Supporting Information - Model Equations

Våge et al. 'Linking internal and external bacterial community control gives mechanistic framework for pelagic virus-to-bacteria ratios'

For a summary of the parameters with descriptions, see Table 1 in article. For a detailed analysis of the dynamic version of the plankton functional type (PFT) food web model, see Thingstad et al (2007). For a summary of the dynamic equations for the PFT food web model, see SI 8. For a detailed analysis of the virus-host model resolving species and strains in a chemostat setting at steady state, see Thingstad et al (2014). Simulations of the evolutionary dynamics of virus-host interactions discussed here go beyond the scope of this study but are subject to ongoing work.

SI 1 - Steady states for PFT food web model including Eq. 1a and 1b

Assuming food intake to be proportional to food concentration and growth being mineral nutrient \( S_m \) limited, the steady state requirement of \( Growth = Loss \) for microbial variables of the PFT food web model (Fig. 1A) gives (from Thingstad et al 2007):

For total bacterial biomass \( B_T \):

\[
\alpha_B S_m B_T = \alpha_H B_T H, \tag{Eq. S1}
\]

where \( \alpha_B \) is the affinity of heterotrophic bacteria for the mineral nutrient \( S_m \) and \( \alpha_H \) is the clearance rate of heterotrophic flagellates \( H \) for bacteria.

For autotrophic flagellates \( A \):

\[
\alpha_A S_m A = \alpha_C AC, \tag{Eq. S2}
\]

where \( \alpha_A \) is the affinity of autotrophic flagellates \( A \) for \( S_m \) and \( \alpha_C \) is the clearance rate of ciliates \( C \) for auto- and heterotrophic flagellates.

For heterotrophic flagellates \( H \):

\[
Y_H \alpha_H B_T H = \alpha_C HC, \tag{Eq. S3}
\]

where \( Y_H \) is the yield from heterotrophic flagellate predation on bacteria.

As long as heterotrophic flagellates are present \( (H > 0) \), bacterial biomass is proportional to the biomass of ciliates \( C \) (from Eq. S3), giving Eqn. 1a in article:

\[
B_T = \frac{\alpha_C C}{Y_H \alpha_H} \tag{Eq. 1a}
\]

As long as autotrophic flagellates are present \( (A > 0) \), ciliates and free mineral nutrients are proportional (from Eq. S2), giving Eqn. 1b in article:
\[ S_m = \frac{\alpha_C C}{\alpha_A} \]  
(Eq. 1b)

For completeness, the equations describing the remaining microbial state variables in the food web model at steady state and the resulting properties when all osmotrophs are mineral nutrient \((S_m)\) limited are given below (for summary of parameters, see Table 1 in article).

As long as bacteria are present \((B > 0)\), heterotrophic flagellates and free mineral nutrients are proportional (from Eq. S1):

\[ S_m = \frac{\alpha_H H}{\alpha_B} \]  
(Eq. S4)

For diatoms \(D\), the steady state requirement of \(Growth = Loss\) in the food web model (Fig. 1A) when food intake is assumed to be proportional to food concentration gives (from Thingstad et al 2007):

\[ \alpha_D S_m D = \alpha_M DM, \]  
(Eq. S5)

where \(\alpha_D\) is the diatom affinity for the mineral nutrient \(S_m\), \(\alpha_M\) is the mesozooplankton clearance rate for diatoms and \(M\) is the mesozooplankton biomass.

For ciliates \(C\), the steady state requirement gives:

\[ Y_C \alpha_C (H + A)C = \tau \alpha_M CM, \]  
(Eq. S6)

where \(Y_C\) is the ciliate yield on auto- \((A)\) and heterotrophic \((H)\) flagellates, \(\alpha_C\) is the ciliate clearance rate for \(A\) and \(H\), \(\tau\) is the selectivity factor of mesozooplankton \((M)\) for ciliates relative to diatoms.

As long as diatoms are present \((D > 0)\), Eq. S5 gives:

\[ S_m = \frac{\alpha_M M}{\alpha_D} \]  
(Eq. S7)

As long as ciliates are present \((C > 0)\), the sum of flagellates \((H + A)\) is proportional to ciliate loss rate \(\tau \alpha_M M\) (from Eq. S6):

\[ H + A = \frac{\tau \alpha_M M}{Y_C \alpha_C} \]  
(Eq. S8)

Inserting Eq. S7 into Eq. S4 and inserting this into Eq. S8 gives:

\[ A = \left[ \frac{\tau}{Y_C \alpha_C} - \frac{\alpha_B}{\alpha_H \alpha_D} \right] \alpha_M M \]  
(Eq. S9)

