased prevalence of the disease and vector.[19] Using well-established climate models, researchers have shown an expansion of competent sand fly vectors into areas farther from the equator.[20] Autochthonous transmission already has a foothold along the United States-Mexican border states because the presence of both an established rodent reservoir for *Leishmania mexicana* and competent sand fly vectors, *Lutzomyia diabolica* and *Lutzomyia anthrophora*, permits a local transmission cycle.[21] Both the rodent reservoir and sand fly are expected to bring *Leishmania* species (spp) northward, potentially even to the United States-Canadian borderlands by 2080.[21]

Hand-foot-and-mouth disease is a classic seasonal enteroviral infection in temperate climates. The incidence correlates with the average temperature and average rainfall.[22] Similar associations with increased average temperature have been shown in an urban population with hand-foot-and-mouth disease, and even stronger associations with another enteroviral infection, herpangina.[23] Using predictive modeling, an increase in weekly average air temperature by 2°C, 2 days per week, will increase the incidence of herpangina by 43%.[23] Humidity is another climate variable associated with epidemics of hand-foot-and-mouth disease.[24]

Dermatologists should be aware of changing seasons and locations along with the typical clinical findings of diseases that are already well established in North America (Supplemental Table I also includes Chagas disease).

ARBOVIRAL DISEASE

Mosquito vectors are unwittingly transported to new geographic footholds through the global used-tire and used-car trade.[25] Dengue, chikungunya, and Zika viruses are spread by the *Aedes aegypti* and *A albopictus* mosquitos. These invasive mosquitos, originally from Africa and Asia, respectively, have spread widely throughout North America.

Chikungunya, caused by a togavirus, can cause severe joint pain, high fever, and a morbilliform

eruption. In infants, chikungunya can cause rusty-brown facial hyperpigmentation. It can leave permanent arthritis. Originally, it had a sylvatic transmission cycle, confined to areas of southeast Africa, involving nonhuman primates and a few species of *Aedes*. Human beings were involved incidentally, but human infection increased as anthropophilic mosquitos, *A aegypti* and *A albopictus*, created a human-mosquito cycle that spread throughout sub-Saharan Africa and countries bordering the Indian Ocean.[26] In 2013, it first appeared in the Western Hemisphere and spread rapidly throughout the Caribbean and Latin America.[27] It experienced rapid geographic spread, and has potential for further migration as the range of *A albopictus* now extends as far north as Connecticut.[28]

Dengue is a flavivirus transmitted by *Aedes* spp mosquitos resulting in a petechial exanthem, fevers, headache with retro-orbital pain, nausea, and severe bone pain. Until recently, there had not been autochthonous transmission in the United States since the 1930s.[29] Now, many locally acquired cases in Hawaii, Texas, and Florida have been reported and up to 38% of residents in Brownsville, TX, and 5.4% in Key West, FL, have serologic evidence of previous dengue infection.[29] These high levels of past infection with the presence of the *Aedes* spp in much of southern and southeastern United States suggest the potential for autochthonous transmission cycle throughout the southeastern United States and likely farther north as well. Although there have been no outbreaks of dengue near major US cities, the combination of a southern population shift and higher temperatures is ominous. For example, in Singapore, the annual number of dengue cases has increased from only a handful in 1980 to 14,000 in 2004 because of population growth and increased temperatures.[30] Increased rainfall has also been associated with dengue.[31]

The Zika virus is an emerging virus at risk for a pandemic, already affecting countries in both hemispheres, including recent expansion from South America up into the United States. It is a flavivirus related to dengue, West Nile, and yellow fever viruses and transmitted also through *Aedes* spp. It was first isolated in a rhesus monkey from Uganda's Zika forest and was thought to be confined to Africa and southeast Asia until it spread to the remote Pacific Islands in 2007.[32] Although the virus was known in Africa as an infrequent cause of a mild exanthem, the current epidemic strains tend to cause disease in 20% of patients after a 3-to 12-day incubation period; patients most commonly present with an exanthem, fever, myalgia, arthralgia, edema, headaches, and conjunctivitis.[33] The exanthem tends to develop over the face and spread caudally, while sparing the palms and soles, and may have a petechial component.[34] By April 2014, 66% of the population of French Polynesia had been infected with the Zika virus.[35] Since 2014, it is now established in more than 27 countries in the Western Hemisphere.[33] The virus likely proliferated in Brazil for a year before it was detected in May 2015, because of its clinical similarity with chikungunya and dengue viruses.[36] Based on imported cases, distribution of competent vectors, and climate changes, Bogoch et al[37] estimate that the Zika virus will establish itself throughout North America with the exception of Canada. The possibility of Zika virus spreading northward appears to rest on the unknown vector competence of *A albopictus*. If efficient, transmission may occur as far north as southern Canada.[38] Dermatologists should also be aware of the potential for semen to harbor and transmit the virus.[39] The biggest threat now appears to be the neurotropic nature of the current virus with multiple early reports indicating virus replication in adult and fetal neural tissue, and epidemiologic studies linking severe neurologic disease to the virus.[33,35,40,41] These arboviral diseases are difficult to distinguish clinically and require serologic or polymerase chain reaction testing.[42] In the face of climate change, global trade, and widespread invasive *Aedes* mosquito species, dermatologists should be ready to identify and diagnose the clinical findings of dengue, chikungunya, and Zika viruses.

