Evolutionarily Optimal Phage Life-History Traits

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Abstract

Optimal phage life-history traits are computed from data on phenotypic tradeoffs presented in De Paepe and Tadei (2006). A parameter is introduced, l_e, that describes the loss of virions in the environment. Hygienic interventions increase l_e. The optimal burst size decreases with l_e and the optimal capsid thickness increases with l_e. The optimal viral fitness also decreases with l_e. An intervention that progressively lowers the transmission of phage in the environment will progressively cause the evolution of decreased burst size and increased capsid thickness until a point at which the virus either goes extinct, if virulent, or is driven to lysogenize, if temperate.

De Paepe and Tadei (2006) published a detailed tabulation of phenotypic tradeoffs for a variety of phages. This note is to synthesize these tradeoffs to predict evolutionarily optimal life-history traits for phage.

The data from De Paepe and Tadei (2006) are fitted by regression to obtain formulas for the tradeoffs. These formulas are substituted into an expression for the fitness of a virion previously derived in Roughgarden (2024). The result is a simple quadratic equation relating phage fitness to burst size. The optimal burst size computed from this equation is then used to predict the remaining life-history traits such as capsid size and decay rate based on the trade-off formulas. The analysis is further applied to predict the optimal phenotypic response of phage to interventions that affect phage transmissibility.

This note highlights numerical data from De Paepe and Tadei (2006). However, the code to generate the predictions is available as a Wolfram Mathematica notebook (.nb) to which alternative data can be supplied.

Table 1 shows tradeoffs among decay rate in laboratory conditions, burst size, latency period, and capsid thickness (expressed as surfacic mass) drawn from De Paepe and Tadei (2006). For the analysis, the time step and latency are normalized to 15 minutes. Regressing decay rate, d, against capsid thickness, s yields

$$d = 0.00445959 - 0.000136736\ s \tag{1}$$

Regressing capsid thickness, s, against burst size, b yields

$$s = 22.8651 - 0.0100168\ b \tag{2}$$

Combining these by eliminating s yields a formula for decay rate, d, *vs.* burst size, b, as

$$d = 0.0013331 + 1.36966 \times 10^{-6}\ b \tag{3}$$

The decay rate, d, is one element in the formula for viral fitness.

The expression for viral fitness is taken from the increase-when-rare condition in a phage-bacteria model (Roughgarden 2024). The virus population can infect the bacterial population if

$$a\,b > R \tag{4}$$

Table 1: Tradeoffs among phage life history traits (De Paepe and Taddei, 2006).

Name	Decay Rate (d)	Burst Size (n)	Latency Period (min)	Surfacic Mass (kDa/(nm^2))
λ	0.072	115	42	22.7
MS2	0.25	400	40	13.7
Mu	0.29	200	60	20.6
P2	0.041	160	48	22.7
P4	0.045	300	60	24.5
ϕ80	0.12	600	55	24.3
ϕX174	0.2	180	15	18.4
PRD1	0.037	50	48	35.5
T2	0.068	135	23	19.9
T3	0.102	200	17	18.1
T4	0.068	150	23	26.9
T5	0.12	290	44	13.7
T7	0.187	260	13	19.4
R17	0.52	3570	53	14.7

where a is the probability of a virion binding when all the binding sites are available; b is the burst size per virus infecting the bacterium, *i.e.* the burst size divided by the multiplicity of infection; and R is the uninfected bacterial population geometric growth factor per time step ($R > 1$). If Eq. 4 is satisfied, the virus can spread into a growing population of bacteria. Conversely, for a virulent virus, if $a\,b < R$, the virus goes extinct because the virus cannot keep up with bacterial population growth. For a temperate virus, if $a\,b < R$, 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.

In light of the model above, the virus fitness, w, is taken as,

$$w = a\,b \tag{5}$$

The a is the probability that a virion lives to be absorbed. This is the probability of *not* decaying, *i.e.* $(1 - d)$. However, d, from Eq. 3 is predicated on laboratory conditions. So in nature, the surviving number of virions is much less and can be influenced by hygienic and other interventions that lower viral transmission. So, let l_e be a parameter denoting environmental loss of virions beyond that experienced in ideal laboratory conditions. Hence the probability of a virion becoming absorbed into a bacterium is taken as

$$a = (1 - l_e\,d) \tag{6}$$

Substituting Eq. 3 into Eq. 6 and then into Eq. 5, yields the viral fitness as a quadratic function of the burst size, b, for a given environmental loss parameter, l_e,

$$w = (1 - l_e\,(0.0013331 + 1.36966 \times 10^{-6}\,b)\,b \tag{7}$$

The optimal burst size, $\hat{b}$, as a function of the environmental loss, l_e, is computed as the value of b that maximizes w in Eq. 7,

$$\hat{b} = \frac{365053}{l_e} - 486.652 \tag{8}$$

Then the optimal values of the other viral traits are calculated successively from the tradeoff formulas, Eq. 2 followed by Eq. 1.

