Trends in
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Review

Plagued by drought: how water scarcity reshapes host–parasite interactions

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Droughts are intensifying worldwide, reorganizing ecosystems and threatening biodiversity and human health. Yet their effects on infectious diseases are poorly understood and sometimes paradoxical, with water limitation often amplifying water-associated infections. This complexity arises because drought not only reduces water availability but also restructures the host–pathogen interactions that govern transmission. Synthesizing theory and empirical evidence from water-associated diseases, we identify three ecological mechanisms through which drought modifies infection dynamics: concentrating organisms into shrinking refugia, selectively favoring drought-tolerant hosts, vectors, and pathogens, and amplifying transmission heterogeneity across landscapes. By linking hydrological change to core epidemiological and ecological processes, this framework highlights parasite traits and strategies likely to be favored during drought while providing a foundation for forecasting infections in a water-limited world.

Highlights

Drought is becoming a defining ecological force, yet its consequences for infectious diseases remain difficult to predict across systems.

Emerging evidence shows that water limitation can intensify many water-associated diseases, challenging assumptions that drying suppresses transmission.

Rather than acting solely as a climatic stressor, drought restructures transmission by altering host aggregation, parasite persistence, community composition, and contact networks across space and time.

Divergent disease responses to drought can be explained through three recurring mechanisms: the concentration of interactions, the ecological filtering of communities, and spatial heterogeneity in transmission.

Viewing drought as a multiscale ecological restructuring process provides a general framework for predicting disease outcomes in an increasingly water-limited world.

Disease in a drying world

Following the worst drought in 40 years, the Horn of Africa suffered major cholera outbreaks as prolonged water scarcity displaced millions of people and forced communities to rely on unsafe water sources. Two years later, an exceptionally dry summer in the western United States coincided with one of the largest avian botulism outbreaks ever recorded, killing more than 94 000 nesting and migratory birds around Tule Lake. Although separated by continents and affecting different hosts, both events illustrate how drought can amplify aquatic diseases through distinct ecological pathways: in Somalia, water shortages and deteriorating sanitation facilitated the transmission of *Vibrio cholerae*, whereas at Tule Lake, shrinking wetlands concentrated birds around warm, hypoxic waters that favored production of the botulinum neurotoxin by *Clostridium botulinum*. Together, these examples highlight some of the diverse mechanisms through which drought can modify disease risk across human and natural systems (see the Supplemental information online).

Globally, droughts represent one of the most destructive forms of natural disaster for agriculture, wildlife conservation, and human health [1–3], costing an estimated US$124 billion and 500 000 human lives since 1998 [4] (see https://news.un.org/en/story/2022/05/1118142). These losses stem from more than just water deficits: droughts often have cascading consequences on the risk of famine, fire, and disease [5–7]. This includes waterborne infections associated with limited access to clean water, along with vector-borne and multihost parasites that thrive in water-limited landscapes [6,8]. However, surprisingly little is known about the ecological pathways through which droughts modify infection [8,9]. This gap reflects the multidimensional nature of drought, which can alter the amount of water, its duration, spatial distribution, and properties such as temperature, salinity, oxygen, pH, and turbidity [2,10]. As a result, identifying causal links to disease outcomes among parasite systems remains difficult, helping explain the relative scarcity of syntheses compared with other climate stressors (e.g., temperature) [6,11].

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Despite these challenges, determining the consequences of drought for disease in humans and wildlife has perhaps never been more important [12,13]. More frequent and more severe droughts are already occurring because of changes in precipitation, human water usage, and especially temperature-driven increases in evaporative water demand [2,14]. Extreme droughts have been linked to ecosystem transformations involving wholesale shifts in ecological communities and processes that are difficult to reverse [14]. To anticipate and ultimately manage infections in a drying world, we need guiding predictions grounded in ecological theory. Here, we synthesize theory and empirical evidence to identify recurrent ecological mechanisms linking drought and water-associated infectious diseases. By evaluating how discrete dimensions of hydrological stress transform host–parasite interactions across spatial and temporal scales, we aim to generate testable predictions about which parasites will be 'winners' and 'losers' in a drying world. Finally, we highlight outstanding questions and future priorities for developing predictive, cross-system frameworks.

A drought–disease paradox?

Drought is intrinsically connected to disease through life's dependence on water. Water both sustains life and provides habitat, such that aquatic systems function as an interaction nexus between aquatic and terrestrial organisms and, by extension, their parasites [15]. Many parasite life cycles have evolved to transmit in water or use hosts found in or near water [16]. Some exclusively infect aquatic hosts (e.g., ciliate pathogens of fish), while others utilize terrestrial hosts but depend on aquatic transmission stages (e.g., waterborne protozoans), aquatic intermediate hosts (e.g., snail-borne trematodes), or semiaquatic vectors (e.g., mosquito-transmitted arboviruses). Indeed, of the 437 parasites known to occur in people [17], nearly 65% depend on water or use water as a medium for infecting hosts.

The strong reliance of parasites on water elicits the expectation that water deficits will reduce parasite populations, which is supported by many observations (Figure 1; see the Supplemental information online) [18]. For instance, human cases of schistosomiasis in Kenya fell dramatically following a prolonged drought due to reductions in infected snail intermediate hosts [19], with similar results for malaria in Zambia [20]. In other cases, drought produces a more paradoxical outcome: infectious diseases that increase following water deficits (Figure 1). This counterintuitive pattern—the **drought–disease paradox** (see Glossary)—has been reported for West Nile virus, St Louis encephalitis virus, anthrax, hemorrhagic disease, cholera, trypanosomiasis, dengue, whirling disease, and black spot disease, among others [6,21–27]. These examples indicate that water availability alone is not the sole lever controlling disease; parasite dynamics and spread are often mediated by additional drought-related changes to habitat characteristics (e.g., enhanced suitability for vectors), trophic interactions (e.g., loss of predators that cull infections), or host contact rates (e.g., aggregation at water bodies).

Progress in understanding alternative drought–disease linkages has been limited because drought is often treated either as a climatic variable in epidemiology or as an ecological disturbance in environmental science, whereas transmission outcomes emerge from the interaction of hydrological change, community interactions, and spatial restructuring. By identifying the processes that generate seemingly paradoxical outcomes, the scales at which they manifest, and the water body and parasite types that experience them, we can better forecast disease risk in a drying world. Although droughts have the potential to enhance or diminish infection (see the Supplemental information online), we pay particular attention to amplifying mechanisms because of their potential to create ecological surprises with consequences for wildlife populations and society.

Glossary

Aggregation: the clustering of species and individuals around aquatic habitats.
Antecedent drought: drought in the previous season or year, often followed by a wet period.
Concentration: elevated densities of hosts and parasites that occur as a consequence of reduced water body volumes.
Desiccation trap: a population sink for parasites (or hosts) caused by the drying of an aquatic habitat.
Disease pathology: harm to a host resulting from infection, often leading to reductions in survival or reproduction.
Drought–disease paradox: a counterintuitive increase in aquatic disease that is associated with the onset or progression of drought.
Ecological filter: the process by which environmental conditions selectively favor organisms with traits that enable persistence under local conditions.
Host–parasite contact rate: the physical encounter between hosts and infective stages that is required to initiate infection. In a simple transmission model, this phenomenon is encapsulated by βSI, where S represents the density of susceptible hosts, I represents the density of infective hosts or stages, and the likelihood of contact is embedded in β. In the context of drought, aggregation and concentration both increase S and I, thereby elevating contact and the potential for transmission.
Host susceptibility: a host's likelihood of becoming infected, given contact with an infective stage. In a simple transmission model, susceptibility is embedded in the transmission coefficient, β. Drought can, therefore, increase or decrease transmission depending on its effects on host susceptibility.
Hydroperiod: the proportion of the year during which a water body holds water. By reducing the hydroperiod, drought can shorten the time available for aquatic transmission.
Landscape scale: a scale based on evaluating disease across a broad spatial extent, that is, a parasite's metapopulation size across the landscape.
Local scale: a scale based on evaluating disease at a fine spatial extent, that is, a parasite's subpopulation size within a single aquatic habitat.

