Evolutionarily Optimal Phage Life-History Traits: Burst Size *vs.* Lysis Time

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Abstract

A single dynamical equation for the virus to microbe ratio (VMR) during log-phase population growth shows that an optimal tradeoff occurs between a short lysis time with low burst size *vs.* a long lysis time with large burst size. The model predicts that interventions lowering phage adsorption by killing free virus and/or limiting their access to bacteria cause the evolution of an increased lysis time and higher burst size until the intervention either drives a virulent phage extinct or, for temperate phage, drives the phage from its lytic phase into its lysogenic phase. The model also predicts the pattern of burst size *vs.* lysis time along environmental gradients in terms of a parameter, Q, that describes the *ratio* of virus productivity to bacterial productivity. High Q environments select for short lysis times and corresponding small burst sizes, and conversely for low Q environments. A correlation between viral burst size and bacterial productivity along environmental gradients in nutrients is interpreted in terms of a gradient in Q.

Introduction

Breitbart (2012) noted that "It is difficult to find a paper in marine viral ecology that does not begin with a statement about the sheer abundance of viruses". The same is true today. Even though abundance estimates for virus have been available for over twenty years, the magnitude still seems incomprehensible. The number of prokaryotes on earth is 4–6 x 10^{30} cells with a production rate of 1.7 x 10^{30} cells/yr (Whitman *et al.* 1998). The number of virus is about ten times the number of prokaryotes (Wommack and Colwell 2000) implying that the number of virus is on the order of 10^{31}. In comparison, the number of grains of sand on all the earth's beaches is only 4 x 10^{20} and the number of stars in the universe a meager 2 x 10^{19} (Plait 2025).

Moreover, marine phage infect approximately 10^{23} bacteria every second, removing 20%–40% of the standing stock of marine bacteria each day (Suttle 2007). They exert top-down control of marine microbial abundance comparable to that of zooplankton grazing (Breitbart *et al.* 2018). Reports of viral abundance also appear for soil (Srinivasiah *et al.* 2008, Williamson *et al.* 2017) and the human gut (Townsend *et al.* 2021, the "phageome").

Furthermore, virus are ancient with differences so great among them that the taxonomic rank of realm has been coined to occupy the top of 15 ranks in total that subsume multiple kingdoms and lower taxa for various viruses (ICTV 2020, Koonin *et al.* 2020, 2021, Harris and Hill 2021). Marine phage represent 94% of the nucleic-acid-containing particles in the oceans (Breitbart *et al.* 2018). Hence, phage represent an enormous number of evolutionary agents whose phenotypes are shaped by natural selection.

What then are the phage phenotypes that natural selection is shaping? Of the many traits phage possess, including their morphology and mechanisms of inheritance and replication, the life-history traits of the lytic/lysogeny switch, the burst size and the latency period are of particular interest because these traits underly the reproductive rate of phages. Ideally, research might offer an evolutionary explanation for such traits across various habitats. A previous paper introduces a model that predicts conditions for the lytic/lysogenic switch to occur and compares the model's predictions with ecogeographic patterns of the ratio of lytic to lysogenic life cycles (Roughgarden 2014). However, information on possible ecogeographic patterns in burst size and latency across habitats is limited. Both traits vary considerably across and within habitats, often in accordance with transient environmental conditions such as temperature (Wommack and Colwell 2000, Weinbauer 2004, Greenrod *et al.* 2024). Parada *et al.* (2006) show a correlation between burst size and bacterial productivity in coastal waters, but do not present data on latency times. Ranasinghe (2019) offers an extended review of burst size and latency across various categories of virus, but does not report the habitats where the virus live. This article extends the model in (Roughgarden 2014) to explain burst size and latency and predicts possible ecogeographic patterns whose discovery would test the model.

Interventions to control microbes with antibiotics have produced antibiotic resistance (Martinez *et al.* 2008, Davies and Davies 2010). These and other interventions might affect virus as well. The model in this article also predicts the evolutionary consequences for phage of interventions that lower the overall absorption rate by virus onto bacteria.

This article explains burst size and latency as an evolutionarily optimal balance between reproducing early to benefit from geometric growth versus reproducing late to benefit from the time to produce a large burst size. The study of an evolutionary tradeoff between the benefits of early and late lysis time enjoys a long history (Abedon 1989, Wang *et al*. 1996, Abedon *et al*. 2001, Abedon *et al*. 2003, Bull *et al*. 2004, Bull 2006, Bull *et al*. 2006, Wang 2006, Heineman and Bull 2007, Bull *et al*. 2011, and Kannoly *et al*. 2022). The models developed in this literature are generally based on a system of three delay-differential equations with interactions represented as mass action terms, that is, as quadratic terms such as $(N_1 \times N_2)$ to represent an interaction between species-1 and species-2, and also include an assumption of density dependence. The analysis of these equations generally features solving for the equilibrium abundance of uninfected bacteria, infected bacteria and free phage.