As long as diatoms are present and all osmotrophs are mineral nutrient \((S_m)\) limited, all state variables \(S_m, B_T, A, H\) and \(C\) are hence proportional to \(M\). Bacterial production \((BP_{S_m})\) therefore becomes proportional to the 2nd power of \(M\) (from inserting Eq. S7 and Eq. 1b into Eq. 1a):
\[ BP_{Sm} = \alpha_B S_m B_T = Y_H^{-1} \frac{\alpha_B \alpha_A}{\alpha_H \alpha_D} \alpha_M^2 M^2 \]  

(Eq. S10)

**SI 2 - Uptake kinetics based on affinity**

Enzymatic rates following Michaelis-Menten kinetics are traditionally expressed as

\[ v = \frac{V_{\text{max}}[S]}{K + [S]} = \frac{v_{\text{max}}}{K} \left( \frac{[S]}{1 + \frac{[S]}{K}} \right) \]  

(Eq. S11)

where \( v_{\text{max}} \) is the maximum rate, \( K \) is the half-saturation constant, and \( S \) is the limiting substrate. For limiting substrate concentrations being small (\( S \to 0 \)), the rate can be approximated as

\[ v \approx \frac{v_{\text{max}}}{K} [S] \]  

(Eq. S12)

In our model, specific growth rates (\( \mu \)) follow Michaelis-Menten kinetics. Under substrate limitation (i.e. low mineral nutrient or dissolved carbon concentrations), the slope of the growth rate at low resource concentration, which is described by the nutrient affinity \( \alpha \), can be used as a linear approximation to the uptake rates. Hence, at resource limitation, \( \alpha \) corresponds to \( \frac{v_{\text{max}}}{K} \) (Eq. S12) in the Michaelis-Menten kinetics, and the specific growth rates in our model get expressed as (from Eq. S11):

\[ \mu = \frac{\alpha[S]}{1 + \frac{\alpha[S]}{\mu_{\text{max}}}} \]  

(Eq. S13)

**SI 3 - Selective grazing**

The presented model can easily be extended to include selective flagellate grazing on bacteria by introducing a selectivity factor \( x \gamma \), which reduces the maximum specific clearance rate of heterotrophic flagellates (\( \alpha_H \)) on a particular bacterial species \( x \). Strains belonging to bacterial species \( x \) would then have a loss rate \( x \delta \) of

\[ x \delta = x \gamma \alpha_H H \]  

(Eq. S14)

The consequence in our model would be that the minimum growth rates required to establish at steady state (i.e. the horizontal lines in Fig. 2A and 2C) would differ between different bacterial species.

**SI 4 - Number of strains in bacterial species \( x \)**

The minimum growth rate that the last established strain of any bacterial species needs to maintain at steady state for a given heterotrophic flagellate abundance (\( \mu_{\text{min}}(H) \)) equals the loss rate through flagellate grazing (\( \delta_B \)):

\[ \delta_B = \alpha_H H = \mu_{\text{min}}(H) \]  

(Eq. 3)
Furthermore, following Eq. S13 and using the cost of resistance function for $\mu^{\text{max}}$ of mutant strains ($\mu^{\text{max}} = \mu^{\text{max}}_0 \nu^i$), the growth rate of the last established strain $n$ of species $x$ at a given ciliate abundance ($\mu_{\text{min}}(H, C)$) equals

$$
\mu_{\text{min}}(H, C) = \frac{x\alpha_0 S_m(C)}{1 + x\alpha_0 S_m(C) \nu^n} 
$$

(Eq. S15)

Solving this for $n$ gives, after some rearrangement, the number of strains $n$ in species $x$:

$$
x n(C, H) = 1 + \log \left( \frac{x\alpha_0 S_m(C) \mu_{\text{min}}}{(x\alpha_0 S_m(C) - \mu_{\text{min}}(H)) \nu^0} \right) 
$$

(Eq. S16)

SI 5 - Upper triangular host-virus infection matrix

As described in Thingstad et al (2014), nested infection structures between host strains and their viruses arrive when a host mutation leading to a new, resistant host strain $B_i$ occurs in host strain $B_{i-1}$ that is only susceptible to the most recently evolved virus $V_{i-1}$, and when the subsequent virus $V_i$ is most likely to arise from a mutation in virus $V_{i-1}$ that already has the ability to attack $B_i$’s closest relative, $B_{i-1}$. These dynamics lead to the most recently evolved viruses being generalists, able to infect most host strains including the more evolved and resistant types, whereas the more ancient viruses are specialists, infecting few, ancient host strains only that generally have low viral resistance (Fig. 1C). Nested infection structures may be prevalent in natural host-virus systems and can arrive from antagonistic coevolution between host strains and their viruses (Flores et al., 2014).