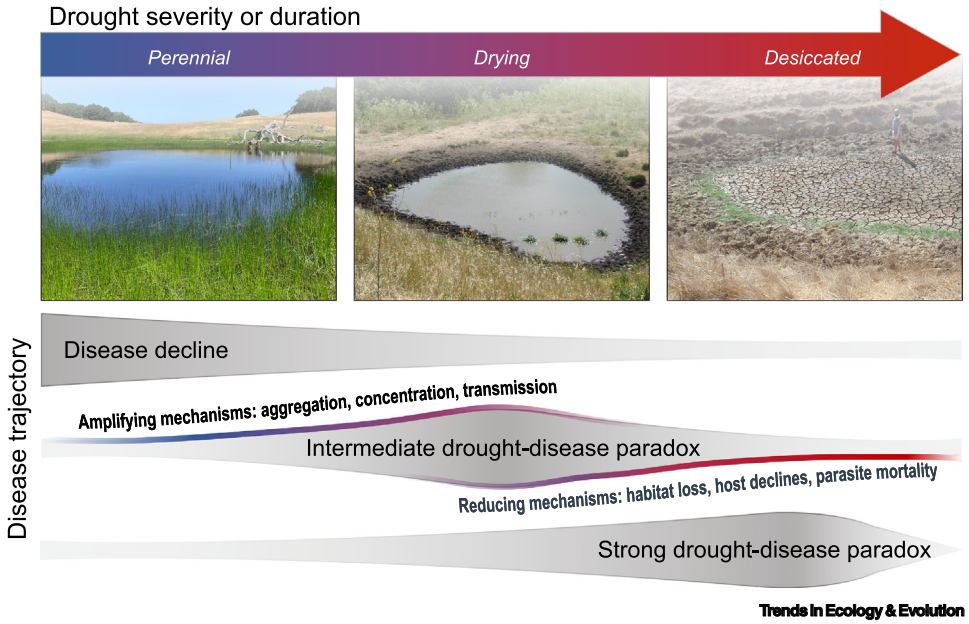

Figure 1. How drought alters disease. Because aquatic parasites depend on water, many water-associated infections are expected to decline with increasing drought severity or duration (Disease decline). However, several infectious diseases exhibit a counterintuitive disease trajectory: the 'drought–disease paradox'. Water-associated infections can increase as aquatic habitats dry (Intermediate drought–disease paradox), and even under desiccated conditions (Strong drought–disease paradox). These outcomes reflect the tension between drought-induced amplifying mechanisms (i.e., increased aggregation of hosts, elevated concentrations of parasite infective stages, and facilitated transmission due to shifting water characteristics) and reducing mechanisms (i.e., loss of aquatic habitat, declines in required hosts, reduced duration for host–parasite interactions, and parasite mortality).

Parasite growth, survival, and reproduction: key components of parasite performance, which, in a simple transmission model, modulate the number of infective stages (I) with transmission potential. Drought can modify parasite growth, survival, and reproduction in ways that can amplify or reduce the likelihood of transmission.
Watering hole hypothesis: drought-driven aggregation of organisms around persisting water bodies, leading to elevated host–parasite contact rates.

Mechanisms through which drought amplifies disease

Understanding how drought restructures transmission across space requires integration of concepts from metacommunity ecology, landscape ecology, and epidemiology. By changing the availability, volume, persistence, and characteristics of water [10], droughts affect host–parasite interactions and resulting disease outcomes through ecological processes, including the **concentration** of organisms in or around shrinking refugia, ecological sorting of host and parasite communities, and increased transmission heterogeneity among environments (Figure 2). From an epidemiological standpoint, these effects manifest by influencing the state variables or parameters within transmission models [38], including (i) **host–parasite contact rates**, (ii) **parasite growth, survival, and reproduction**, (iii) **host susceptibility** and **disease pathology**, and (iv) the abundance or composition of host and vector communities (Figure 2).

Changes in host aggregations and host–parasite contact rates

Water limitation across landscapes often causes organisms to aggregate around persisting aquatic systems, while declines in the size or connectivity of remaining aquatic habitats leads to **concentration** of resident hosts (Figure 2; see the Supplemental information online). Resulting increases in contacts among hosts, between species, and with water-associated infective stages can elevate transmission (**watering hole hypothesis**), (e.g., [21,39–43]). In East Africa, droughts enhance the **aggregation** of wild and domestic herbivores around watering holes that function as hotspots for gastrointestinal nematode transmission, while reductions in rainfall or river flows can elevate chytrid fungal infections by increasing amphibian contacts within confined

Mechanism		Host	Parasite	Habitat	Region	Study
Higher host aggregation and parasite contact		Large mammals	Gastrointestinal nematodes	Watering hole	Central Kenya	[8]
		Amphibian	Chytrid fungus	Atlantic forest	São Paulo, Brazil	[9]
		Fish	Trematodes	Rivers, lakes, streams	Western Amazon, Brazil	[34]
Altered water quality amplifies parasite growth or survival		Oyster	Dermo (protozoan)	Estuaries	Texas, USA	[28]
		Fish	Black spot (trematode)	Rivers	Ontario, Canada	[35]
		Salmonids	Whirling disease (cnidarian)	Streams	Oregon, USA	[36]
Increased host susceptibility or disease pathology		Perch	Ulcerative disease (oomycete)	Lakes	England, UK	[37]
		Birds	Avian botulism (bacterium)	Lakes and reservoirs	Great Lakes, USA	[29]
		Octopus	Marine protozoan	Coastal ocean	Firth of Forth, Scotland, UK	[18]
Transformation of host or parasite communities		Mosquito vectors and birds	West Nile virus, St. Louis encephalitis virus	Wetlands	Florida, USA	[31-32]
		Humans	Malaria (protozoan)	Streams	New Guinea, Indonesia	[33]
		Fish	Copepod ectoparasite	Streams	Paraíba State, Brazil	[30]

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Figure 2. Cross-system evidence for disease-altering mechanisms during drought. For each of the major identified ecological pathways linking drought and disease, illustrative examples are included that encompass a range of parasite types, host taxa, aquatic habitats, and geographic regions. Image credits: Wikimedia Commons. For a more complete listing of examples, see the Supplemental information online. (See [8,9,18,28–37].)

water sources—despite evidence that the pathogen fares poorly in dry conditions [9,44–47]. In humans, reduced access to clean water sources during drought can increase the use of contaminated water sources, promoting waterborne diseases [6,7,12,48].

Aggregations of different species together can likewise enhance the transmission of parasites with multihost life cycles by amplifying interactions among host species or parasite stages. Indeed, droughts have frequently been linked to greater helminth infections in fishes, snails, amphibians, and humans [21,24,49–51] (see the Supplemental information online). During a severe drought in England, cestode and trematode infections in fish increased dramatically in response to higher densities and diversity of parasite-carrying waterbirds [17]. In rivers and streams, reductions in water flow and flushing rates during drought can increase the retention of waterborne infective stages and enhance transmission of bacteria, copepods, myxozoans, trematodes, monogeneans, and nematodes [7,21,23,50], including fish infections by *Myxobolus cerebralis*, the causative agent of whirling disease [21,50,52]. By reducing **hydroperiod**, however, droughts also constrain the amount of time over which hosts and pathogens interact,

thereby magnifying both temporal and spatial heterogeneity in infection outcomes following drought (Figure 1).

Effects of water quality on parasite growth, survival, and reproduction

Droughts often lead to fundamental changes in water quality that can be consequential for parasite growth, survival, and reproduction, including warmer temperatures, higher salinity, less dissolved oxygen, and more nutrients or contaminants (Figure 2). Reduced rainfall and greater evaporation, for instance, elevate the salinities of freshwater and marine ecosystems, which can adversely impact the survival of ectoparasites and parasite infective stages (e.g., eggs, spores, and cercariae) [53,54]. In South Africa, long-term drought increased salinity beyond the tolerance of an invasive ectoparasitic copepod, eliminating infection and improving the body condition of fish hosts [55]. Drought-induced salinity increases can also promote infection [56], including those of economically important diseases such as bitter crab disease and perkinsosis (Dermo) in oysters [28,57,58]. Importantly, such relationships are often nonlinear: for instance, while moderate increases in salinity promote *Vibrio vulnificus* bacterial infections in oysters, a major cause of seafood-borne illness [59], sustained drought can elevate salinity beyond the pathogen's tolerance and extirpate the infection [60].