This article differs from the preceding literature in being based on a new model that is defined in discrete time and features viral colonization of bacteria as a Poisson process rather than as a product of viral and bacterial abundances. The model here pertains to a virus-bacteria system in the log phase, where both populations grow exponentially rather than exist at equilibrium in a stationary phase. The model's analysis rests on a single dynamical equation for the virus to microbe ratio (VMR) rather than a system of three coupled equations for uninfected bacteria, infected bacteria and free phage. Thus, the situation in which the optimal life history evolves is envisioned differently here as compared to the earlier literature—stationary phase versus log phase and the mathematical approach is different. The Discussion section features a more detailed comparison of the differing intuition behind the prediction of an optimal life history found in earlier models compared to the present model.

Two experimental studies (Wang 2006, Kannoly *et al*. 2022) demonstrate that an optimal tradeoff between lysis time and burst size exists. Hence, the reality of an optimal tradeoff can now be taken as given. Instead, this article concerns how to predict *what* that optimal tradeoff is. In nature, phage may not possess the optimal balance between latency and burst size for their present circumstances, but knowing the optimal balance in those circumstances reveals the direction toward which evolutionary change will occur.

The article proceeds in five stages: The first stage defines the basic population-dynamic model. The second stage extends the population-dynamic model to predict the optimal lysis-time/burst size tradeoff. The third stage compares the predicted the optimal lysis-time/burst size tradeoff to data from experiments with λ-phage and *Escherichia coli* (Wang 2006). The fourth stage predicts the evolutionary impact of interventions that affect the absorption rate. The fifth stage develops predictions for possible ecogeographic patterns.

Model

Population Dynamics of Phage and Bacteria

The phage/bacteria population-dynamic model used here is defined in discrete time with the time step being the viral lysis time period (Roughgarden 2024). A time step begins with the state variables, $N(t)$ and $V(t)$ for the number of bacteria and free virus particles (virions) in their environmental source pools at time t. Then the virus infect the uninfected bacteria with Poisson sampling such that some bacteria remain uninfected, some have only one infection, some have two infections *etc*. (Ellis and Delbrück 1939).

The Poisson distribution describing the infection process has density parameter, $\mu(t) \equiv a(v(t))\, v(t)$ where $v(t) \equiv V(t)/N(t)$ is the ratio of virus to bacteria in their source pools (the environmental virus to microbe ratio, VMR). The $a(v(t))$ is the adsorption function—it refers to the probability that a viral particle comes in contact with a bacterium, binds to surface molecules, and then is incorporated into the bacterial cell. adsorption is a function of $v(t)$ because the number binding sites on the surface of the bacterium is finite and the sites may become saturated. Thus, $a(v(t))$ includes information about the binding affinity of virus particles to receptors in the bacterial surface and also about the number of binding sites. Furthermore, $a(v(t))$ also includes information about the accessibility of the bacterium to virus particles. For a given ratio of virus to bacteria, $v(t)$, if the environment is such that virus are prevented from reaching the bacteria, then $a(v(t))$ is low compared to $a(v(t))$ in environments that allow ready access of the virus to the bacteria. Environments may differ in how viscous the media is, or for airborne virus particles, whether air filters are installed.

According to a Poisson density function, the fraction of uninfected bacteria that remain uninfected, $P_0(\mu(t))$, is $e^{-\mu(t)}$ and the fraction of bacteria colonized by m virus, $P_m(\mu(t))$, is $\frac{e^{-\mu(t)}\mu(t)^m}{m!}$ for $m \geq 0$ where $\mu(t) = a(v(t))\, v(t)$. The m indexes the number of virus per bacterium. The fraction of bacteria that are infected by one or more virus is $1 - P_0(\mu(t))$.

The uninfected bacteria population increases by a geometric factor of increase per time step, R. Necessarily, $R > 1$, for the bacteria to be able to increase in the absence of the virus. Accordingly, the uninfected bacteria resupply the bacterial source pool as

$$N(t+1) = R\, P_0(\mu(t))\, N(t) \tag{1}$$

where $P_0(\mu(t))$ is the Poisson probability that a bacterium is uninfected. Thus, the population size of bacteria at time $t+1$ equals R times the number of bacteria that are not infected. Meanwhile, the virus in the infected bacteria produce new virus particles as

$$V(t+1) = \Big(b(1)\, P_1(\mu(t)) + b(2) P_2(\mu(t)) + \ldots\Big)\, N(t) \tag{2}$$

where $b(m)$ is the burst size from a bacterium infected with m virus particles (the multiplicity of infection, MOI) and $P_m(\mu(t))$ is the Poisson probability that a bacterium is infected by m virus particles. Thus, the population size of virions at time $t+1$ equals the number of virions produced from bacteria infected by

one phage, plus those produced from bacteria infected by two phage, plus those from bacteria infected with three phage, and so forth. Together, Eqs. 1 and 2 constitute a dynamical system for phage and bacteria.

The adsorption is taken as density-dependent and the burst size is assumed to depend linearly on the number of virus infecting a bacterium

$$a(v(t)) = a_o\,(1 - \frac{v(t)}{\kappa}) \tag{3}$$

where κ is the saturation density—the viral density at which all the binding sites per bacterium are occupied and a_o is the maximal probability of binding when all the binding sites are available, given the environmental conditions.