DISEASE ASSOCIATED WITH WATER WARMING AND FLOODING

Major water-associated aspects of climate change include rising sea levels, flooding, increased water temperature, decreased ocean pH (because of carbon dioxide dissolution in oceans), and invasions of nonnative species.[43] Skin diseases, along with respiratory and diarrheal illnesses, are the most likely diseases seen after natural disasters associated with flooding (Supplemental Table II).[44]

Warming and acidification of the oceans contribute to increased jellyfish populations.[45,46] Only recently have large Portuguese man-of-war aggregations been found along the southeastern US coastline with large outbreaks of envenomizations.[47] Similarly, a Pacific jellyfish, *Porpita pacifica*, was first reported to cause direct envenomation in 2005 and since has skyrocketed off Japanese beaches.[48] Indeed, a systematic review of jellyfish populations in marine ecosystems across the globe has confirmed the worldwide increase in abundance.[49]

Seabather eruption, caused by jellyfish larva (planulae), most commonly of *Linuche unguiculata*, has become increasingly common potentially because of increased ocean temperatures.[50] This eruption can occur in up to 16% of patients swimming during peak seasons in southeast Florida.[50] There is a cold water variant seen along the Northeast's Atlantic coast caused by *Edwardsiella lineata*, a parasitic jellyfish.[51] Because of warming water temperatures, milder winters in estuaries, and downstream effects on the food chain, the host ctenophore species have bloomed earlier,[52,53] likely allowing for increasing frequency of Northern seabather eruption.

Swimmer itch, or cercarial dermatitis, occurs in about 7% of patients exposed to northern US freshwater.[54] Increased water temperatures and increased nitrogen and phosphorus runoff may lead to an abundance of avian schistosomes (commonly *Trichobilharzia* spp). The same factors improve survival of their intermediate snail host, to improve the schistosome life-cycle efficiency and risk of human cercarial dermatitis.[55] Shallow freshwater lakes and ponds are higher risk.[56] Even arid regions, such as the US Southwest, can host outbreaks of cercarial dermatitis, particularly in man-made lakes and irrigation ponds.[57] With climate change, parts of North America, particularly the Great Lakes region, are expected to have substantially higher precipitation leading to more outbreaks of waterborne disease.[58]

Vibrio parahaemolyticus and *V vulnificus* reproduce and proliferate more rapidly in warmer waters, and higher than average temperatures in the Gulf of Mexico contribute to higher numbers of *V vulnificus* illnesses from consuming raw oysters.[59] Even small changes in peak water temperature have been correlated with local hospital admissions for *V vulnificus*–associated wound infections, cellulitis, and sepsis.[60]

Extreme climactic events have also been associated with *Burkholderia pseudomallei*, the cause of melioidosis (which affects the skin and soft tissue as a localized infection, abscess, ulceration, cellulitis, or necrotizing fasciitis).[61] Melioidosis is typically sporadic but several recent clusters of disease are clearly associated with unusually high precipitation and hurricanes, floods, and tornados may allow for outbreaks.[62,63] The CDC reports 37 confirmed cases in the United States between 2008 and 2013. At least 5 cases were confirmed to have no travel outside of the United States; 3 of those 5 occurred since 2010.[64,65]

SKIN CANCER

Ozone depletion by chlorofluorocarbons has resulted in an increased risk of skin cancer for the foreseeable future.[66] Fortunately, the Montreal Protocol on Substances that Deplete the Ozone Layer, implemented internationally in 1987,[67] restricted ozone-depleting aerosols, leading to a gradual ozone recovery. Unfortunately 33,000 additional melanoma and nonmelanoma skin cancers are seen annually in the United States based on current ozone damage.[68] In addition, elevated temperatures alone may result in increased ultraviolet damage from the same ultraviolet light dose.[69] Thus a 2°C temperature increase may increase the number of skin cancers yearly by 10%.[70] In addition, warmer temperatures may influence sun exposure and ultraviolet-protective behaviors. Not surprisingly, higher temperatures are associated with increased time spent outdoors and without protective clothing in children and adults.[71]