Water temperatures frequently increase with drought due to heightened ambient temperatures and reductions in waterbody volume [2]. Aquatic hosts and parasites are particularly sensitive to warming, for which biological performance (e.g., activity, growth, or reproduction) initially rises with temperature, peaks at a thermal optimum, and then rapidly declines [58,61–63]. For example, trematode cercariae from the Baltic Sea exhibit a unimodal thermal performance curve, with both their release from snails and infection success in subsequent hosts peaking around 22°C [61]. Recent integration of thermal performance curves into epidemiological models demonstrates that the transmission of waterborne infections exhibits similar thermal optima [64,65]. These nonlinear relationships are essential for guiding predictions: transmission is likely to increase when warming shifts host–parasite interactions toward their thermal optimum, whereas temperatures above that optimum can inhibit transmission or drive parasite loss [63–65].

Changes in host susceptibility and disease pathology resulting from water shortages

For aquatic animals, physiological stress resulting from high temperatures, salinity, and hypoxia associated with droughts can amplify host susceptibility or pathology (Figure 2) [41,66]. A recent meta-analysis found that environmental stressors, such as elevated temperatures and salinities, often lead to greater pathogen intensity and lower host survivorship [67]. For example, stress associated with pond drying, warming, and faster development can inhibit amphibian immunity and increase vulnerability to chytrid infections [9,66,68]. Drought-induced stress can also enhance host pathology from disease. For instance, ectoparasitic copepods and monogenetic trematodes on the gills of fishes can accelerate asphyxiation and cause die-offs during drought [69], enhancing or suppressing population-level transmission depending on how host losses affect the parasite. Opportunistic pathogens, including some bacteria and protozoans, are buffered against negative feedback mechanisms and are thus frequently implicated in drought mortalities. During a severe drought in the United Kingdom, ulcerative disease caused by an opportunistic oomycete likely caused the deaths of more than 1 million fish in association with warmer water, reduced water quality, and host stress [18]. In the mountains of Oregon, USA, drought-induced reductions in water depth exposed eggs of a declining amphibian to more UV-B radiation, amplifying their susceptibility to a water mold (*Saprolegnia ferax*) and resulting in substantial mortality [70].

The cumulative effects of drought-driven changes in water characteristics, physiological stress, and host aggregations are well illustrated by epizootic die-offs in migratory waterfowl, which

are caused by mycobacteriosis, avian plague, parasitic infections, and especially avian botulism [29]. Such die-offs may involve thousands of birds and are tightly linked to drought-induced crowding around water sources, higher host susceptibility associated with fewer food resources, and warm, anoxic water conditions that favor pathogen growth. The toxin-producing *C. botulinum*, which thrives in anaerobic environments, poisons birds as they feed on organic matter. Decomposing bird carcasses, in turn, fuel additional bacterial growth, leading to a deadly positive feedback loop [71].

Transformations of host and parasite communities

Finally, droughts act as an **ecological filter**. By selectively promoting drought-tolerant hosts, vectors, and pathogens, droughts often cause wholesale changes in the abundance or composition of aquatic communities and types of infectious organisms (Figure 2). Progressive reductions in water flows can transform lotic systems into standing-water environments that favor different host, vector, and parasite communities. For instance, the drying phase of Brazilian intermittent streams involves abrupt changes in physical habitat (isolated pools with warmer temperatures and lower water quality) and biotic interactions (higher competition and crowding), leading to sharp increases in ectoparasitic copepod infections in fish [30]. This phenomenon is also aptly demonstrated by drought and mosquito-borne illnesses. Although water losses reduce aquatic habitat availability, remaining environments may be especially conducive to vector larvae due to reduced flushing, higher organic matter, and fewer aquatic predators [20]. Such relationships are often strongest for **antecedent drought** (i.e., drought in the previous season or year), which is a highly influential climatic predictor of West Nile virus, St Louis encephalitis virus, and hemorrhagic disease in North America [26,31,32,72,73].

Experimental research provides mechanistic insight into how drought-driven simplifications of aquatic food webs can promote vectors. Chase and Knight [74] found that wetland hydroperiod interacted with drought to regulate mosquito emergence: wetlands with intermediate hydroperiods supported lower predator biomass (relative to perennial systems) and fewer competitors (relative to temporary wetlands). This trophic simplification led to 20-fold higher mosquito emergence in mesocosms with intermediate hydroperiods. However, such responses vary among mosquito species depending on their ecology and breeding site preferences, with some studies reporting that drought-related increases in transmission are due to the crowding of vectors with reservoir hosts, such as birds, amplifying vector infection prevalence independent of changes in vector abundance [25,72]. Changes in vector communities and infection can have important consequences for public health. In Indonesia, a severe drought sharply increased malarial deaths through two mechanisms. Water and food shortages first pushed people to lower elevations where malaria is more common. Reductions in flow then transformed streams into standing pools conducive to larval mosquitoes, enhancing vector production, transmission opportunities, and ultimately resulting in 554 deaths [33].

How drought restructures transmission: scale- and habitat-dependent outcomes of drought

Our review indicates that a key feature of drought is increased transmission heterogeneity across landscapes through the formation of transmission hotspots (‘watering holes’) and dry spots (**desiccation traps**). How this heterogeneity shapes the spatial distribution of diseases and their temporal dynamics will depend on the duration of water shortages, the type of aquatic habitat, and the degree to which local hotspots compensate for lost or degraded habitats (Box 1). In a landscape of shifting water availability, water-associated infections are likely to respond to drought at both the **local** and **landscape scales**, with key linkages between them (Figure 3).

Box 1. Amphibian–trematode interactions during a severe drought

Data collected during the 2013–2015 drought in California, USA, revealed how trematode parasites in amphibians respond to changes in water availability. In the dry conditions of 2014, annual measures of infection for three trematode parasites in Pacific chorus frogs (*Pseudacris regilla*) exhibited increased mean infection (and variance) following the onset of the drought. By the third year of reduced water in 2015, however, nearly all parasites exhibited a pronounced decrease in infection. The stark and rapid increase in mean infection is congruent with enhanced local transmission, while the subsequent decline points to landscape-wide reductions in the parasites' populations.

These outcomes likely reflect the multidimensional impacts of drought. While low water availability can increase host crowding and amplify contact with infective stages in the short term, sustained drought can reduce infection in the long term due to elevated host mortality, adverse water conditions, and low survival of infected intermediate hosts (i.e., snails). Our data help to differentiate among these possibilities. In 2014, drought resulted in the desiccation of 25% of monitored wetlands (Figure IA, bars). For each parasite, the proportion of wetlands that could support transmission (i.e., those holding water, infected snails, and susceptible amphibians) consequently declined (Figure IA, points), reducing the total production of parasites from the monitored region. Contrasted against these losses were simultaneous increases in mean infection that occurred for all three parasites (Figure IB–D), which could offset wetland losses if persisting wetlands became 'hotter' (facilitated higher transmission) due to aggregation of definitive hosts and enhanced trematode–amphibian contact rates. Yet, despite the initial rise in infection, mean infections plummeted in 2015, suggesting locally elevated infection loads could not compensate for wetland losses (Figure IB–D). Using a few simplifying assumptions, we simulated how 'hot' transmission of the pathogenic trematode *Ribeiroia ondatrae* would have to be for increased infection loads to compensate for wetland losses (Figure IE). For ponds persisting in 2014 to achieve equivalent parasite production as estimated from ponds monitored in 2013, mean infection loads in 2014 would have had to double. While possible, it is unlikely that such high infections can persist to the point of transmission, given known intensity-dependent mortality of *R. ondatrae* in amphibians [75]. This highlights disease pathology as a potentially important regulating mechanism, illustrating the tension between amplifying and reducing forces as local infections feed into parasite populations.

