SUPPLEMENTARY INFORMATION

Supplementary methods

Microbial food web model

To illustrate the potential impacts of temperature, microbial food web structure, and viral infection on the carbon and nutrient cycling, we developed a simple mathematical model to study the dynamics of an assortment of organisms that exist at different trophic levels and play distinct functional roles within microbial food webs—including N-fixers (*NF*), decomposers (*D*), eukaryotic algae (*A*), protist grazers (*G*), protist top predators (*P*), and viruses (V_i) that exclusively infect each organism (Box 1, Figure B1). The model also includes pools (external to organisms) of relevant essential elements—including, inorganic nitrogen (N_I; converted from N_2 by N-fixers), inorganic carbon (C_I; i.e., carbon fraction of CO_2), and organic carbon (C_O; dead organic matter)). These pools of essential elements are available for use by organisms and their concentrations are influenced by biological processes (e.g., photosynthesis, respiration, and mortality). Biological populations and elemental pools are referred to in terms of mass concentrations standardized by units of peat mass (units of μg / g of peat). The dynamics of all components are governed by a system of ordinary differential equations (Eqns. S1-S13). Variable and parameter definitions, units, and values used for analysis are given in Table S2. Parameter values were chosen such that all organisms exhibited non-zero equilibrium densities using the same parameter values across all biological scenarios shown in Figure B2, allowing for more direct comparison of biological scenarios.

In this model, all basal organisms (i.e., organisms that do not consume other organisms; *NF*, *D*, *A*) grow logistically and consume elements from external pools (N_I, C_I, C_O) according to their modes of energy acquisition: autotrophs (*NF* and *A*) use C_I, non-N-fixers (*D* and *A*) use N_I, and decomposers (*D*) use C_O. Element uptake rates follow Michaelis-Menton kinetics. Biomass production rates in all organisms is reduced by inefficient conversion of resources (ε_i). Conversion efficiency in consumers is also reduced according to the lowest stoichiometric ratio (carbon or nitrogen) between a given resource organism and its consumer ($q_{element,resource}$ / $q_{element,consumer}$; i.e., Leipig's law of the minimum). All organisms are infected by viruses that are specific to each host. All elemental pools operate as chemostats with an inflow rate (α_k) and an outflow rate (δ_k). Inorganic nitrogen (N_I) increases with respiration and decreases with growth of decomposers (*D*) and eukaryotic algae (*A*). Inorganic carbon (C_I) increases with respiration and decreases with growth of N-fixers (*NF*) and eukaryotic algae (*A*). Organic carbon increases with mortality (*m*, organisms and viruses) and viral lysis (ϕ) and decreases with growth of decomposers (*D*). All temperature dependencies follow Sharpe-Schoolfield functional forms[1] (Eqn. S14) with activation energies that are specific to each rate: respiration (0.65eV[2]), photosynthesis (0.32eV[3]), mortality (0.45eV[4]), and consumption (0.65eV[2,5]). Viral lysis rates and burst sizes were assumed to follow established activation energies of consumption (0.65eV).

