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Temperature as a Driver of Phage Ecology and Evolution

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Abstract

Bacteriophages (phages) are virtually ubiquitous and play a fundamental role in the ecological and evolutionary dynamics of their bacterial hosts. While phages are found across many thermal environments, they can be highly sensitive to changes in temperature. Moreover, phages are expected to face increasingly frequent and intense thermal perturbations with global climate change. In this review, we combine theoretical and empirical evidence to assess the impact of the thermal environment on phage biology at the global scale. We identify key thermal environments that phages inhabit, and we discuss the role of temperature in determining phage life-history strategies, ecological interactions, and evolutionary dynamics. We then explore the potential effects of thermal variation on phage functions in natural microbial communities and the application of phages as biomedical therapeutics.

Contents

INTRODUCTION

Bacteriophages (phages), which are viruses that infect bacteria, are the most abundant biological entity on the planet (reviewed in 160 and 181). Phages can be found in nearly all habitats permissive to life, and their abundance and diversity typically track those of their bacterial hosts. A primary impact of phages in microbial communities occurs through their lysis (killing) of bacterial cells. Phage lysis significantly affects the abundance of bacterial genotypes and species and, thus, microbial community composition and structure. Importantly, phages disproportionately target the most abundant bacterial taxa and genotypes, and this targeting leads to negative, frequency-dependent selection (114). These antagonistic interactions can be reciprocal and can drive rapid coevolution (95).

In natural populations, phages are often exposed to extremes of salinity, pH, and temperature (reviewed in 84). Temperature is a particularly important environmental factor that affects processes at all biological levels, from enzyme kinetics (134) to ecological and evolutionary dynamics (12, 64, 94). Phages are highly vulnerable to temperature because of their dependence on the activity of replicative and lytic enzymes and on the growth rates of their bacterial hosts (123). Given that phages also rapidly deplete their host populations and so exist predominantly in the environment as noninfecting particles (178), phages are susceptible to thermal or UV degradation (24, 182). Temperature changes can drive shifts in phage persistence (16) and growth rates (43).

Horizontal gene transfer: the transfer of genetic material between bacterial cells that are not parent and offspring

By altering the mode of phage behavior upon infection, environmental temperatures determine whether phages act as bacterial parasites or as mutualists. Lytic phages replicate immediately, and then they kill their bacterial hosts upon infection; conversely, some phages follow a lysogenic life cycle, and they integrate their genetic material into the bacterial chromosome and replicate in synchrony without inducing cell death (78). Integrative (also known as temperate) phage transmission among cells can facilitate bacterial horizontal gene transfer (165) through the carriage of genes involved in bacterial symbiotic mutualism (126) as well as in pathogen virulence, metabolism, and competitiveness (reviewed in 162). Finally, phages can follow pseudolysogenic life cycles, in which they exist in a dormant, nonintegrative state postinfection (146). The three

phage life-cycle strategies are not mutually exclusive; many phages can swap between lytic and lysogenic/pseudolysogenic life cycles in response to changes in temperature (127, 185).

Phages have been identified across many environments that have unique thermal distributions (**Figure 1** and **Table 1**; see also **Supplemental Text 1** for a detailed overview and **Supplemental Data and Code** for data analysis). Phage habitats include extreme thermal environments such as polar ice and Antarctic soils in which temperatures regularly fall below 0°C (75). Phage habitats also include hydrothermal vents and hot springs in which temperatures often rise above 100°C (35). In hot deserts, phages can experience high diurnal fluctuations between extreme hot and cold temperatures (189). In between thermal extremes is a continuum of intermediate thermal environments more permissive to life. These intermediate environments include lower temperature habitats such as soils and oceans; increasingly warm habitats such as ectotherm or plant hosts, whose thermoregulation depends on external sources; and endotherm animal hosts, who can regulate their body temperatures internally (11, 111, 160, 180). By comparing environments across the thermal continuum, we can determine the breadth of thermal effects in phage systems.

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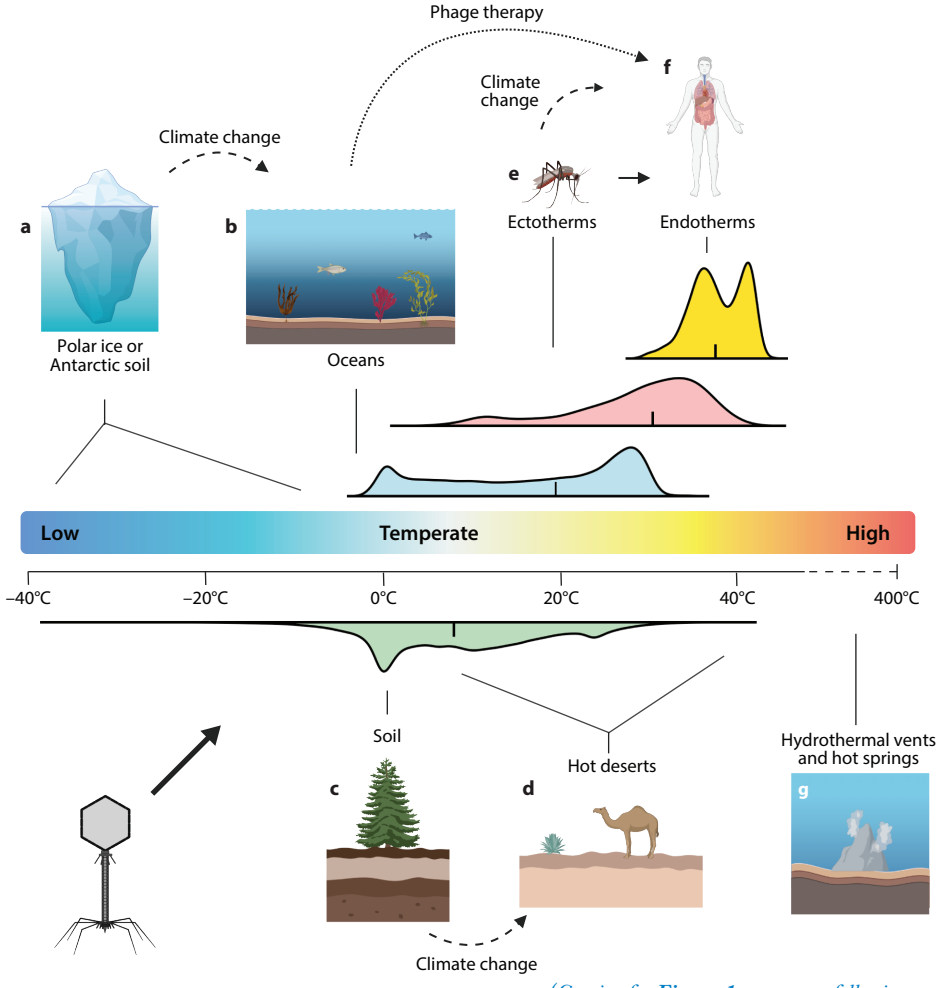
In this review, we assess how thermal variation within and between phage thermal environments affects bacteria-phage interactions at the global level. First, we provide a mechanistic