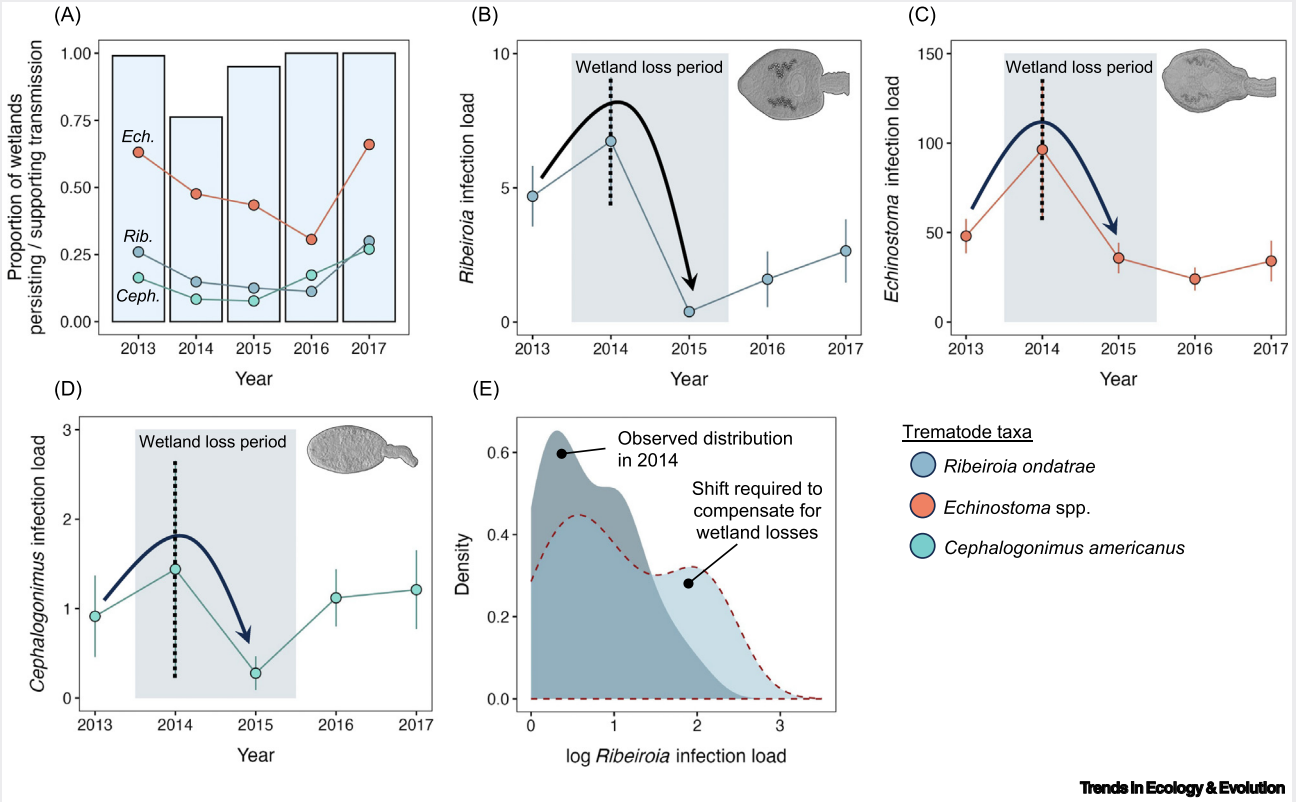

Figure I. An unfolding drought–disease relationship. (A) The proportion of wetlands holding water (bars) and supporting transmission (points) declined during drought for three trematode parasite taxa in chorus frogs. (B)–(D) Amphibian infections increased initially during drought and then declined as dry conditions persisted. (E) For the remaining ponds to compensate for wetland losses, amphibian infection loads would have had to nearly double relative to 2013 (contrast the observed infection load of *Ribeiroia ondatrae* with the estimated distribution necessary to yield total parasites comparable to those detected in 2013 before severe drought effects).

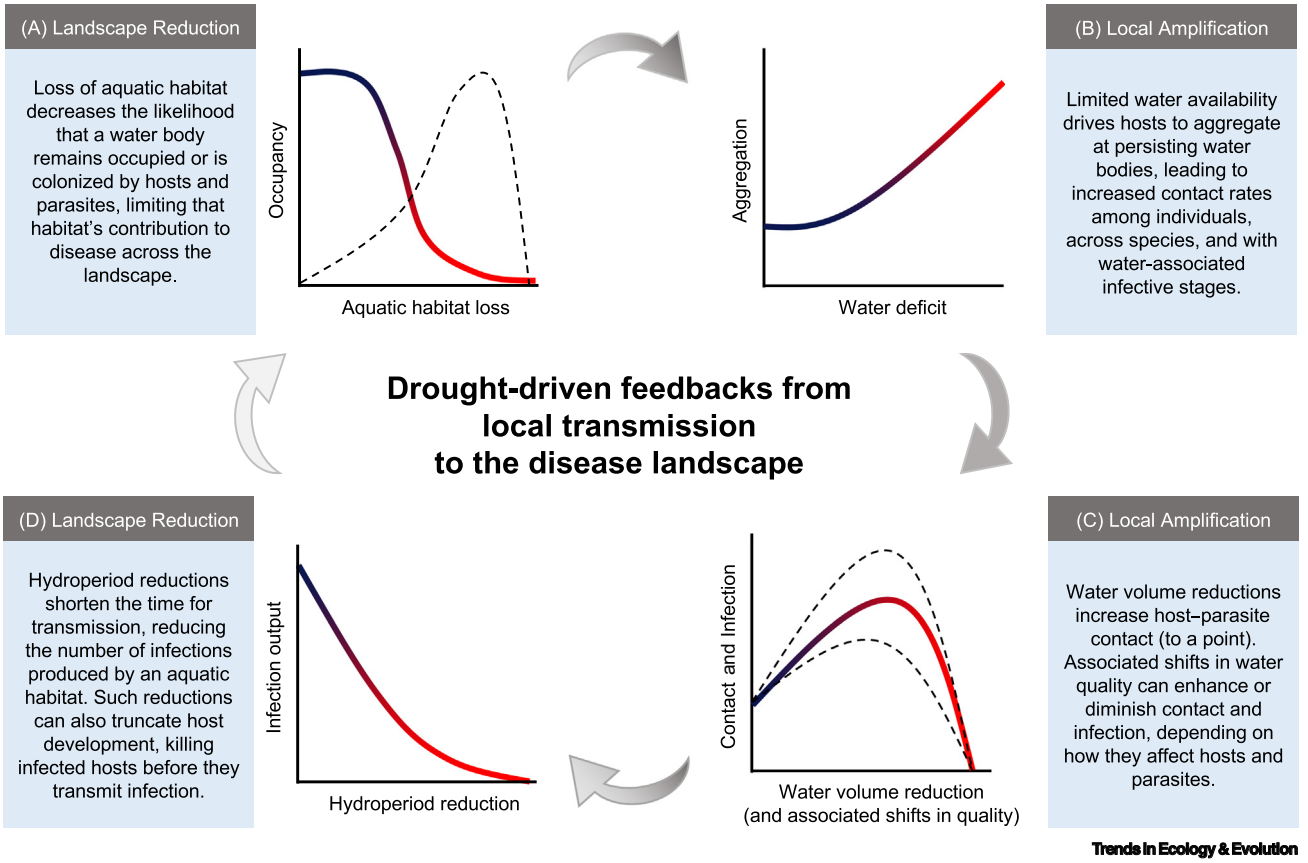

Figure 3. Droughts affect disease over space and time by altering local-to-landscape feedbacks. Potential hypotheses for how feedbacks manifest across different dimensions of drought are illustrated, with solid line color gradients reflecting drought severity increasing from low (blue) to high (red). (A) Aquatic habitat loss limits occupancy by hosts and parasites, potentially reducing parasite populations. This relationship may vary across systems (e.g., the dashed line represents a vector that thrives in low-volume aquatic habitats). (B) When less water is available, hosts may aggregate at persisting water bodies, amplifying local transmission. (C) By concentrating hosts and parasites, water loss can amplify density-dependent transmission. Concurrent shifts in water characteristics may either support hosts and parasites, increasing contact and infection (top dashed line), or be adverse, reducing contact and infection (bottom dashed line). (D) Shortened hydroperiods can diminish parasite output from an aquatic habitat or limit opportunities to infect hosts, thereby reducing the parasite population.