VEGETATION-RELATED DERMATOLOGIC CONSEQUENCES

Phytodermatoses may also be impacted with clinical and experimental reports of poison ivy growth and urushiol potency associated with changing temperatures and increasing carbon dioxide within the atmosphere,[72,73] and northward expansion for giant hogweed in the northern United States and Canada.[74,75]

Conclusion

Implementation of mitigation strategies are critical in following the lowest-risk models for climate change that otherwise will diverge between 2020 and 2050.[76] Although worst-case scenarios such as re-emergence of smallpox from frozen victims in the arctic tundra are unlikely,[77] dermatologists should be aware of changing patterns and types of diseases we may encounter in clinical practice.

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Supplemental Table I. Changing demographics of North America endemic diseases

Disease	Current range	New projected range	Organism	Vector	Cutaneous findings	Treatment
Lyme disease	Northeast United States, northwest United States	Northeast Canada, northwest Canada	*Borrelia burgdorferi*	*Ixodes scapularis* (east) and*I pacificus* (west)	Erythema chronicum migrans	Doxycycline
Coccidioidomycosis	Southwest United States	Same, increased risk	*Coccidioides* spp	Soil fungus/ aerosolization	Directly inoculated nodules, erythema nodosum, lymphadenopathy, "valley fever"	Itraconazole
Chagas disease	Primarily Latin and South America	Vector and organism now present in 26 states	*Trypanosoma cruzi*	*Triatoma* spp (reduviid bug), may also be spread via transfusions but screened for in United States	Evanescent periorbital edema (Romaña sign)	Benznidazole or nifurtimox
New World Leishmaniasis	Latin America, northern South America	Extension further from equatorial regions	*Leishmania* spp	*Lutzomyia* spp sandfly	Inflamed or eczematous nodule or plaque in an exposed body part	Pentavalent antimonials, amphotericin B, local destructive methods
Hand-foot-and-mouth disease, herpangina, and enteroviral disease	Ubiquitous although different patterns based on climate	Same but less seasonal in temperate regions	*Enterovirus*, coxsackievirus	—	Oval vesicles in acral-shaped distribution, oral vesicles, generalized in coxsackie A6 infection	Supportive care

Supplemental Table II. Diseases associated with water warming and flooding potentially impacted by climate change

Disease	Current range	Change in demographic feature	Organism	Reservoir/means of disease	Cutaneous findings	Treatment
Jellyfish envenomations	Worldwide	Increased density in existing range	Numerous	Direct toxic effect	Linear streaks, edema, pain, rarely shock	Supportive care, some suggest warm water rinses, vinegar soaks, topical corticosteroids
Leptospirosis	Throughout North America	Associated flooding	*Leptospira interrogans*	Infected animal urine	Petechiae of palette, conjunctival injection, combined with flu-like symptoms or even sepsis	Penicillin
Vibrio wound infection/sepsis	Eastern US salt water, bays, estuaries	More frequent infections	*Vibrio vulnificus*	Direct inoculation, oysters	Necrotizing skin/soft-tissue infection	Surgical debridement, combined third-generation cephalosporin and doxycycline
Melioidosis	Typically southeast Asia, northern Australia	Associated with severe weather events (heavy rain, floods, cyclones, hurricanes), now reported in Western Hemisphere	*Burkholderia pseudomallei*	Soil and shallow water, often abrasions	Acute suppurative nodule ± sepsis or chronic draining suppurative sinuses	Surgical drainage, third- and fourth-generation cephalosporins typically for long-term treatment, supportive care
Seabather eruption	Northeast US and Florida saltwater	More frequent, early season of involvement in Northeast	*Linuche unguiculata*, *Edwardsiella lineata* (planula larva, jellyfish family)	Discharge of larval venom from organisms upon leaving salt water	Itchy/painful papules within a bathing suit distribution	Topical corticosteroids; removing swim suit before bathing which may trap larval elements and trigger release by exposure to freshwater
Cercarial dermatitis (swimmer itch)	Northern freshwater lakes	Longer season of infection with warmer waters	*Trichobilharzia* spp (schistosome)	Migrating bird excrement in lakes infecting natural snails with human beings as a dead-end host	Itchy papules sparing the bathing suit distribution	Topical corticosteroids