Several choices are available for the burst size from a bacterium as a function of the number of virus in it, $b(m)$. The burst size might increase linearly with m so that $b(m) = b_o\,m$ where b_o is the contribution to the overall burst made by a single viral particle (Patel and Rao 1984, Figures 1–2). This case is for no density dependence in $b(m)$. Another possibility is for $b(m)$ to be a concave increasing function of m (Gadagkar and Gopinathan 1980, Figures 4–5). In this case, the burst-size function might be taken as $b(m) = b_o\,m^{\alpha}$ where α is an exponent between 0 and 1, say 1/2. This case shows limited density dependence in $b(m)$. Still a third possibility is that $b(m)$ is a constant, $b(m) = b_o$, indicating that the burst size does not increase for m beyond the initial infection. A constant $b(m)$ indicates strong density dependence. A final possibility is to allow α to be negative, say $\alpha = -1$, to indicate that burst size varies inversely with the multiplicity of infection, indicating extremely strong density dependence as a response to "superinfection" (Brown and Bidle 2014). All these relations between burst size and m may be subsumed by taking $b(m) = b_0\,m^{\alpha}$ with α as 1, 1/2, 0, or -1. The cases of $\alpha = 1$ and $\alpha = 0$ were presented in Roughgarden (2014) and results for other α were noted but not shown. All in all, the evidence shows that density dependence in burst size is weak (*cf.* Gadagkar and Gopinathan 1980, Figs 4–5; Patel and Rao 1984, Figs 1–2). Therefore, the burst size as a function of the multiplicity of infection is taken as

$$b(m) = b_o\,m \tag{4}$$

where b_o is the burst per virus infecting the bacterium. Even if $b(m)$ is not quite linear throughout the range of possible m, the linear form can be viewed as a Taylor approximation in the limit as $m \to 0$. This approximation is appropriate here because the analysis focusses on the conditions for the virus to increase when rare into a bacterial population that is growing in its log phase—in such a circumstance m is small.

Next, identify $v(t+1)$ with $\frac{V(t+1)}{N(t+1)}$ and $v(t)$ with $\frac{V(t)}{N(t)}$. Then doing the sum yields

$$v(t+1) = \frac{b_o}{R}\,\frac{a_o\,(1 - \frac{v(t)}{\kappa})\,v(t)}{e^{-a_o\,(1-\frac{v(t)}{\kappa})}}\,v(t) \tag{5}$$

Equation 5 is a dynamical equation in one variable, $v(t)$.

In the limit, $v(t) \to 0$, the model in Eq. 5, upon suppressing the subscripts, reduces to

$$v(t+1) = \frac{a\,b}{R} v(t) \tag{6}$$

The virus population can infect the bacterial population if it can increase when rare, *ie.*, if $v(t) \approx 0$, then $v(t+1) > v(t)$. Inspection of Eq. 6 shows that if

$$\frac{a\,b}{R} > 1, \tag{7}$$

then the virus can spread into a population of increasing bacteria. Alternatively, for a virulent virus, if $a\,b/R < 1$, the virus goes extinct because the virus cannot keep up with bacterial population growth. But for a temperate virus, if $a\,b/R < 1$, the virus switches to a prophage because its population grows faster as part of the bacterial genome than by bursting new virions each time step.

Optimal Tradeoff Between Burst Size and Lysis Time

This section applies the phage-bacteria population-dynamic model of the previous section to the tradeoff between burst size and lysis time. Wang (2006) experimentally determined a functional relation between burst size and lysis time as

$$\begin{aligned} b &= \beta\,(l-\epsilon) \quad \text{for} \;\; l > \epsilon \\ &= 0 \quad \text{for} \;\; l \leq \epsilon \end{aligned} \tag{8}$$

where l is the lysis time, ϵ is the eclipse, which is the time needed for the viral replication to commence, and β is the phage production rate (also called the phage maturation rate) once viral production has begun. The burst size increases linearly with the lysis time period after the eclipse has transpired.

To find the optimal lysis time period, a maximum time step, L, is stipulated that is longer than the largest feasible lysis time period. Then the model is iterated for the number of periods, l, that fit within L, given the eclipse. For example, suppose the eclipse is 0 and L is 100. If l is also 100, then only one iteration of the model takes place within L, whereas if l is 1, then 100 iterations fit within L, and if l is 25, then four iterations fit within L, and so forth. Thus, the v after the number of iterations that fit within L is

$$v(t+L) = \left(\frac{a}{R}\,\beta\,(l-\epsilon)\right)^{\lfloor \frac{L}{l} \rfloor}\, v(t) \tag{9}$$

for l = (ϵ+1, ϵ+2 ... L) where $\lfloor \frac{L}{l} \rfloor$ indicates the integer part of $\frac{L}{l}$. The exponent in Eq. 9 is the number of iterations of Eq. 6 that fit into L. The optimal l, and corresponding b, is that with the highest viral production after L has elapsed.