(*Caption for* ***Figure 1*** *appears on following page*)

Figure 1 (*Figure appears on preceding page*)

Examples of phage thermal environments spanning a thermal gradient. (*a*) Extreme cold phage environments such as polar ice or Antarctic soil span a range of subzero temperatures (9). (*b*) Ocean phages (160) inhabit a more temperate environment, although temperatures are bimodal, with prevalence peaks at 0°C and 30°C. Global sea surface temperature data for the year 2023 were obtained from Reference 124. (*c*) Soil phages (180) have similar general thermal ranges to those of ocean phages, with temperatures largely occurring at or above 0°C. Thermal data were obtained from Reference 101. (*d*) Desert phages (189) experience the highest daily thermal variation, with diurnal temperatures ranging from those of high heat to those of low cold (194). (*e*) Phages associated with ectotherms (167) experience higher temperatures and less thermal variation than do free-living phages; the latter difference is due to behavioral thermoregulation. (*f*) Endotherms also carry phages (111) and have higher but more stable body temperatures than do ectotherms. Endotherm body temperatures are bimodal within a small range because of diel and nocturnal lifestyle differences. Ectotherm and endotherm body temperature data were obtained from Reference 120. (*g*) Phages have been found in hydrothermal vents and hot springs (35), which often have temperatures above 90°C (198). The blue-to-red bar shows the thermal gradient. Environmental temperatures for soil, oceans, ectotherms, and endotherms are presented as smoothed density plots. Plots were generated using R (143) and RStudio (149) (see **Supplemental Data and Code**). The vertical lines within the plots show median values. The thermal ranges that are shown for extreme cold and desert environments reflect values reported in the literature. The dashed arrows show expected thermal environment changes with climate change such as polar ice melting, soil desertification, and ectotherm vector range expansion (82). The block arrow between ectotherms and endotherms highlights potential ectotherm-endotherm phage transmission (3). The dotted arrow shows the thermal shift of marine phages deployed as therapeutics (81). **Supplemental Text 1** provides a detailed overview of phage thermal environments. Figure adapted from images created in BioRender; MacLean C. 2025. **https://BioRender.com/s5dfzfd**.

Supplemental Material >

analysis of thermal effects on phage life-history traits and trade-offs. We then discuss the potential implications of thermal change for phage ecology and evolution. Finally, we provide an overview of how thermal environments affect phages in natural communities. Thermal effects are discussed in the context of global climate change, animal and plant microbiomes, and a topic of growing interest—the use of phages as antibacterial therapeutics.

TEMPERATURE AND PHAGE INFECTION

Phage Infectivity on Bacteria

Phage thermal sensitivity is illustrated by the observed temperature dependence of the sequential phage infection steps that are essential for replication (reviewed in 32): phage adsorption to the

Table 1 Estimated thermal profiles of phage environments

Thermal environment	Thermal range (°C)	Upper and lower quartiles (°C)	Median temperature (°C)	Phage reference	Temperature reference
Antarctic soil	ND	ND	∼−24.9	18	116
Glacial ice	−50 to −9	ND	ND	195	141
Soil	−36.3 to 39.9	1.00 to 15.5	8.04	180	101
Ocean	−1.85 to 35.0	7.76 to 26.8	19.7	160	124
Ectotherm	6 to 40.8	25.4 to 34.5	30.7	100	120
Endotherm	30.2 to 44.6	36.5 to 41.5	38.4	111	120
Desert	−10 to 60	ND	ND	140	194
Hot spring	10 (or 36.7)[a] to 108	28.7 to 55.0	∼40	28	161
Hydrothermal vent	2 to 400	ND	ND	35	198

Abbreviation: ND, not determined.

[a]There is not unanimous agreement on the defined temperature range of hot springs (132).

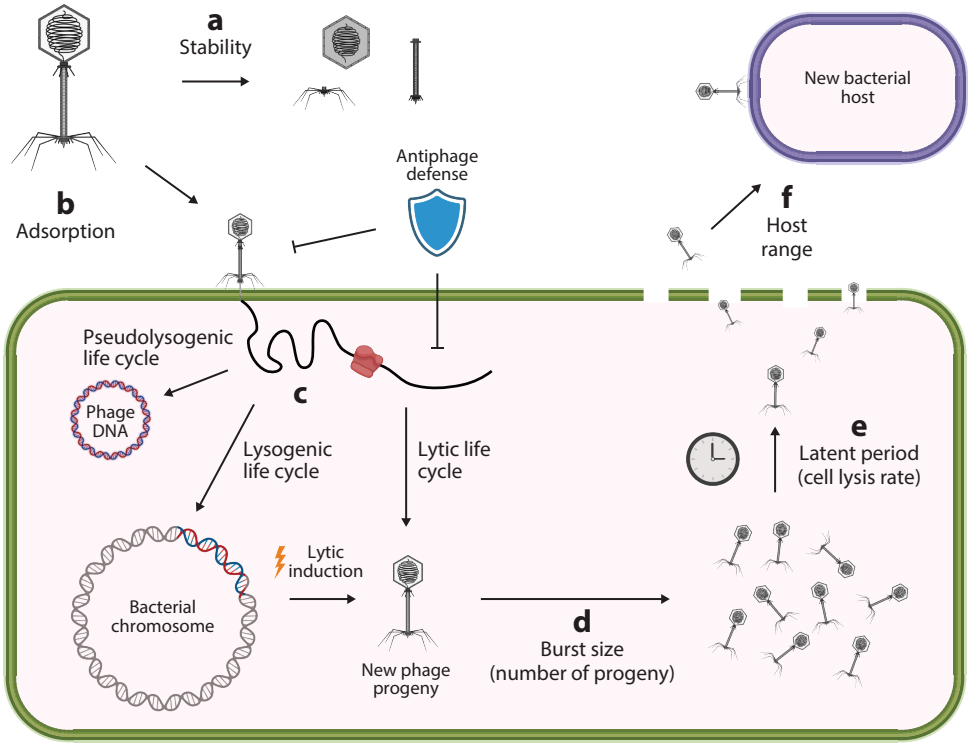


Figure 2

Phage life-history traits, with those that contribute to phage fitness labeled with letters. (*a*) Phage stability is a combination of the phage's intrinsic and extrinsic death rates (i.e., particle decay). (*b*) Phage adsorption reflects the rate of attachment to bacterial cells. (*c*) The lytic and lysogenic life cycles refer to the ability of phages to either start replicating immediately upon infection or integrate into the bacterial chromosome. The pseudolysogenic life cycle refers to the ability of phages to adopt a dormant, nonintegrative state postinfection. (*d*) Burst size is the number of phage progeny (particles) released from a single cellular infection. (*e*) Latent period is the time that passes from the point of infection to the point of cellular lysis. Burst size and latent period are coupled, as short latent periods restrict the time available to produce phage progeny. (*f*) Host range represents the diversity and number of bacterial hosts that the phage can infect. Finally, antiphage defense is the ability of the bacterial host to resist phage infections. While antiphage defense is a bacterial trait rather than a phage trait, it was included because of its importance in the success of phage infection. The labels correspond to those in **Table 2**. Figure adapted from images created in BioRender; MacLean C. 2025. **https://BioRender.com/swjx07z**.

bacterial cell, injection of phage genetic material, production of new phage particles (lytic life cycle) or integration of genetic material (lysogeny), and release of phage progeny through lytic enzyme activity. Phage infection steps can be grouped into life-history traits that contribute to phage fitness (**Figure 2**). Phage life-history traits include particle stability, adsorption rate, life-cycle preference (lytic versus lysogenic), burst size (number of phage progeny per infected cell), latent period (time from infection to cell lysis), and host range. Temperature can affect each of these traits independently (**Table 2**) and can help shape phage life-history strategies across thermal environments.