Across landscapes, the availability and distribution of water is a principal factor governing aquatic host and parasite population sizes and their interactions. As drought eliminates aquatic habitats, occupancy by hosts and parasites may decline (Figure 3) [67], as observed for *Bulinus* snails and their *Schistosoma* trematodes during an almost decade-long drought in Kenya [19]. Reductions in the duration of water availability can similarly reduce disease if there is insufficient time for transmission, parasite development, or overlap between hosts and parasites (Figure 3) [76], particularly for parasites with limited dispersal (see Box 2 for a discussion of parasite traits predicted to affect drought resilience). Increased stream salinity led to a spatial decoupling between salt-tolerant pupfish and snail intermediate hosts, reducing trematode infections and enhancing fish body condition [72,81]. These disease-reducing mechanisms are balanced against local amplifying mechanisms, such as host aggregation or changes in water conditions that favor transmission (Figure 3). In the St Lawrence River, Canada, for instance, the prevalence and richness of myxozoan infections in fish correlated locally with wastewater outflow, whereas annual variation at the landscape scale tracked hydrological flow rates [82]. Predicting parasites' responses to drought thus requires answers to two central questions: first, during water deficit,

Box 2. Which parasites will be 'winners' or 'losers' in a drying world?

Which parasites will be winners or losers in a drying world will depend largely on three interacting trait axes: the ability to persist locally during hydrological stress, the capacity to recolonize following disturbance, and the extent to which drought alters transmission opportunities. Although systematic comparative data on parasite responses to water shortage remain limited [18], drought is expected to favor parasites with resistant resting stages, drought-tolerant hosts or vectors, or strong recolonization capacity, whereas those reliant on drought-sensitive hosts or habitats, narrow ecological specialization, or limited dispersal are predicted to be especially vulnerable to drying conditions [50,77]. For example, some parasites of zooplankton produce resting stages that survive in sediment, buffering them against periods of environmental stress or host shortages [78], while a monogenean parasite of desert toads precisely synchronizes its reproduction with the single night each year that hosts breed in temporary wetlands [79]. Parasites transported via mobile hosts or transport vectors (e.g., flying insects, birds, bats, and humans) may also rapidly recolonize habitats following drought, increasing resilience relative to those dependent exclusively on aquatic taxa (Figure I).

Beyond traits of parasite persistence and dispersal, drought can directly influence transmission opportunities by altering host density, movement, and environmental suitability for infectious stages. Infections involving fast-growing, drought-tolerant intermediate hosts or vectors are especially likely to thrive in drought-affected aquatic environments. Some of the intermediate snail hosts of human blood flukes, for instance, survive extended desiccation [24,76,80], while mosquito vectors for important zoonotic diseases can proliferate rapidly within post-drought aquatic environments [73]. By crowding hosts while decreasing water flow, drought is hypothesized to favor parasites with density-dependent transmission, including those with free-living stages requiring contact with a suitable host [50]. Yet these same stages can also be highly sensitive to fluctuations in temperature, salinity, and pH [54], emphasizing likely trade-offs and nonlinearities in transmission responses as a function of drought severity/duration and the type of aquatic system under consideration. Together, these patterns suggest that drought responses can be anticipated from a small set of ecological and transmission traits governing persistence, dispersal, and host aggregation, while emphasizing that trade-offs between environmental suitability and transmission are likely to generate nonlinear and context-dependent disease responses under increasing hydrological stress.

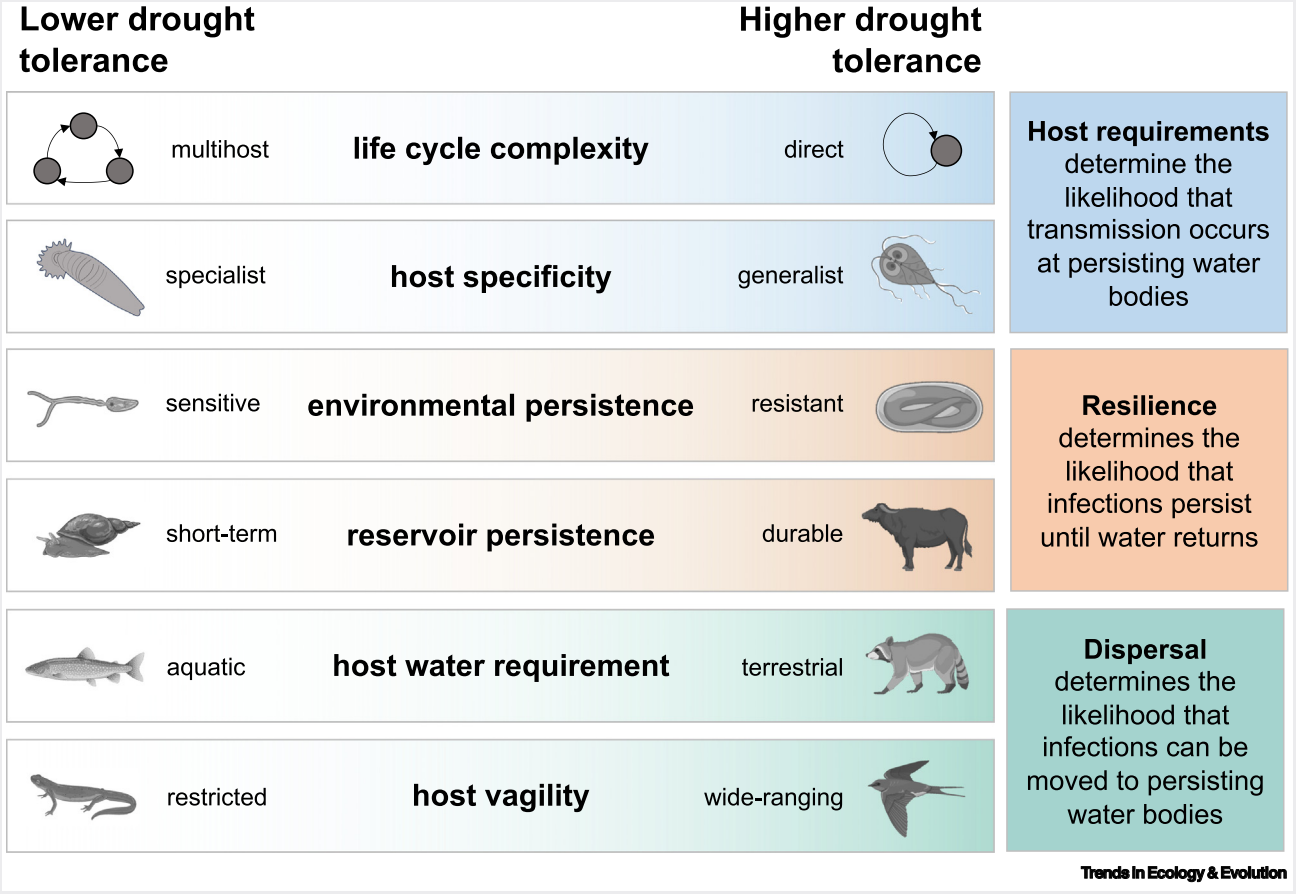

Figure I. Parasite traits that determine drought tolerance. Droughts are expected to selectively favor parasites that can withstand water shortages, quickly recolonize habitats, or experience enhanced transmission under drought conditions. These are predicted to be infections with simple life cycles involving drought-tolerant hosts or vectors, environmentally persistent stages or reservoirs, or parasite taxa that can rapidly recolonize following the easing of water shortages, such as highly mobile hosts (i.e., high host vagility).

can required host species persist in or disperse to remaining aquatic habitats? And second, how conducive are those remaining habitats for subsequent transmission?

