Ecological Archives E090-095-A1

Meghan A. Duffy, Spencer R. Hall, Carla E. Cáceres, and Anthony R. Ives. 2009. Rapid evolution, seasonality, and the termination of parasite epidemics. *Ecology* 90:1441–1448.

Appendix A. Supplementary methods and results.

Derivation of evolutionary effects on transmission (Eq. 1c)

The equations we use to model epidemics, repeated from the main text, are

$$I_t = \beta_{t-L} S_{t-L}^{b_s} I_{t-L}^{b_t} \tag{A.1a}$$

$$\boldsymbol{\beta}_t = \boldsymbol{\beta}_t^o \boldsymbol{e}^{\boldsymbol{b}_T T} \tag{A.1b}$$

$$\boldsymbol{\beta}_{t}^{0} = \boldsymbol{\beta}_{t-1}^{0} - \boldsymbol{v} \frac{\boldsymbol{I}_{t}}{\boldsymbol{S}_{t}}, \tag{A.1c}$$

where S_t , and I_t are the densities of susceptibles and infecteds in the population at time t, β_t is the transmission rate, β_t^0 is the nominal transmission rate that is independent of temperature T, and v is a measure of the clonal genetic variation in resistance to infection. Exponents b_s and b_t allow infection rates to scale nonlinearly with densities of susceptible and infected hosts. Transmission rate β_t can depend on temperature T, as governed by the exponential function in Eq. A.1b; larger values of b_T result in larger declines in transmission with decreasing temperature, as compared to its nominal, temperature independent value, β_t^0 . Here, we derive Eq. A.1c. Although not used for fitting data, we need to specify a general equation for the dynamics of susceptible *Daphnia* to derive Eq. A.1c. We assume the dynamics of the susceptible population are governed by the equation

$$S_{t} = f(N_{t-L}) - \beta_{t-L} S_{t-L}^{b_{s}} I_{t-L}^{b_{t}}, \qquad (A.2)$$

where f() is some function giving the dynamics of an uninfected *Daphnia* population, and N_t is the total population density $(N_t = S_t + I_t)$. The per capita fitness, $W_{t-L}(\widetilde{\beta}_{t-L})$, for a given individual between times t - L and t as a function of transmission rate is

$$W_{t-L}(\widetilde{\beta}_{t-L}) = \frac{f(N_{t-L}) - \widetilde{\beta}_{t-L} S_{t-L}^{b_s} I_{t-L}^{b_t}}{N_{t-L}}.$$
(A.3)

Here, we have used $\tilde{\boldsymbol{\beta}}_t$ to denote the transmission rate of a particular individual. Assume that the transmission rate among individuals is distributed with mean β_t and additive genetic variance $V(\beta_t)$. We allow the variance to depend on the mean, because the transmission rate cannot be negative; therefore, as the transmission rate decreases, the variance must as well. In particular, we assume that $V(\beta_t)$ is proportional to the mean, $V(\beta_t) = v\beta_t$.

Assuming that the distribution of transmission rates is symmetrical and the additive genetic variance is not too large (Iwasa 1991, Abrams et al. 1993, Abrams 2001), we can use a common quantitative genetic based recursion equation to represent change in mean phenotype of

a trait (here, transmission rate, β_t) that is under selection. Therefore, change in mean transmission rate equals the additive genetic variance of that trait, V(β_t), times the slope of an individual's fitness with respect to its own transmission rate when evaluated at the value of the population mean, all divided by mean fitness (\overline{W}_{t-L}), or

$$\Delta \beta_{t} = \frac{V(\beta_{t-L})}{\overline{W}_{t-L}} \frac{\partial W(\widetilde{\beta}_{t-L})}{\partial \widetilde{\beta}_{t-L}} \bigg|_{\widetilde{\beta}_{t-L} = \beta_{t-L}}$$

$$= -\frac{V(\beta_{t-L})}{\overline{W}_{t-L}} \frac{S_{t-L}^{b_{s}} I_{t-L}^{b_{t}}}{N_{t-L}}.$$
(A.4)

If we combine Eq. A.2 and Eq. A.3, we find that mean fitness (\overline{W}_{t-L}) equals the density of susceptible hosts at time *t* divided by total host density at the previous lagged time, or $\overline{W}_{t-L} = S_t / N_{t-L}$. Using this simplification, Eq. A.1, and the assumption that genetic variance in transmission rate (β_t) is proportional to the mean, $V(\beta_t) = v\beta_{t,t}$, we can derive a dynamic equation for transmission rate. Below, we show the steps of the derivation for interested readers:

$$\beta_t = \beta_{t-L} + \Delta \beta_t \tag{A.5a}$$

$$= \beta_{t-L} - \frac{V(\beta_{t-L})}{\overline{W}_{t-L}} \frac{S_{t-L}^{b_s} I_{t-L}^{b_t}}{N_{t-L}}$$
(A.5b)

$$=\beta_{t-L} - V(\beta_{t-L}) \frac{N_{t-L}}{S_t} \frac{S_{t-L}^{b_s} I_{t-L}^{b_t}}{N_{t-L}}$$
(A.5c)

$$=\beta_{t-L} - V(\beta_{t-L})\frac{1}{S_t}\frac{I_t}{\beta_{t-L}}$$
(A.5d)

$$=\beta_{t-L} - v \frac{I_t}{S_t}$$
(A.5e)

We start with a very general form of the equation (Eq. A.5a). Then, we substitute in our derived, quantitative-genetics based expression for $\Delta\beta_t$ (from Eq. A.4) to produce Eq. (A.5b), which is further simplified with the derived expression for mean fitness, \overline{W}_{t-L} (yielding Eq. A.5c). After a little bit of algebra (moving to Eq. A.5d) and substitution of our assumption about genetic variance (yielding Eq. A.5e), the derivation is complete. Note that the final equation provided by this process (Eq. A.5e) differs from Eq. A.1c by the inclusion of the lag *L* in β_{t-L} on the righthand side of the equation, rather than β_{t-1} . For the statistical analyses, we lagged the changes in transmission rate by only a single sample rather than L = 3 samples in order to assure smooth changes in transmission rates. This will not change the general results.