Particle stability. Reviews that explore phages and temperature have primarily focused on phage inactivation through thermal effects on phage stability (15, 84, 119), likely because phage

Table 2 Examples of studies reporting the impact of temperature increase on phage life-history traits[a]

Phage life-history trait	Bacteria used	Phage used	Phage family	Genome length (kB)	Genome type	Temperatures used (°C)	Effect of warming on trait	Reference(s)
Stability	*Escherichia coli*	φX174	Microviridae	~5.4	ssDNA	37 to 44	Restricted particle formation	31
	Pseudomonas aeruginosa	φPEV2	Schitoviridae	~72.7	dsDNA	37 to 42	Increased particle decay	67
		φLUZ19	Autographiviridae	~43.5	dsDNA			
		φ14-1 (PB1-like)	Myoviridae*	~66.2	dsDNA			
Adsorption rate	*Listeria monocytogenes*	φLP-026 (P70-like)	Siphoviridae	~67.1	dsDNA	6.5 to 37	Reduced phage binding affinity	163
		φLP-037 (P70-like)	Siphoviridae	~64.8	dsDNA			
		φLP-048 (P100-like)	Herelleviridae	~133	dsDNA			
		φLP-0125 (P100-like)	Herelleviridae	~135	dsDNA			
		φA511 (P100-like)	Herelleviridae	~134	dsDNA			
	Yersinia pestis	φvB_YpM_3 (Mu-like)	Myoviridae*	~39.4	dsDNA	26 and 37	Reduced phage receptor expression	117
		φvB_YpM_5 (Mu-like)	Myoviridae*	~37.2	dsDNA			
		φvB_YpM_6 (Mu-like)	Myoviridae*	~37.2	dsDNA			
		φvB_YpM_23 (Mu-like)	Myoviridae*	~38.7	dsDNA			
	P. aeruginosa; *Vibrio cholerae*	Not tested	NA	NA	NA	23 and 37; 22 to 37	Altered receptor access through biofilm production	23, 46, 170

(*Continued*)

Table 2 (*Continued*)

Phage life-history trait	Bacteria used	Phage used	Phage family	Genome length (kB)	Genome type	Temperatures used (°C)	Effect of warming on trait	Reference(s)
Life-cycle strategy	*Lactococcus lactis*	φLC3 (P335-like)	Siphoviridae*	~32.1	dsDNA	30 to 34.5	Lytic induction (thermal stress)	108
	Burkholderia pseudomallei	φBp-AMP1	Autographiviridae	~45	ND	25 to 37	Selection for lytic infection (virulence-transmission trade-off)	7, 155
Burst size	*Lactobacillus paracasei*	φiLp84 (P22-like)	Siphoviridae	~39.4	dsDNA	30 to 37	Reduced burst size	118
		φiLp1308 (P22-like)	Siphoviridae	~34.1	dsDNA			
	L. paracasei	φiLp84 (P22-like)	Siphoviridae	~39.4	dsDNA	20 to 37	Increased burst size	190
		φiLp1308 (P22-like)	Siphoviridae	~34.1	dsDNA			
		φB1	Siphoviridae	~38	dsDNA			
		φLDG	Siphoviridae	~26.6	dsDNA			
Latent period	*L. paracasei*	φiLp84	Siphoviridae	~39.4	dsDNA	30 to 37	Reduced latent period	118
		φiLp1308	Siphoviridae	~34.1	dsDNA			
Host range	*E. coli*	φWG01	Straboviridae	~170	dsDNA	28 to 42	Expanded host range	34
		φQL01	Straboviridae	~171	dsDNA			

Abbreviations: NA, not applicable; ND, not determined.

Asterisk indicates families that were inferred from similar phages.

[a] The National Center for Biotechnology Information database or discovery publications were used to determine phage families.

persistence in the environment is crucial for population survival during periods when bacterial densities are limiting. Additionally, there is growing interest in the use of phages as biocontrols and therapeutics, which must remain stable during storage and which may be used for products that will experience thermal extremes, such as foods undergoing pasteurization (6, 112).

Phage thermal stability is largely determined by the physical capsid structure of free phage particles. For example, as shown by Bull et al. (31), the evolution of phage thermal tolerance can occur through mutations in genes involved in capsid formation, likely because thermal destabilization of phage proteins may disrupt their folding and assembly into phage particles. High temperatures can also select for mutations in structural genes (41, 76) or in their promoters (29). These results are supported by findings that the genomes of natural phages isolated from hot springs exhibit preferences for GNA (glycine-*N*-alanine) sequences that promote the formation of thermostable disulfide bridges (113). In some phages, thermal decay is determined by the stability of capsid-tail connector proteins that degrade at high temperatures; this degradation results in DNA expulsion (6, 171). Phage stability can be enhanced by the presence of "decoration" proteins that stabilize phage capsids and support particle assembly (45).

Adsorption. The first stage of phage infection involves adsorption to the bacterial cell (reviewed in 14). Briefly, phages collide initially with bacterial cells through Brownian motion and then bind reversibly to common cell surface components. Through periodic adsorption and desorption, phages conduct a "random walk" across the bacterial surface until they find preferred receptors. Once target receptors are identified, phages bind irreversibly before injecting their genetic material into the cell. Bacterial surface receptors targeted by phages are diverse and often have roles in bacterial metabolism (14).

Rising temperatures reduce phage adsorption efficiencies (154, 163, 164). However, the mechanisms behind reduced adsorption can vary. Thermal increases may alter the ability of phages to bind to their target receptor—for example, because of reduced phage binding affinities (40, 138, 151). Consistent with this possibility, thermal adaptation in some phages has been linked to mutations in tail fiber genes that support phage attachment to bacterial surface receptors (39). Theoretically, reduced binding may also reflect changes in the conformation of bacterial structures that are used as surface receptors, although studies demonstrating this hypothesis are lacking. Alternatively, warming may alter the expression of bacterial surface receptors (102) and so change the availability of attachment areas. Receptor access may be further affected by biofilm production (170), which can increase (22, 23, 30) or decrease (46) in response to temperature. Notably, the adsorption of some phage taxa appears to be resilient to warming, and so thermal effects may be taxon dependent (21, 67, 164). Whether such effects are primarily driven by changes in phage binding ability or bacterial susceptibility to phage attachment is unclear.