Further analysis is aided by taking a continuous approximation in Eq. 9, as though a fractional iteration can fit into L,

$$v(t+L) = \left(\frac{a}{R}\,\beta\,(l-\epsilon)\right)^{\frac{L}{l}}\, v(t) \tag{10}$$

for $l > \epsilon$. Solutions for the optimal lysis time from Eq. 10 can be rounded down to the nearest integer if needed. The expression in parentheses in Eq. 10 is the fitness of a lytic phage over the period, L, as a function of l

$$W_L(l) = \left(\frac{a}{R}\,\beta\,(l-\epsilon)\right)^{\frac{L}{l}} \tag{11}$$

The optimal lysis time, $\hat{l}$, is found by maximizing $W_L(l)$ with respect to l. The corresponding optimal burst size, $\hat{b}$, is found from Eq. 8, evaluated at $l = \hat{l}$.

To find the optimal lysis time, differentiate W_L with respect to l. Then the quantity, $Y(l)$, then emerges, independent of L, identified as

$$Y(l) = \frac{1}{(l-\epsilon)} - \frac{\log\left(\frac{a}{R}\,\beta\,(l-\epsilon)\right)}{l} \tag{12}$$

The optimal lysis time, $\hat{l}$ is the root of $Y(l) = 0$.

Table 1: Parameters

Name	Value	Notes
β	7.7	Wang 2006
ϵ	28	Wang 2006
R	1.035	$e^{2.04/60}$, Wang 2006
a	0.1	Schwartz 1976, Philipson 1983
L	100	(time step = minutes)

For comparison, the fitness over the same time interval of a prophage that has incorporated into the bacterial chromosome and reproduces with the bacteria, w_L, is

$$w_L = R^L \tag{13}$$

The critical lysis time, l_c, at which being lytic becomes a better strategy than being a prophage is found by setting

$$W_L(l) = w_L \tag{14}$$

Predictions

Comparison of Model with Laboratory Data

Table 1 gives the parameters for an illustration. The β and ϵ, in units of min$^{-}1$ and phage/min, respectively, pertain to isogenic λ-phages with different lysis times from Wang (2006). The bacterial intrinsic rate of increase, R, from Wang (2006) for *Escherichia coli* K-12, is given as 2.04/hr—this converts to the value of R in the table in minutes. The adsorption parameter, a, is the product of the probability that a collision between a phage and a bacterial binding site results in a binding (10^{-4}, Philipson 1983) times the number of binding sites per bacterium (10^3, Schwartz 1976) leading to the value in the table. The adsorption parameter can vary considerably depending on environmental conditions affecting the accessibility of the bacteria to the virus and furthermore, the number of binding sites per bacterium varies with nutrient conditions (Schwartz 1976). The value used can be considered a nominal value.

To find the optimal lysis time, using the NSolve[] function in Mathematica with $Y(l)$ in Eq. 12, together with the parameters from Table 1, returns the value,

$$\hat{l} = 45.7 \tag{15}$$

independent of L. The corresponding optimal burst size from Eq. 8 is

$$\hat{b} = 136.5 \tag{16}$$

and the corresponding maximal fitness from Eq. 11 is

$$\widehat{W_{100}} = 281.7 \tag{17}$$

For comparison, the fitness of a prophage from Eq. 13 is

$$w_{100} = 31.2 \tag{18}$$

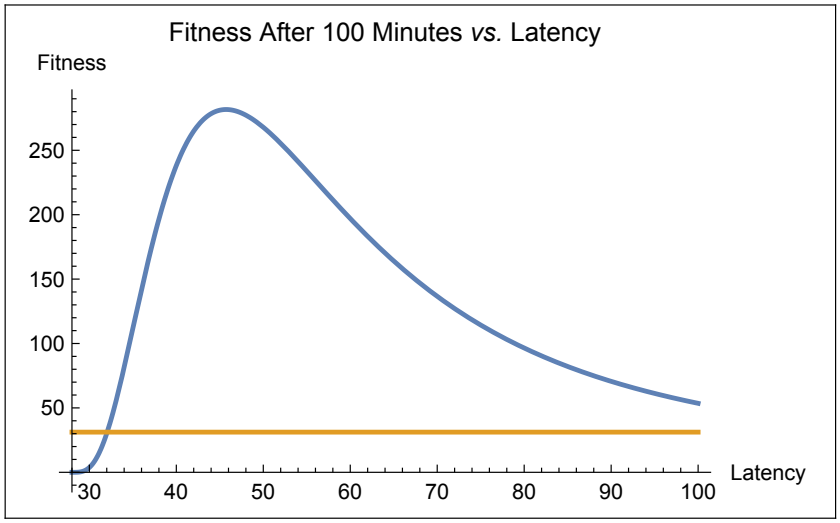

Figure 1: *Fitness of lytic phage after 100 minutes, W_{100}, as a function of the lysis time, l, is shown as a humped-shaped curve starting from the eclipse period of 28 minutes. The optimal lysis time occurs at $\hat{l}$ = 45.7 which yields a maximum lytic fitness, $\widehat{W_{100}}$, of 281.7. Fitness after 100 minutes as a prophage incorporated into bacterial genome, w_{100} = 31.2, is shown as horizontal line. The hump-shaped curve for W_{100} intersects the line at $l_c = 32.0$. Thus, the fitness of a lytic phage exceeds that of a prophage when the lysis time exceeds 32.0. If the lysis time of the phage is between 28 and 32.0, the phage cannot coexist with the bacteria unless it becomes a prophage.*