Parasite life cycles will be reinforced when (i) loss of aquatic habitat during drought aggregates host individuals or species together [8,23,83]; (ii) spatial overlap increases host–parasite contact rates and transmission [9,84]; and (iii) remaining aquatic habitat supports the survival of susceptible hosts and parasite infective stages. If these criteria are met, the broader parasite population is likely to remain stable or even increase during low water availability (Figure 1). Conversely, parasite life cycles will be disrupted if (i) required hosts are missing, insufficiently abundant, or cannot disperse to remaining habitat and (ii) such habitat is not suitable or sufficiently durable for transmission [54,76]. Tension between local epidemiological processes and landscape ecological patterns can generate discrepancies between host infections and parasite population trajectories, ultimately leading to nonlinearities or lags in how disease dynamics unfold (see Outstanding questions).

The relative influence of landscape factors (i.e., water availability and distribution) and local factors (i.e., temperature, oxygen, and salinity) on parasite metapopulations will further depend on the type of water body and the distribution of the parasite population. If the parasite population is largely contained within a single, large body of water (e.g., lake, estuary, or river), drought will primarily affect disease through localized changes to the water body. If the parasite metapopulation is distributed across aquatic patches (e.g., ponds, streams, or rock pools), drought can additionally influence water quantity and distribution across patches, such that drought–disease outcomes emerge from the connection between within-patch transmission models and across-patch metapopulation models [85] (via effects on patch colonization, extinction, and parasite output). To illustrate the tension between landscape and local processes, we provide a case study of trematode infections in California amphibians during a severe drought (Box 1).

Toward a mechanistic approach

Important frontiers in advancing our understanding of drought–disease linkages include the application of quantitative comparisons across disease systems and the integration of multifactorial experiments with mechanistic models. Continued observational studies that extend beyond the short-term and local-scale effects of drought are essential for detecting lag effects and ecological transformations. To complement observational studies, we advocate for a combination of standardized performance assays in response to specific drought dimensions with larger-scale experiments that incorporate species interactions and disease outcomes both within and across water bodies. Analogous to thermal performance approaches, 'hydric performance curves' could be used to characterize nonlinear responses of hosts, vectors, or free-living parasite stages to multiple dimensions of hydrological stress, including humidity, desiccation rate, salinity, and hydroperiod duration [86,87]. Beyond physiological traits alone, such curves could be applied to epidemiologically relevant traits and processes, including host susceptibility, vector survival and biting activity, parasite survival, infectivity, and transmission, thereby identifying hydric optima and thresholds that regulate disease risk under drought [62].

At larger scales relevant for species interactions, aquatic research methodology includes an established array of experimental tools ideally suited for evaluating drought-driven changes in disease responses, including mesocosms, artificial stream channels, *in situ* enclosures, rain-out shelters, and whole-ecosystem manipulations [88–91]. While such approaches have provided key insights into how changes in water volume, hydroperiod, and flow affect aquatic communities, they remain rare in studies of drought and disease [8,9,51,70,74]. Multifactorial designs offer a valuable opportunity to test the hypothesized mechanisms outlined here and quantify

alternative or interacting pathways linking drought and infection. Distributed experiments applying the same design across different diseases, aquatic habitats, or locations can likewise yield powerful insights into response variation [92]. Integrating these approaches together provides a foundation for spatially explicit epidemiological models that can capture nonlinear and disease-specific responses to future droughts.

Concluding remarks: consequences for wildlife, conservation, and disease spread

Drought-driven ecological transformations are becoming more frequent, extensive, and consequential for both humans and wildlife [13,14], with nearly two-thirds of the contiguous United States experiencing drought conditions in 2026 alone. In a drying world, some water-associated infections will decline or disappear, whereas others may intensify, emerge in new locations, or show greater temporal variability (Box 2). Linking drought to disease dynamics remains challenging because drought acts as a multifactor, multiscale stressor whose effects on host–parasite interactions propagate through interacting physical, ecological, and biological pathways, often in complex and sometimes counterintuitive ways (Figure 2). Consequently, predictions based on water limitation alone are unlikely to capture the full spectrum of disease responses. Reliable forecasts will require integrating the multidimensional nature of drought with ecological theory that accounts for community interactions, spatial structure, and nonlinear responses.

We identify three primary pathways through which drought reshapes disease risk: by amplifying interactions among hosts, parasites, and other species; by acting as an ecological filter that alters the survival, composition, or abundance of hosts, parasites, and vectors; and by reducing aquatic habitat connectivity in ways that increase transmission heterogeneity. Variation in how disease systems respond along these pathways helps explain why drought can paradoxically amplify some water-associated infections while suppressing others. Such processes may be intensified by oscillations between drought and flooding, an 'extreme event syndrome' that can further destabilize ecological equilibria and promote disease outbreaks [93]. Because nearly all life requires water, aquatic habitats in drying landscapes serve as interaction nexuses where organisms and parasites converge, and where tensions between wildlife conservation and human water needs are most acute [94,95]. Progress toward a predictive framework will require experimental manipulations, long-term monitoring, and comparative analyses to quantify pathogen and host sensitivity to water limitation and hydrological variability (see Outstanding questions).

From a management perspective, identifying which host–parasite systems are most likely to increase during drought is critical to minimizing ecological surprises, including pathogen spillover from novel species interactions [6]. Water management strategies that maintain habitat connectivity, prevent transmission hotspots, and preserve refugia for vulnerable species may benefit both biodiversity conservation and public health. Because drought simultaneously modifies hydrological systems, wildlife communities, livestock management, and human water use, anticipating disease responses to water limitation may benefit from integrated approaches spanning ecological, veterinary, and public health systems [96–98]. Efforts that explicitly link hydrological change to ecological and epidemiological processes can help guide interventions to reduce disease risk while sustaining ecosystems in an increasingly water-limited world.

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Outstanding questions

Drought–disease interactions across space, time, and ecological systems

What time lags occur between drought onset, ecological changes, and shifts in disease risk?

When will increased landscape heterogeneity under drought generate transmission hotspots versus suppress infection through habitat fragmentation?

What drought conditions push host–parasite systems toward alternative disease states (i.e., endemic vs. epizootic, sublethal vs. mass mortality) or pathogen types?

How do drought effects differ between marine and freshwater ecosystems and across lotic and lentic habitats?

When will drought-altered landscapes increase the likelihood of pathogen spillover?Toward predictive theory and mechanistic approaches

How can drought–disease relationships be captured using generalizable theoretical frameworks, analogous to thermal performance approaches in temperature–disease studies?

How do specific components of drought (e.g., water volume, hydroperiod, temperature, salinity, oxygen, or desiccation stress) individually and interactively influence the transmission of different parasites?

When will infections increase host vulnerability to drought stress, creating feedback loops that amplify disease impacts during extreme water limitation?

Alternatively, can periodic droughts benefit hosts by removing or reducing infections?

How do oscillations between drought and flooding ('climate whiplash') shape pathogen persistence, amplification, or emergence?

Declaration of interests

The authors declare no competing interests.