Infection and genome stability. While phages may be able to attach to hosts at high temperatures, the injection of phage genetic material into the target bacterium can depend on temperature. For instance, during injection, phage λ DNA undergoes a density transition that facilitates successful infection at 37°C, and this process can be hindered at higher or lower temperatures (169). Similarly, under elevated temperatures, phage Qβ rapidly evolves mutations in a protein involved in DNA transfer; this finding suggests strong temperature dependence of genome injection (77, 98). For the successful establishment of infections, phages must be able to both adsorb to bacterial cells and transfer their genetic material at different temperatures.

Theoretically, injection and persistence of phage genetic material inside the bacterial cell may depend on the phage genome type: DNA versus RNA and double-stranded (ds) versus single-stranded (ss) molecules. For example, dsRNA is considered more thermostable than is dsDNA (85), and so dsRNA phage genomes are expected to be at lower risk of thermal decay than

are dsDNA phage genomes. Similarly, dsDNA is thought to be more thermostable than is ssDNA (130); correspondingly, ssDNA and potentially ssRNA phage genomes may be at higher risk of thermal decay. Notably, RNA and ssDNA phages have higher mutation rates than do dsDNA phages (52). Rising temperatures may increase RNA or dsDNA phage prevalence if genome stability is under selection. However, if thermal change selects for different phage traits, RNA or ssDNA phages may become more prevalent given their greater access to mutational diversity.

Lysis and lysogeny: alternative phage life cycles wherein phages either immediately kill their hosts upon infection (lysis) or integrate into the bacterial chromosome (lysogeny)

Life-cycle strategy. Phage life-cycle strategies (lytic, lysogenic, or pseudolysogenic; see **Figure 2**) are not mutually exclusive, and phages can move among them depending on environmental conditions (127, 185). While phages following a lysogenic life cycle are often under selection to preserve and even to protect host cells (25), high temperatures can induce integrated phages into a lytic life cycle through the threat of cell death (103, 108). Temperature modulates lysis and lysogeny decisions during phage infections, with higher temperatures generally encouraging phages to follow a lytic life cycle (117, 155, 185). Phage preference for lysis at high temperatures may follow the same mechanism as lytic induction, with phages attempting to "jump ship" before the death of their hosts.

Lysis and lysogeny decisions are also affected by temperature-mediated changes in bacterial growth rates and population densities (136). At high temperatures, bacterial growth rates are high, and so an abundance of bacterial hosts are available for phage infection (145). High host densities select for virulent, lytic phages, which rapidly replicate through host killing (2). In contrast, poor bacterial growth at low temperatures selects for low phage virulence through long latent periods or lysogeny, in which phages replicate in tandem with their hosts (2). Phage life-cycle decisions vary in response to host densities; phages following lysogenic life cycles often possess quorum-sensing systems that allow them to become induced into a lytic life cycle when susceptible bacterial host densities increase (5). **Supplemental Text 2** presents a detailed analysis of phage virulence and life-cycle preferences across phage thermal environments.

Supplemental Material >

Latent period and burst size. Following successful adsorption and DNA injection, the phage lytic life cycle involves the production of phage particles, the replication and packaging of phage genetic material, and lysis of the bacterial cell. The success of these steps can be exemplified by two correlated, temperature-dependent phage traits: latent period and burst size. The latent period represents the time from cell infection to cell lysis and is determined by the expression and activity of lytic enzymes (187). The burst size represents the number of viral particles produced from a single infection cycle and is determined by the replicative period length (latent period) and the reaction rates of phage replication proteins (86). Given that more rapid cell lysis shortens the time available to replicate, a longer latent period allows for a larger burst size.

Theory suggests that, by altering lytic enzyme activity, thermal change will either increase or reduce latent periods and burst sizes. High temperatures have indeed been found to shorten latent periods and reduce burst sizes; these findings indicate higher lytic enzyme activity (118). Warming temperatures can also reduce latent periods and burst sizes through host density effects that increase bacterial growth rates and densities (136). High host densities select for phages with shorter latent periods (2), as the benefit of ongoing transmission outweighs the cost of a smaller burst size.

Some studies have found that thermal change disrupts the coupling between latent period and burst size, with higher temperatures generally shortening the latent period but increasing the burst size (121, 123, 190). One explanation for this decoupling is that lytic enzymes and replicative/packaging proteins may have different responses to temperature. If replicative proteins have steeper thermal performance curves than do lytic enzymes, rising temperatures may cause phage particle production rates to rise more rapidly than do lysis rates. Given that latent period and burst

size determine phage virulence and transmission potential (2), further investigation of the tripartite relationship among temperature, burst size, and latent period is needed to better understand how phage fitness varies in natural environments.

Darwinian demon: a hypothetical organism that can achieve maximal fitness in all life-history traits and so outcompete all other organisms; the existence of Darwinian demons is restricted by fitness trade-offs among life-history traits

Thermal optimum: the temperature at which a bacterium or phage grows at its maximum rate

Host range. Changes in phage host ranges across temperatures can alter phage population dynamics and competition outcomes (96). In microbial communities, phages are often surrounded by diverse bacteria, including many they are unable to infect. The presence of unavailable hosts creates a strong selection pressure for host range expansion as phage host range generalists have higher host encounter rates (152). Accordingly, some phages have broad host ranges and can infect multiple unrelated bacterial species (33). Yet broad host ranges come with fitness trade-offs (139), and so most phages have species- or even strain-specific associations with bacterial hosts (135). By expanding or narrowing phage host ranges, temperature change could either increase bacterial host availability or reduce phage competitive fitness.

Phage host range expansion typically occurs through mutations in tail fiber genes, which are responsible for phage adsorption to the bacterial cell surface (152, 186). Interestingly, tail fiber mutations are also frequent targets when phages adapt to thermal stress (39); this observation raises the possibility that thermal adaptation may have pleiotropic effects on host range. For example, Chen et al. (34) found that tail fiber mutations that widen phage thermal tolerance ranges also expand phage host range. Yehl et al. (186) provided further details and found that changes in phage host range and thermal tolerance range depend on thermally adaptive mutations occurring in the "host range–determining region" of the tail fiber gene. However, thermal adaptation in other traits, such as phage particle stability, can restrict phage host range evolvability (159). Different modes of thermal adaptation may exert opposing pressures on phage host range.

High host diversity selects for phage host range expansion (152). Given that bacterial diversity often increases with temperature (197), co-selection for host range expansion and thermal tolerance may be frequent under warming conditions. Selection for host range shifts is exacerbated by temperature-mediated changes in bacterial community composition (59). While overall species richness may increase with warming, some bacterial taxa may be excluded, with this exclusion forcing phages to change their focal host to avoid extinction.