Nitrogen-fixer:	$\dot{NF} = NF\left(\varepsilon_N \mu_{NF}(T) \frac{C_I}{h_{C_I,NF} + C_I}\left(1 - \frac{NF}{K_{NF}}\right) - a_{NF,G}(T)G - a_{NF,P}(T)P - \phi_{NF}(T)V_{NF} - r_{NF}(T) - m_{NF}(T)\right)$	(S1)
Decomposer:	$\dot{D} = D\left(\varepsilon_D \mu_D(T) \frac{N_I}{h_{N_I,D} + N_I} \frac{C_O}{h_{C_O,D} + C_O}\left(1 - \frac{D}{K_D}\right) - a_{D,G}(T)G - a_{D,P}(T)P - \phi_D(T)V_D - r_D(T) - m_D(T)\right)$	(S2)
Eukaryotic Algae:	$\dot{A} = A\left(\varepsilon_A \mu_A(T) \frac{N_I}{h_{N_I,A} + N_I} \frac{C_I}{h_{C_I,A} + C_I}\left(1 - \frac{A}{K_A}\right) - a_{A,P}(T)P - \phi_A(T)V_A - r_A(T) - m_A(T)\right)$	(S3)
Grazer:	$\dot{G} = G\left(\varepsilon_G min\left(\frac{q_{N,NF}}{q_{N,G}}, \frac{q_{C,NF}}{q_{C,G}}\right) a_{NF,G}(T)NF + \varepsilon_G min\left(\frac{q_{N,D}}{q_{N,G}}, \frac{q_{C,D}}{q_{C,G}}\right) a_{D,G}(T)D - a_{G,P}(T)P - \phi_G(T)V_G - r_G(T) - m_G(T)\right)$	(S4)
Predator:	$\dot{P} = P\left(\varepsilon_P min\left(\frac{q_{N,NF}}{q_{N,P}}, \frac{q_{C,NF}}{q_{C,P}}\right) a_{NF,P}(T)NF + \varepsilon_P min\left(\frac{q_{N,D}}{q_{N,P}}, \frac{q_{C,D}}{q_{C,P}}\right) a_{D,P}(T)D + \varepsilon_P min\left(\frac{q_{N,A}}{q_{N,P}}, \frac{q_{C,A}}{q_{C,P}}\right) a_{A,P}(T)A + \varepsilon_P min\left(\frac{q_{N,G}}{q_{N,P}}, \frac{q_{C,G}}{q_{C,P}}\right) a_{G,P}(T)G - \phi_P(T)V_P - r_P(T) - m_P(T)\right)$	(S5)
Virus (N-fixer):	$\dot{V}_{NF} = V_{NF}\left(\beta_{NF}(T)\phi_{NF}(T)NF - m_V(T)\right)$	(S6)
Virus (Decomposer):	$\dot{V}_D = V_D\left(\beta_D(T)\phi_D(T)D - m_V(T)\right)$	(S7)
Virus (Algae):	$\dot{V}_A = V_A\left(\beta_A(T)\phi_A(T)A - m_V(T)\right)$	(S8)
Virus (Grazer):	$\dot{V}_G = V_G\left(\beta_G(T)\phi_G(T)G - m_V(T)\right)$	(S9)
Virus (Predator):	$\dot{V}_P = V_P\left(\beta_P(T)\phi_P(T)P - m_V(T)\right)$	(S10)
Inorganic Nitrogen (N_I):	$\dot{N}_I = \alpha_{N_I} + r_{NF}(T)q_{N,NF}NF + r_D(T)q_{N,D}D + r_A(T)q_{N,A}A + r_G(T)q_{N,G}G + r_P(T)q_{N,P}P - q_{N,D}\mu_D(T)\frac{N_I}{h_{N_I,D} + N_I}\frac{C_O}{h_{C_O,D} + C_O}D - q_{N,A}\mu_A(T)\frac{N_I}{h_{N_I,A} + N_I}\frac{C_I}{h_{C_I,A} + C_I}A - \delta_{N_I}N_I$	(S11)
Inorganic Carbon (C_I):	$\dot{C}_I = \alpha_{C_I} + r_{NF}(T)q_{C,NF}NF + r_D(T)q_{C,D}D + r_A(T)q_{C,A}A + r_G(T)q_{C,G}G + r_P(T)q_{C,P}P - q_{C,NF}\mu_{NF}(T)\frac{C_I}{h_{C_I,NF} + C_I}NF - q_{C,A}\mu_A(T)\frac{N_I}{h_{N_I,A} + N_I}\frac{C_I}{h_{C_I,A} + C_I}A - \delta_{C_I}C_I$	(S12)
Organic Carbon (C_O):	$\dot{C}_O = \alpha_{C_O} + m_{NF}(T)q_{C,NF}NF + m_D(T)q_{C,D}D + m_A(T)q_{C,A}A + m_G(T)q_{C,G}G + m_P(T)q_{C,P}P + m_V(T)q_{C,V}(V_{NF} + V_D + V_A + V_G + V_P) + \phi_{NF}(T)V_{NF}q_{C,NF}NF + \phi_D(T)V_Dq_{C,D}D + \phi_A(T)V_Aq_{C,A}A + \phi_G(T)V_Gq_{C,G}G + \phi_P(T)V_Pq_{C,P}P - q_{C,D}\mu_D(T)\frac{N_I}{h_{N_I,D} + N_I}\frac{C_O}{h_{C_O,D} + C_O}D - \delta_{C_O}C_O$	(S13)

Supplementary Tables

Table S1. Detailed description and summarized results for select published studies of temperature effects on viruses.