Estimation of two types of R^2 using the Kalman Filter

The Kalman filter uses a two-step procedure for estimating the log density of infected individuals. First, it takes the estimated value of I_{t-L} , denoted \hat{I}_{t-L} , along with the associated estimate of the transmission rate, to project the dynamics 3 samples forward using Eqs. 1, thereby giving \hat{I}_t^p . It then updates this predicted value by comparing it to the observed value I_t . This produces the updated estimate \hat{I}_t that moves closer to I_t to the extent allowed by the measurement error η_t . Thus, there are two estimates of I_t : \hat{I}_t^p and \hat{I}_t . Because \hat{I}_t^p predicts I_t using only information from generation t, the errors $\varepsilon_t = I_t - \hat{I}_t^p$ include process errors $\varepsilon_{t,t}$ and measurement error $\eta(t)$. Because \hat{I}_t predicts I_t after factoring out measurement error, the errors $\hat{\varepsilon}_t = \hat{I}_t - \hat{I}_t^p$ include only process error. The two R^2 values are: (i) prediction R^2 for $I_t = 1 - \text{var } \varepsilon_t$ /var[$I_t - I_{t-l}$] (Harvey 1989), and (ii) process error prediction R^2 for $\hat{I}_t = 1 - \text{var } \hat{\varepsilon}_t$ /var[$\hat{I}_t - \hat{I}_{t-L}$]. The former is the equivalent of the R^2 for *L*-step-ahead predictions, and the latter is equivalent to the *L*-step-ahead predictions after factoring out unavoidable measurement error.

Transmission of Metschnikowia

Our analysis indicates that transmission is nonlinear in both *S* and *I*. In Eq. 1a, the exponents b_S and b_I allow for transmission that is not directly proportional to *S* or *I*, respectively. Our estimate for b_S is -0.07 indicating that the density of infected individuals is slightly negatively related to the density of susceptible hosts when infection would have occurred, that is, at time t - L where *L* is the time lag required for the incubation of infections (9-12 d). Our estimate of $b_I = 0.91$ indicates that the density of infected individuals increases almost linearly with the density of infected individuals at a lag of *L*. Infected individuals at time t - L are unlikely to live to time *t*, so this suggests individuals who are infected at time t - L infect other (susceptible) hosts at that time, and those hosts then appear as infected at time *t*.

Effect of temperature on transmission rate

In an earlier laboratory study, we showed that temperature influences transmission rate (Hall et al. 2006). That study included two experiments with four temperature treatments, and we fit the infection data produced from each to an Arrhenius function. Because those Arrhenius-function-based results eventually pointed to a role for temperature in disease dynamics, our present model includes an effect of temperature on transmission rate -- but using an exponential rather than an Arrhenius function (b_T ; Eq. 1b). Our current estimate of b_T for the model that includes temperature but not evolution is 0.17 (Table A1). Since this exponential function differed from the one used in the original paper (Arrhenius), we re-estimated b_T from those lab

experiments. More specifically, we fit an exponential temperature-based differential equation model using a binomial likelihood function (see Hall et al. 2006 Appendix B for more methods). The estimates for the experiments from Hall et al. (2006) are 0.20 (for all temperature treatments) and 0.23 (excluding the 10°C treatment) from "experiment 1", and 0.18 (all temperature treatments) and 0.075 (excluding the 10°C treatment) from "experiment 2". Thus, there is a remarkable concordance between our lab and field estimates of this exponent relating transmission rate with temperature.

LITERATURE CITED

- Hall, S. R., A. J. Tessier, M. A. Duffy, M. Huebner, and C. E. Cáceres. 2006. Warmer does not have to mean sicker: Temperature and predators can jointly drive timing of epidemics. Ecology 87:1684–1695.
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TABLE A1: Parameter estimates for the three models of epidemic dynamics. "TR" = transmission rate.

		Full	Excluding	Excluding
Parameter	Symbol	model	evolution $(v = 0)$	temperature ($b_T = 0$)
TR exponent, infected class	b_I	0.92	0.91	0.91
TR exponent, susceptible class	b_S	-0.08	0.01	-0.1
TR exponent, temp. function	b_T	0.10	0.17	-
Genetic (clonal) variance in TR	ν	0.48	-	0.82
Baseline TR, Baker '03	${eta_{1,0}}^0$	3.84	1.44	5.4
Baseline TR, Bassett '03	${eta_{2,0}}^0$	4.18	1.59	5.92
Baseline TR, Bassett '04	$eta_{3,0}{}^0$	5.99	2	8.57
Baseline TR, Bristol '04	$eta_{4,0}{}^0$	6.21	1.88	9.11
Baseline TR, Warner '03	$eta_{5,0}{}^0$	5.35	1.97	7.44
Process errors, infected class	σ_{I}^{2}	0.31	0.34	0.32