Life-history trade-offs. Life-history theory posits that life-history traits must have fitness trade-offs to explain the absence of Darwinian demons, which are hypothetical organisms capable of achieving maximal fitness for all traits (99). Such trade-offs are frequently found in phage systems (58, 60, 91, 129), and some have been shown to be temperature dependent (65). For example, rising temperatures can reduce phage stability but increase replication rates, thereby modulating phage population dynamics (47). Similarly, selection for increased phage thermal stability can lead to a reduction in replication rates (48). Thermal trade-offs and trade-ups have also been found among phage stability and adsorption rate, latent period, and burst size (87, 88). The evolution of trade-offs is restricted by thermal variation; fluctuating temperatures that alternately select for thermal stability and replication rate can remove the stability-replication trade-off (115).

Bacterial Resistance to Phages

Virulent phage infections create a strong selection pressure for the evolution of phage resistance in bacterial hosts, although the mechanisms and costs of phage resistance vary across temperatures. In single-species experiments, phage resistance typically arises through mutations in surface receptors (17, 183). These mutations prevent phages from adsorbing to the bacterial membrane but generally come with high fitness costs through pleiotropic effects on growth rates, virulence, and antibiotic susceptibility (110). Padfield et al. (128) showed that the costs of receptor mutation–based phage resistance on bacterial growth rates were highest at the bacterial thermal optimum.

These findings likely reflect the fact that mutated receptors generally have functions in bacterial metabolism (14); receptor mutations restrict growth at the thermal optimum by reducing metabolic rates.

Resistance can alternatively arise through the evolution or horizontal acquisition of phage defense systems (51, 166), which also have temperature-dependent costs (4). However, Aframian et al. (4) found that the costs of certain phage defense systems, in contrast to those of receptor-based mutations, decrease closer to the bacterial thermal optimum. These findings suggest that phage defense systems may be preferred at the thermal optimum, while receptor mutations are favored away from the optimum. The best route for phage resistance may depend on the level of warming experienced and on phage defense system availability.

The relative benefits of receptor mutations and phage defense systems can be further influenced by the effects of temperature on bacterial growth rates. At low temperatures, longer bacterial generation times increase the probability of multiple phage infections within a single bacterial reproductive cycle. As a result, selection at low temperatures favors phage defense systems that respond to ongoing infections, such as CRISPR-Cas, over receptor mutations (79). In addition, temperature can alter the expression of phage defense systems (20), although such temperature-dependent expression is not universal across all bacterial taxa (173).

Phage defense system: a bacterial cellular mechanism used to restrict phage growth during infections by either blocking phage replication inside the cell or restricting phage transmission

Thermal tolerance limits: the minimum and maximum temperatures at which a bacterium or phage can replicate

MISMATCHES IN BACTERIA-PHAGE THERMAL RESPONSES

The thermal sensitivity of phage life-history traits ultimately leads to temperature dependence in fitness, typically measured through phage growth rates (128). Changes in fitness with temperature can be visualized via thermal performance curves consisting of upper and lower thermal limits and of a thermal optimum in which fitness is highest. Thermal performance curves across biological systems generally follow the same pattern: Fitness increases gradually with temperature up to a thermal optimum, after which fitness rapidly decreases (153).

Mismatches in Ecological Time

Hosts and parasites often differ in the shape and positioning of their thermal performance curves (37). Generally, parasites are thought to have broader thermal tolerance limits than do their hosts; parasite burden is highest on either side of the host thermal optimum. However, experimental studies frequently highlight cases in which parasites have narrower thermal limits than do their hosts (61, 128). Depending on the extent and position of host-parasite thermal performance mismatches, changes in temperature can either disrupt or exacerbate parasite infections.

Phages are obligate parasites and so tend to have narrower thermal limits than do their bacterial hosts (**Figure 3*a***). In addition, studies generally find that phage thermal optima are either the same (128) or lower than bacterial thermal optima (93). The mismatch among bacterial and phage thermal optima and upper thermal limits represents a thermal refuge in which bacteria can grow in the absence of phage infection (128). Using a panel of 15 G4-like *Escherichia coli* phages, Knies et al. (93) found that phages infecting the same bacterial strain exhibit considerable thermal optimum and tolerance limit variation. As observed in bacteria (50), rising temperatures are expected to lead to the extinction of thermally sensitive phages and in turn to a decrease in phage diversity. Reduced phage predation will increase the relative abundance of dominant bacterial genotypes (114), although resource availability will decrease because of reduced nutrient cycling (179). Phage predation can be maintained at high temperatures by the presence of thermally tolerant phage community members (67).

Phages generally follow a "hotter-is-better" thermal strategy whereby higher thermal optima tend to coincide with higher maximum population growth rates (93) (**Figure 3*b***). The