and the critical lysis time at which being lytic becomes a better strategy than being a prophage works out to be

$$l_c = 32.0 \tag{19}$$

Figure 1 shows the fitness of a lytic phage after a period of 100 minutes as a function of the lysis time, l, based on the parameters in Table 1. For comparison, the fitness over the same time interval of a prophage that has incorporated into the bacterial chromosome and reproduces with the bacteria is shown as a horizontal line. If l is greater than 32.0, then the lytic phase has a higher fitness than the lysogenic phase. If l is between ϵ and l_c, *i.e.* between 28 and 32.0 minutes, then the phage cannot coexist with the bacteria in its lytic phase—the bacteria population grows faster than the viral population, leaving it behind, so to speak. Instead, the phage must lysogenize—incorporate into the bacterial genome to acquire the population growth rate of the bacteria.

Wang (2006) measured the fitness of 11 strains of λ phage whose latencies varied from 28.3 to 63.0 minutes. The study found that the strain whose lysis time was 51.0 minutes had the highest fitness, a value quite close to that predicted in the model here, 45.7, based on the parameters in Table 1. The burst size for the strain with the highest fitness was 170.0, also fairly close to the value predicted here, 136.5. This initial degree of agreement between theory and experiment is likely coincidental because the nominal value of the adsorption parameter, a, in Table 1 was not experimentally determined. Instead, if a is lowered to 0.054, then $\hat{l}$ works out to be 50.9 and $\hat{b}$ to 176.6, almost exactly the same as Wang(2006) found experimentally. Thus, lowering a from the nominal value of 0.100 to 0.054, while keeping the other parameters from Table 1 the same, brings the predicted optimal lysis time and burst size into close agreement with the experimental results. The next section considers further implications of variation in a.