Type of Study	Location or Host-Virus system	Observed Temperature Effects	Reference
Environmental	Backwater system of Danube River	· Higher temperature induced higher viral decay rates · Viral abundance was tightly correlated with seasonal bacterial abundance one year, but not the next · The lowest percentage of bacteria infected by phage were observed at 23-26°C, the highest at 6-22°C, and between at ≤ 5°C · Burst size was temperature dependent	Mathias *et al.* (1995)[6]
Laboratory	*Heterosigma akashiwo* (H93616, NM96) / Hav (HaV01, HaV08)	· Decay rates increased with increasing temperature · Latent phase decreased with increasing temperature · Thermal ranges of lysis by virus were unique for different host-virus pairs	Nagasaki and Yamaguchi (1998)[7]
Laboratory	Bacteriophage 9A isolated from Arctic seawater	· The half-life of infective phages decreased with increasing temperature	Wells and Deming (2006)[8]
Laboratory	Samples from Western Pacific Ocean	· Increases in temperature and photosynthetic radiation resulted in higher virus decay rates · Low fluorescence viruses were more sensitive to warming and increased PAR than high fluorescence viruses	Wei *et al.* (2018)[9]
Metadata	*N/A*	· Temperatures at which most marine viruses are inactivated fall outside of the host temperature range	Mojica and Brussaard (2014)[10]
Laboratory	*Escherichia coli* / coliphage isolates from the River Swift	· Temperature range of phages were independent of host growth temperature · Temperature was seen to affect the adsorption of 2 phages and the multiplication of another 2	Seeley and Primrose (1980)[11]
Laboratory	*Escherichia coli* / T4	· Adsorptions rates increased with increasing growth rate and positively correlated with cell size · The rate of phage release and burst size increased with growth rate, but the length of the eclipse and latent periods decreased with growth rate · Burst size was dependent on both growth rate and time until lysis	Hadas *et al.* (1997)[12]
Laboratory	*Emiliana huyxleyi* CCMP374 / EhV86	· 3°C increase in temperature induces a viral resistant host phenotype	Kendrick *et al.* (2014)[13]
Laboratory	*Chaetoceros tenuissimus* / Cten DNAV and Cten RNAV	· Susceptibility of all strains to Cten DNAV increased with temperature up to T_{opt} · Temperature range and degree of susceptibility to Cten RNAV was strain dependent	Tomaru *et al.* (2014)[14]

		· Maximum burst size of Cten DNAV and minimum burst size of Cten RNAV were both observed between 15-20°C	
Laboratory	*Staphylococcus aureus* / *S. aureus* phage	· The rate of phage production is related to the growth rate of the host. Higher growth rates up to T(opt) result in shorter latency periods, though T > T(opt) result in longer latency periods	Krueger and Fong (1937)[15]
Laboratory	*Escherichia coli* / coliphage	· Latency period decreases with increasing temperature and is directly inversely proportional to the division rate of bacteria	Ellis *et al.* (1939)[16]
Laboratory	*Micromonas* sp. MicA, MicB, MicC / MicVA, MicVB, MicVC	· At temperatures < T_{opt}, latent periods were increased, host cell lysis was delayed, and viral yield was reduced · Cell lysis did not usually occur at temperatures > T_{opt} · At temperatures slightly above T_{opt}, chronic infection (viral production with no cell lysis) was observed · At temperatures much above T_{opt}, no viral progeny were produced	Demory *et al.* (2017)[17]
Laboratory	*Micromonas polaris* / MpoV	· Higher temperatures resulted in shorter latent periods and increased burst sizes	Maat *et al.* (2017)[18]
Laboratory	*Micromonas polaris* strain RCC2257, strain RCC2258 / Mpov-45T	· Higher temperature (7°C vs. 3°C) caused earlier cell lysis and increased burst size, except in low light conditions	Piedade *et al.* (2018)[19]
Environmental	Southern Beaufort Sea and Amundsen Gulf	· Seasonal and spatial variation in virus concentrations were correlated with Chl-a concentration, bacterial abundance and composition, temperature, salinity, and depth · Percentage of variance explained by temperature was inconsistent between seasons	Payet and Suttle (2007)[20]
Environmental	Lake Pavin	· Virus abundances correlated most closely with host abundance · Surface bacterial abundances were largely influenced by temperature while monimolimnion bacterial abundances likely influenced by organic matter export during surface blooms	Colombet *et al.* (2009)[21]
Metadata	N/A	· Positive relationships were observed between viral abundance and temperature within all distinct oceanic regions examined, however a global decreasing trend was seen across these regions when all data was assessed together · Water column viral production increased with temperature in polar and cold temperate regions, but decreased with temperature in warm temperate systems	Danovaro *et al.* (2011)[22]
Environmental	Japanese paddy field flood waters	· Viral abundance changed seasonally, but was highly correlated with bacterial abundance	Nakayama *et al.* (2007)[23]