Supplemental information

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References

1. Cook, B.I. *et al.* (2018) Climate change and drought: from past to future. *Curr. Clim. Chang. Rep.* 4, 164–179
2. Crausbay, S.D. *et al.* (2020) Unfamiliar territory: emerging themes for ecological drought research and management. *One Earth* 3, 337–353
3. Vicente-Serrano, S.M. *et al.* (2022) Global drought trends and future projections. *Philos. Trans. R. Soc. A* 380, 20210285
4. Below, R. *et al.* (2007) Documenting drought-related disasters: a global reassessment. *J. Environ. Dev.* 16, 328–344
5. Littell, J.S. *et al.* (2016) A review of the relationships between drought and forest fire in the United States. *Glob. Chang. Biol.* 22, 2353–2369
6. Mora, C. *et al.* (2022) Over half of known human pathogenic diseases can be aggravated by climate change. *Nat. Clim. Chang.* 12, 869–875
7. Stanke, C. *et al.* (2013) Health effects of drought: a systematic review of the evidence. *PLoS Curr.* 5 ecurrents.dis
8. Titcomb, G. *et al.* (2021) Water sources aggregate parasites with increasing effects in more arid conditions. *Nat. Commun.* 12, 7066
9. Buttimer, S. *et al.* (2025) Experimental drought suppresses amphibian pathogen yet intensifies transmission and disrupts protective skin microbiome. *Glob. Chang. Biol.* 31, e70275
10. Van Loon, A.F. (2015) Hydrological drought explained. *WIREs Water* 2, 359–392
11. Levy, K. *et al.* (2016) Untangling the impacts of climate change on waterborne diseases: a systematic review of relationships between diarrheal diseases and temperature, rainfall, flooding, and drought. *Environ. Sci. Technol.* 50, 4905–4922
12. Cann, K. *et al.* (2013) Extreme water-related weather events and waterborne disease. *Epidemiol. Infect.* 141, 671–686
13. Maxwell, S.L. *et al.* (2019) Conservation implications of ecological responses to extreme weather and climate events. *Divers. Distrib.* 25, 613–625
14. Moss, W.E. *et al.* (2024) Drought as an emergent driver of ecological transformation in the twenty-first century. *Bioscience* 74, 524–538
15. Johnson, P.T.J. and Paull, S.H. (2011) The ecology and emergence of diseases in fresh waters. *Freshw. Biol.* 56, 638–657
16. Lafferty, K.D. (2017) Marine infectious disease ecology. *Annu. Rev. Ecol. Evol. Syst.* 48, 473–496
17. Ashford, R., Crewe, W., eds (2003) *Parasites of Homo sapiens: An Annotated Checklist of the Protozoa, Helminths and Arthropods for Which We are Home*, CRC Press
18. Morley, N. and Lewis, J. (2014) Extreme climatic events and host–pathogen interactions: the impact of the 1976 drought in the UK. *Ecol. Complex.* 17, 1–19
19. Mutuku, F.M. *et al.* (2011) Impact of drought on the spatial pattern of transmission of *Schistosoma haematobium* in coastal Kenya. *Am. J. Trop. Med. Hyg.* 85, 1065–1070
20. Kent, R.J. *et al.* (2007) Seasonality, blood feeding behavior, and transmission of *Plasmodium falciparum* by *Anopheles arabiensis* after an extended drought in southern Zambia. *Am. J. Trop. Med. Hyg.* 76, 267–274
21. Marcogliese, D.J. (2001) Implications of climate change for parasitism of animals in the aquatic environment. *Can. J. Zool.* 79, 1331–1352
22. Jones, N. *et al.* (2020) Water, sanitation and hygiene risk factors for the transmission of cholera in a changing climate: using a systematic review to develop a causal process diagram. *J. Water Health* 18, 145–158
23. Lymbery, A.J. *et al.* (2020) Fish out of water: aquatic parasites in a drying world. *Int. J. Parasitol. Parasites Wildl.* 12, 300–307
24. Parietti, M. *et al.* (2021) Effects of droughts on the larval digenean assemblage that parasitizes the snail *Biomphalaria peregrina* (Planorbidae): a 4-year study in a temporary pond from Argentina. *Parasitol. Res.* 120, 1909–1914
25. Sambado, S. *et al.* (2025) The paradoxical impact of drought on West Nile virus risk: insights from long-term ecological data. *Proc. R. Soc. B* 292, 2054
26. Christensen, S.A. *et al.* (2020) The role of drought as a determinant of hemorrhagic disease in the Eastern United States. *Glob. Chang. Biol.* 26, 3799–3808
27. Hugh-Jones, M.E. and de Vos, V. (2002) Anthrax and wildlife. *Rev. Sci. Tech.* 21, 359–383
28. Savage, K.B. (2025) Effects of climate-driven salinity regimes on disease dynamics of the Eastern oyster, a key estuarine resource and bioindicator. In *Freshwater Inflows to Texas Bays and Estuaries: A Regional-scale Review, Synthesis, and Recommendations* (Montagna, P.A. *et al.*, eds), pp. 295–307, Springer
29. Alexander, N. *et al.* (2024) Waterbird disease in the United States Laurentian Great Lakes under climate change. *J. Great Lakes Res.* 50, 102450
30. Medeiros, E.S. and Maltchik, L. (1999) The effects of hydrological disturbance on the intensity of infestation of *Lernaea cyprinacea* in an intermittent stream fish community. *J. Arid Environ.* 43, 351–356
31. Shaman, J. *et al.* (2002) Drought-induced amplification of Saint Louis encephalitis virus, Florida. *Emerg. Infect. Dis.* 8, 575–580
32. Shaman, J. *et al.* (2005) Drought-induced amplification and epidemic transmission of West Nile virus in southern Florida. *J. Med. Entomol.* 42, 134–141
33. Bangs, M.J. and Subianto, D.B. (1999) El Niño and associated outbreaks of severe malaria in highland populations in Irian Jaya, Indonesia: a review and epidemiological perspective. *Southeast Asian J. Trop. Med. Public Health* 30, 608–619
34. Virgilio, L.R. *et al.* (2023) Endoparasite communities of fish at different trophic levels in the Western Brazilian Amazon: human, environmental and seasonal influence. *Acta Parasitol.* 68, 612–636
35. Steedman, R.J. (1991) Occurrence and environmental correlates of black spot disease in stream fishes near Toronto, Ontario. *Trans. Am. Fish. Soc.* 120, 494–499
36. Hallett, S.L. and Bartholomew, J.L. (2008) Effects of water flow on the infection dynamics of *Myxobolus cerebralis*. *Parasitology* 135, 371–384
37. Bucke, D. *et al.* (1979) Further studies of an epizootic of perch *Perca fluviatilis* L., of uncertain aetiology. *J. Fish Dis.* 2, 297–311
38. McCallum, H. *et al.* (2001) How should pathogen transmission be modelled? *Trends Ecol. Evol.* 16, 295–300
39. Paull, S.H. *et al.* (2012) Temperature-driven shifts in a host-parasite interaction drive nonlinear changes in disease risk. *Glob. Chang. Biol.* 18, 3558–3567
40. Turner, W.C. *et al.* (2016) Lethal exposure: an integrated approach to pathogen transmission via environmental reservoirs. *Sci. Rep.* 6, 27311
41. Lennox, R.J. *et al.* (2019) Toward a better understanding of freshwater fish responses to an increasingly drought-stricken world. *Rev. Fish Biol. Fish.* 29, 71–92
42. Ndachena, N. *et al.* (2025) Effect of drought on wildlife activity at artificial waterholes. *Biol. Conserv.* 310, 111370
43. Payne, A. *et al.* (2017) Wildlife interactions on baited places and waterholes in a French area infected by bovine tuberculosis. *Front. Vet. Sci.* 3, 122
44. Adams, A.J. *et al.* (2017) Extreme drought, host density, sex, and bullfrogs influence fungal pathogen infection in a declining lotic amphibian. *Ecosphere* 8, e01740