In our model, a possible fractional reduction in effective adsorption coefficient ($\beta_{ij}$) for each mutation leading to a new virus-host pair (i.e. increasing step in $j$) is modeled by a parameter $\rho$. This means that viruses evolving to infect increasingly defensive host strains have a cost in the form of a reduced effective adsorption coefficient. A possible fractional reduction in $\beta_{ij}$ for previously established virus-host pairs (i.e. each decreasing step in $i$ away from $j$) is modeled by a parameter $\sigma$. This means that new virus mutants lose some of their ability to infect previously established hosts and $\sigma$ thus referred to as a “memory” coefficient. This gives the following effective adsorption coefficient for virus-host pairs $ij$, where $\beta_0$ is the effective adsorption coefficient of the original virus on the original parent strain:

$$
\beta_{ij} = \beta_0 \sigma^{j-i} \rho^i 
$$

(Eq. S17)

An example of a resulting nested infection network is shown below for three host strains (representing columns) and three viruses (representing rows). The nested structure is characterized by the upper triangular $\beta$ matrix:

$$
\beta = \beta_0 \begin{bmatrix}
1 & \sigma \rho & \sigma^2 \rho^2 \\
0 & \rho & \sigma \rho^2 \\
0 & 0 & \rho^2 
\end{bmatrix}
$$

For a memory coefficient $\sigma = 0$, this nested infection model contains the simple one-virus-to-one-host infection network, where all off-diagonal elements are zero.
SI 6 - Number of individuals in strains and total abundance of bacterial species $x$

The steady-state abundance for the undefended parental strain of bacterial species $x$ ($x B_0$), when controlled by virus $x V_0$, is given by the requirement that production of viruses $x V_0$ through host lysis (left hand side in Eq. S18) equals the sum of loss by infection and decay (right hand side in Eq. S18):

$$m_0 x \beta_0 x V_0 x B_0 = x \beta_0 x V_0 x B_0 + \delta_{V_0} x V_0.$$  \hspace{1cm} (Eq. S18)

where $m_0$ is the burst size of $x V_0$ in $x B_0$, $x \beta_0$ is the effective adsorption coefficient of $x V_0$ and $\delta_{V_0}$ is the decay rate of $x V_0$.

This can be solved for $x B_0$ to give the abundance of the parent strain of species $x$:

$$x B_0 = \frac{\delta_{V_0}}{(m_0 - 1) x \beta_0}.$$  \hspace{1cm} (Eq. S19)

In analogy to Eq. S18, host abundance of any subsequent host strain of bacterial species $x$ can be derived from the requirement that all virus populations are produced at a rate balancing the sum of losses by infection and decay. The equilibrium condition for virus $x V_j$ thus gives (from Eq. S18):

$$\delta_{V_j} x V_j = \sum_{i=0}^{j} (m_{ij} - 1) x \beta_{ij} x B_i x V_j.$$  \hspace{1cm} (Eq. S20)

Assuming all viral strains to have the same decay rate $\delta_{V_j} = \delta_{V}$ and assuming the same burst size in all host strains $m_{ij} = m_0$, this can be solved for the vector $x B$ of bacterial host strains belonging to species $x$ to give

$$x B = \frac{\delta_{V}}{m_0 - 1} (x \beta^T)^{-1} U,$$  \hspace{1cm} (Eq. S21)

where $U$ is a column vector with all elements equal to 1. Combining Eq. S19 and Eq. S21 for the example of 3 strains thus gives

$$\begin{bmatrix} \delta_{V} \\ (m_0 - 1) x \beta_0 \end{bmatrix} \begin{bmatrix} 1 \\ 1 \\ 1 \end{bmatrix} = \begin{bmatrix} 1 & 0 & 0 \\ x \sigma x \rho & x \rho & 0 \\ x \sigma x \rho^2 & x \sigma x \rho^2 & x \rho^2 \end{bmatrix} \begin{bmatrix} x B_0 \\ x B_1 \\ x B_2 \end{bmatrix}.$$  \hspace{1cm} (Eq. S22)

This is directly solvable by forward substitution. Starting from the first line, this gives again the abundance of the parent strain of species $x$ (Eq. S19):

$$x B_0 = \frac{\delta_{V}}{(m_0 - 1) x \beta_0}.$$  \hspace{1cm} (Eq. S19)