Environmental	North Atlantic Ocean	· Shift from virus-induced to grazing-induced phytoplankton mortality with increased latitude (decreased temperature)	Mojica *et al*. (2016)[24]
Environmental	Michigan agricultural soils	-Viral abundance changed seasonally; abundance was highly correlated to bacterial abundance, organic carbon content and total nitrogen	Roy et al. 2020[25]
Metadata	Global	-Viral abundances are several orders of magnitude higher in cold deserts compared to hot deserts	Williamson et al. 2017[26]

Table S2. Variables and parameters used in the microbial food web model. For parameters that are functions of temperature ($f(T)$), values are given at a reference temperature of 20°C.

Variable/Parameter	Definition	Units	Value
(NF, D, A, G, P)	Biomass conc.	µg g_{peat}^{-1}	na
(N_I, C_I, C_O)	Nutrient conc.	µg g_{peat}^{-1}	na
ε_i	Production efficiency	na	0.8
$\mu_i(T)$	Max growth rate	d^{-1}	2.5
$h_{k,i}$	Half-saturation constant	g	10
K_i	Carrying capacity	µg g_{peat}^{-1}	K_{NF}, K_A = 500 K_D = 1000
$a_{i,j}(T)$	Consumption rate	d^{-1} (µg g_{peat})$^{-1}$	$a_{NF,G}, a_{D,G}$ = 0.01 $a_{NF,P}, a_{D,P}$ = 0.0001 $a_{A,P}$ = 0.001 $a_{G,P}$ = 0.08
$\phi_i(T)$	Lysis rate	d^{-1} (µg g_{peat})$^{-1}$	0.01
$r_i(T)$	Respiration rate	d^{-1}	r_{NF}, r_A = 0.05 r_D = 0.09 r_G = 0.2 r_P = 0.3
$m_i(T)$	Mortality rate	d^{-1}	m_{NF} = 0.05 m_D = 0.01 m_A, m_G, m_P = 0.1
$q_{k,i}$	Elemental content	g g^{-1}	$q_{N,NF}, q_{N,D}$ = 0.05 $q_{N,A}, q_{N,G}$ = 0.03 $q_{N,P}$ = 0.08 q_C = 0.5
$\beta_i(T)$	Burst size	d^{-1} (µg g_{peat})$^{-1}$	β_{NF}, β_D = 0.05 $\beta_A, \beta_G, \beta_P$ = 0.03
α_k	Inflow rate	µg g_{peat}^{-1} d^{-1}	α_{N_I} = 6 α_{C_I} = 100 α_{C_O} = 30
δ_k	Outflow rate	d^{-1}	$\delta_{N_I}, \delta_{C_I}, \delta_{C_O}$ = 0.01

References

1. Schoolfield, R. M., Sharpe, P. J. H. & Magnuson, C. E. Non-linear regression of biological temperature-dependent rate models based on absolute reaction-rate theory. *J. Theor. Biol.* **88**, 719–731 (1981).
2. Brown, J. H., Gillooly, J. F., Allen, A. P., Savage, V. M. & West, G. B. Toward a Metabolic Theory of Ecology. *Ecology* **85**, 1771–1789 (2004).
3. Allen, A. P., Gillooly, J. F. & Brown, J. H. Linking the global carbon cycle to individual metabolism. *Funct. Ecol.* **19**, 202–213 (2005).
4. Savage, V. M., Gilloly, J. F., Brown, J. H. & Charnov, E. L. Effects of body size and temperature on population growth. *Am. Nat.* **163**, 429–441 (2004).
5. Dell, A. I., Pawar, S. & Savage, V. M. Systematic variation in the temperature dependence of physiological and ecological traits. *Proc. Natl. Acad. Sci.* **108**, 10591–10596 (2011).
6. Mathias, C. B., Kirschner, A. & Velimirov, B. Seasonal variations of virus abundance and viral control of the bacterial production in a backwater system of the danube river. *Appl. Environ. Microbiol.* **61**, 3734–3740 (1995).
7. Nagasaki, K. & Yamaguchi, M. Effect of temperature on the algicidal activity and the stability of HaV (Heterosigma akashiwo virus). *Aquat. Microb. Ecol.* **15**, 211–216 (1998).
8. Wells, L. E. & Deming, J. W. Effects of temperature, salinity and clay particles on inactivation and decay of cold-active marine Bacteriophage 9A. *Aquat. Microb. Ecol.* **45**, 31–39 (2006).
9. Wei, W., Zhang, R., Peng, L., Liang, Y. & Jiao, N. Effects of temperature and photosynthetically active radiation on virioplankton decay in the western Pacific Ocean. *Sci. Rep.* **8**, 1525 (2018).
10. Mojica, K. D. A. & Brussaard, C. P. D. Factors affecting virus dynamics and microbial host-virus interactions in marine environments. *FEMS Microbiol. Ecol.* **89**, 495–515 (2014).
11. Seeley, N. D. & Primrose, S. B. Y. 1980. The Effect of Temperature on the Ecology of Aquatic Bacteriophages. *J. Gen. Virol.* **46**, 87–95 (1980).