45. Longo, A.V. *et al.* (2010) Seasonality of *Batrachochytrium dendrobatidis* infection in direct-developing frogs suggests a mechanism for persistence. *Dis. Aquat. Org.* 92, 253–260
46. Moura-Campos, D. *et al.* (2021) Fungal disease cluster in tropical terrestrial frogs predicted by low rainfall. *Biol. Conserv.* 261, 109246–109252
47. Raffel, T.R. *et al.* (2015) Temperature variability and moisture synergistically interact to exacerbate an epizootic disease. *Proc. R. Soc. B* 282, 20142039
48. Levy, J.I. *et al.* (2023) Wastewater surveillance for public health. *Science* 379, 26–27
49. Kennedy, C. and Burrough, R. (1981) The establishment and subsequent history of a population of *Ligula intestinalis* in roach *Rutilus rutilus* (L.). *J. Fish Biol.* 19, 105–126
50. Marcogliese, D.J. (2025) Climate change and parasitism in aquatic ecosystems. In *Aquatic Parasitology: Ecological and Environmental Concepts and Implications of Marine and Freshwater Parasites* (Smit, N.J. and Sures, B., eds), pp. 547–593, Springer
51. Kiesecker, J.M. and Skelly, D.K. (2001) Effects of disease and pond drying on gray tree frog growth, development, and survival. *Ecology* 82, 1956–1963
52. Alexander, J.D. and Bartholomew, J.L. (2020) Myxoboliosis (*Myxobolus cerebralis*). In *Climate Change and Infectious Fish Diseases* (Woo, P.T.K. *et al.*, eds), pp. 381–403, CABI
53. Bommarito, C. *et al.* (2020) Freshening rather than warming drives trematode transmission from periwinkles to mussels. *Mar. Biol.* 167, 46
54. Pietrock, M. and Marcogliese, D.J. (2003) Free-living endohelminth stages: at the mercy of environmental conditions. *Trends Parasitol.* 19, 293–299
55. Welicky, R.L. *et al.* (2017) Drought-associated absence of alien invasive anchorworm, *Lernaea cyprinacea* (Copepoda: Lernaeidae), is related to changes in fish health. *Int. J. Parasitol. Parasites Wildl.* 6, 430–438
56. Rocker, M.M. *et al.* (2026) Salt in the wound: consequences of changing salinities on marine disease. *Philos. Trans. R. Soc. B* 381, 20240324
57. La Peyre, M.K. *et al.* (2016) Analysis of environmental factors influencing salinity patterns, oyster growth, and mortality in Lower Breton Sound Estuary, Louisiana, using 20 years of data. *J. Coast. Res.* 32, 519–530
58. Shields, J.D. (2019) Climate change enhances disease processes in crustaceans: case studies in lobsters, crabs, and shrimps. *J. Crustac. Biol.* 39, 673–683
59. Archer, E.J. *et al.* (2023) Climate warming and increasing *Vibrio vulnificus* infections in North America. *Sci. Rep.* 13, 3893–3903
60. Froelich, B.A. *et al.* (2012) Apparent loss of *Vibrio vulnificus* from North Carolina oysters coincides with a drought-induced increase in salinity. *Appl. Environ. Microbiol.* 78, 3885–3889
61. Díaz-Morales, D.M. *et al.* (2022) Heat sensitivity of first host and cercariae may restrict parasite transmission in a warming sea. *Sci. Rep.* 12, 1174
62. Kirk, D. *et al.* (2022) Scaling effects of temperature on parasitism from individuals to populations. *J. Anim. Ecol.* 91, 2087–2102
63. Gehman, A.L.M. *et al.* (2018) Host and parasite thermal ecology jointly determine the effect of climate warming on epidemic dynamics. *Proc. Natl. Acad. Sci. U. S. A.* 115, 744–749
64. Aslan, I.H. *et al.* (2024) Re-assessing thermal response of schistosomiasis transmission risk: evidence for a higher thermal optimum than previously predicted. *PLoS Negl. Trop. Dis.* 18, e0011836
65. Nguyen, K.H. *et al.* (2021) Interventions can shift the thermal optimum for parasitic disease transmission. *Proc. Natl. Acad. Sci. U. S. A.* 118, e2017537118
66. Kohli, A.K. *et al.* (2019) Disease and the drying pond: examining possible links among drought, immune function, and disease development in amphibians. *Physiol. Biochem. Zool.* 92, 339–348
67. Vicente-Santos, A. *et al.* (2023) Host–pathogen interactions under pressure: a review and meta-analysis of stress-mediated effects on disease dynamics. *Ecol. Lett.* 26, 2003–2020
68. Rollins-Smith, L.A. and Le Sage, E.H. (2023) Heat stress and amphibian immunity in a time of climate change. *Philos. Trans. R. Soc. B* 378, 20220132
69. Kuperman, B.I. *et al.* (2001) Parasites of fish from the Salton Sea, California, USA. *Hydrobiologia* 466, 195–208
70. Kiesecker, J.M. *et al.* (2001) Complex causes of amphibian population declines. *Nature* 410, 681–684
71. Rocke, T.E. and Bollinger, T.K. (2007) Avian botulism. In *Infectious Diseases of Wild Birds* (Thomas, N.J. *et al.*, eds), pp. 377–416, Blackwell Publishing
72. Paull, S.H. *et al.* (2017) Drought and immunity determine the intensity of West Nile virus epidemics and climate change impacts. *Proc. R. Soc. B* 284, 20162078
73. Wang, G. *et al.* (2010) Dry weather induces outbreaks of human West Nile virus infections. *BMC Infect. Dis.* 10, 1–7
74. Chase, J.M. and Knight, T.M. (2003) Drought-induced mosquito outbreaks in wetlands. *Ecol. Lett.* 6, 1017–1024
75. Wilber, M. *et al.* (2020) Disease hotspots or hot species? Infection dynamics in multi-host metacommunities controlled by species composition, not source location. *Ecol. Lett.* 23, 1201–1211
76. Starkloff, N.C. *et al.* (2024) Spatio-temporal variability in transmission risk of human schistosomes and animal trematodes in a seasonally desiccating East African landscape. *Proc. R. Soc. B* 291, 20231766
77. Lafferty, K.D. and Holt, R.D. (2003) How should environmental stress affect the population dynamics of disease? *Ecol. Lett.* 6, 654–664
78. Decaestecker, E. *et al.* (2002) In deep trouble: habitat selection constrained by multiple enemies in zooplankton. *Proc. Natl. Acad. Sci. U. S. A.* 99, 5481–5485
79. Tinsley, R.C. (1999) Parasite adaptation to extreme conditions in a desert environment. *Parasitology* 119, S31–S56
80. Rubaba, O. *et al.* (2016) The role of snail aestivation in transmission of schistosomiasis in changing climatic conditions. *Afr. J. Aquat. Sci.* 41, 143–150
81. Rogowski, D.L. and Stockwell, C.A. (2006) Parasites and salinity: costly tradeoffs in a threatened species. *Oecologia* 146, 615–622
82. Marcogliese, D.J. *et al.* (2009) Impact of municipal effluents and hydrological regime on myxozoan parasite communities of fish. *Int. J. Parasitol.* 39, 1345–1351
83. Cable, J. *et al.* (2017) Global change, parasite transmission and disease control: lessons from ecology. *Philos. Trans. R. Soc. B* 372, 20160088
84. Merino, S. (2010) Host-parasite interactions and climate change. In *The Effects of Climate Change on Birds* (Møller, A.P. *et al.*, eds), pp. 213–226, Oxford University Press
85. Borer, E.T. *et al.* (2016) A multiscale approach to plant disease using the metacommunity concept. *Annu. Rev. Phytopathol.* 54, 397–418
86. Delgado-Suazo, P. and Burrowes, P.A. (2022) Response to thermal and hydric regimes point to differential inter- and intraspecific vulnerability of tropical amphibians to climate warming. *J. Therm. Biol.* 103, 103148
87. Anderegg, W.R.L. *et al.* (2018) Hydraulic diversity of forests regulates ecosystem resilience during drought. *Nature* 561, 538–541
88. Aspin, T.W. *et al.* (2023) Extra terrestrials: drought creates niche space for rare invertebrates in a large-scale and long-term field experiment. *Biol. Lett.* 19, 20230381
89. Marino, A. *et al.* (2025) Experimental droughts in mesocosms reveal a gradient of tolerance in Alpine stream macroinvertebrates. *Limnol. Oceanogr.* 70, S352–S363
90. Wilbur, H.M. (1997) Experimental ecology of food webs: complex systems in temporary ponds. *Ecology* 78, 2279–2302
91. Schindler, D.W. (1998) Whole-ecosystem experiments: replication versus realism: the need for ecosystem-scale experiments. *Ecosystems* 1, 323–334
92. Beier, C. *et al.* (2012) Precipitation manipulation experiments – challenges and recommendations for the future. *Ecol. Lett.* 15, 899–911
93. MacDonald, A.J. *et al.* (2025) Extreme climate whiplash events drive divergent responses of mosquito-borne disease. *PNAS Nexus* 4, pgaf223
94. Abrahms, B. (2021) Human-wildlife conflict under climate change. *Science* 373, 484–485

95. Calhoun, K.L. *et al.* (2025) Human-wildlife conflict is amplified during periods of drought. *Sci. Adv.* 11, eadx0286
96. Merrill Stewart, T. (2025) Ecosystems and infectious disease. In *Global One Health and Infectious Diseases* (Sander, W.E. *et al.*, eds), pp. 164–191, CRC Press
97. Carlson, C.J. *et al.* (2022) Climate change increases cross-species viral transmission risk. *Nature* 607, 555–562
98. Kelly, T.R. *et al.* (2020) Implementing One Health approaches to confront emerging and re-emerging zoonotic disease threats: lessons from PREDICT. *One Health Outlook* 2, 1