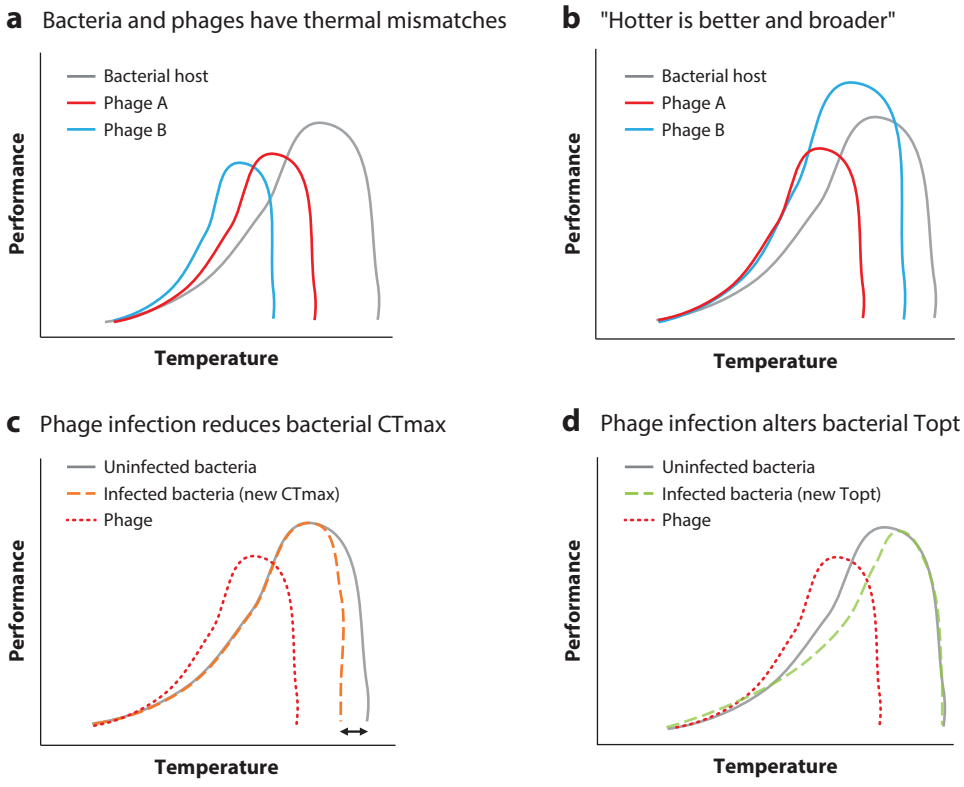


Figure 3

Examples of bacteria and phage thermal performance curves. (*a*) Bacteria and phages have thermal mismatches. Phages generally have lower thermal optima and thermal maxima than do their bacterial hosts (128). However, thermal responses vary among phages (93). (*b*) Phages generally follow "hotter-is-better" and "hotter-is-broader" thermal strategies whereby phages with higher thermal optima tend to have higher maximum performance and higher upper thermal limits (93). (*c*) Phage infection may reduce bacterial upper thermal limits (CTmax) through the metabolic costs associated with antiphage defense (4, 74). The two-sided arrow shows the shift in the bacterial upper thermal limit. (*d*) Phage infection can lead to shifts in the bacterial thermal optimum (Topt) through higher growth at phage nonpermissive temperatures and temperature-dependent fitness costs of phage resistance (93). Figure adapted from images created in BioRender; MacLean C. 2025. **https://BioRender.com/r1w4qkz**.

Thermal mismatch: the difference in thermal performance curve shape, including differences in thermal optima and thermal tolerance limits, between two organisms

"hotter-is-better" strategy stems from the fact that rising temperatures increase reaction rates and allow for faster growth. The relationship between maximum growth and thermal optima exacerbates bacteria-phage thermal mismatches as phages adapted to low temperatures will have lower fitness relative to their bacterial hosts. While a "hotter-is-better" strategy is also observed in insects (56), a recent meta-analysis found only limited support for the existence of the strategy across species (109). One reason that evidence from phages may show stronger support for the strategy could be because phage thermal responses depend on the reaction norms of individual proteins involved in phage infection and replication (92). More complex organisms may also be able to maintain high fitness at low temperatures through thermal acclimation, behavioral modifications, or internal thermoregulation. In addition to "hotter is better," phages also follow a "hotter-is-broader" strategy whereby phages with higher thermal optima tend to have broader thermal tolerance limits (93). A correlation between thermal optimum, thermal tolerance range,

and maximum fitness contradicts specialist-generalist theory, which states that an increase in thermal tolerance range should lead to a trade-off with maximum performance (89). However, the strategy means that cold-adapted phages will rapidly lose activity as temperatures increase, with this loss leading to phage community shifts and a decrease in phage diversity.

Host-parasite thermal performance mismatches are not fixed. For example, Hector et al. (74) found that parasite infection can reduce host upper thermal limits through exploitation of host resources and costly activation of immune responses. Additionally, parasites, being smaller than their hosts, are expected to rapidly acclimate to thermal change, with this rapid acclimation potentially reducing mismatches (131, 148). In bacteria-phage systems, the activation of phage defense systems has temperature-dependent fitness costs linked to autoimmunity (4). To what extent phages are capable of plastic responses to thermal change is unclear. However, one mechanism for phage acclimation could include temperature-dependent expression of decoration or structural proteins to stabilize phage particles (29, 192). Through the activation of bacterial immune responses and phage thermal acclimation, bacterial and phage upper thermal limits may become increasingly aligned (**Figure 3*c***). The alignment of bacterial and phage thermal responses will remove the high-temperature bacterial thermal refuge and could increase the probability of bacterial population collapses during warming events.

Mismatches in Evolutionary Time

Bacteria-phage thermal mismatches can change over time as thermal performance evolves. Phage infection has been shown to select for upshifts in bacterial host thermal optima (128, 142). In some bacteria-phage systems, the evolution of phage resistance results in temperature-dependent fitness costs, and these costs are highest at the bacterial thermal optimum (128). As bacterial fitness at the thermal optimum decreases following resistance evolution, relative bacterial fitness above the thermal optimum increases, creating a new, higher thermal optimum (128). An upshift in bacterial thermal optima is expected to exacerbate bacteria-phage thermal mismatches as bacteria start to specialize at high temperatures (**Figure 3*d***).

Phage thermal adaptation can reduce thermal mismatches by aligning both thermal optima and thermal tolerance limits to those of their bacterial hosts. By reanalyzing the evolved lines from Holder & Bull (76), Knies et al. (92) showed that the "hotter-is-better" and "hotter-is-broader" strategies are evolvable; selection for extended upper thermal limits resulted in an increase in phage thermal optima and maximum performance (**Figure 3*b***). A correlation between phage thermal optima and upper thermal limits may be expected when thermal stress affects proteins directly involved in phage replication. For example, fitness-reducing phage polymerase mutations have been shown to simultaneously reduce phage replication at both the thermal optimum and upper thermal limit (97). The evolvability of phage thermal optima and upper thermal limits means that thermal adaptation may reduce the size of bacterial thermal refuges. In addition, heat stress that reduces bacterial growth may still permit phage infections and lead to reduced phage infectious periods and low bacterial host availability. We might predict that reduced phage virulence will evolve under extreme warming (2, 73).

The evolution of thermal performance curves depends on thermal variability. Static temperatures are expected to select for specialists with narrow thermal ranges, in which performance is highest at the evolved temperature. In contrast, fluctuating temperatures favor thermal generalists with wider thermal tolerance limits but lower maximum fitness (63). Studies in phages suggest that selection under fluctuating temperatures is primarily driven by the most extreme temperature (10). However, other studies have found that fluctuations can reduce the strength of selection for phage high-temperature adaptation (70) and restrict coevolutionary dynamics with bacterial hosts (53). The lack of consensus among studies on the relationship between fluctuating temperatures

and thermal adaptation likely reflects the different fluctuation conditions and phages used. While slow fluctuations facilitate the fixation of thermally adapted genotypes, rapid fluctuations increase the impact of genetic drift relative to selection and thereby reduce fixation rates and slow adaptation (42). In addition, some phages can rapidly adapt to high temperatures, although the evolution of other phages is impeded because of inhibited growth (76).

Phageome: the total community of phage populations within a specific area

PHAGE COMMUNITIES AND THERMAL SENSITIVITY

Phageome Composition and Dynamics

Phages exist in diverse communities, termed phageomes (reviewed in 36), whose compositions are expected to change with temperature because of phage-specific thermal responses (93). Theoretically, a change in temperature should select for phages whose thermal optima most closely match the new environmental temperature (59, 157). Thermal selection should also favor phages that can rapidly adapt to thermal change through altered thermal optima or extended thermal tolerance limits (76). Consistent with this premise, rising temperatures alter relative phage growth rates and allow weaker phage competitors to become more dominant (67). Thermal increases also cause some phage species to decay more rapidly than others and potentially lead to the exclusion of these phages from the community (6, 67).