The next line gives

$$x \sigma x \rho x B_0 + x \rho x B_1 = \frac{\delta_{V}}{(m_0 - 1) x \beta_0},$$  \hspace{1cm} (Eq. S23)

which by insertion for $x B_0$ gives
\[
x B_1 = \frac{\delta_V}{(m_0 - 1)\beta_0} (1 - x \sigma x \rho) x \rho^{-1}.
\]  
(Eq. S24)

Line 3 then gives
\[
x \sigma^2 x \rho^2 x B_0 + x \sigma x \rho^2 x B_1 + x \rho^2 x B_2 = \frac{\delta_V}{(m_0 - 1)\beta_0},
\]  
(Eq. S25)

which by insertion for \(x B_0\) and \(x B_1\) gives
\[
x B_2 = \frac{\delta_V}{(m_0 - 1)\beta_0} (1 - x \sigma x \rho) x \rho^{-2}
\]  
(Eq. S26)

In the general case for \(i > 0\), this gives abundance of strain \(i\) in species \(x\):
\[
x B_i = \frac{\delta_V}{(m_0 - 1)\beta_0} (1 - x \sigma x \rho) x \rho^{-i} = x B_0 (1 - x \sigma x \rho) x \rho^{-i}
\]  
(Eq. S27)

Summing up the \(n\) strains of species \(x\) using Eq. S16 and Eq. S27 to get the total abundance of species \(x\) gives
\[
x B_T = x B_0 + \sum_{i=1}^{n-1} x B_i = x B_0 \left( 1 + (1 - x \sigma x \rho) \sum_{i=1}^{n-1} x \rho^{-i} \right)
\]  
(Eq. S28)

or
\[
\left( \frac{x B_T}{x B_0} - 1 \right) = \frac{1 - x \sigma x \rho}{x \rho} \sum_{i=1}^{n-1} x \rho^{-i+1}.
\]  
(Eq. S29)

Since
\[
\sum_{i=1}^{n-1} x \rho^{-i+1} = (x \rho^{-1})^{i-1} = \frac{1 - (x \rho^{-1})^{n-1}}{1 - x \rho^{-1}},
\]  
(Eq. S30)

this gives
\[
\left( \frac{x B_T}{x B_0} - 1 \right) = \frac{1 - x \sigma x \rho}{x \rho} \frac{1 - (x \rho^{-1})^{n-1}}{1 - x \rho^{-1}}.
\]  
(Eq. S31)

The total abundance in species \(x\) is thus after some rearrangement given by:
\[
x B_T = x B_0 \left[ 1 + \frac{(1 - x \rho x \sigma)}{x \rho} \frac{1 - (x \rho^{-1})^{n-1}}{1 - x \rho^{-1}} \right] = x B_0 \left[ 1 + \frac{(1 - x \rho x \sigma)}{1 - x \rho} (x \rho^{-(n-1)} - 1) \right],
\]  
(Eq. 4)

where \(n\) is the number of strains established in species \(x\).
SI 7 - Virus abundance belonging to species x

Abundance in the viral strains $xV_j$ belonging to species $x$ can be calculated from the requirement that each host strain $xB_i$ has a growth rate ($x\mu(S)$) balancing the sum of losses due to flagellate grazing ($\delta_B$) and its loss to viral lysis. The difference $xc_i(S) = x\mu(S) - \delta_B = x\mu_i(S) - \alpha_HH$ thus needs to be equal to the sum of losses over the $k = i...n - 1$ viruses infecting $xB_i$: 

$$xc_i(S) = \sum_{k=1}^{n-1} x\beta_{ij}xV_k, \quad i = 0...n - 1$$  \hspace{1cm} (Eq. S32)

Solving this for the vector $xV$ gives in matrix notation:

$$xV = x\beta^{-1}xc$$  \hspace{1cm} (Eq. S33)

Using backward substitution, the n-dimensional matrix equation $\beta xV = xc$, where $\beta$ is the upper triangular infection matrix, can be solved starting with the last row (using Eq. S17):

$$x\beta_0x^0-1xV_{n-1} = xc_{n-1}(S).$$  \hspace{1cm} (Eq. S34)

The next-to-last row gives

$$x\beta_0(x\rho^{n-2}xV_{n-2} + x\sigma_xx\rho^{n-1}xV_{n-1}) = xc_{n-2}(S),$$  \hspace{1cm} (Eq. S35)

giving

$$xV_{n-2} = \frac{xc_{n-2}(S) - x\sigma_xxc_{n-1}(S)}{x\beta_0x\rho^{n-2}}.$$  \hspace{1cm} (Eq. S36)