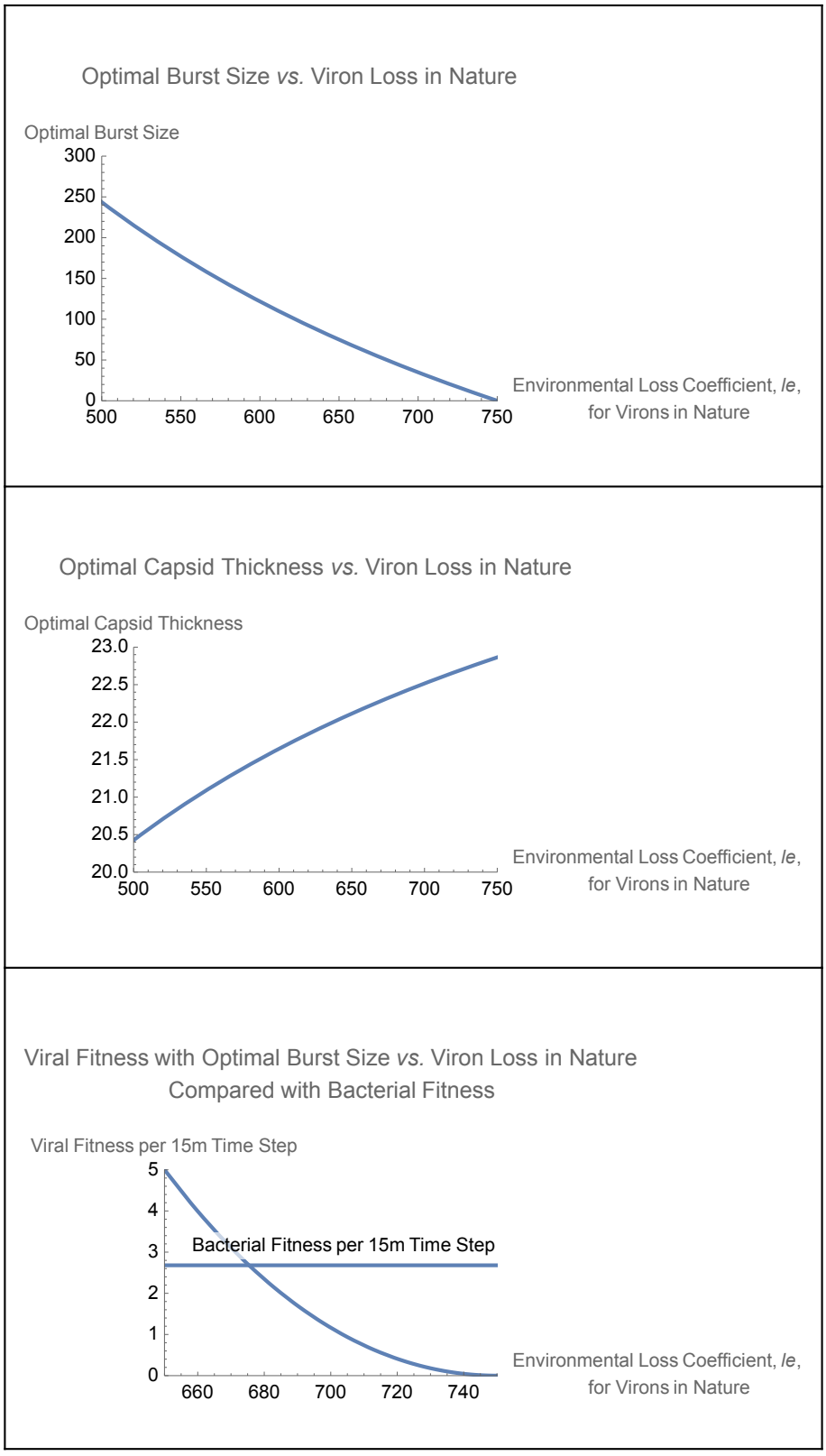

Figure 1: *Optimal burst size, optimal capsid thickness and optimal viral fitness as a function of environmental loss, l_e. In bottom panel, extinction of virulent phage occurs at degree of environmental loss where curve for viral fitness intersects line for bacterial fitness. For temperate phage, a switch from lytic to lysogeny occurs at the intersection.*

Figure 1 shows that the optimal burst size, $\hat{b}$, decreases as a hyperbola with l_e and the optimal capsid thickness increases with l_e. The optimal viral fitness, *i.e.* the viral fitness if the virus has the optimal burst size and capsid thickness, decreases with l_e. The optimal viral fitness intersects a horizontal line representing the bacterial fitness, R. (The R is taken as 2.68 for *E. coli* in laboratory conditions, following Campbell, 1961)). For virulent virus, extinction occurs at the intersection whereas for temperate virus, a switch from lytic to lysogeny occurs at the intersection.

An intervention that progressively lowers the transmission of virus in the environment will progressively cause the evolution of decreased burst size and increased capsid thickness until a point at which the virus either goes extinct, if virulent, or is driven to lysogenize, if temperate. For aerosol born virus, such interventions might include changes to ventilation and humidity (*e.g.* Beltrán *et al,* 2023, *cf.* Stacy 2024).

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