Phageome composition is also expected to change with temperature because of thermal sensitivity in the bacterial host community. In bacterial communities, species vary in their thermal tolerance ranges and thermal optima (59). Accordingly, thermal change generally leads to changes in bacterial community composition (150, 157), including in animal microbiomes (104). Bacteria and phages have modular interaction networks in which each phage generally targets a small subset of bacterial community members (135). Changes in bacterial relative abundances—for example, through dysbiosis of gut microbiota with warming—are expected to lead to phageome composition shifts (177). The effects of temperature on phage community composition likely depend on the thermal regime. Static thermal shifts generally reduce genotypic and species diversity by selecting for a few thermal specialists (59). In both bacterial and phage communities, thermal fluctuations may promote the maintenance of species and genetic diversity through selection for a range of thermal optima (193).

Inter-Phage Competition

Phage competition outcomes depend on both relative phage growth rates and the rate of host population depletion (virulence) (13, 69, 71). When host densities are high, phages maximize their host share by rapidly depleting the host population. Rapid host depletion benefits fast-growing phages by reducing the availability of hosts for slow-growing competitors. Conversely, when host densities are low, high-virulence phages rapidly run out of available hosts, and so selection favors low-virulence phages, which maximize within-host replication and transmission potential (2).

The rate of bacterial clearance by phages is determined by temperature-dependent life-history traits including adsorption rate, latent period, and burst size. Rapid adsorption and short latent periods mean host cells are lysed quickly at the cost of a reduced burst size. High temperatures have been shown to reduce latent periods in some phages (118), with this reduction potentially making the phages more virulent (2). In addition, phage virulence may increase with temperature via pleiotropy with thermal adaptation. For example, Kashiwagi et al. (87) found that mutations arising from thermal selection resulted in more rapid adsorption, reduced latent periods, and extended burst sizes. Given that thermal adaptation occurs more rapidly in some phages than in others (76), pleiotropy may allow phages to improve competition outcomes as host availability rises during warming events.

The interaction between thermal adaptation and competition is bidirectional. While thermal adaptation can alter competitiveness, competition has the potential to restrict species' thermal adaptation (172). Competition restricts thermal adaptation by reducing population growth rates and restricting the generation of adaptive mutations (83). Competition can also reduce the time available for thermal adaptation by promoting the exclusion of thermally maladapted taxa (44). The presence of competitors is expected to restrict phage thermal adaptation. However, some studies have suggested that competition can promote thermal adaptation when both selection pressures are acting in the same direction (109, 176). For instance, in some phage systems, both competition and high temperatures select for shorter latent periods (2, 87). Synergistic selection between competition and thermal adaptation may help to stabilize phageomes during thermal perturbations.

Viral shunt: the release of carbon and other nutrients from bacterial cells after their lysis by a phage

WIDER IMPLICATIONS AND CONCLUDING REMARKS

Climate Change and Phage Thermal Environments

Anthropogenic climate change is a growing threat to global ecology via the alteration of both average temperatures and thermal variability (82). In an early review of marine viruses and climate change, Danovaro et al. (43) highlighted that rising temperatures in polar regions generally lead to an increase in bacterial growth rates and a rise in viral activity. In contrast, temperate ocean heating leads to a decoupling of bacterial growth and viral activity due to reduced bacterial growth efficiency. Polar regions are at the greatest risk of warming due to climate change (144). As a result, microbial communities in polar regions will likely see a rise in phage lytic activity and nutrient cycling (viral shunt) (156). Conversely, warming in temperate oceans may lead to a decrease in phage activity. Warming, especially in polar regions, is also expected to lead to changes in phageome composition. In a recent study, Zhong et al. (196) showed that glacial phage communities from warm and cold climatic periods have unique compositions that possibly reflect community temperature responses.

In soil environments, climate change is expected to drive desertification through an increase in soil surface temperatures and a reduction in soil moisture content (27). Desertification reduces soil carbon and nitrogen levels and leads to reduced bacterial diversity and altered community composition (19, 191). As phage diversity largely tracks bacterial diversity (68, 177), desertification is expected to indirectly reduce phage diversity. Phage abundance and diversity may be further restricted by elevated phage particle decay under dry, thermally variable conditions (66). Supporting these hypotheses is the observation that hot desert soils tend to have lower phage abundances than do temperate soils (181).

Finally, the thermal variation experienced by phages that are associated with ectotherm hosts is expected to increase with climate change. While ectotherms can acclimate in response to thermal change (148), ectothermic animals generally maintain stable body temperatures through behavioral thermoregulation. Rising temperatures due to climate change are expected to reduce the efficacy of behavioral thermoregulation by limiting access to thermal refuges (90). Uncontrolled body temperature increases may contribute to ectotherm microbiome dysbiosis (104) and to the disruption of phage activity. However, for ectothermic disease vectors such as mosquitoes, rising temperatures are predicted to increase their reproduction rates and expand their inhabitable geographic ranges (147). Phages are known constituents of the mosquito microbiome (105) and are expected to be transferred with the microbiome during feeding (3, 49). With a changing climate, phages may experience more frequent thermal shifts through transmissions to animal and plant hosts.

Phage-Microbiome Interactions Under Rapid Thermal Change

Phage therapy: the deployment of phages in plants and animals to treat bacterial infections

Eukaryotic microbiome diversity and composition are highly sensitive to thermal shifts (104), which may arise through extreme weather events (e.g., in plants and ectothermic animals) or the induction of fevers (e.g., in endothermic animals). The impact of temperature on microbiomes is generally considered with regard to bacterial thermal responses. In a recent review, Huus & Ley (80) highlighted that commensal bacteria often occupy different thermal niches and that external thermal stress can directly lead to bacterial microbiome composition shifts. They also suggested that fevers may provide protective effects by increasing the fitness of bacterial microbiome members relative to invading pathogens. Studies to date have generally disregarded the role of the phageome in modulating bacterial microbiota during thermal change.

Phages often form associations with animals or plants as members of their microbiomes (11, 111). Phage lytic activity is crucial for maintaining microbiome diversity through negative, frequency-dependent selection and nutrient cycling (114, 179). Microbial diversification increases the resilience of microbiomes to environmental perturbations and reduces the impact of thermal change (107). However, the stabilizing effects of the phageome may be disrupted by temperature if phages vary in their thermal responses. Thermal upregulation of phages targeting keystone microbiome members may reduce microbiome stability and remove the microbiome's protective effects against pathogens (8, 158). Additionally, diverse phageomes may provide their own protective effects by actively lysing infecting pathogens. For example, Yang et al. (184) showed that plant rhizospheres contain pathogen-targeting phages that can protect plants from infections. However, plant pathogen–targeting phages can be temperature sensitive (173).

Phages also contribute to microbiome resilience by facilitating horizontal gene transfer through phage lysogeny. Phages often encode genes that contribute to bacterial environmental stress tolerance (174) and that support the stabilization of bacterial heat shock proteins (133). While rising temperatures generally support a lytic life cycle (117), temporary heat stress (such as that caused by fevers or heat waves) may increase the transfer of heat tolerance genes among bacteria through prophage mobilization (108, 188). An important caveat is that phages also often carry genes involved in pathogen virulence and antimicrobial resistance (38, 55). While phages may buffer against microbiome destabilization, heat stress may facilitate the acquisition of phage-encoded virulence or antimicrobial resistance genes by pathogens (122).