12. Hadas, H., Einav, M., Fishov, I. & Zaritsky, A. Bacteriophage T4 development depends on the physiology of its host Escherichia coli. *Microbiol. Read. Engl.* **143 (Pt 1)**, 179–185 (1997).
13. Kendrick, B. J. *et al.* Temperature-Induced Viral Resistance in Emiliania huxleyi (Prymnesiophyceae). *PLOS ONE* **9**, e112134 (2014).
14. Tomaru, Y., Kimura, K. & Yamaguchi, H. Temperature alters algicidal activity of DNA and RNA viruses infecting Chaetoceros tenuissimus. *Aquat. Microb. Ecol.* **73**, 171–183 (2014).
15. Krueger, A. P. & Fong, J. THE RELATIONSHIP BETWEEN BACTERIAL GROWTH AND PHAGE PRODUCTION. *J. Gen. Physiol.* **21**, 137–150 (1937).
16. Ellis, E. L. & Delbrück, M. THE GROWTH OF BACTERIOPHAGE. *J. Gen. Physiol.* **22**, 365–384 (1939).
17. Demory, D. *et al.* Temperature is a key factor in Micromonas-virus interactions. *ISME J.* **11**, 601–612 (2017).
18. Maat, D. S. *et al.* Characterization and Temperature Dependence of Arctic Micromonas polaris Viruses. *Viruses* **9**, 134 (2017).
19. Piedade, G. J., Wesdorp, E. M., Montenegro-Borbolla, E., Maat, D. S. & Brussaard, C. P. D. Influence of Irradiance and Temperature on the Virus MpoV-45T Infecting the Arctic Picophytoplankter Micromonas polaris. *Viruses* **10**, 676 (2018).
20. Payet, J. & Suttle, C. Physical and biological correlates of virus dynamics in the southern Beaufort Sea and Amundsen Gulf. *J Mar Syst* **74**, (2007).
21. Colombet, J. *et al.* Seasonal Depth-Related Gradients in Virioplankton: Standing Stock and Relationships with Microbial Communities in Lake Pavin (France). *Microb. Ecol.* **58**, 728–736 (2009).
22. Danovaro, R. *et al.* Marine viruses and global climate change. *FEMS Microbiol. Rev.* **35**, 993–1034 (2011).

23. Nakayama, N., Okumura, M., Inoue, K., Asakawa, S. & Kimura, M. Seasonal variations in the abundance of virus-like particles and bacteria in the floodwater of a Japanese paddy field. *Soil Sci. Plant Nutr.* **53**, 420–429 (2007).
24. Mojica, K. D. A., Huisman, J., Wilhelm, S. W. & Brussaard, C. P. D. Latitudinal variation in virus-induced mortality of phytoplankton across the North Atlantic Ocean. *ISME J.* **10**, 500–513 (2016).
25. Roy, K. *et al.* Temporal Dynamics of Soil Virus and Bacterial Populations in Agricultural and Early Plant Successional Soils. *Front. Microbiol.* **11**, 1494 (2020).
26. Williamson, K. E., Fuhrmann, J. J., Wommack, K. E. & Radosevich, M. Viruses in Soil Ecosystems: An Unknown Quantity Within an Unexplored Territory. *Annu. Rev. Virol.* **4**, 201–219 (2017).