Continuing, this gives the general formula for abundance of viral strain $k$ of species $x$

$$xV_k = \frac{xc_k(S) - x\sigma_xxc_{k+1}(S)}{x\beta_0x\rho^k}, \quad k = 0...n - 1.$$  \hspace{1cm} (Eq. S37)

Summing over all $n$ strains in species $x$ gives after some rearrangement:

$$xV_T = x\beta_0^{-1}[xc_0(S) + \sum_{k=1}^{n-1} \frac{xc_k(S) - x\sigma_xxc_{k+1}(S)}{\rho^k}] = x\beta_0^{-1}[xc_0(S) + (1 - x\sigma_x)\sum_{k=1}^{n-1} \frac{xc_k(S)}{\rho^k}].$$  \hspace{1cm} (Eq 5)

SI 8 - Dynamic equations for PFT food web model (from Thingstad et al 2007)

The steady state requirements for the PFT model used to derive the relationships discussed here are based on the 'minimum' microbial food web model by Thingstad et al 2007. Although the differential equations for this model used to simulate mesocosm dynamics in Thingstad et al 2007 are not used in the present analysis, they are given to the interested reader for completeness. Assuming food intake to be proportional to food concentration gives:
For heterotrophic bacteria B:
\[ \frac{dB}{dt} = \alpha_B S_m B - \alpha_H BH \]  
(Eq. S38)
where \( \alpha_B \) is the nutrient affinity of bacteria on the limiting mineral nutrient and other symbols and parameters are as in Table 1.

For autotrophic flagellates A:
\[ \frac{dA}{dt} = \alpha_A S_m A - \alpha_C AC \]  
(Eq. S39)
where symbols and parameters are as in Table 1.

For heterotrophic flagellates H:
\[ \frac{dH}{dt} = Y_H \alpha_H BH - \alpha_C HC \]  
(Eq. S40)
where symbols and parameters are as in Table 1.

For ciliates C:
\[ \frac{dC}{dt} = Y_C \alpha_C \left( D + \tau C \right) C - \tau \alpha_CM \]  
(Eq. S41)
where \( Y_C \) is the yield from ciliate predation on auto- and heterotrophic flagellates, \( \tau \) is the mesozooplankton selectivity factor for ciliates relative to diatoms, \( \alpha_M \) is the clearance rate of mesozooplankton for diatoms and other symbols and parameters are as in Table 1.

For diatoms D:
\[ \frac{dD}{dt} = \alpha_D S_m D - \alpha_M DM \]  
(Eq. S42)
where \( \alpha_D \) is the nutrient affinity of diatoms for the limiting mineral nutrient \( S_m \), \( \alpha_M \) is the clearance rate of mesozooplankton for diatoms and other symbols and parameters are as in Table 1.

For mesozooplankton M:
\[ \frac{dM}{dt} = Y_M \alpha_M \left( D + \tau C \right) M - \delta_M M \]  
(Eq. S43)
where \( Y_M \) is the mesozooplankton yield on ciliates and diatoms, \( \alpha_M \) is the clearance rate of mesozooplankton for diatoms, \( \tau \) is the mesozooplankton selectivity factor for ciliates relative to diatoms, \( \delta_M \) is the mortality rate of mesozooplankton and other symbols and parameters are as in Table 1.

For the limiting mineral nutrient \( S_m \), assuming that non-assimilated biomass from predation gets immediately remineralized:
\[ \frac{dS_m}{dt} = (1 - Y_H)\alpha_H BH + (1 - Y_C)\alpha_C (A + H) C + (1 - Y_M)\alpha_M (D + \tau C) M - \alpha_B S_m B - \alpha_A S_m A - \alpha_D S_m D + E_{S_m} \]  
(Eq. S44)
where $Y_C$ is the ciliate yield on auto- and heterotrophic flagellates, $Y_M$ is the mesozooplankton yield on ciliates and diatoms, $\alpha_M$ is the clearance rate of mesozooplankton for diatoms, $\tau$ is the mesozooplankton selectivity factor for ciliates relative to diatoms, $E_{Sm}$ is the experimental input rate of $S_m$ in the mesocosm treatment and other symbols and parameters are as in Table 1.

References

Thingstad TF, Havskum H, Zweifel UL, Berdalet E, Sala MM, Peters F et al (2007). Ability of a "minimum" microbial food web model to reproduce response patterns observed in mesocosms manipulated with N and P, glucose, and Si. *J Mar Syst* **64**:15-34.

Thingstad TF, Våge S, Storesund JE, Sandaa R-A, Giske J (2014). A theoretical analysis of how strain-specific viruses can control microbial species diversity. *PNAS USA* **111**:7813-7818.