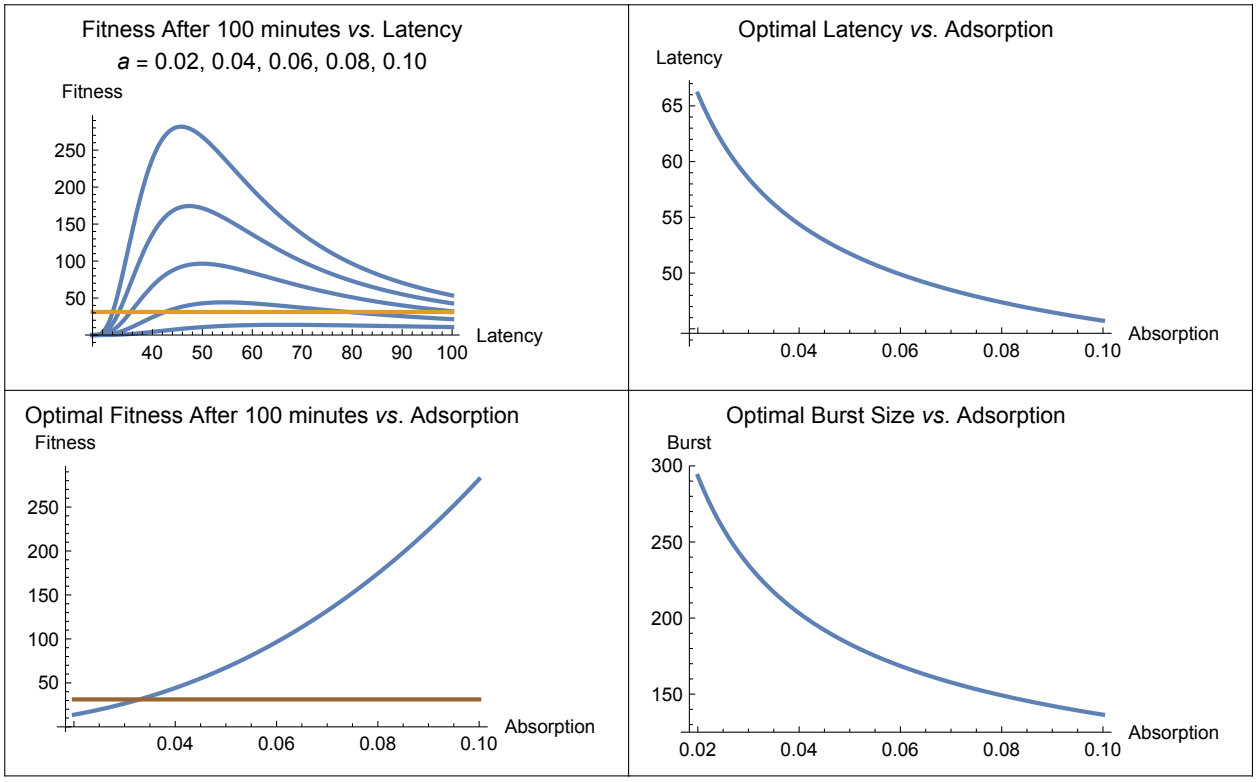

Figure 2: *Impact of varying adsorption on phage evolution. Top left: Fitness of the lytic phase* vs. *lysis time for several values of the adsorption coefficient,* a*. The top curve is for* $a = 0.10$ *and is identical to that shown in Figure 1. The curves below the top are successively for* $a = 0.08, 0.06, 0.04,$ *and 0.02. The horizontal line is the fitness of the lysogenized phage incorporated into the bacterial genome as a prophage. As* a *decreases, the peaks of the curves for the lytic phase progressively become lower and shift to the right. Top right: Optimal lysis time* vs. *the adsorption parameter,* a*. As* a *decreases from 0.10, the optimal lysis time increases. Bottom right: Optimal burst size* vs. *the adsorption parameter,* a*. As* a *decreases from 0.10, the optimal burst size also increases, reflecting the increasingly longer lysis time. Bottom left: Optimal fitness of lytic phase* vs. *the adsorption parameter,* a*. The horizontal line is the fitness of the lysogenized phage incorporated into the bacterial genome as a prophage. As* a *decreases from 0.10, the optimal lytic fitness decreases and intersects the horizontal line at* $a = 0.033$*, at which point the phage lysogenizes, switching from its lyric phase to being a prophage.*

Interventions that Impact Adsorption

Figure 2 shows the impact of varying adsorption on phage evolution. Top left: As a decreases from the nominal value of 0.10, the peaks of the curves for the lytic phase progressively become lower and shift to the right. Top right: As a decreases from 0.10, the optimal lysis time increases. Bottom right: As a decreases from 0.10, the optimal burst size also increases, reflecting the increasingly longer lysis time. Bottom left: As a decreases from 0.10, the optimal lytic fitness decreases and intersects the horizontal line representing the prophage fitness at $a = 0.033$, at which point the phage lysogenizes, switching from its lyric phase to being a prophage.

Many interventions to control phages, and viruses more generally, involve techniques such as air or water filters, or controlling humidity that lower the adsorption of phage by bacteria (*cf.* Sobsey *et al.* 2008, Clokie and Kropinski 2009, Wommack *et al.* 2009, Moriyama *et al.* 2020, Curtius *et al.* 2021, Van Charante *et al.* 2021, Beltrán *et al.* 2023). Overall, the model predicts that interventions lowering a cause the evolution of an increased lysis time and correspondingly higher burst size until

the intervention is sufficient either to drive a virulent phage extinct, or for temperate phage, to drive the phage from its lytic phase into its lysogenic phase.

Ecogeographic Patterns

To see how the optimal viral fitness might vary along environmental gradients, begin by rewriting Eq. 11 for the viral fitness in terms of an important lumped parameter, Q, as

$$W_L(l) = (Q\ (l - \epsilon))^{\frac{L}{l}} \tag{20}$$

where Q is

$$Q \equiv \frac{a\,\beta}{R} \tag{21}$$

For a given habitat, Q describes the ratio of a virion's production, $(a\,\beta)$, to a bacterium's production, R. The higher the Q, the better the habitat is for the virus relative to the bacteria. The response of the virus along environmental gradients depends on how the ratio of its productivity to that of the bacteria, as measured by Q, varies along the gradient.

The eclipse period, ϵ, tends to be small relative to the overall latency, l, and qualitative insight can be gained by letting ϵ be zero. Accordingly, Eq. 20 becomes

$$W_{Lo}(l) = (Q\ l)^{\frac{L}{l}} \tag{22}$$

By differentiating $W_{Lo}(l)$ with respect to l, setting the result equal to zero and solving for l, yields an explicit result for the optimal latency

$$\hat{l_o}(Q) = \frac{e}{Q} \tag{23}$$

where e refers to Euler's constant (not the eclipse, ϵ). Thus, the optimal latency for the virus varies inversely with the ratio of how good the environment is for it relative to the bacteria. A high Q environment selects for short latencies.

The optimal fitness is found by combining Eq. 22 with Eq. 23, leading to

$$\widehat{W_{Lo}}(Q) = e^{(\frac{L\,Q}{e})} \tag{24}$$

Thus, the optimal viral fitness increases exponentially with the ratio of how good the environment is for it relative to the bacteria.

All in all, provided the eclipse is zero, the environmental Q determines both the optimal latency according to Eq. 23 and fitness realized at the optimal latency according to Eq. 24.

The top panel of Figure 3 shows the fitness as a function of the latency, $W_{Lo}(l)$ from Eq. 22, for several values of Q. The curves for the fitness show a peak at optimal latency. The middle panel of the figure shows how the optimal latency, $\hat{l_o}(Q)$ from Eq. 23, depends on Q. And the bottom panel shows the fitness realized when the latency is optimal from Eq. 24.