High Temperatures Can Reduce Phage Therapy Efficacy

Because of their lysis of bacterial cells, phages are increasingly being viewed as a potential supplement or alternative to antibiotics in the treatment of animal and plant bacterial infections (137, 175). In the selection of candidates for phage therapies, efforts are made to ensure that phages can operate at host body temperatures. For example, phages used in human therapies are tested for in vitro lytic potential at 37°C (resting human body temperature) (168). In contrast, phages targeting plant pathogens are tested at lower, more physiologically relevant temperatures (57, 175). Phage candidates that perform well at in situ temperatures may be disrupted by thermal upshifts caused by fevers (e.g., in endothermic animals) or heat waves (e.g., in plants). Greenrod et al. (67) showed that thermal increases above 37°C can inactivate phages previously used in human phage therapy treatments (e.g., φ14-1; see 168).

One approach to improving phage performance across a breadth of host temperatures is to preevolve, or "train," phages with their bacterial targets (26, 54). Phage training at or above in situ temperatures could expand phage upper thermal limits, improve the position of phage thermal optima, and increase maximum performance (93). Phage cocktails, which are combinations of multiple phages, are a potential alternative to phage thermal training (1, 62). Phage cocktails could

be designed to withstand thermal increases via the use of phages that span a range of thermal optima and thermal limits. However, thermal increases may inactivate some cocktail members and lead to weaker suppression and to faster and stronger phage resistance evolution (17, 183).

CONCLUSIONS

Phages are crucial for the maintenance of microbial diversity, the functioning of microbiomes, and the suppression of bacterial pathogens (137, 160, 199). Thus, large temperature changes shaping phage ecology and evolution could dramatically affect ecosystem stability and the health of crops, livestock, and humans. Phages can be grouped based on the thermal environments in which they are found. Phage thermal environments encompass a broad temperature range and include both relatively stable environments (e.g., polar ice, hydrothermal vents, and endothermic hosts) and highly variable environments (e.g., deserts, oceans, and soil). To accommodate such thermal breadth, phages often exhibit different life-history strategies that are specific to their thermal environments.

Experimental studies have shown that phages vary considerably in their thermal responses (67, 93). While such studies highlight that thermal change will alter phage community dynamics, these studies have a limited ability to predict overall thermal effects in natural phageomes because they omit the largely unexplored diversity of environmental phage taxa (72). Emerging technologies such as machine/deep learning could support phage thermal performance predictions and have recently been used to predict other phage traits such as host range from phage DNA/protein

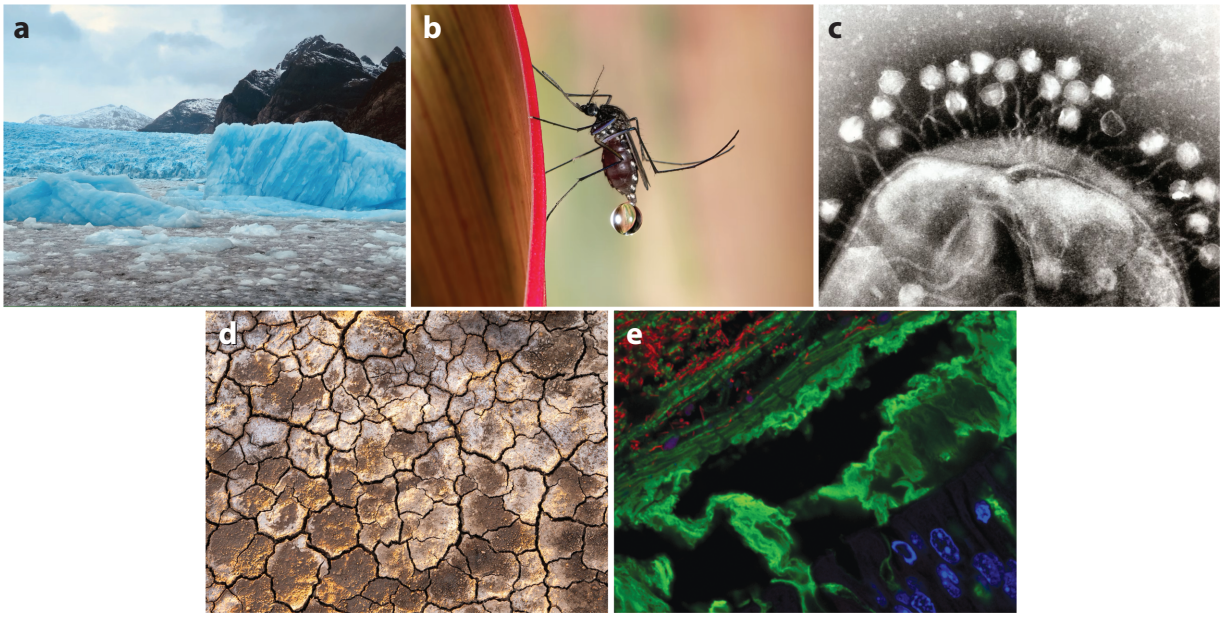


Figure 4

Examples of changing phage thermal environments. (*a*) Polar ice melts into ocean water. Photo by Jairo Gallegos, Unsplash. (*b*) Mosquito vectors often transmit microbes to endotherms. Photo by David Clode, Unsplash. (*c*) Phages isolated from soil or water can be deployed as therapeutics in animals or plants. Photo from **https://commons.wikimedia.org/wiki/File:Phage.jpg** (CC BY-SA 3.0). (*d*) Desertification of soils leads to increased thermal variability. Photo by Zetong Li, Unsplash. (*e*) The animal gut microbiome can experience thermal change through the induction of fevers or microbial immigration from ingested soil or water. In the confocal microscopy image, bacteria are red, gut mucus is green, and the gut epithelium is blue. Panel adapted with permission from Reference 125.

sequences (106). The combination of these technologies with environmental DNA/RNA sequencing may provide an important step toward the determination of thermal effects in phage communities at a global scale.

Phages are becoming increasingly at risk of thermal perturbations caused by global climate change (82) or by their movement between thermal environments (81) (**Figure 4**). While rising temperatures may lead to increased phage activity in some environments, such as polar oceans (43), thermal environment transitions through desertification are expected to lead to the loss of phage diversity and activity (191). Rapid thermal change should also be considered in the context of animal and plant health. The functioning of animal and plant microbiota, the efficacy of phage therapies, and the success of vector-borne disease control programs all depend on the ability of phages to withstand thermal variation caused by fevers and heat waves. The impact of climate warming on ecosystems and human health will fundamentally depend on the ecological and evolutionary responses of phages and on the effects rippling up levels of biological organization.

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The authors are not aware of any affiliations, memberships, funding, or financial holdings that might be perceived as affecting the objectivity of this review.

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