Environmental gradients in Q will produce biogeographic patterns in viral latency and the corresponding viral fitness. The burst size along the gradient depends on both β and the latency according to Eq. 8.

Various physical gradients within the environment underlie gradients in Q. One possibility is a physical gradient in nutrient availability leading to a gradient in bacterial productivity. Parada *et al.* (2006, Fig. 5) showed a positive relationship between bacterial productivity and viral burst size for virus in the North Sea. The effect of a gradient in bacterial productivity affects Q differently depending on whether both the virus and bacteria benefit from the nutrients or whether only the bacteria benefit.

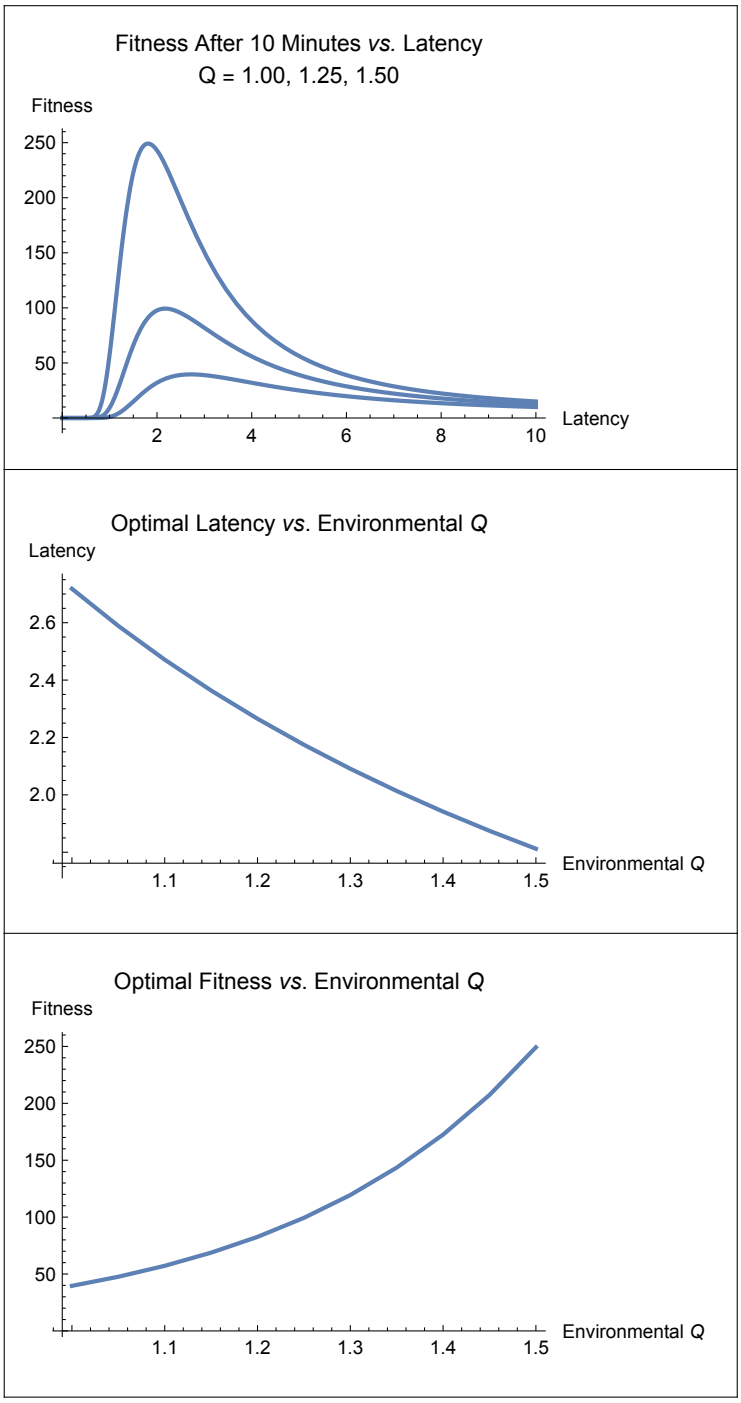

Figure 3: *Impact of varying environmental Q on phage evolution. The top panel shows the fitness as a function of the latency, $W_{Lo}(l)$. The curves from top to bottom are for values of Q = 1.50, 1.25, and 1.00. The peaks of the curves correspond to the optimal latency for each Q. The middle panel shows the optimal latency, $\hat{l}_o(Q)$ as a function of Q. The bottom panel shows the fitness realized when the latency is optimal. The graphs assume $L = 10$, and the eclipse, $\epsilon = 0$.*

If the nutrients increase both the bacterial R and also the number of virions produced per infection from within the bacterial cell, β, then these changes tend to cancel out leaving Q relatively unchanged

along the gradient. Therefore, the optimal latency will also be relatively unchanged along the gradient. However, the increase in β from the nutrients does increase the burst size along the gradient because of Eq. 8. Thus, the latency will be relatively constant while the burst size will increase along this type of gradient from low to high nutrient availability. The explanation for a correlation between viral burst size and bacterial productivity in this case rests with the ability of the virus to produce more virions in the nutrient enriched intracellular environment of the bacteria.

Alternatively, the nutrients may increase solely the bacterial R while leaving the viral β unchanged. In this case, Q declines along the nutrient gradient because the environment is becoming progressively worse for the virus relative to the bacteria. Hence, the latency increases along the gradient from low to high bacterial productivity. The burst size also increases along the gradient because as the latency increases, so does the burst size according to Eq. 8. In this case the explanation for a correlation between viral burst size and bacterial productivity is a side effect of the increasing latency time the virus evolves as a response to how its Q declines as productivity increases. However, if the bacterial R is increased enough along the gradient that Q drops below 1, then the virus cannot increase when rare and either disappears, if lytic, or enters the bacterial chromosome as a prophage, if temperate. When Q drops below 1, the virus reproduces faster by joining the bacterial chromosome than by replicating itself through producing virions. Accordingly, high bacterial growth in the microbializaton environment on coral reefs promotes the occurrence of lysogeny (Silveira *et al*. 2021).

A gradient that affects solely the adsorption, a, is covered in the previous section about the effect of interventions.

Discussion

This article provides a mathematical model for the evolutionarily optimal tradeoff between lysis time and burst size. Experimental evidence from two studies demonstrates that an optimal tradeoff exists. Wang (2006) demonstrated the optimal tradeoff with measurements on strains of λ phage infecting *E. coli*. Kannoly *et al*. 2022) also measured strains of λ phage infecting *E. coli*. The value of the optimal lysis time differed between these studies because the environmental conditions in the experiments differed. In Wang's (2006) study the optimal lysis time was 51 minutes, and in the Kannoly *et al*. (2022) study the optimal lysis time was about 80 minutes. Given the demonstrated reality of an optimal lysis time that depends on environmental conditions, the issue is how to explain it.

This article explains the optimal lysis time by finding the optimal tradeoff between rapid reproduction with a short lysis time at the cost of a low burst size *vs*. slow reproduction with a long lysis time with the benefit of a large burst size. The optimal tradeoff between these two considerations is derived using a single dynamical equation in discrete time for the virus to microbe ratio (VMR) during the log phase of phage/bacteria population growth (Roughgarden 2024).

Previous models for the optimal tradeoff use three delay-differential equations for the dynamics of uninfected bacteria, infected bacteria and free phage, respectively, in the stationary phase of virus-bacterial population growth (*eg*. Bull 2006, Bull *et al*. 2006). The three-equation system is an extension of the original two delay-differential equations introduced by Campbell (1961). The original Campbell model is critiqued in detail in Roughgarden (2024, incl. Supplementary Material). That critique carries over to the three-equation extension and, to avoid redundancy, is not repeated here.

In the present context, the relevant distinction between the one-equation analysis in this paper and the three-equation analysis in previous papers pertains to the intuition behind what is optimal about the lysis time *vs*. burst size tradeoff. The Eq. 4 in Bull (2006) gives a formula for the optimal lysis time (called, L). When the phage is at population-dynamic equilibrium (called $\hat{\lambda} = 0$), Bull's Eq. 4 predicts that the optimal lysis time varies inversely with the parameter called d, the death rate of the bacterial

cells. The intuition is that a high bacterial death rate implies a short life span for a bacterium leaving only a short time to for an infecting virus to make new virions. Thus, the tradeoff in Bull's (2006) model is between the time needed to produce a burst and the time available within a bacterial cell to do it. This explanation for the optimal tradeoff is conceptually different from that offered by this paper's model that focusses on the tradeoff between rapid reproduction with low burst *vs*. slow reproduction with high burst.

Grounds for preferring this paper's single-equation model over the previous three-equation model, in addition to its relative simplicity and better tractability, perhaps rest on which environmental situation is more common—log phase growth or stationary growth for the phage-bacteria pair. The three-equation model, as with the earlier two-equation model of of Campbell (1961), is directed to experimental situations such as a chemostat where a stationary phase can be realized. But in nature it is hard to imagine phage and bacteria in equilibrium. In nature the populations are invariably either recovering from or being decimated by environmental events such as storms *etc*. Natural populations of phage and bacteria are likely continually in a bust-boom regime where a log-phase model is preferable to a stationary-phase model.

Unlike the three-equation model, the single-equation of this paper is able to predict the evolutionary impact of interventions to control phage and predict possible ecogeographic patterns of latency and burst size along environmental gradients. The model predicts that interventions lowering phage adsorption cause the evolution of an increased lysis time and correspondingly higher burst size until the intervention is sufficient either to drive a virulent phage extinct, or for temperate phage, to drive the phage from its lytic phase into its lysogenic phase. Additionally, the model predicts ecogeographic patterns based on a parameter, Q, that describes the ratio of a virion's productivity to a bacterium's productivity. Various physical gradients in the environment, such as in the nutrients that promote bacterial productivity, produce various predicted ecogeographic patterns in latency and burst size.

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